ESSENCE OF ANESTHESIA PRACTICE
ESSENCE OF ANESTHESIA PRACTICE

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To Renee and Nancy, thanks for the inspiration
CoNTRiBuToRS

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Chromium

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Hypoaontria

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Carcinoid, Excision of  
Patent Ductus Arteriosus  
Ventricular Septal Defect, Repair of

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Conn’s Syndrome  
Saw Palmetto

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Circumcision

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Patent Ductus Arteriosus
Ventricular Septal Defect, Repair of

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Burn Injury, Electrical
Burn Injury, Flame
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University of Pennsylvania
Philadelphia, Pennsylvania
Pacemaker Implantation for Sick Sinus Syndrome
Exercise Stress Testing
<table>
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<tr>
<th>Name</th>
<th>Title and Affiliation</th>
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<tbody>
<tr>
<td><strong>Charles Weissman, MD</strong></td>
<td>Professor and Chair, Department of Anesthesiology and Critical Care Medicine</td>
<td>Diabetes Mellitus</td>
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<tr>
<td><strong>Nathanael Jeong, MD</strong></td>
<td>Assistant Professor, Department of Anesthesiology</td>
<td>Diverticulosis, Transjugular Intrahepatic Portosystemic Shunt (TIPS)</td>
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<td><strong>Nancy C. Wilkes, MD</strong></td>
<td>Professor of Anesthesiology, Department of Anesthesiology Medical Director</td>
<td>Diabetes, Type II (Gestational Diabetes Mellitus)</td>
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<td><strong>Michael Williams, MD</strong></td>
<td>Assistant Clinical Professor, Department of Anesthesiology</td>
<td>Epiglottitis, Transjugular Intrahepatic Portosystemic Shunt (TIPS)</td>
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<td><strong>Edward Yudkowitz, MD</strong></td>
<td>Associate Professor, Department of Anesthesiology and Pediatrics</td>
<td>Depression, Unipolar</td>
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<td><strong>Francine S. Yudkowitz, MD, FAAP</strong></td>
<td>Associate Professor, Department of Anesthesiology and Pediatrics</td>
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<td><strong>Gina Whitney, MD</strong></td>
<td>Associate Professor of Anesthesiology and Pediatrics</td>
<td>Endocardial Cushion Defect</td>
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<td><strong>Robert B. Wittenberg, MD</strong></td>
<td>Associate Professor, Department of Anesthesiology</td>
<td>Strabismus Repair</td>
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<td><strong>Andrew A. Zaidan, MD</strong></td>
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<td><strong>Andrew K. Wong, MD</strong></td>
<td>Assistant Professor, Department of Anesthesiology</td>
<td>Protein C Deficiency</td>
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<td><strong>A.J. Wright III, MLS</strong></td>
<td>Associate Professor, Department of Anesthesiology</td>
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<td><strong>Zhihui Xu, MD, PhD</strong></td>
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<td><strong>Christopher C. Young, MD, FCCM</strong></td>
<td>Associate Professor of Anesthesiology, Assistant Professor of Surgery</td>
<td>Chondroitin Sulfate</td>
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<tr>
<td><strong>Maurice S. Zwass, MD</strong></td>
<td>Professor of Anesthesiology and Pediatrics, Department of Anesthesiology</td>
<td>Chondroitin Sulfate</td>
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</table>

**Contributors:**

- Charles Weissman, MD
- Nathanael Jeong, MD
- Nancy C. Wilkes, MD
- Michael Williams, MD
- Edward Yudkowitz, MD
- Francine S. Yudkowitz, MD, FAAP
- Gina Whitney, MD
- Robert B. Wittenberg, MD
- Andrew K. Wong, MD
- A.J. Wright III, MLS
- Zhihui Xu, MD, PhD
- Christopher C. Young, MD, FCCM
- Maurice S. Zwass, MD
Lee Fleisher and Michael Roizen have updated and expanded the second edition of *Essence of Anesthesia Practice*, which ingeniously encapsulated information important for any anesthesia consultant. Having been their associates at UCSF and Yale, we respect their clinical judgments, the fruit of years of experience in the practice of anesthesia. This book reflects the innovative yet comprehensive approach that they often take. They are no ivory tower practitioners—they work “in the trenches.” We think that they have succeeded well in summarizing the pertinent aspects of the disease process, as well as the procedures, drugs, alternative medicines, and tests that are considered before a patient is anesthetized. Each chapter succinctly points the reader toward optimal care of a patient, by exploring the pathophysiology of a disease process and the management appropriate to specific conditions, clinical situations, and drug interactions. The intent is to help the physician rapidly and comprehensively plan perioperative management.

This is not a how-to-do-it book or “recipes” for perioperative care. Rather, it suggests that the pathophysiology of a disease or the physiologic imbalance caused by an operation should influence our thinking about therapeutic options. It offers a method for setting priorities to facilitate exemplary performance as a consultant in anesthesia. *Essence of Anesthesia Practice* has proven useful not only to anesthesiologists but also to our colleagues in other specialties who interface with the surgical patient.

The third edition expands on their previous success by including additional disease, drug, procedure, and laboratory testing topics and a section on alternative medicines. Interaction between herbal medicines and anesthetics is becoming increasingly important, and this text will serve as a handy reference.

The editors are to be congratulated for improving on their innovative clinical and educational format to serve both residents and practicing clinicians.

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**Ronald D. Miller, MD**
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It has been 9 years since the last edition of *Essence of Anesthesia Practice* was published and 14 years since the first edition. The goal of this text was, and continues to be, to provide a concise summary of the pathophysiology of both common and rare conditions seen in the perioperative period, medications used to treat these conditions, and the surgical procedures performed. These summaries are structured in a defined way to focus the clinician on the key facts and issues as well as the anticipated concerns regarding these conditions, medications, and procedures. Treatments, including medications for chronic conditions, continue to evolve, and it is difficult to keep up with the perioperative implications and the appropriate preoperative evaluation. Additionally, surgery has advanced and become more noninvasive over time. We therefore enrolled more than 500 authors, some of whom wrote the original chapters and many of whom are new and have either updated the original chapters or added new topics to address these concerns in the third edition of *Essence of Anesthesia Practice*.

This edition continues to improve and update the material that went before and to add the most up-to-date topics and new medications. We continue to include a large section on herbal medications, given their popularity and common use by our surgical patients. Mobile computing continues to advance and we are currently working on iPhone and Android applications that we hope will be available in the near future. We believe that the current format lends itself to quick review and orientation of the practitioner to perioperative implications at the point of care.

We wish to thank Natasha Andjelkovic, PhD, our publisher at Elsevier, and her editorial assistant, Brad McIlwain, for ensuring that our book received appropriate editing and development as well as providing the relentless support for this text to be published in a timely manner. We also wish to thank Eileen O’Shaughnessy, Lee’s executive assistant, who managed the contributions of more than 500 authors, a herculean task.

Lee A. Fleisher, MD
Michael F. Roizen, MD
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<td>?</td>
<td>questionable</td>
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<td>alveolar-arterial oxygen delivery</td>
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<tr>
<td>BMT</td>
<td>bone marrow transplantation</td>
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<tr>
<td>BO</td>
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<tr>
<td>BOOP</td>
<td>bronchitis obliterator with cryptogenic organizing pneumonia</td>
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<td>BP</td>
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<td>BPD</td>
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<td>BPH</td>
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<td>bpm</td>
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<td>BS</td>
<td>breath sounds</td>
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<td>BSA</td>
<td>body surface area</td>
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<td>Abbreviation</td>
<td>Full Form</td>
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<tr>
<td>BT</td>
<td>bleeding time; Blalock-Taussig (shunt)</td>
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<td>C</td>
<td>cancer, cold agglutinins</td>
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<td>ca.</td>
<td>about (L., circa)</td>
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<td>Ca2+</td>
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<td>CAB</td>
<td>coronary artery bypass</td>
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<td>CAHS</td>
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<td>cAMP</td>
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<td>CaO2</td>
<td>arterial oxygen concentration</td>
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<td>CAS</td>
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<td>CASS</td>
<td>Coronary Artery Surgery Study</td>
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<td>cath</td>
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<td>CBC</td>
<td>complete blood count</td>
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<td>CBF</td>
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<td>CBV</td>
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<td>CGL</td>
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<td>cGMP</td>
<td>cyclic guanosine monophosphate</td>
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<td>C-GSF</td>
<td>granulocyte colony-stimulating factor</td>
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<td>CHD</td>
<td>congenital heart disease; congenital heart defect</td>
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<td>CI</td>
<td>cardiac index; confidence interval</td>
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<td>cervical intraepithelial neoplasia</td>
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<td>circ</td>
<td>circulation; circulatory</td>
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<td>cis-DDP</td>
<td>cis-diaminodichloroplatinum</td>
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<td>CK-MB</td>
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<td>CNS</td>
<td>central nervous system</td>
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<td>carbon dioxide</td>
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<td>cyclooxygenase-2</td>
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<td>cP</td>
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<td>CP</td>
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<td>cephalopelvic disproportion</td>
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<td>CRI</td>
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<td>CSF</td>
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<td>CSH</td>
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<td>CSM</td>
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<td>CSS</td>
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<tr>
<td>CT</td>
<td>computed tomography; connective tissue</td>
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<td>CTX</td>
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<td>CVA</td>
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<td>CVP</td>
<td>central venous pressure</td>
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<td>D</td>
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<td>D2,3-DPG</td>
<td>two-dimensional</td>
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<td>d</td>
<td>day</td>
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<td>D and T</td>
<td>diphtheria and tetanus</td>
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<td>dilatation and curettage</td>
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<td>D1</td>
<td>dextrose 5% in water</td>
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<tr>
<td>DA</td>
<td>dopamine</td>
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<td>DBP</td>
<td>diastolic blood pressure</td>
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<tr>
<td>DC</td>
<td>direct current</td>
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<tr>
<td>DCM</td>
<td>dilated cardiomyopathy</td>
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<td>DDHAVP</td>
<td>1-deamino(8-D-arginine) vasopressin; desmopressin acetate</td>
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<td>dichlormethyltrichloroethane</td>
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<td>Drug Enforcement Agency</td>
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<td>DEB</td>
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<td>DFA</td>
<td>direct immunofluorescent assay</td>
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<td>DFT</td>
<td>defibrillation threshold</td>
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<td>DGL</td>
<td>deglucuronidized licorice</td>
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<tr>
<td>DGLA</td>
<td>dihydro-beta-linolenic acid</td>
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<td>DHT</td>
<td>dehydroepiandrosterone</td>
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<td>DI</td>
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<tr>
<td>DIC</td>
<td>diabetes insipidus</td>
</tr>
<tr>
<td>DVT</td>
<td>disseminated intravascular coagulation</td>
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<tr>
<td>Dig</td>
<td>digoxin</td>
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<td>DJD</td>
<td>degenerative joint disease</td>
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<td>DKA</td>
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<tr>
<td>DLco</td>
<td>carbon monoxide diffusion capacity in the lungs</td>
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<tr>
<td>DM</td>
<td>diabetes mellitus</td>
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<tr>
<td>DMD</td>
<td>Duchenne muscular dystrophy</td>
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<tr>
<td>DMT</td>
<td>dimethyltryptamine</td>
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<tr>
<td>DNR</td>
<td>do not resuscitate</td>
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<td>Do,</td>
<td>oxygen delivery</td>
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<td>DOB</td>
<td>dobutamine</td>
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<tr>
<td>Abbreviation</td>
<td>Definition</td>
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<td>--------------</td>
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<tr>
<td>DOE</td>
<td>dyspnea on exertion</td>
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<tr>
<td>dP/dT</td>
<td>ratio of change in ventricular pressure to change in time</td>
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<tr>
<td>DPNB</td>
<td>dorsal penile nerve block</td>
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<tr>
<td>dsSEEP</td>
<td>dermatomal somatosensory evoked potentials</td>
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<tr>
<td>DTIC</td>
<td>dimethyltriazenoimidazole carboxamide (dacarbazine)</td>
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<td>DTPA</td>
<td>diethylenetriaminepenta-acetic acid</td>
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<tr>
<td>DTR</td>
<td>deep tendon reflex</td>
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<td>DTs</td>
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<td>DVT</td>
<td>deep vein thrombosis</td>
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<td>Dx</td>
<td>diagnosis; diagnostic</td>
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<tr>
<td>EACA</td>
<td>epsilon-aminocaproic</td>
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<tr>
<td>EBL</td>
<td>estimated blood loss</td>
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<tr>
<td>EBV</td>
<td>Epstein-Barr virus</td>
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<tr>
<td>EC</td>
<td>ecchymasia</td>
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<tr>
<td>ECA</td>
<td>ethacrynic acid</td>
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<td>ECC</td>
<td>extracorporeal circulation</td>
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<td>ECD</td>
<td>endocardial cushion defect</td>
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<tr>
<td>ECFV</td>
<td>extracellular fluid volume</td>
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<td>ECG</td>
<td>electrocardiogram</td>
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<tr>
<td>ECHO</td>
<td>echocardiogram</td>
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<tr>
<td>ECMO</td>
<td>extracorporeal membrane oxygenation</td>
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<td>ECoG</td>
<td>electrocorticography</td>
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<td>ECT</td>
<td>electroconvulsive therapy</td>
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<td>ED&lt;sub&gt;50&lt;/sub&gt;</td>
<td>median effective dose</td>
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<td>end-diastolic volume</td>
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<td>electroencephalogram</td>
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<tr>
<td>EENT</td>
<td>eyes, ears, nose, throat</td>
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<td>EF</td>
<td>ejection fraction</td>
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<td>EGD</td>
<td>esophagogastroduodenoscopy</td>
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<td>E-L</td>
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<td>ELBW</td>
<td>extremely low birth weight</td>
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<td>ELISA</td>
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<td>EMD</td>
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<td>electromyography</td>
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<tr>
<td>EMI</td>
<td>electromagnetic interference; electromechanical interference</td>
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<td>EMLA</td>
<td>eutectic mixture of local anesthetics</td>
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<td>endocrine</td>
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<tr>
<td>ENT</td>
<td>ear, nose, and throat</td>
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<td>EP</td>
<td>electrophysiologic</td>
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<td>EPA</td>
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<td>ERV</td>
<td>expiratory reserve volume</td>
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<td>Eisenmenger’s syndrome</td>
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<td>ethosuximide</td>
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<td>erythrocyte sedimentation rate</td>
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<td>ETN&lt;sub&gt;2&lt;/sub&gt;</td>
<td>end-tidal nitrogen</td>
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<td>ethanol</td>
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<td>ETT</td>
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<td>exercise</td>
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<td>female(s)</td>
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<tr>
<td>Fa/Fi</td>
<td>fraction alveolar/fraction inspired</td>
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<tr>
<td>Fab</td>
<td>fragment, antigen-binding</td>
</tr>
<tr>
<td>FAD</td>
<td>flavin adenine dinucleotide</td>
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<tr>
<td>FBS</td>
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<tr>
<td>FDA</td>
<td>food and Drug Administration</td>
</tr>
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<td>FDP</td>
<td>fibrin-degradation product</td>
</tr>
<tr>
<td>Fe</td>
<td>iron</td>
</tr>
<tr>
<td>Fe&lt;sup&gt;2+&lt;/sup&gt;</td>
<td>ferrous</td>
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<tr>
<td>Fe&lt;sup&gt;3+&lt;/sup&gt;</td>
<td>ferric</td>
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<td>FEN&lt;sub&gt;2&lt;/sub&gt;</td>
<td>excreted fraction of filtered sodium</td>
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<td>FES</td>
<td>fat embolism syndrome</td>
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<td>FEV</td>
<td>forced expiratory volume</td>
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<td>FEV&lt;sub&gt;1&lt;/sub&gt;</td>
<td>Forced expiratory volume in 1 second</td>
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<td>FFP</td>
<td>fresh frozen plasma</td>
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<td>FHP</td>
<td>fulminant hepatic failure</td>
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<td>FHT</td>
<td>fetal heart tone</td>
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<td>glucose-6 phosphate dehydrogenase</td>
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<td>GA</td>
<td>general anesthesia</td>
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<td>GBL</td>
<td>gamma butyrolactone</td>
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<tr>
<td>G-CSF</td>
<td>granulocyte colony-stimulating factor</td>
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<tr>
<td>GDM</td>
<td>gestational diabetes mellitus</td>
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<tr>
<td>GE</td>
<td>gastroesophageal</td>
</tr>
<tr>
<td>GER</td>
<td>gastroesophageal reflux</td>
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<tr>
<td>GERD</td>
<td>gastroesophageal reflux disease</td>
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<tr>
<td>GETA</td>
<td>general endotracheal anesthesia</td>
</tr>
<tr>
<td>GFR</td>
<td>glomerular filtration rate</td>
</tr>
<tr>
<td>GGTP</td>
<td>gamma-glutamyl-transpeptidase</td>
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<tr>
<td>GH</td>
<td>growth hormone</td>
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<tr>
<td>GHB</td>
<td>gamma hydroxybutyrate</td>
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<tr>
<td>Gi</td>
<td>inhibitory G protein</td>
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<tr>
<td>GI</td>
<td>gastrointestinal</td>
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<tr>
<td>GIFT</td>
<td>gamete intrafallopian transfer</td>
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<td>GLA</td>
<td>γ-linolenic acid</td>
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<tr>
<td>glu</td>
<td>glucose</td>
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</table>
ABBREVIATIONS

GMP  guanosine monophosphate
Gn-RH  gonadotropin-releasing hormone
GRAS  generally recognized as safe
GTP  guanosine triphosphate
GTG  glucose tolerance test
GU  genitourinary
GVHD  graft vs. host disease
gyn  gynecologic

H
5-HIAA  5-hydroxyindoleacetic acid
5-HT  5-hydroxytryptamine
H & N  head and neck
H & P  history and physical
H1  histamine receptor type 1
H2  histamine receptor type 2
H2O  water
HAF-PCM  hypoalbuminemic form of protein-calorie malnutrition
HAV  hepatitis A virus
HB  heart block
HbA1c  glycosylated hemoglobin
HbAA  hemoglobin homozygous for A
HbM  hemoglobin Milwaukee
HbO2  oxyhemoglobin
HbsAg  hepatitis B surface antigen
HbSS  homozygosity for hemoglobin S (sickle cell anemia)
HBV  hepatitis B virus
HCFA  Health Care Financing Administration
hCG  human gonadotropin hormone
HCM  hypertrophic cardiomyopathy
HCO3  bicarbonate
Hct  hematocrit
HCV  hepatitis C virus
HD  heart disease; Hodgkin’s disease
HDL  high-density lipoprotein
HDL-C  HDL cholesterol
He  helium
HEENT  head, eyes, ears, nose, throat
HELLP  hemolysis, elevated liver enzymes, and low platelet count (syndrome)
heme  hematology
Hg  mercury
Hgb  hemoglobin
HGPRT  hypoxanthine-guanine-phosphoribosyltransferase
HHV-3-6  human herpes viruses
HIV  human immunodeficiency virus
HLA  human leukocyte antigen
hLH  hemophagocytic lymphohistiocytosis
HLHS  hypoplastic left heart syndrome
HMD  hyaline membrane disease
HMG CoA  3-hydroxy-3-methylglutaryl
HN2  nitrogen mustard
hosp  hospitalization
HPV  hypoxic pulmonary vasoconstriction
hr  hour(s)
HR  heart rate
HSV  herpes simplex virus
HSV-1  HSV type 1
HSV-2  HSV type 2
ht  height
Htm  hypertension
HUS  hemolytic uremic syndrome
Hx  history

I
I & D  incision and drainage
I/O  intake-output
IABCP  intra-aortic balloon counterpulsation
IABP  intra-aortic balloon pump
IADH  inappropriate antidiuretic hormone
IBD  inflammatory bowel disease
ICA  internal carotid artery
ICD  implantable cardioverter defibrillator
ICGA  immunochromatographic assay
ICH  intracranial hypertension
ICMA  immunochemiluminometric assay
ICP  intracranial pressure
ICU  intensive care unit
ID  infectious disease
IDCM  idiopathic dilated cardiomyopathy
IDDM  insulin-dependent diabetes mellitus
IDL  intermediate-density lipoprotein
IE  inspiratory/expiratory ratio
IFN  interferon
Ig  immunoglobulin
IGF  insulin-like growth factor
IGF-I  insulin-like growth factor I
IHHD  ischemic heart disease
IHSS  idiopathic hypertrophic subaortic stenosis
IL  interleukin
IM  intramuscular
immuno  immunologic
in.  inch
incl  including
inf  inferior
info  information
INH  isoniazid
INR  International Normalized Ratio
insp  inspiratory
intox  intoxication
intraop  intraoperative
IOL  intraocular lens
IOP  intraocular pressure
IP  impedance plethysmography; intraperitoneal; intraperitoneally
IPPB  intermittent positive pressure breathing
IPPV  intermittent positive pressure ventilation
IQ  intelligence quotient
IRDS  infant respiratory distress syndrome
IRMA  immunoradiometric assay
ITP  immune thrombocytopenic purpura
I-V  interventricular
IV  intravenous
infravenous
inferior vena cava
intravenous fluid
intracranial/intraventricular hemorrhage
intravenous pyelogram

K
K+  potassium
Kr  krypton
KSS  Kearns-Sayre syndrome
KUB  kidney, ureter, bladder
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>L</td>
<td>left</td>
</tr>
<tr>
<td>L→R</td>
<td>left to right</td>
</tr>
<tr>
<td>LA</td>
<td>left atrial; left atrium; linoleic acid; local anesthetic</td>
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<tr>
<td>lab</td>
<td>laboratory</td>
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<tr>
<td>LAD</td>
<td>left anterior descending (coronary artery)</td>
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<tr>
<td>LAFB</td>
<td>left anterior fascicular block</td>
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<td>LAO</td>
<td>left anterior oblique</td>
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<tr>
<td>LAP</td>
<td>left atrial pressure</td>
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<tr>
<td>lat</td>
<td>lateral</td>
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<tr>
<td>LBBB</td>
<td>left bundle branch block</td>
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<tr>
<td>LBO</td>
<td>large-bowel obstruction</td>
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<tr>
<td>LCAT</td>
<td>lecithin-cholesterol acyltransferase</td>
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<td>LCH</td>
<td>Langerhans cell histiocytosis</td>
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<td>LDH</td>
<td>lactate dehydrogenase</td>
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<td>LDL</td>
<td>low-density lipoprotein</td>
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<td>LDL-C</td>
<td>LDL cholesterol</td>
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<td>LE</td>
<td>lower extremity</td>
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<td>LEA</td>
<td>lower extremity amputation</td>
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<td>LES</td>
<td>lower esophageal sphincter</td>
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<td>LFT</td>
<td>liver function test</td>
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<td>LGL</td>
<td>Lown-Ganong-Levine syndrome</td>
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<td>LH</td>
<td>luteinizing hormone</td>
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<td>LLQ</td>
<td>left lower quadrant</td>
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<tr>
<td>LMA</td>
<td>laryngeal mask airway</td>
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<tr>
<td>LMP</td>
<td>last menstrual period</td>
</tr>
<tr>
<td>LMW</td>
<td>low molecular weight</td>
</tr>
<tr>
<td>LMWH</td>
<td>low molecular weight heparin</td>
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<tr>
<td>LOC</td>
<td>level of consciousness; loss of consciousness</td>
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<tr>
<td>LOS</td>
<td>length of stay</td>
</tr>
<tr>
<td>LP</td>
<td>lumbar puncture</td>
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<td>Lp(a)</td>
<td>lipoprotein(a)</td>
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<td>L-PAM</td>
<td>melphalan (Alkeran)</td>
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<td>LPFB</td>
<td>left posterior fascicular block</td>
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<td>LPO</td>
<td>left posterior oblique</td>
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<tr>
<td>LR</td>
<td>lactated Ringer’s (solution)</td>
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<td>LRI</td>
<td>lower respiratory tract infection</td>
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<tr>
<td>LSB</td>
<td>lumber sympathetic block</td>
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<td>LSD</td>
<td>lysergic acid diethylamide</td>
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<td>LTB₁</td>
<td>leukotriene B₁</td>
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<td>LUQ</td>
<td>left upper quadrant</td>
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<td>LV</td>
<td>left ventricle</td>
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<td>LVAD</td>
<td>left ventricular assist device</td>
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<td>LVEDP</td>
<td>left ventricular end-diastolic pressure</td>
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<td>LVEF</td>
<td>left ventricular ejection fraction</td>
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<td>LVET</td>
<td>left ventricular ejection time</td>
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<td>left ventricular failure</td>
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<td>LVH</td>
<td>left ventricular hypertrophy</td>
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<td>LVOT</td>
<td>left ventricular outflow tract</td>
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<td>lytes</td>
<td>electrolytes</td>
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<td>M:F</td>
<td>male to female ratio</td>
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<td>M2</td>
<td>muscarinic</td>
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<td>MAC</td>
<td>minimum alveolar concentration; monitored anesthesia care</td>
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<td>MALA</td>
<td>metformin-associated lactic acidosis</td>
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<tr>
<td>MAO</td>
<td>monoamine oxidase</td>
</tr>
<tr>
<td>MAOI</td>
<td>MAO inhibitor</td>
</tr>
<tr>
<td>MAP</td>
<td>mean arterial pressure</td>
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<tr>
<td>MAST</td>
<td>medical antishock trousers</td>
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<td>MAT</td>
<td>multifocal atrial tachycardia</td>
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<tr>
<td>max</td>
<td>maximum; maximal</td>
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<tr>
<td>MBC</td>
<td>maximal breathing capacity</td>
</tr>
<tr>
<td>MCA</td>
<td>middle cerebral artery</td>
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<tr>
<td>MD</td>
<td>muscular dystrophy</td>
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<tr>
<td>MEA</td>
<td>multiple endocrine adenomas</td>
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<tr>
<td>mech</td>
<td>mechanical; mechanism</td>
</tr>
<tr>
<td>med</td>
<td>medication</td>
</tr>
<tr>
<td>MEN</td>
<td>multiple endocrine neoplasia</td>
</tr>
<tr>
<td>MEN I</td>
<td>multiple endocrine neoplasia type I</td>
</tr>
<tr>
<td>MEN II</td>
<td>multiple endocrine neoplasia type II</td>
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<tr>
<td>MEP</td>
<td>motor/multimodality evoked potential</td>
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<tr>
<td>MET</td>
<td>metabolic equivalent</td>
</tr>
<tr>
<td>metab</td>
<td>metabolism; metabolic</td>
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<tr>
<td>methHb</td>
<td>methemoglobin</td>
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<td>mets</td>
<td>metastases</td>
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<tr>
<td>MF-PCM</td>
<td>marasmic form of PCM</td>
</tr>
<tr>
<td>Mg⁺⁺</td>
<td>magnesium</td>
</tr>
<tr>
<td>MgSO₄</td>
<td>magnesium sulfate</td>
</tr>
<tr>
<td>MH</td>
<td>malignant hyperthermia</td>
</tr>
<tr>
<td>MI</td>
<td>myocardial infarction</td>
</tr>
<tr>
<td>MIDCAB</td>
<td>minimally invasive direct coronary artery bypass</td>
</tr>
<tr>
<td>min</td>
<td>minimal; minimum; minute</td>
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<tr>
<td>MIsch</td>
<td>myocardial ischemia</td>
</tr>
<tr>
<td>mIU</td>
<td>milli-International unit</td>
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<tr>
<td>MIV</td>
<td>mivacurium</td>
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<tr>
<td>MLAP</td>
<td>mean left atrial pressure</td>
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<tr>
<td>MLD</td>
<td>median lethal dose</td>
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<tr>
<td>MMEFR</td>
<td>maximal midexpiratory flow rate</td>
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<tr>
<td>MMFR</td>
<td>masseter muscle rigidity</td>
</tr>
<tr>
<td>mo</td>
<td>month</td>
</tr>
<tr>
<td>mo wt</td>
<td>molecular weight</td>
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<tr>
<td>MODS</td>
<td>multiorgan dysfunction syndrome</td>
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<tr>
<td>MPS</td>
<td>mucopolysaccharide</td>
</tr>
<tr>
<td>MP</td>
<td>mean pulmonary artery pressure</td>
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<tr>
<td>MPAP</td>
<td>mast cell proliferative disorder</td>
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<tr>
<td>MR</td>
<td>mitral regurgitation</td>
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<td>MRA</td>
<td>magnetic resonance angiography</td>
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<td>MRI</td>
<td>magnetic resonance imaging</td>
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<tr>
<td>MS</td>
<td>mental status; mitral stenosis; multiple sclerosis; musculoskeletal</td>
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<tr>
<td>ms</td>
<td>milliseconds</td>
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<tr>
<td>MSLT</td>
<td>Multiple Sleep Latency Test</td>
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<td>MSOF</td>
<td>multisystem organ failure</td>
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<tr>
<td>MTX</td>
<td>methotrexate</td>
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<td>MU</td>
<td>million units</td>
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<td>mucocut</td>
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<td>MUGA</td>
<td>multiple gated acquisition</td>
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<td>musc</td>
<td>muscular</td>
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<tr>
<td>MVD</td>
<td>microvascular decompression</td>
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<tr>
<td>MVI</td>
<td>multiple vitamin infusion</td>
</tr>
<tr>
<td>Mvo₂</td>
<td>minute venous oxygen</td>
</tr>
<tr>
<td>MVP</td>
<td>mitral valve prolapse</td>
</tr>
<tr>
<td>MW</td>
<td>molecular weight</td>
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<tr>
<td>MYL</td>
<td>Myleran (busulfan)</td>
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<tr>
<td>N</td>
<td>nitrogen</td>
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<tr>
<td>n.</td>
<td>nerve</td>
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<tr>
<td>n-MPTP</td>
<td>1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine</td>
</tr>
<tr>
<td>N/A</td>
<td>not applicable</td>
</tr>
<tr>
<td>N/S</td>
<td>normal saline</td>
</tr>
<tr>
<td>N/V</td>
<td>nausea/vomiting</td>
</tr>
<tr>
<td>N₂O</td>
<td>dinitrogen monoxide (nitrous oxide)</td>
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<tr>
<td>Na⁺</td>
<td>sodium</td>
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<tr>
<td>NAC</td>
<td>N-acetyl-L-cysteine</td>
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<tr>
<td>NADH</td>
<td>nicotinamide adenine dinucleotide reduced form</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
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<tr>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>pos</td>
<td>positive</td>
</tr>
<tr>
<td>poss</td>
<td>possible; possibly</td>
</tr>
<tr>
<td>postop</td>
<td>postoperative</td>
</tr>
<tr>
<td>PPAR</td>
<td>peroxisome proliferator-activated receptor</td>
</tr>
<tr>
<td>PPD</td>
<td>purified protein derivative (tuberculin)</td>
</tr>
<tr>
<td>PPH</td>
<td>persistent pulmonary hypertension</td>
</tr>
<tr>
<td>Pplat</td>
<td>plateau pressure</td>
</tr>
<tr>
<td>ppm</td>
<td>parts per million</td>
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<tr>
<td>PPV</td>
<td>positive predictive value; positive pressure ventilation</td>
</tr>
<tr>
<td>PR</td>
<td>per rectum</td>
</tr>
<tr>
<td>PRA</td>
<td>plasma renin activity</td>
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<tr>
<td>prb</td>
<td>problem</td>
</tr>
<tr>
<td>PRBCs</td>
<td>packed red blood cells</td>
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<tr>
<td>preg</td>
<td>pregnancy; pregnant</td>
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<tr>
<td>premed</td>
<td>premedication</td>
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<td>preop</td>
<td>preoperative</td>
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<td>preparation</td>
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<td>PRL</td>
<td>prolactin</td>
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<td>prn</td>
<td>as needed</td>
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<td>PS</td>
<td>pulmonary stenosis</td>
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<td>PSA</td>
<td>prostate-specific antigen</td>
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<td>PSVT</td>
<td>paroxysmal supraventricular tachycardia</td>
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<td>psych</td>
<td>psychological</td>
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<td>pt</td>
<td>patient</td>
</tr>
<tr>
<td>PT</td>
<td>physical therapy; prothrombin time</td>
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<tr>
<td>PTCA</td>
<td>percutaneous transluminal coronary angioplasty</td>
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<tr>
<td>PTH</td>
<td>parathyroid hormone</td>
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<tr>
<td>PTLD</td>
<td>post transplant lymphoproliferative disease</td>
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<tr>
<td>pts</td>
<td>patients</td>
</tr>
<tr>
<td>PTS</td>
<td>posttraumatic stress disorder</td>
</tr>
<tr>
<td>PTT</td>
<td>partial thromboplastin time</td>
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<tr>
<td>PTU</td>
<td>propylthiouracil</td>
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<tr>
<td>PUD</td>
<td>peptic ulcer disease</td>
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<td>pulm</td>
<td>pulmonary</td>
</tr>
<tr>
<td>PUVA</td>
<td>psoralens plus ultraviolet A</td>
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<tr>
<td>PVC</td>
<td>polyvinyl chloride; premature ventricular contraction</td>
</tr>
<tr>
<td>PVD</td>
<td>peripheral vascular disease</td>
</tr>
<tr>
<td>PVO₂</td>
<td>partial pressure of oxygen, venous</td>
</tr>
<tr>
<td>PVR</td>
<td>pulmonary vascular resistance</td>
</tr>
<tr>
<td>Q</td>
<td>perfusion</td>
</tr>
<tr>
<td>q</td>
<td>every</td>
</tr>
<tr>
<td>q.a.m.</td>
<td>every morning</td>
</tr>
<tr>
<td>q.n.</td>
<td>every night</td>
</tr>
<tr>
<td>q.p.m.</td>
<td>every evening</td>
</tr>
<tr>
<td>qhs</td>
<td>every hour of sleep</td>
</tr>
<tr>
<td>qid</td>
<td>four times per day</td>
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<tr>
<td>Qp:Qs</td>
<td>ratio of pulmonary blood to systemic blood flow</td>
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<tr>
<td>QRS</td>
<td>Q wave, R wave, S wave</td>
</tr>
<tr>
<td>R</td>
<td>right</td>
</tr>
<tr>
<td>R/O</td>
<td>rule out</td>
</tr>
<tr>
<td>RA</td>
<td>rheumatoid arthritis; right atrial; right atrium</td>
</tr>
<tr>
<td>RAAS</td>
<td>renin-angiotensin-aldosterone system</td>
</tr>
<tr>
<td>RAD</td>
<td>reactive airway disease</td>
</tr>
<tr>
<td>RAE</td>
<td>right atrial enlargement</td>
</tr>
<tr>
<td>RAH</td>
<td>right atrial hypertrophy</td>
</tr>
<tr>
<td>RAI</td>
<td>resting ankle index</td>
</tr>
<tr>
<td>RAO</td>
<td>right anterior oblique</td>
</tr>
<tr>
<td>RAP</td>
<td>right atrial pressure</td>
</tr>
<tr>
<td>RAST</td>
<td>radioallergosorbent test</td>
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<tr>
<td>RBBB</td>
<td>right bundle branch block</td>
</tr>
<tr>
<td>RBC</td>
<td>red blood cell</td>
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<td>RBF</td>
<td>renal blood flow</td>
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<tr>
<td>RCM</td>
<td>congenital methemoglobinemia of the recessive type</td>
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<tr>
<td>RDA</td>
<td>recommended daily allowance</td>
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<td>RDS</td>
<td>respiratory distress syndrome</td>
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<td>reg</td>
<td>regular</td>
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<td>rehab</td>
<td>rehabilitation</td>
</tr>
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<td>REM</td>
<td>rapid eye movement</td>
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<td>reprod</td>
<td>reproductive (system)</td>
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<td>resp</td>
<td>respiratory</td>
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<td>RH</td>
<td>releasing hormone</td>
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<td>RHD</td>
<td>rheumatic heart disease</td>
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<td>RHF</td>
<td>right heart failure</td>
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<tr>
<td>RIA</td>
<td>radioimmunoassay</td>
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<td>RII</td>
<td>right internal jugular</td>
</tr>
<tr>
<td>RIMA</td>
<td>reversible inhibitor of monoamine</td>
</tr>
<tr>
<td>RIND</td>
<td>reversible ischemic neurologic deficit</td>
</tr>
<tr>
<td>RLD</td>
<td>restrictive lung disease</td>
</tr>
<tr>
<td>ROM</td>
<td>range of motion</td>
</tr>
<tr>
<td>ROP</td>
<td>retinopathy of prematurity</td>
</tr>
<tr>
<td>ROS</td>
<td>review of systems</td>
</tr>
<tr>
<td>ROSC</td>
<td>return of spontaneous circulation</td>
</tr>
<tr>
<td>RPO</td>
<td>right posterior oblique</td>
</tr>
<tr>
<td>RR</td>
<td>respiratory rate</td>
</tr>
<tr>
<td>R→L</td>
<td>right to left</td>
</tr>
<tr>
<td>RSD</td>
<td>reflex sympathetic dystrophy</td>
</tr>
<tr>
<td>RSV</td>
<td>respiratory syncytial virus</td>
</tr>
<tr>
<td>RT</td>
<td>radiation therapy</td>
</tr>
<tr>
<td>RTA</td>
<td>renal tubule acidosis</td>
</tr>
<tr>
<td>RUQ</td>
<td>right upper quadrant</td>
</tr>
<tr>
<td>RV</td>
<td>residual volume; right ventricle</td>
</tr>
<tr>
<td>RVE</td>
<td>right ventricular enlargement</td>
</tr>
<tr>
<td>RVEDP</td>
<td>right ventricular end-diastolic pressure</td>
</tr>
<tr>
<td>RVH</td>
<td>right ventricular hypertrophy</td>
</tr>
<tr>
<td>Rx</td>
<td>therapy; treatment; therapeutic</td>
</tr>
<tr>
<td>S</td>
<td>Svedberg unit</td>
</tr>
<tr>
<td>S/P</td>
<td>status post</td>
</tr>
<tr>
<td>SA</td>
<td>sinoatrial; beta S/beta A globin gene</td>
</tr>
<tr>
<td>SAH</td>
<td>subarachnoid hemorrhage</td>
</tr>
<tr>
<td>SAM</td>
<td>systolic anterior motion</td>
</tr>
<tr>
<td>SAMe</td>
<td>S-adenosyl-L-methionine</td>
</tr>
<tr>
<td>SaO₂</td>
<td>oxygen saturation in arterial blood</td>
</tr>
<tr>
<td>SAP</td>
<td>systematic arterial pressure</td>
</tr>
<tr>
<td>SAS</td>
<td>sleep apnea syndrome</td>
</tr>
<tr>
<td>sat</td>
<td>saturation</td>
</tr>
<tr>
<td>SBE</td>
<td>standard base excess; subacute bacterial endocarditis</td>
</tr>
<tr>
<td>SBO</td>
<td>small-bowel obstruction</td>
</tr>
<tr>
<td>SBP</td>
<td>systolic blood pressure</td>
</tr>
<tr>
<td>SCD</td>
<td>sudden cardiac death</td>
</tr>
<tr>
<td>SCH</td>
<td>succinylcholine</td>
</tr>
<tr>
<td>SD</td>
<td>standard deviation(s)</td>
</tr>
<tr>
<td>SEB</td>
<td>simplex epidermolysis</td>
</tr>
<tr>
<td>sec</td>
<td>second(s)</td>
</tr>
<tr>
<td>SEP</td>
<td>sensory evoked potential</td>
</tr>
<tr>
<td>seroneg</td>
<td>seronegative</td>
</tr>
<tr>
<td>SG</td>
<td>specific gravity</td>
</tr>
<tr>
<td>SGOT</td>
<td>serum glutamic-oxaloacetate transaminase</td>
</tr>
<tr>
<td>SGPT</td>
<td>serum glutamate pyruvate transaminase</td>
</tr>
<tr>
<td>SIADH</td>
<td>syndrome of inappropriate secretion of antidiuretic hormone</td>
</tr>
<tr>
<td>SICU</td>
<td>surgical ICU</td>
</tr>
<tr>
<td>SIDS</td>
<td>sudden infant death syndrome</td>
</tr>
<tr>
<td>SIRS</td>
<td>systemic inflammatory response syndrome</td>
</tr>
</tbody>
</table>
ABBREVIATIONS

SL  sublingual
SLE  systemic lupus erythematosus
SMA  superior mesenteric artery
SMA-20 Sequential Multiple Analyzer
SNS  sympathetic nervous system
SOB  shortness of breath
soln solution
SPECT single-photon emission computed tomography
SPK simultaneous pancreas-kidney
SpO₂ oxygen saturation as measured by pulse oximetry
spont spontaneously
SQ  subcutaneous; subcutaneously
SEEP somatosensory evoked potential
SSRI selective serotonin reuptake inhibitor
SS  sick sinus syndrome
STD sexually transmitted disease
STP 2,5-dimethoxy-4-methylamphetamine
STSG split-thickness skin graft
Stz streptozocin
sup superior
surg surgery; surgical
SV  stroke volume
SVC superior vena cava
SVO₂ mixed venous continuous oxygen saturation
SVR systemic vascular resistance
SVT supraventricular tachycardia
Sx signs and symptoms
Sz seizure
TN  trigeminal neuralgia
TNF  tumor necrosis factor
TNM  tumor, nodes, and metastasis
TOF  train-of-four; tetralogy of Fallot
TP  total protein
t-PA tissue plasminogen activator
TPN total parenteral nutrition
TR  tricuspid regurgitation
TRH thyrotropin-releasing hormone
TRUS transrectal ultrasonography
TSH thyroid stimulating hormone
TT  thrombin time
TTE thoracostomy echocardiography
T-TEPA triethylene-thiophosphoramide (thiotepa)
TTP thrombotic thrombocytopenic purpura
TURBT transurethral resection of bladder tumor
TRUP transurethral resection of the prostate
TV tidal volume
TVH total vaginal hysterectomy
Tx transplant; transfusion
TXA₁ thromboxane A₁
TXA₂ thromboxane A₂
TVH total venous hystereotemy
U urinalysis
UA upper extremity
U GI upper gastrointestinal
UK United Kingdom
U-lytes urine electrolytes
UO urine output
Urticaria pigmentosa
UPIJ ureteropelvic junction
URI upper respiratory tract infection
urology, urologic
US ultrasound
USA United States of America
UT urinary tract
UTI urinary tract infection
UV ultraviolet

T

T⁻¹ temperature
T&C type and crossmatch
T₀ half-life
T₁ triiodothyronine
T₂ thyroxine
TA tricuspid atresia
TAH total abdominal hysterectomy
TAPVD total anomalous pulmonary venous drainage
TB tuberculosis
TCA tricyclic antidepressant
TCD transcerebral Doppler
TDP torsades de pointes
TEE transesophageal echocardiography
TEF transesophageal fistula
TEG thromboelastography
temp temperature
TENS transcutaneous electrical nerve stimulation
tet tetralogy of Fallot
TFA trifluoroacetic acid
TFT thyroid function test
TGA transposition of the great arteries
TGV transposition of great vessels
THC delta-9-tetrahydrocannabinol
THR total hip replacement
TIA transient ischemic attack
tid three times per day
TIPS transjugular intrahepatic portosystemic shunt
TJC The Joint Commission
TKR total knee replacement
TLC total lung capacity/compliance
Tm maximal tubular excretory capacity (of kidney)
TM temporomandibular
TMEP telangiectasia macularis eruptive perstans
TMJ temporomandibular joint
TMP/SMX trimethoprim/sulfamethoxazole

T

V ventilation
V/Q ventilation-perfusion
VACTERL vertebral, anal, cardiac, tracheal, esophageal, renal, and limb
VE venous air embolism
VAE ventilator-associated lung injury
VALI Visual Analogue Scale
VAS vascular
VATER vertebral anomalies, anal atresia, tracheoesophageal fistula, esophageal atresia, radial dysplasia
Vc vital capacity; vocal cord
VCO₂ carbon dioxide consumption per unit time
VD volume of distribution
VD/s volume of distribution in a steady state
vent ventilation
VFIB ventricular fibrillation
VFP ventricular filling pressure
VIPoma vasoactive intestinal peptide-secreting tumors
vit vitamin
VLBW very low birth weight
VLNL very low density lipoprotein
VM-26 teniposide
VMA vanillylmandelic acid
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO_{2}</td>
<td>oxygen consumption per unit time</td>
</tr>
<tr>
<td>vol</td>
<td>volume</td>
</tr>
<tr>
<td>VP-16</td>
<td>etoposide</td>
</tr>
<tr>
<td>VPA</td>
<td>valproic acid</td>
</tr>
<tr>
<td>VR</td>
<td>venous return</td>
</tr>
<tr>
<td>VS</td>
<td>vital signs</td>
</tr>
<tr>
<td>vs.</td>
<td>versus</td>
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<tr>
<td>VSD</td>
<td>ventricular septal defect</td>
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<tr>
<td>VSM</td>
<td>vascular smooth muscle</td>
</tr>
<tr>
<td>VTach</td>
<td>ventricular tachycardia</td>
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<tr>
<td>VUR</td>
<td>vesicoureteral reflux</td>
</tr>
<tr>
<td>VVB</td>
<td>venovenous bypass</td>
</tr>
<tr>
<td>VVI</td>
<td>ventricular inhibited</td>
</tr>
<tr>
<td>vWF</td>
<td>von Willebrand factor</td>
</tr>
<tr>
<td>w/</td>
<td>with</td>
</tr>
<tr>
<td>w/o</td>
<td>without</td>
</tr>
<tr>
<td>WBC</td>
<td>white blood cell</td>
</tr>
<tr>
<td>wk</td>
<td>week(s)</td>
</tr>
<tr>
<td>WNL</td>
<td>within normal limits</td>
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<tr>
<td>WPW</td>
<td>Wolff-Parkinson-White syndrome</td>
</tr>
<tr>
<td>wt</td>
<td>weight</td>
</tr>
<tr>
<td>Xe</td>
<td>xenon</td>
</tr>
<tr>
<td>XS</td>
<td>excessive</td>
</tr>
<tr>
<td>y</td>
<td>year(s)</td>
</tr>
</tbody>
</table>
Abruptio Placentae

Risk
- People within USA: 1:200 of the approximately 4 million births/y
- Races with highest prevalence: African-Americans and Caucasians vs. Asian and Hispanic
- Increased prevalence with preeclampsia, chronic hypertension, multiple gestations, LBW, hydramnios, thrombophilia, cocaine use, trauma, increased age and parity, smoking, premature rupture of membranes, and prior abruptio

Perioperative Risks
- Maternal: Antepartum and postpartum hemorrhage, DIC, and death
- Fetal: Hypoxia due to maternal hypotension and/or decreased area for placental exchange; usually there is minimal bleeding from the fetus but it can occur

Worry About
- Concealed hemorrhage behind the placenta that does not manifest as vaginal bleeding, which may be considerable
- Postpartum hemorrhage refractory to usual oxytocic agents; some believe old blood can infiltrate into and between uterine muscle fibers and decrease the effectiveness of uterine contractions (Couvelaire uterus)
- Fetal distress and/or death
- Need for cesarean hysterectomy due to previous concerns

Overview
- Along with placenta previa, a major cause of antepartum hemorrhage, maternal mortality and perinatal mortality
- Maternal mortality: 1.8–2.8%
- Perinatal mortality: 30–40%
- Morbidity: ~20% of survivors have some form of neurologic deficit
- Abruptio placentae is the most common cause of DIC in pregnant patients; 20% with clinically significant abruptio develop clotting defects. DIC is probably due to the release of thromboplastin by placenta and damaged tissues at abruptio site.
- Postpartum hemorrhage correlates directly with severity of coagulopathy

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hemorrhage</td>
<td>Vaginal bleeding and abdominal pain</td>
<td>Vaginal bleeding and firm, tender uterus; hypotension, tachycardia, low CVP and wedge pressures, decreased urine output</td>
<td>Hct</td>
</tr>
<tr>
<td>HEME</td>
<td>Hypovolemia; acute anemia</td>
<td>Bleeding diathesis</td>
<td>Hypotension, tachycardia, bleeding from puncture sites, easy bruisability</td>
<td>Hgb, Hct, clotting evaluation that includes platelets, fibrinogen, and fibrin split products</td>
</tr>
<tr>
<td>RENAL</td>
<td>Oliguria and/or acute renal failure</td>
<td>Urine output</td>
<td>Signs of hypovolemia</td>
<td>Urinalysis to include specific gravity and sodium excretion, possibly in addition to central hemodynamic monitoring values</td>
</tr>
<tr>
<td>UTERUS/ VAGINA</td>
<td>Abruption; hemorrhage</td>
<td>Painful vaginal bleeding</td>
<td>Tender, firm uterus; vaginal bleeding may be &lt; CV signs and symptoms, indicating concealed hemorrhage</td>
<td>Hct and hemodynamic monitoring values</td>
</tr>
<tr>
<td>FETUS</td>
<td>Fetal distress and/or demise</td>
<td>Presence or absence of fetal movement</td>
<td>Fetal movement, heart rate</td>
<td>Electronic fetal monitoring</td>
</tr>
</tbody>
</table>


Perioperative Implications—for Labor and Vaginal Delivery

Preinduction/Induction/Maintenance
- Epidural analgesia appropriate if volume status can be maintained and if hemorrhage controllable
- Optimize cardiovascular and fetal status and evaluate coagulation system
- Technique not different from that for normal labor and vaginal delivery except that the smallest effective doses should be used; combined spinal/epidural with narcotics and local anesthetic may be useful
- Electronic fetal monitoring is essential
- CV monitoring appropriate for volume and bleeding status

Perioperative Implications—for Cesarean Section

Preinduction/Induction/Maintenance
- Optimize CV and fetal status, usually by means of appropriate volume replacement

Monitoring
- All cases will require electronic fetal monitoring as well as intrauterine pressure monitoring
- Urine output
- Hct and clotting studies as above
- Consider CVP and/or PA catheter depending on severity of hemorrhage and decreased urine output not responsive to simple fluid challenges

General Anesthesia
- Probably required for massive hemorrhage and/or acute fetal distress
- Aspiration prophylaxis
- Rapid-sequence induction with cricoid pressure
- Consider ketamine 1 mg/kg and large-bore lines
- Watch for continued hemorrhage after delivery of infant. Uterus may not respond to usual tocolytic agents. For hemorrhage control:
  - Oxytocin 20–40 mU in 1 L of balanced salt solution
  - Methergine 0.2 mg IM; not in the presence of hypertension
  - Prostaglandin F2α 250 µg IM or intramyometrial. May cause bronchospasm and decrease in SaO2
  - Hypogastric artery ligation
  - Uterine, hypogastric artery embolization
  - Cesarean hysterectomy
  - Awake extubation

Regional Anesthesia
- Appropriate in the absence of severe hemorrhage and/or acute fetal distress
- Aspiration prophylaxis
- Optimize volume status
- Epidural preferred over spinal because the level can be raised slowly, but could do same with continuous spinal

- Blood and blood clots in muscle fibers may inhibit ability of uterus to contract, which leads to more blood loss

ICD-9-CM Code: 641.2

Etiology
- Separation of placenta from uterine wall along decidual plane between membranes and uterus

Usual Treatment
- Maintenance of volume status and fetal surveillance
- If fetus is premature and hemorrhage is not great, careful observation would be appropriate to allow for fetal growth
- If at term and volume status OK, labor with vaginal delivery is optimal
- If hemorrhage continues and/or fetal distress occurs, C-section is necessary. If fetus is at term and doing well, then may elect for ceserean section to prevent fetal harm or death from sudden increase in abruptio process

Anticipated Problems/Concerns
- Amount of bleeding may be considerably greater than what is evident per vagina. A significant amount of blood can be trapped behind the abrupeted placenta.
- Be alert to the need for immediate cesarean section for fetal distress and/or dramatic increase in hemorrhage
- Best therapy for DIC is removal of the placenta by C-section or vaginal delivery
- Hemorrhage may continue postpartum from an atomic uterus that is refractory to the usual oxytocic agents
- C-section hysterectomy may be necessary, which may in itself be accompanied by large blood loss
- If multiple blood units are transfused, watch for dilutional thrombocytopenia.
Achondroplasia, Dwarfism

Minh Chau Joe Tran

**Risk**
- 1 per 15,000–40,000 births worldwide
- Females ≥ males
- No race predilection
- Most common type of dwarfism

**Perioperative Risks**
- Cervical spine instability
- Foramen magnum and cervical spine stenosis
- Restrictive pulmonary disease
- Thoracolumbar kyphosis

**Worry About**
- Central apnea
- Obstructive sleep apnea
- Cervicomedullary compression
- Cauda equina syndrome
- Paresthesia or paraplegia
- Nerve root compression

**Overview**
- Results from failure in development and premature ossification of bones that form cartilage. This leads to the characteristic frontal bossing, short arms and legs, maxillary hypoplasia, depressed nasal bridge, and trident hands.
- Other major features incl cervicomedullary compression; foramen magnum stenosis; small, flattened chest; RVH; restrictive lung disease; pulmonary hypertension; apraxia; thoracolumbar spinal stenosis; scoliosis; thoracolumbar kyphosis; and lumbar hyperlordosis
- Brainstem compression contributes to central apnea, whereas obstructive apnea is from midface structural abnormalities
- Mean adult male height is 131 ± 5.6 cm; the mean adult female height is 124 ± 5.9 cm (about 4 feet for both)
- Mean adult male weights 120 lbs (55 kg); the mean adult female weights 100 lbs (45 kg)
- Trunk length and intelligence are normal; life expectancy is normal
- Obesity is present in both sexes

**ICD-9-CM Code: 756.4 Chondrodystrophy**

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Megaloecephaly with frontal bossing, large mandible, chronic otitis media with hearing loss, choanal stenosis, narrow nasopharynx, cervical kyphosis with limited neck extension, cervical neck instability, foramen magnum stenosis, dental malocclusion and crowding</td>
<td>Recurrent otitis media Conductive hearing loss Apnea with cyanotic spells Speech problems</td>
<td>Limited neck ROM Limited ability to visualize glottic opening Nasopharyngoscopy</td>
<td>Cervical flexion/ extension neck films Hearing test</td>
</tr>
<tr>
<td>CV</td>
<td>Pulm Htn, RVH, RV strain</td>
<td>SOB with routine activities, fatigue, dizziness, syncope, supplemental oxygen use</td>
<td>JVD distention, lower extremity edema, hypoxia, cyanosis, arrhythmia</td>
<td>EKG, ECHO, heart catheterization</td>
</tr>
<tr>
<td>PULM</td>
<td>Restrictive lung disease, decreased FRC, OSA, hypoxemia, hypercapnia, apnea</td>
<td>Apnea with cyanotic spells, Daytime somnolence, SOB Recurrent respiratory infections Pneumothorax from positive pressure ventilation</td>
<td>Rib hypoplasia, pectus carinatum or excavatus</td>
<td>CXR, ABG, PFT, sleep study</td>
</tr>
<tr>
<td>GI</td>
<td>Obesity Gastric hypomobility</td>
<td>Reflux symptoms, recurrent aspiration, dysphagia, globus hystericus</td>
<td>BMI</td>
<td>EGD, CXR</td>
</tr>
<tr>
<td>CNS</td>
<td>Hydrocephalus, elevated ICP, hyperreflexia, hypertonia, clonus, hypotonia (in infancy and early childhood), central apnea from brainstem compression at the level of the foramen magnum, cervicomedullary compression</td>
<td>Headaches, vertigo, dizziness, paresis, pyramidal signs, cervical myelopathy, ataxia, incontinence, snoring, daytime somnolence, depression is common</td>
<td>Cranioventricular stenosis, foramen magnum stenosis, kinking of the medulla, increased lateral ventricular size, hypoplasia of the corpus callosum</td>
<td>Axial head MRT, MRI, MEP, SSEP, sleep study</td>
</tr>
<tr>
<td>MS</td>
<td>Pectus carinatum or excavatum, genu varum, narrow spinal canal, rhizomelic shortening of arms and legs, small thoracic cage</td>
<td>Delayed motor milestones, premature degenerative joint disease</td>
<td>Thoracolumbar kyphoscoliosis, proximal limbs &lt; distal limbs, brachydactyly and trident hand configuration, hyperextensibility of most joints (knees in particular), limited elbow extension and rotation</td>
<td>Spine films Bone scans</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- GI prophylaxis
- Review airway films and studies
- Assess pt using systems base approach
- Assume unstable cervical neck

**Monitoring**
- Standard ASA monitors

- Foley catheter; A-line; CVP; and frequent H/H checks for invasive cases with major fluid shifts
- MEP, SSEP for spinal cord surgeries

**Induction**
- Careful IV induction with controlled airway
- Prevent hypoxia, which can worsen pulm Htn

- No guidelines for ETT size and depth placement; have different sized ETTs on hand
- Keep neck neutral and avoid hyperextension or hyperflexion
• Prevent sudden drops in SVR, which hypo-perfuse the brain through the stenotic foramen magnum
• RA rarely indicated and can be anatomically challenging

**Maintenance**
• Pressure-controlled ventilation with careful attention to PAP
• MEP and SSEP with spinal surgeries
• Careful positioning

• OG tube for gastric decompression
• Increased sensitivity to muscle relaxants
• Use peripheral nerve stimulator

**Postoperative Period**
• Resp insufficiency with frequent ABG checks
• CXR
• Pain control
• ICU monitoring

**Anticipated Problems/Concerns**
• Difficult IV access
• SIDS: 2–5%
• Neurologic impairment
• Pain control and resp depression
• Postop ventilation
Acidosis, Lactic/Metabolic

Peter Schulman
Jeffrey Mako

Perioperative Implications

Preoperative Preparation
- Pts with metabolic acidosis may be hemodynamically unstable and demonstrate decreased responsiveness to inotropes and vasopressors
- Consider postponing surgery until the underlying cause is corrected, unless treatment requires immediate surgical intervention
- If surgery is urgent or emergent, consider ways to optimize the pt preop

Intraoperative
- Invasive monitoring may be indicated, depending on the severity of illness
- Goal for induction is hemodynamic stability
- Inotropes and vasopressors should be readily available
- Consider the need for pt to remain intubated postop

Postoperative Period
- Pt may require postop ICU care and prolonged mechanical ventilation

Anticipated Problems/Concerns
- Hemodynamic instability with decreased responsiveness to inotropes and vasopressors
- Compensation for profound metabolic acidosis may lead to acute resp failure
- Treatment with bicarbonate may paradoxically increase Paco, and worsen intracellular acidosis and respiratory status

Risk
- Incidence in USA: Unknown
- Present in a variety of disease states, from mild to severe systemic illness

Perioperative Risks
- Hemodynamic instability (due to arteriolar vasodilation and decreased cardiac output)
- Hyperkalemia
- Insulin resistance and hyperglycemia
- Acute respiratory failure

Worry About
- Decreased responsiveness to vasopressors and inotropes
- Decreased activity of local anesthetic agents
- Arhythmias

Overview
- Physiologic disturbance resulting from excess acid production, failure of organic acid excretion, or inappropriate bicarbonate loss causing increased serum acidity
- Marker of an underlying disease process
- Severe when, in the presence of resp compensation
- Marker of an underlying disease process
- Delta gap (ΔA): Used to determine the presence of concomitant metabolic derangements and calculated as: ΔAGoduca [HCO3]-, where ΔAG = (calculated AG - expected AG). ΔAG is used for clinical use.
- Normal AG metabolic acidosis: Associated with excess HCO3- loss from the kidney or Gl tract, failure of the kidney to excrete H+, or rapid IV infusion of bicarbonate-free solutions (e.g., normal saline)
- High AG metabolic acidosis: Results from an accumulation of excess acid in the serum. Specific causes are due to production of lactate or ketones (diabetic, alcoholic, or starvation ketoacidosis), toxic ingestion (methanol, ethylene glycol, salicylates), uremia, or medication side effects (propofol infusion syndrome, lactic acidosis associated with metformin)

ASSESSMENT POINTS

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<thead>
<tr>
<th>System</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEURO</td>
<td>Altered mental status, seizures</td>
<td>Level of consciousness, delirium, somnolence, nausea/vomiting, seizures, toxic ingestion</td>
<td>Obtunded, confused, somnolent</td>
<td>Toxicology screen, osmolar gap, serum lyses</td>
</tr>
<tr>
<td>CV</td>
<td>Arteriolar vasodilatation, hypotension, response to vasopressors &amp; inotropes, arrhythmias, hypokontracility</td>
<td>Signs of end-organ hyperperfusion</td>
<td>Tachycardia, hypotension, poor peripheral pulses, cold extremities, poor capillary refill</td>
<td>Invasive hemodynamic monitoring, ECHO, ECG</td>
</tr>
<tr>
<td>PULM</td>
<td>Hypoxemia, hyperventilation, resp failure</td>
<td>Tachypnea, dyspnea</td>
<td>Rapid &amp; shallow breathing, accessory muscle use, hypoxia, hypercarbia</td>
<td>CXR, ABG, pulse oximetry</td>
</tr>
<tr>
<td>RENAL</td>
<td>Oliguria, acute kidney injury, ATN</td>
<td>Urine output, chronic renal disease</td>
<td>Signs of hypo- or hyperperovlemia</td>
<td>UO, Cr, BUN, urine lytes, UA, serum lyses</td>
</tr>
<tr>
<td>GI</td>
<td>Nausea, vomiting, diarrhea, melena, abdominal pain</td>
<td>Abdominal pain to palpation</td>
<td>Serum lactate, radiographic imaging, upper/lower endoscopy</td>
<td></td>
</tr>
<tr>
<td>ID</td>
<td>Hyperglycemia, insulin resistance</td>
<td>DM, polyuria, polydipsia, hyperphagia</td>
<td>Signs of dehydration, signs of focal infection</td>
<td>Blood glucose, serum ketones</td>
</tr>
</tbody>
</table>

Acromegaly

**Overview**
Acromegaly is a slowly progressive, debilitating endocrinopathy resulting from excess secretion of growth hormone, usually from a benign macroadenoma of the pituitary gland, and characterized by overgrowth of soft tissues and bone and cartilage of skeleton (nose, jaw, hands, fingers, feet, toes). Excess growth hormone before puberty (epiphyseal closure) leads to gigantism (<5% of acromegalics)

**ICD-9-CM Code: 253.0**

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Bone and soft tissue overgrowth of head and neck</td>
<td>TMJ arthritis, Hoarseness, Deep voice</td>
<td>Enlarged frontal, nasal bones, Enlarged sinuses, Macroglossia with glossoptosis, Prognathism, Hypertrophy of larynx, Vocal cord thickening &amp; edema, Subglottic narrowing, Enlarged thyroid gland (25%) with possible tracheal compression/deviation</td>
<td>Indirect laryngoscopy, Lateral neck x-rays, CT of neck</td>
</tr>
<tr>
<td>CV</td>
<td>CAD, PVD, LV dysfunction, Cardiomyopathy</td>
<td>Chest pain, Htn, CHF, Dysrhythmias, Diastolic dysfunction</td>
<td>Htn, CHF, Dysrhythmias, Cardiomegaly, Diastolic dysfunction</td>
<td>CXR, ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Airway soft tissue overgrowth, Upper airway and small airway narrowing</td>
<td>Obstructive sleep apnea (60% of patients)</td>
<td>Barrel chest with kyphosis</td>
<td>PFTs (if indicated), Sleep study</td>
</tr>
<tr>
<td>RENAL</td>
<td>↑GI Ca²⁺ absorption, Hypercalciuria, ↑Total body Na⁺</td>
<td>Urolithiasis</td>
<td>Peripheral edema</td>
<td>To diagnose acromegaly: ↑24 h GH levels, Best screening test: ↑serum IGF I (insulin-like growth factor), Definitive test: Oral glucose tolerance test (GH levels do not ↓)</td>
</tr>
<tr>
<td>ENDO</td>
<td>↑BMR</td>
<td>Heat intolerance</td>
<td>Hyperhidrosis</td>
<td>TFTs</td>
</tr>
<tr>
<td>CNS</td>
<td>Pituitary mass effect</td>
<td>Headache, Hypersomnolence, Visual disturbances</td>
<td></td>
<td>CT, MRI (with gadolinium) to determine tumor size +/- extracranial expansion</td>
</tr>
<tr>
<td>PNS</td>
<td>Carpal tunnel syndrome</td>
<td>Paresthesias</td>
<td>Median nerve compression</td>
<td>EMG, NCVs</td>
</tr>
<tr>
<td>MS</td>
<td>Bone and soft tissue overgrowth, Osteoporosis, Myopathy</td>
<td>Arthralgias, Osteoarthritis (knees, hips, shoulders, LS spine), Fatigue, weakness</td>
<td>Enlarged hands and feet, Hip, knee, shoulder, low back pain, Muscle weakness</td>
<td>X-rays</td>
</tr>
</tbody>
</table>

**Risk**
- People within USA:
  - Prevalence is 40 cases/million; incidence is 3 new cases/million/y
  - Occurs with equal frequency in men and women and most frequently diagnosed in third to fifth decades of life (5–20 y lag between onset of symptoms and diagnosis)

**Perioperative Risks**
- Common conditions increasing periop risk incl airway abnormalities, cardiovascular dysfunction (Htn), resp impairment (obstructive sleep apnea), endocrine abnormalities (hyperglycemia)

**Worry About**
- Difficulty or inability to ventilate and/or intubate
- Extent of CV disease

**Postop airway obstruction**

**Usual Treatment**
- Surgery—primary therapy
- Transphenoidal pituitary microsurgery versus transcranial; transphenoidal more common and preferred, with less morbidity. Smaller tumors (<10 mm diameter) yield probable cure
- Pituitary radiation—reserved for persistent postsurgical disease or when surgery is contra-indicated
- Medical—adjuvant therapy or for nonsurgical candidates, effective if adenoma cells have dopamine and/or somatostatin receptors
  - Dopamine agonists—bromocriptine and cabergoline
  - Somatostatin analogue—octreotide and lanreotide

Perioperative Implications

Preoperative Preparation
- Optimize hemodynamics—BP control, no CHF
- Somatostatin analogue (octreotide) may shrink large macroadenoma

Monitoring
- Pulse oximeter may be difficult to fit (large fingers, toes); recommend A-line, brachial or femoral preferable

Airway
- Large masks, airways, blades, intubating LMA, tracheostomy equipment available
- Consider awake fiberoptic endotracheal intubation

Induction
- If GA, anticipate airway obstruction
- If hypopituitarism from mass effect, then may need hydrocortisone

Maintenance
- Possible lumbar drain if suprasellar extension
- Prophylactic antibiotics

Postoperative Period
- Transient diabetes insipidus (20%), permanent 1-9%
- CSF rhinorrhea <5% of patients
- Anterior pituitary insufficiency (ACTH, TSH, gonadotropins) (20%)—hormonal replacement with tapered cortisol therapy if necessary
- Meningitis, sinusitis, hematoma, cranial nerve palsy (III, IV, VI), nasal septal perforation, visual disturbances <1% each

Anticipated Problems/Concerns
- Airway management
- Hemodynamic stability
Acute Respiratory Distress Syndrome (ARDS)

Risk
- Incidence estimated at 15,000–140,000 cases per year in the USA. True incidence is unknown due to difficulty in defining the disease and making the diagnosis.
- Mortality rates vary from 25–40%. Mortality rate strongly influenced by associated conditions (e.g., higher when associated with sepsis, liver disease, and advanced age; lower with trauma, transfusion related lung injury, drug overdose, or other reversible conditions)

Perioperative Risks
- Increased risk of sudden and profound hypoxia secondary to loss of alveolar recruitment
- Worsening respiratory status due to effects of anesthesia and surgery
- Difficult balance between maintaining adequate intravascular volume and avoiding right ventricular dysfunction or worsening pulmonary edema leading to decreased oxygenation and ventilation

Worry About
- Maintaining required PEEP during pt transport with Ambu bag or Mapleson circuit. Transport with ICU ventilator may be necessary.
- Inability of standard OR ventilators to deliver required minute ventilation, high inspiratory pressures, and advanced modes of ventilation (bi-level, APRV, inverse ratio ventilation, high-frequency oscillatory ventilation)

Overview
- Defined as acute onset lung injury with Pao/FIO₂ ratio ≤ 200 mmHg (regardless of PEEP level), bilateral infiltrates on CXR, PCWP ≤ 18 when measured or no clinical evidence of cardiogenic edema. Criteria do not correlate well with lung histology and do not account for the effects of ventilator settings
- Though classically defined by severe hypoxia, also can be associated with profound hypercarbia due to elevated alveolar dead space
- Associated with low pulm compliance and lung volumes (due to alveolar edema and atelectasis) and, in certain pts, with abnormally low chest wall compliance
- Most deaths are from sepsis or multisystem organ failure (more rarely from refractory hypoxemia or hypercarbia)

ICD-9-CM Code: 518.81 (With respiratory failure)

Etiology
- Direct or indirect lung injury leading to acute inflammatory alveolar injury characterized by increased microvascular permeability with interstitial and alveolar edema and often progressing to fibrosis
- Precipitants incl aspiration, pneumonia, sepsis, massive transfusion, pancreatitis, trauma, ischemia-reperfusion, opiate or cocaine overdose, CNS injury, air embolism, cardiopulmonary bypass
- Mechanical ventilation may worsen lung injury through alveolar overdistension and shear forces from cyclic opening and closing of collapsed alveoli (ventilator-associated lung injury)

ASSESSMENT POINTS

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<tr>
<td>CV</td>
<td>Pulm Htn</td>
<td>Hypotension, ↓ renal and hepatic function, metabolic acidosis</td>
<td>Cool extremities, narrow pulse pressure, JVD, RV heave, peripheral edema, enlarged liver, abdominal distension</td>
<td>PA catheter, ECHO, mixed venous oxygen saturation</td>
</tr>
<tr>
<td>RESP</td>
<td>Ventilator-associated lung injury Pneumothorax</td>
<td>Airway pressures, impaired respiratory mechanics, worsening blood gases</td>
<td>Bilateral rhonchi, crackles decreased or absent breath sounds, tracheal deviation</td>
<td>CXR, CT chest</td>
</tr>
<tr>
<td>ID</td>
<td>Ventilator associated pneumonia Line sepsis</td>
<td>↑ WBC/bandemia, new infiltrates, hypotension</td>
<td>Fever, purulent secretions</td>
<td>CXR, CT chest, blood and sputum culture</td>
</tr>
<tr>
<td>GI</td>
<td>Hemorrhage</td>
<td>↓ Hct</td>
<td>Melena, bloody NG output</td>
<td>Esophagogastroduodenoscopy</td>
</tr>
<tr>
<td>GU</td>
<td>Acute kidney injury</td>
<td>Oliguria, increased creatinine</td>
<td>Peripheral edema</td>
<td>Serum creatinine</td>
</tr>
<tr>
<td>MS</td>
<td>Prolonged weakness Diaphragm atrophy</td>
<td>Pharmacologic paralysis, high-dose steroids, sepsis, prolonged ventilation</td>
<td>Polynuropathy, myopathy</td>
<td>Electromyography, muscle biopsy</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Assess current ventilator mode and settings in ICU and review last blood gas
- Assess pt preop hemodynamic and intravascular volume status
- Use PEEP valve for pt transport or consider transportation to OR on ICU ventilator
- Consider use of ICU ventilator intraop with concurrent total intravenous anesthesia, particularly when very high minute volumes and airway pressures are required
- Maintain comparable levels of mean airway pressure and minute volume when transitioning between modes or ventilators and when paralyzing the patient

Airway
- Avoid suctioning and unnecessary ETT disconnection. Even transient loss of PEEP may result in lung derecruitment and severe hypoxemia that is difficult to correct.

Monitoring
- CVP, PA catheter, or intraop TEE may be helpful in estimating intravascular volume status and ventricular function
- Closely monitor airway pressures (peak, plateau, mean airway), tidal volumes, minute ventilation
- Monitor oxygen saturation and obtain frequent blood gases. ETCO₂ may not be representative of arterial Pco₂ when dead space is high

Preinduction/Induction
- Expect increased shunt with increased FIO₂ and/or PEEP requirements due to loss of hypoxic pulmonary vasoconstriction caused by anesthetics
- Prepare for worsening respiratory mechanics and decreased ventilation in spontaneously breathing pt given anesthetics, narcotics, or muscle relaxants
- Lying pt in full supine position associated with elevated mean airway pressures and increased risk of aspiration (suction stomach via NG/OG tube before lying supine)

Maintenance
- Attention to fluid management to avoid right ventricular dysfunction or worsening pulmonary edema from excessive fluid administration
- Avoid decreased oxygen delivery due to low cardiac output and anemia
- Treat worsened hypoxemia with recruitment maneuvers (apply continuous airway pressure of 40 to 50 cm H₂O for 40 seconds) followed by increased PEEP

Postoperative Period
- Continued careful monitoring of hemodynamic and volume status
- Reduce FIO₂ and airway pressures as tolerated

Anticipated Problems/Concerns
- Sudden and profound hypoxia can occur if lung recruitment is lost during transport, movement, positioning, or surgical retraction.
Addison’s Disease

**Risk**
- Incidence in USA: 60–110 cases/million
- Incidence 5 or 6/million/y
- M:F ratio: 1:1.5–3.5

**Perioperative Risks**
- Hypotension, distributive shock, hyperkalemia
- Muscle weakness, anorexia, vomiting, diarrhea, decreased level of consciousness

**Worry About**
- Acute adrenal insufficiency leading to hypotension and refractory distributive shock
- Cardiac dysrhythmia caused by hyperkalemia
- Hypovolemia, electrolyte imbalance

**Overview**
- Addison disease is adrenal insufficiency due to primary undersecretion of glucocorticoids and mineralocorticoids by the adrenal cortex or decreased ACTH secretion
- A normal adult will secrete 20 mg of cortisol daily and up to 100 mg/m² daily during stress.
- A normal adult will secrete 0.1 mg of aldosterone daily.
- Addison disease may be subtle or overlooked by the pt until the stress of surgery leads to adrenal crisis

**Etiology**
- Most frequently due to idiopathic adrenal insufficiency secondary to autoimmune destruction of the adrenal gland (80% of cases)
- Other causes incl bacterial, fungal and viral infection, TB, HIV, sepsis, hemorrhage into the adrenal gland, cancer, amyloid disease, and chronic corticosteroid therapy

**ASSESSMENT POINTS**

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</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hypotension</td>
<td>Low blood pressure</td>
<td>Pale, diaphoresis</td>
<td>BP</td>
</tr>
<tr>
<td>HEME</td>
<td>Low cortisol and aldosterone</td>
<td>Muscle weakness, anorexia, vomiting, diarrhea</td>
<td>Decreased level of consciousness, hypotension, shock</td>
<td>ACTH stimulation test</td>
</tr>
<tr>
<td>GI</td>
<td>Hypovolemia, electrolyte abnormalities</td>
<td>Anorexia, vomiting, diarrhea</td>
<td>Poor skin turgor, orthostatic vital signs, poor capillary refill, dry mucous membranes</td>
<td>Chemistry panel</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperkalemia, hyponatremia, hypoglycemia</td>
<td>Weakness, cardiac disrhymia</td>
<td>Inability to stand from seated position</td>
<td>Chemistry panel</td>
</tr>
<tr>
<td>SKIN</td>
<td>Increased ACTH leading to increased melanocytes</td>
<td></td>
<td>Hyperpigmentation</td>
<td>ACTH stimulation test</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preinduction/Induction/Maintenance**
- Glucocorticoid and mineralcorticoid levels should be checked
- Glucocorticoid and mineralcorticoid treatment should be optimized
- Potassium level should be checked and replaced as needed
- Glucose level should be checked and replaced as needed

**Monitoring**
- Standard ASA monitors
- Arterial line and central line may be necessary in acute adrenal insufficiency

**General Anesthesia**
- Pre-induction: Confirm that pt corticosteroid and mineralocorticoid levels are optimized. Elective cases should be postponed until levels are optimized.
- Induction: Avoid etomidate as it suppresses adrenal function
- Maintenance: Monitor for hypotension, cautious use of muscle relaxants as reduced dose may be necessary
- Emergence: Emergency can be prolonged

**Postoperative Period**
- Must monitor pts for adrenal insufficiency as there have been reports of significant adrenal insufficiency into the postop period
- Continue steroid replacement for at least 24 hr postop
- Watch for complications of steroid use such as ulcers, infection, poor wound healing, glucose intolerance

**Regional Anesthesia**
- Effective in postponing the increase in cortisol

**Usual Treatment**
- Glucocorticoid, mineralocorticoid and electrolyte replacement. For example, prednisone 5 mg q am and 2.5 mg q pm or hydrocortisone 20 mg q am 10 mg q pm for glucocorticoid replacement and fludrocortisone 0.05 mg–0.1 mg daily for mineralocorticoid replacement.
- Acute adrenal insufficiency treatment: Supportive treatment with rapid isotonic solution, hydrocortisone IV 100 mg q 8 hr and electrolyte replacement

**Anticipated Problems/Concerns**
- The greatest danger comes from undiagnosed Addison disease. These pts may present with acute adrenal insufficiency intraop or postop secondary to surgical stress.
- Accurate diagnosis and treatment can be life saving. Refractory hypotension should alert clinicians to the possibility that the pt is adrenal insufficient.
- Glucocorticoid replacement and supportive care are the mainstays of treatment in the periop period
Adrenal Insufficiency, Acute or Secondary

Charles B. Hantler

Risk
- Risk of adrenal insufficiency: 1/1000–1/10,000 (if steroids used in prior year)
- With steroids >20 mg/d (cortisol equivalent), 57-14 d within 1 y (large variability in patient response to dose duration and timing of prior steroid use)
- Clinical signs worsen with stress, such as trauma, surgery, or infection

Perioperative Risks
- Increases CV instability, fever, CHF, electrolyte abnormalities
- High cardiac output failure, or low-output state (hypovolemia) with signs of tissue hypoperfusion
- Often evidence of systemic vasodilation with decreased reactivity to vasoconstrictors

Worry About
- GI; N/V; dehydration
- Anemia, neutropenia with androgen deficiency: rare
- CV response; decreased SVR, decreased left ventricular stroke work index and decreased vascular responsiveness to maintain perfusion pressure; steroids necessary for blood vessel responsiveness to catecholamines
- Hyperkalemia with or without hypotension (usually aldosterone deficiency); hypoglycemia, acidosis, hypercalcemia, and anemia; cardiac conduction abnormalities

Overview
- Adrenal insufficiency results from inadequate production of glucocorticoids (cortisol), mineralocorticoids (aldosterone), and/or androgens
- Adrenal insufficiency can be acute or chronic, primary or secondary

Primary adrenal insufficiency: Associated with >90% destruction of the adrenal glands and deficiency in both cortisol and aldosterone
- Secondary adrenal insufficiency develops from the hypothalamic-pituitary-adrenal axis dysfunction or failure
- May present without symptoms until stress
- Acute adrenal (addisonian) crisis may develop in periop period when another stress is present (infection, hemorrhage, or major or prolonged surgery), leading to hypotension, hyperkalemia, dehydration, and shock
- Adrenals secrete around 150 mg of cortisol in periop period, the production may increase up to 300 mg during the maximal stress
- Recovery of the adrenal function may take up to 9–12 m after withdrawal of exogenous steroids and the supplementation of the daily cortisol production is advised
- Critical illness–related corticosteroid insufficiency may develop from inadequate corticosteroid activity in relation to the severity of the patient’s illness
- Pts with community-acquired pneumonia, severe pancreatitis, acute or chronic liver failure, post-venous thrombosis, pts who underwent trauma with hemorrhagic shock or cardiac surgery, or pts who are being weaned from mechanical ventilation may benefit from glucocorticoid therapy, dosing recommendation requires further investigation
- Chronic adrenal insufficiency from use of steroids in prior year may manifest as weakness, fatigue, nausea, emesis, weight loss, and a variety of psychiatric disturbances
- Inadequate mineralocorticoid production can cause hyperkalemia, hyponatremia, and metabolic acidosis, with or without signs of dehydration

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<tr>
<td>CV</td>
<td>Dehydration, hypotension, high-output failure</td>
<td>Postural symptoms, fatigue; Wt loss, Hx of surgery on adrenals, pituitary</td>
<td>Low BP, postural drop, signs of dehydration</td>
<td>Hct BUN/Cre, adrenal, ACTH stimulation, insulin tolerance, metyrapone test</td>
</tr>
<tr>
<td>RESP</td>
<td>CHF (high or low output)</td>
<td>DOE, SOB</td>
<td>S$_{2}$, rules</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Dehydration, nausea, emesis</td>
<td>Appetite, Hx of emesis</td>
<td>See CV</td>
<td>Lytes</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, neutropenia</td>
<td>Hyperpigmentation (excess corticortropin)</td>
<td>Hct</td>
<td>WBC</td>
</tr>
<tr>
<td>CNS</td>
<td>Depression, confusion, psychosis</td>
<td></td>
<td>Reverses with replacement</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Weakness, potentiation of neuromuscular blockade</td>
<td></td>
<td>Nerve stimulator</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Consider periop steroid coverage if benefits outweigh risks if high index of suspicion of adrenal depression (e.g., supraphysiologic doses of steroids for >1 wk within last y)
- Correct electrolyte abnormalities, hypoglycemia, and dehydration prior to elective surgery
- Fluorocortisone with resistant aldosterone (K+ and Na+) abnormalities; glucose for hypoglycemia

Monitoring
- ECG for signs of abnormal conduction (QRS duration, u waves)
- Consider CVP, PCWP, or TEE if fluid/electrolyte and hemodynamic abnormalities
- Sodium, potassium, bicarbonate, and glucose

Airway
- None

Premedication/Induction
- Consider volume status with regard to hydration and choice of agents

Maintenance
- No hemodynamic instability: Follow electrolytes and glucose as needed
- Hemodynamic instability (hypotension)
  - R/O other causes, then consider hydrocortisone hemisuccinate, 25–100 mg IV, then 100 mg q 12–24 hr for 2 or 3 d
  - Fluid resuscitation as needed

Exubation
- Possible potentiation of nondepolarizing muscle relaxants with use of high-dose steroids; ensure adequate muscle relaxant reversal

Adjuvants
- Glucose, fluids, careful monitoring of temperature to avoid hyperthermia

Postoperative Period
- Stress steroids possibly required several days postop
- High steroid doses may be associated with decreased wound healing and immunosuppression with increased infection risk
- Consider prolonged steroid coverage if severe stress continues (e.g., severe trauma with multiple operations)
- Mineralocorticoid administration as needed; usually glucocorticoids have significant mineralocorticoid action

Anticipated Problems/Concerns
- Severe resistant hypotension, hyperthermia, and CNS abnormalities, such as confusion, coma, lethargy, may occur intraop or postop and may be unpredictable
- Syndrome may occur in severely traumatized pts without history of steroid use, with clinical picture of sepsis and associated abnormalities in adrenal function; Rx is life saving
Alcohol Abuse

Risk
- Incidence in USA: 10 percent of Americans, including physicians, will abuse alcohol at some point in their lives
- Third leading cause of death and disability, including 30% of traffic fatalities
- Male gender and family Hx major risk factors

Perioperative Risks
- Severe malnutrition as significant as ethanol-induced end-organ injury
- Risk of Htn, CVA, diabetes, GI disease
- Liver most severely affected organ
- Dilated cardiomyopathy
- Withdrawal symptoms can themselves be life-threatening

Worry About
- Concomitant use of other drugs: Amphetamines, cocaine, benzodiazepines
- Affects of chronic smoking, such as COPD and emphysema

Overview
- Disease characterized by addiction (compulsion and craving despite consequences) to alcohol
- Clinical syndromes related to direct effect of ETOH and secondary adaptive response to excess ETOH exposure
- ETOH rapidly absorbed and metabolized
- Hepatic dysfunction usually takes 10–15 y to develop
- Cirrhosis may develop after 1 or more acute episodes

ICD-9-CM Code: 303.0 (Acute)

Etymology
- Unknown: Likely multifactorial with environmental, genetic, and psychosocial components

Usual Treatment
- Recovery involves some or all of the following:
  - Detoxification: Inpatient, residential, day treatment, or outpatient
  - Evaluation for comorbid psychiatric disorder
  - Referral to Alcoholics Anonymous or other alcohol programs
  - Pharmacotherapy to help with Withdrawal and prevent relapse
    - Disulfiram (Antabuse): Acetaldehyde dehydrogenase inhibitor
    - Naltrexone (Revia): Pure opioid receptor antagonist, blunts ETOH’s pleasurable effects and reduces craving. Available as monthly IM depot.
    - Acamprosate (Campral): A synthetic derivative of homotaurine, a structural analog of gamma-aminobutyric acid (GABA). Decreases excitatory glutamatergic neurotransmission during alcohol withdrawal

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<td>CV</td>
<td>Cardiomyopathy, arrhythmias, hypertension</td>
<td>Orthopnea, nocturnal urination, coughing, leg swelling</td>
<td>Dypsnea</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>GI</td>
<td>Erosive gastritis, hepatic cirrhosis, acute hepatitis, pancreatitis, fatty liver</td>
<td>Hx of bleeding, easily bruised, anorexia, N/V</td>
<td>Ascites, jaundice Hepatomegaly, “spider” angiomias Abdominal pain, hepatomegaly</td>
<td>Upper endoscopy, stool guaiac LFTs Serum amylase Mg++; K+</td>
</tr>
<tr>
<td>ENDO</td>
<td>Gynecomastia, testicular atrophy, irregular menses</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Leukopenia, anemia, thrombocytopenia</td>
<td></td>
<td></td>
<td>CBC with differential</td>
</tr>
<tr>
<td>CNS</td>
<td>Wernicke’s syndrome Korsakoff’s syndrome Peripheral polyneuropathy Cerebellar degeneration</td>
<td>Amnesia, impaired reasoning</td>
<td>Sixth nerve palsy, ataxia CNS exam Distal numbness and paresthesias Unsteady gait</td>
<td>MRI or CT scan</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Gastric prophylaxis
- Blood ETOH and toxicology screen if indicated

Monitoring
- Standard ASA monitors
- Consider invasive monitors for cardiomyopathy, hepatic dysfunction, and/or end-organ compromise

Airway
- Consider full stomach in acute intoxication

Preinduction/Induction
- Consider long-acting benzodiazepine, barbiturate, or $\alpha_2$-adrenergic agonist
- Anesthetic doses increased in chronic disease
- Decreased dose in acute intoxication

Postoperative Period
- Provide adequate analgesia in PACU
- Anxiety can worsen withdrawal symptoms
- Withdrawal syndrome may develop within 6–8 hr; treat with IV ETOH, $\beta$-adrenergic agonist, $\alpha_2$-adrenergic agonist, benzodiazepines, PO ETOH
- DTs develop in 5% of pts in withdrawal

- Rapid sequence in acute intoxication
- Consider Rx of nutritional/metabolic deficiencies

Maintenance
- Requirements vary by age, general health, nutrition and hydration status, concomitant disease

Extubation
- Ensure return of airway reflexes

Adjuvants
- Long-term consumption of ETOH impairs hepatic metabolism
- Short-term consumption inhibits drug metabolism
- Polyneuropathy a relative contraindication to regional anesthesia
- Consider periop clonidine patch

Anticipated Problems/Concerns
- Recognition and treatment of withdrawal important, as significant mortality occurs if inadequately treated

10% mortality secondary to hypotension, arrhythmias; treat with diazepam, $\beta$-adrenergic agonist
Allergy

Jerrold H. Levy

Diseases

<table>
<thead>
<tr>
<th>Risk</th>
<th>Overview</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Incidence in USA: 5% of adults in are allergic to one or more drugs</td>
<td>• IgE anaphylaxis (type I immediate hypersensitivity reaction): Adverse response of host; mediated by antibodies, the antigen bridges with two IgE on the surface of basophils and mast cells; can be reproduced if foreign substance reinjected</td>
<td>• Clinical history of allergy or perianesthetic allergic reaction considered to put patient at increased risk for a reaction from neuromuscular blocking agents and induction agents</td>
</tr>
<tr>
<td>• During surgery, the risk of anaphylaxis is 1:3500–1:20,000, with a mortality rate of 4%</td>
<td>• Anaphylactoid reactions or histamine release: Describes a clinically indistinguishable syndrome probably involving similar mediators but not mediated by IgE antibody and not necessarily requiring previous exposure to the inciting substance, associated with vancomycin, benzylisoquinolinium-derived muscle relaxants, but term should be avoided.</td>
<td>• Preventive therapy with corticosteroids and antihistamines is of unproven value</td>
</tr>
<tr>
<td>• Females &gt; males (1.6:1)</td>
<td>• Clinical implications based on the reaction</td>
<td>• Severe allergic therapy: Stop antigen, maintain the airway with 100% O₂ and intubate if necessary; discontinue all anesthetic drugs, volume expansion, epinephrine (5–10 µg IV boluses as starting doses and titrate upward), antihistamines, β-sympathomimetic if bronchospasm, arginine vasopressin for refractory shock, phosphodiesterase inhibitors for RV dysfunction, airway evaluation prior to extubation, ICU observation</td>
</tr>
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Perioperative Risks

<table>
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<tr>
<th>Perioperative Risks</th>
<th>Worry About</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Intensity of sx variable: From an isolated cutaneous eruption to CV collapse and death</td>
<td>• Patient’s Hx: Knowledge of prior allergic event leads to avoiding drugs or other components involved</td>
</tr>
<tr>
<td>• CV, cutaneous, resp systems are mostly involved</td>
<td>• Hypotension, bronchospasm, and angioedema may become life-threatening events</td>
</tr>
<tr>
<td>• Increased morbidity and hospitalization time if intensive care required</td>
<td></td>
</tr>
</tbody>
</table>

Overview

• IgE anaphylaxis (type I immediate hypersensitivity reaction): Adverse response of host; mediated by antibodies, the antigen bridges with two IgE on the surface of basophils and mast cells; can be reproduced if foreign substance reinjected

Etiology

• Clinical history of allergy or perianesthetic allergic reaction considered to put patient at increased risk for a reaction from neuromuscular blocking agents and induction agents

Usual Treatment

• Preventive therapy with corticosteroids and antihistamines is of unproven value
• Severe allergic therapy: Stop antigen, maintain the airway with 100% O₂ and intubate if necessary; discontinue all anesthetic drugs, volume expansion, epinephrine (5–10 µg IV boluses as starting doses and titrate upward), antihistamines, β-sympathomimetic if bronchospasm, arginine vasopressin for refractory shock, phosphodiesterase inhibitors for RV dysfunction, airway evaluation prior to extubation, ICU observation

Anticipated Problems/Concerns

• For each pt who has a periop allergic reaction, consider evaluation 1 mo after with skin testing, antigen-specific IgE level dosage (radioallergosorbent test, ELISA).
• Measure tryptase if anaphylactic reaction within 1–2 hr of reaction, then 24 hr later to support diagnosis.
• Latex allergy incidence is increasing. Healthcare workers at greater risk, and Hx has to be evoked at the preanesthetic evaluation.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hypotension, tachycardia, dysrhythmias</td>
<td>BP</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>Pulm Htn</td>
<td>PA pressure</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cardiac arrest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Dyspnea, sneezing</td>
<td>Chest exam</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>Coughing, wheezing</td>
<td></td>
<td>PA catheter</td>
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<tr>
<td></td>
<td>Laryngeal edema</td>
<td></td>
<td>ETCO₂</td>
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<tr>
<td></td>
<td>Fulminant pulm edema</td>
<td></td>
<td>ABGs</td>
</tr>
<tr>
<td></td>
<td>Acute resp failure</td>
<td></td>
<td></td>
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<tr>
<td>DERM</td>
<td>Urticaria, flushing</td>
<td>Skin exam</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Perioral, periorbital edema</td>
<td></td>
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</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation

• Prick tests, intradermal testing: Anesthetic drugs (neuromuscular blocking agents)
• Most of the allergic reactions are unexpected. In case of established allergy, those drugs or latex should be strictly avoided.

Monitoring

• Routine

If major anaphylaxis occurs, consider pulm and radial arterial catheterization to guide therapeutic interventions.

Airway

• None, except specific care for the asthmatic patient

Preinduction/Induction/Maintenance/Extubation

• Slow injection of drugs. Avoid histamine-releasing drugs in high-risk pts.
## Amniotic Fluid Embolism

### Overview
- Amniotic fluid going to central circulation
- There are three necessary conditions:
  - Amniotomy (breach in the barrier between the intact fetal membranes that isolate amniotic fluid from the maternal circulation)
  - Laceration of endocardial or uterine vessels
  - Traditionally, it was thought that a pressure gradient (intrauterine pressure > CVP or uterine venous pressure) was needed, but the presence of an electrochemical gradient can provide the means for mediators of AFE to inflict damage
- Immunological factors may also be involved as complement activation may play a role in the pathophysiology of AFE

### ICD9-CM: 673.1

### Etiology
- Postulated mechanism of action: Powerful contractions force amniotic fluid into the maternal circulation through a defect in the fetal membranes, placenta, or elsewhere
- Risk factors incl turbulent labor; cesarean delivery; advanced maternal age; multiparity; meconium (present in 75% of cases); intrauterine fetal demise (present in 40% of cases); male fetus; sudden fetal expulsion; meconion staining of the amniotic fluid; chorioamnionitis; and macrosomia.

### Perioperative Risks
- Amniotic fluid embolism accounts for approx 10% of maternal deaths in the USA
- Mortality has been reported to be as high as 61–86% but more recent registries have reported mortality between 27–37% of pts
- Morbidity is also high as it is suggested that only 15% of survivors are neurologically intact

### Worry About
- Hypoxia
- Hypotension/cardio pulmonary collapse
- Heart failure (can have both right and left ventricular failure)
- DIC: Occurs in nearly all survivors of the initial catastrophic event
- Hemorrhage: 40% of amniotic fluid embolism-associated deaths are due to hemorrhage
- Altered mental status
- Seizures

### Key Reference:

## ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
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</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Tachycardia, Hypotension</td>
<td>Assessment by Hx</td>
<td>PE</td>
<td>Test</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxygenation, Pulmonary edema</td>
<td>Hypoxygenation</td>
<td>PE</td>
<td>Test</td>
</tr>
<tr>
<td>GI</td>
<td>Nausea</td>
<td>Vomiting</td>
<td>Excessive bleeding</td>
<td>PT, PTT, PL, fibrinogen, FSP</td>
</tr>
<tr>
<td>HEME</td>
<td>DIC</td>
<td>Anxiet</td>
<td>Convulsions</td>
<td>Shivering, Sweating</td>
</tr>
</tbody>
</table>

### Monitoring
- If amniotic fluid embolism is suspected, consider PA catheter to aspirate blood; hemodynamic management

### Maintenance
- Usually resuscitative with support of breathing and circulation
- Case reports of use of CPB, inhaled nitric oxide, ventricular assist devices

### Extubation
- If pt survives, keep intubated until stable

### Perioperative Implications
- Most common presentation is hemodynamic collapse

### Preoperative Preparation
- Maximize maternal oxygen delivery
- Place several large-bore IVs, consider central access for inotrope administration and fluid resuscitation
- Notify blood bank of anticipated coagulopathy and cross-match for several units of packed RBCs, FFP, platelets, and cryoprecipitate
- Consider preparing for cardiopulmonary bypass, if an option

### Usual Treatment
- Usually supportive to maintain oxygenation, circulatory support, and correct coagulopathy
- Case reports of successful treatment with cardiopulmonary bypass (both thrombectomy and placement of ventricular assist devices) have been reported in the literature
- Employ left uterine displacement to prevent aortocaval compression
- Stop oxytocin infusion if present
- Cardiopulmonary resuscitation, often requiring intubation with 100% O2/PEEP. Inhaled nitric oxide has also been described.
- Pressors and inotropes will often be required
- Delivery of fetus as soon as is practical; may require operative or cesarean delivery
- Replacement of clotting factors if pt develops DIC. Recent case reports describe use of recombinant factor VII (rFVIIa).

### Anticipated Problems/Concerns
- Not all sudden deaths during the peripartum period are due to amniotic fluid embolism. The pathologic diagnosis is quite specific (finding hair, mucin, or nucleated squamous cells in the maternal circulation), but its sensitivity is unknown.
- Even with early and aggressive intervention, AFE carries a high maternal and fetal mortality. Given that an AFE can occur unpredictably and then has such a high risk for morbidity and mortality, it can be devastating for the pt's families and healthcare providers. Psychological counseling for all parties involved should be considered to deal with any posttraumatic stress.
# Amyloidosis

**Kenneth J. Holroyd**

## Risk
- Incidence in USA: 1:50,000
- Race with highest prevalence: Unknown

## Perioperative Risks
- Increased risk of periop renal failure, CHF, bleeding from coagulopathy
- Autonomic neuropathy

## Worry About
- Signs of CHF
- Decreasing urine output

## Overview
- Extracellular deposition of amyloid-type proteins
- Congo-red stain of tissue reveals green birefringence in a polarizing microscope
- Associated end-stage renal, myocardial, and neuropathic disease
- Best diagnosed by subcutaneous abdominal fat pad aspirate or rectal biopsy

**ICD-9-CM Code:** 277.3

## Etiology
- Both acquired and hereditary forms exist
- Major risk factors for acquired disease: multiple myeloma, chronic infectious or inflammatory disease (osteomyelitis, rheumatoid arthritis)
- Hereditary forms very rare

## Usual Treatment
- Acquired: Treat underlying disease, stem cell transplant, Chem RX
- Hereditary: Colchicine, liver transplantation

## Key Reference:

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<tr>
<td>HEENT</td>
<td>Macroglossia</td>
<td>Enlarged tongue</td>
<td>Macroglossia</td>
<td>CT scan</td>
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<tr>
<td></td>
<td>Tracheal stenosis</td>
<td>Dyspnea</td>
<td>Stridor</td>
<td>Flow-volume loop</td>
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<tr>
<td>CV</td>
<td>Restrictive myopathy</td>
<td>Exercise tolerance</td>
<td>Dyspnea</td>
<td>S&lt;sub&gt;3&lt;/sub&gt;</td>
</tr>
<tr>
<td></td>
<td>LV and RV dysfunction</td>
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<td>Syncope</td>
<td>Bradycardia</td>
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<td>Conduction abnormalities</td>
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<td>ECHO</td>
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<td>RESP</td>
<td>CHF</td>
<td>Cough</td>
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<td></td>
<td>Lung nodules</td>
<td>Chest wall pain</td>
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<td>ECG</td>
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<td>GI</td>
<td>Autonomic dysfunction</td>
<td>Wt loss</td>
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<td>CXR</td>
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<td></td>
<td>Diarrhea</td>
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<tr>
<td>HEME</td>
<td>Factor X deficiency</td>
<td>Brusing</td>
<td></td>
<td>Factor X assay</td>
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<tr>
<td>RENAL</td>
<td>Decreased renal perfusion</td>
<td></td>
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<td>BUN/Cr urine</td>
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</tr>
<tr>
<td>CNS</td>
<td>Autonomic neuropathy</td>
<td>Inability to sweat; hoarseness; early satiety; postural dizziness</td>
<td>Orthostasis</td>
<td>Biopsy</td>
</tr>
</tbody>
</table>

## Perioperative Implications

### Preoperative Preparation
- Optimize treatment of heart failure
- Avoid dehydration (renal failure)

### Monitoring
- Consider PA catheter for large fluid shift operations or patients with severe LV dysfunction

### Airway
- Macroglossia or tracheal stenosis
- Increased risk of bleeding into airway from capillary fragility and possible coagulopathy

### Preinduction/Induction
- May develop reduced CO and hypotension
- Coagulopathy may contraindicate regional anesthesia

### Maintenance
- No agent or technique shown superior
- Maintain adequate urine output

### Extubation
- Patient fully awake to minimize risk of reintubation
- Use caution with nasal airway—may cause hemorrhage

### Postoperative Period
- Close monitoring of CV and renal status
- Consider ICU setting for postop care

### Adjuvants
- Avoid digoxin: not usually helpful in treating amyloid CHF, associated with increased arrhythmias

### Anticipated Problems/Concerns
- Difficult airway
- CHF
- Hypotension
- Renal failure
Amyotrophic Lateral Sclerosis

Todd A. Bromberg
Richard L. Boortz-Marx

Risk
- Estimated incidence of 1 to 3 per 100,000
- Mean age of onset is in the sixties, but ALS can occur as early as the twenties
- Disease duration is approximately 3 y from the time of diagnosis to death
- Slight male predominance of sporadic spinal ALS, slight female predominance of bulbar ALS
- Most cases are sporadic but 5–10% are familial
- The risk of anesthesia increases as the FVC falls below 50% such that ALS patients can be stratified as low risk if the FVC is greater than 50%, moderate risk if the FVC is 30–50%, and high risk if the FVC is less than 30%

Perioperative Risks
- Aspiration
- Resp depression
- Inability of pt to communicate secondary to bulbar weakness

Worry About
- Succinylcholine induced hyperkalemia
- Prolonged resp depression with inability to extubate, even without use of muscle relaxants
- Hypersensitivity to non-depolarizing neuromuscular blockers

Perioperative Implications

Preinduction/Induction/Maintenance
- Succinylcholine is contraindicated as it can cause hyperkalemia
- Non-depolarizing agents may be used, but anticipate prolonged weakness
- Short-acting muscle relaxants should be used when necessary

Preoperative Considerations
- Preop pulm function tests may help to predict anesthetic risk
- Consider aspiration prophylaxis
- Avoid opioids and benzodiazepines if possible

Monitoring
- Routine
- Anesthesia should be performed in an inpatient setting

General Anesthesia
- Avoid if possible
- May cause significant postop resp depression
- Diaphragmatic pacing stimulation may improve resp compliance and stimulate respirations
- Extubate when pt is fully awake

Regional Anesthesia
- May be preferred compared to general anesthesia
- Case reports have documented successful use of epidural anesthesia
- Minimize neural extent of blockade to reduce risk of resp depression

Postoperative Period
- Anticipate prolonged postop ventilation
- Use nonnedsing medications for pain control

Progressive bulbar palsy: Progressive motoneuron loss from lower cranial nerve nuclei and cervical spine

ICD9-CM: 335.20

Etiology
- Familial ALS caused by gene mutations: 14 mutations described. Most studied occurs in the gene encoding superoxide dismutase: forms aggregates leading to mitochondria and muscle complex dysfunction.
- Etiology of sporadic ALS remains uncertain, but autoimmune, infectious, and neurotoxic mechanisms likely contribute. An interaction between a genetic susceptibility and environmental exposure likely leads to the disease.

Usual Treatment
- Care is mainly supportive consisting of psychological therapy, symptom management, physical therapy, and palliative care
- Care in a multidisciplinary clinic is associated with prolonged survival and improved quality of life
- Riluzole, which inhibits glutamate release, is the only drug shown to improve survival. On average, patients live 2–3 mo longer on riluzole versus a placebo.

ASSESSMENT POINTS

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<thead>
<tr>
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<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Dysarthria, dysphagia, sialorrhea</td>
<td>Slurred speech, coughing with eating, drooling</td>
<td>Decreased gag reflex</td>
<td>Swallow study</td>
</tr>
<tr>
<td>CV</td>
<td>Reduced sympathetic tone</td>
<td>Syncope</td>
<td>Decreased breath sounds</td>
<td>Prolonged QTc</td>
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<tr>
<td></td>
<td>Vagal dysfunction</td>
<td>Cardiac arrest</td>
<td></td>
<td>Tachycardia</td>
</tr>
<tr>
<td>PULM</td>
<td>Aspiration; nocturnal apnea; weak cough</td>
<td>Recurrent pneumonia; nighttime arousals; lethargy</td>
<td></td>
<td>PFTs</td>
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<tr>
<td></td>
<td></td>
<td>Decreased breath sounds; coarse breath sounds</td>
<td></td>
<td>Nocturnal oximetry</td>
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<td>CXR</td>
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<td></td>
<td></td>
<td>ABG</td>
</tr>
<tr>
<td>GI</td>
<td>Malnourished</td>
<td>Caloric intake</td>
<td>BMI</td>
<td>Albumin</td>
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<tr>
<td></td>
<td></td>
<td>Food journal</td>
<td></td>
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</tr>
<tr>
<td>CNS</td>
<td>Motor neuron loss in spinal cord and brain</td>
<td>Weakness Pseudobulbar affect</td>
<td>Weakness Fasciculations Atrophy</td>
<td>EMG/NCS</td>
</tr>
</tbody>
</table>

Anaphylaxis

**Risk**
- Approximately 1 in 5000 anesthetic procedures
- Females outnumber males 3:1
- No prospective data to suggest an increased risk of generalized allergy, although Hx of atopy is overrepresented in several series of life-threatening anaphylaxis to anesthetic agents

**Perioperative Risks**
- Significant risks of life-threatening airway compromise, CV collapse, and bronchospasm—particularly severe in patients on β-blockers

**Worry About**
- Pts with pre-existing ASCVD tolerate CV sequelae poorly
- Pts with Hx of allergy to anesthetics
- Antibodies (and potentially anaphylaxis) to muscle relaxants may persist for >25 y

**Overview**
- The body’s response to what is perceived to be a foreign substance

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</tr>
</thead>
<tbody>
<tr>
<td><strong>HEENT</strong></td>
<td>Head and neck swelling, and potential glottic edema</td>
<td>Will occur suddenly</td>
<td>Swelling</td>
<td>Clinically obvious</td>
</tr>
<tr>
<td><strong>CV</strong></td>
<td>↑ HR, ↓ BP and SVR, ↑ ectopy, change in P-R interval, coronary vasospasm</td>
<td>Hypotension, tachycardia</td>
<td>ECG may reveal PVCs or change in P-R interval, CV collapse may ensue</td>
<td></td>
</tr>
<tr>
<td><strong>RESP</strong></td>
<td>Bronchospasm</td>
<td>Wheezing</td>
<td>↑ Peak insp pressure, ↓ O2 saturation</td>
<td></td>
</tr>
<tr>
<td><strong>SKIN</strong></td>
<td>Urticaria or other cutaneous manifestations, generalized edema with fluid leakage</td>
<td>Body rash</td>
<td>Not needed, CVP or PA pressures or TEE</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Monitoring**
- It is important to distinguish from drug effects or mechanical problems
- CV collapse with or without associated bronchospasm or cutaneous manifestations during induction, but without evidence of mechanical problems, suggest anaphylaxis
- Prophylactic H1 and H2 antagonists may attenuate the severity, although not the incidence
- The airway may swell, making intubation very difficult

**Induction**
- Reactions usually occur during induction. Consider administering antibiotics in the preop holding area or after rather than during induction.

**Maintenance**
- Perpetuation of reaction can occur, particularly if due to latex
- Significant cross-reactivity between myorelaxants (approaching 80%)  
- Avoid all muscle relaxants in pts with prior reactions

**Exubation**
- Ensure stable from a cardiorespiratory viewpoint
- Assess for airway edema

**Adjuvants**
- Epinephrine is drug of choice in true anaphylaxis, even in the face of tachycardia

**Postoperative Period**
- Blood should be drawn for possible tryptase levels. Although histamine measurements during the acute event can assist in Dx, they can be difficult to perform. Tryptase can be drawn up to 2 hr afterward and may be positive in anaphylaxis, but is not elevated in chemically mediated reactions.
- Skin testing may be done several weeks after initial event to assess etiologic agent

**Usual Treatment**
- IV fluids (put in large-bore IV), often to 7 L in adults
- Epinephrine even in the face of significant tachycardia
- O2 and supportive measures
- Possible H1, and H2 antagonists

**Anticipated Problems/Concerns**
- Early aggressive treatment may be critical
- Advise pts exactly what drugs they have received for future anesthetics
Anemia, Aplastic

**Risk**
- Incidence in USA: 2000 new cases/y
- 1:1 per million up to age 9
- Southeast Asia and South Africa have 10–20 times higher incidence
- Within USA, related to agricultural areas or petrochemical industry and chemical exposures

**Perioperative Risks**
- Infection
- Hemorrhage
- LV dysfunction due to high-output state and fluid overload

**Worry About**
- Sepsis
- Co-existing congenital anomalies, especially renal and cardiac
- Concomitant GI and intracranial hemorrhage
- Difficulty cross-matching blood products after previous multiple transfusions

**Overview**
- Self-perpetuating disorder resulting in pancytopenia due to a congenital or acquired loss of hemopoietic pluripotent stem cells

**ASSESSMENT POINTS**

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<tr>
<td>HEENT</td>
<td>Epistaxis</td>
<td>Headache</td>
<td>Stomatitis</td>
<td>CBC, differential, plt PT, PTT, CT scan</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm embolism</td>
<td>Dyspnea</td>
<td>Tachypnea Lung field consolidation Wheezing</td>
<td>CXR, V/Q scan CT scan ABGis, bronchoscopy</td>
</tr>
<tr>
<td>CV</td>
<td>LV failure</td>
<td>Dyspnea</td>
<td>Tachycardia, S</td>
<td>ECG</td>
</tr>
<tr>
<td>GI</td>
<td>GI bleeding</td>
<td>N/V, diarrhea</td>
<td>Acute abdomen</td>
<td>Endoscopy, bleeding scan</td>
</tr>
<tr>
<td>GI</td>
<td>GI GVHD</td>
<td>Melena</td>
<td>Hypoactive bowel sounds</td>
<td>Selective angiography</td>
</tr>
<tr>
<td>CNS</td>
<td>Microcephaly, meningitis, intracranial hemorrhage</td>
<td>Irritability, lethargy</td>
<td>Meningismus Papilledema</td>
<td>Lumbar puncture after coagulopathy treated, head CT, MRI</td>
</tr>
<tr>
<td>HEME</td>
<td>Pancreatitis</td>
<td>Bleeding gums, infections</td>
<td>Petechiae</td>
<td>CBC, differential</td>
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<tr>
<td></td>
<td>Leukemia</td>
<td>Easy bruising Fatigue</td>
<td>Retinal hemorrhage</td>
<td>Reticulocyte count</td>
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<td>Paroxysmal nocturnal hemoglobinuria</td>
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<td>Pallor</td>
<td>BM biopsy</td>
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<tr>
<td>METAB</td>
<td>Electrolyte abnormalities</td>
<td>Long-term hyperalimentation</td>
<td>Electrolytes</td>
<td>Ca++, Mg++, phosphate, albumin, transferrin</td>
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<td></td>
<td>Glucose intolerance</td>
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<td>Hypoproteinemia</td>
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</table>


**Perioperative Implications**

**Preoperative Preparation**
- Reverse isolation precautions
- Adequacy of blood products
- Severe neutropenia, co-existing congenital heart disease (HD) may warrant prophylactic anti-microbial therapy
- Avoid IM and rectal sedation
- Concomitant steroid therapy and necessity of stress doses should be considered

**Monitoring**
- Arterial line if indicated
- Consider CVP or PA catheter as indicated
- Urine output for new-onset hemoglobinuria as first sign of transfusion reaction

**Airway**
- Avoid nasal manipulation

**Use extreme caution with friable oral and pharyngeal mucosal surfaces**

**Preinduction/Induction**
- May exhibit hypotension and excessive fluid requirements to maintain adequate CO
- Central neuraxial blockade contraindicated in ongoing thrombocytopenia requiring transfusion
- Peripheral neural blockade may be approached cautiously if coagulation status is judged adequate

**Maintenance**
- PEEP assures adequate tissue oxygenation at lower FIO2 as hyperoxia depresses normal erythropoiesis and marrow function
- Nitrous oxide depresses bone marrow function even after brief exposure; best to use O2-air mixture
- Normothermia promotes coagulation

**Usual Treatment**
- Pts <55 y are managed with HLA-matched BMT or hematopoietic stem-cell transplant
- Pts >55 y or those unable to find HLA-matched donor receive immunosuppression and immunomodulation Rx incl ATG, cyclosporine, steroids, androgens, and G-CSF
- Hematopoietic growth factors such as G-CSF and GM-CSF may improve the short-term hematological recovery at the risk of long-term clonal evolution to myelodysplastic syndrome and AML.

**Drug-induced:** Chloramphenicol, NSAIDs, anti-epileptics, gold and sulfapyridine

**DISEASES**

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<td>RESP</td>
<td>Pulm embolism</td>
<td>Dyspnea</td>
<td>Tachypnea Lung field consolidation Wheezing</td>
<td>CXR, V/Q scan CT scan ABGis, bronchoscopy</td>
</tr>
<tr>
<td>CV</td>
<td>LV failure</td>
<td>Dyspnea</td>
<td>Tachycardia, S</td>
<td>ECG</td>
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<tr>
<td>GI</td>
<td>GI bleeding</td>
<td>N/V, diarrhea</td>
<td>Acute abdomen</td>
<td>Endoscopy, bleeding scan</td>
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<tr>
<td>GI</td>
<td>GI GVHD</td>
<td>Melena</td>
<td>Hypoactive bowel sounds</td>
<td>Selective angiography</td>
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<tr>
<td>CNS</td>
<td>Microcephaly, meningitis, intracranial hemorrhage</td>
<td>Irritability, lethargy</td>
<td>Meningismus Papilledema</td>
<td>Lumbar puncture after coagulopathy treated, head CT, MRI</td>
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<tr>
<td>HEME</td>
<td>Pancreatitis</td>
<td>Bleeding gums, infections</td>
<td>Petechiae</td>
<td>CBC, differential</td>
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<tr>
<td></td>
<td>Leukemia</td>
<td>Easy bruising Fatigue</td>
<td>Retinal hemorrhage</td>
<td>Reticulocyte count</td>
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<td></td>
<td>Paroxysmal nocturnal hemoglobinuria</td>
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<td>Pallor</td>
<td>BM biopsy</td>
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<tr>
<td>METAB</td>
<td>Electrolyte abnormalities</td>
<td>Long-term hyperalimentation</td>
<td>Electrolytes</td>
<td>Ca++, Mg++, phosphate, albumin, transferrin</td>
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<tr>
<td></td>
<td>Glucose intolerance</td>
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<tr>
<td></td>
<td>Hypoproteinemia</td>
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</tbody>
</table>

Anemia, Chronic Disease/Inflammation

Hayden R. Hughes

Risk

• Incidence in USA: All anemia, 8%; ACD/I second most common form
• Having chronic infectious, inflammatory, or malignant conditions
• ≥130 million Americans living with chronic diseases

Perioperative Risks

• Risks related to underlying diseases
• Transfusion related risks; e.g., TRALI, hemolytic reactions, immunosuppression
• Risks related to compensatory mechanisms for increasing O₂ delivery; e.g., angina, heart failure, dysrhythmias

Overview

• WHO definition of anemia: Children 6 mo–6 y; Hgb <11 g/dl; 6–14 y; Hgb <12 g/dl; nonpregnant females: Hgb <12 g/dl; pregnant females: Hgb <11 g/dl; males: Hgb <13 g/dl
• Normochromic, normocytic with low reticulocyte count
• ACD/I due to disturbances of Fe homeostasis due to diversion of Fe from the circulation into storage sites within the reticuloendothelial system
• Usually mild with Hgb 8–11 g/dl

ICD-9-CM Code: 285.21 Anemia in chronic kidney disease

Etiology

• Relative Fe deficiency
• Certain treatments for chronic conditions

Usual Treatment

• Treatment of underlying disease
• Fe, folic acid, cobalamin supplementation
• Uman recombinant erythropoietin
• Allogeneic blood transfusion

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hyperdynamic circulation</td>
<td>Palpitation</td>
<td>Tachycardia</td>
<td>ECG</td>
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<tr>
<td></td>
<td>Myocardial ischemia</td>
<td>Pounding pulse</td>
<td>Wide pulse pressure</td>
<td>Exercise ECG</td>
</tr>
<tr>
<td></td>
<td>CHF</td>
<td>Angina Sx, dyspnea</td>
<td>Exercise intolerance</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Dyspnea</td>
<td>Blood in stool</td>
<td>Occult blood in stool</td>
<td>See CV</td>
</tr>
<tr>
<td>Gl</td>
<td>Chronic blood loss</td>
<td>Angina equivalent (pain, nausea, indigestion)</td>
<td></td>
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<td></td>
<td>Hypoperfusion</td>
<td></td>
<td></td>
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<tr>
<td>HEME</td>
<td>Hgb below WHO definition level (see Overview)</td>
<td>↓ In exercise tolerance</td>
<td>Hgb</td>
<td></td>
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<tr>
<td>RENAL</td>
<td>Chronic renal failure</td>
<td>↓ Urine output</td>
<td>Shunt</td>
<td>Cr</td>
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<tr>
<td></td>
<td>Dialysis</td>
<td></td>
<td></td>
<td>K⁺</td>
</tr>
<tr>
<td>CNS</td>
<td>Decreased cerebral O₂ delivery</td>
<td>Dizziness</td>
<td></td>
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<td></td>
<td>Headache</td>
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<tr>
<td></td>
<td>Transient cerebral ischemia</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>MS</td>
<td>Low exercise capacity</td>
<td>Fatigue</td>
<td></td>
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</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation

• Standard monitoring
• Warm the room
• CVP, Hgb, electrolytes
• ST-segment analysis in pts with signs of CAD
• PA catheter for large fluid shifts or pts with signs of LV dysfunction or advanced renal failure
• ABG

Airway

• None

Preinduction/Induction

• Prehydrate liberally if CV status will tolerate
• Avoid CO reduction

• Avoid hypoxemia
• Choose drugs according to underlying conditions

Maintenance

• Avoid hypoxemia
• Maintain CO
• Avoid hypovolemia
• Keep pt warm
• Maintain Hgb above critical level for pt taking comorbidities into account

Extubation

• Keep pt warm
• Maintain high Paco₂
• In pt with CAD, this is the period of greatest risk for ischemia

Postoperative Period

• Keep pt warm, prevent shivering
• Maintain high Paco₂

Adjuvants

• According to underlying disorder

Anticipated Problems/Concerns

• Myocardial ischemia/infarction or CHF in pts with concomitant CAD
• Deterioration of renal function in pts with CRI
• Prolonged effects of drugs in pts with impaired renal and/or hepatic function
Anemia, Hemolytic

**Risk**
- Autoimmune disorders (SLE, RA, scleroderma, cold agglutinin disease)
- Lymphoproliferative disorders (CLL, NHL)
- Prosthetic heart valves (ball-and-cage, and bileaflet valves). Usually subclinical, but can be severe in up to 15% of pts
- Family history of hemoglobinopathies or RBC membrane defects (thalassemia, sickle cell disease, G6PD deficiency, spheroctysis)
- Exposure to drugs (cephalosporins, penicillins, NSAIDs) or other chemicals (naphthalene, fava beans)

**Perioperative Risks**
- Anemia, hypoxia
- Underlying CV compromise
- Splenomegaly in pts with extravascular hemolysis (within the reticuloendothelial system).
- Renal failure due to massive hemolysis (cold agglutinin hemolysis, sickling, drug reaction, etc.)
- Varying levels of liver disease depending on type of hemolytic anemia. Synthetic function of liver is usually normal, but in severe cases can be compromised.

**Worry About**
- Uncompensated anemia in pts with sub-acute hemolysis
- Periop hemolysis and/or hypoxia
- Need for transfusion and/or fluids

**Overview**
- Pts with hemolytic anemia may present with any of the following: fatigue, angina, SOB, tachypnea, tachycardia, or jaundice. The hemolysis can lead to changes in blood viscosity, gallstone production, splenomegaly, and renal failure in severe cases. Many pts will be both iron and folate deficient.
- Epidemiology varies by population. For example, G6PD is an X-linked condition and its prevalence is near 50% in Kurdish Jews, but around 1:1000 in North American and European populations
- Other things to consider incl monitoring periodic Hct levels, and administering prophylactic antibiotics/vaccinations to pts who have had a splenectomy.

**ICD-9-CM Code: 282., 283.**

**Etiology**
- Multiple causes; see Risk section (RBC structural abnormalities, autoimmune reaction, enzyme deficiency, hemoglobinopathies, mechanical heart valves, drugs, etc.)

**Usual Treatment**
- Treatment depends on etiology
  - Autoimmune: corticosteroids, plasmapheresis, packed RBC: transfusion for symptomatic pts, supportive care
  - Drug induced: Discontinuation of offending medication, corticosteroids, supportive care
  - Prosthetic valve: Cardiology consult and transfusion if symptoms rapidly worsen
  - RBC membrane defect: Splenectomy and supportive care
  - Enzyme deficiency: Avoidance of triggers, splenectomy, supportive care

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Dehydration</td>
<td>Fatigue, dizziness</td>
<td>Hypotension, weak pulses, increased capillary refill</td>
<td>CBC, BNP</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td>Fatigue, SOB, dizziness</td>
<td>Jaundice, pallor, splenomegaly</td>
<td>Hgb, Hct, reticulocyte count, indirect bilirubin, LDH</td>
</tr>
<tr>
<td>RENAL</td>
<td>Hemoglobinuria, acute renal failure</td>
<td>Dark urine (episodic)</td>
<td>Possible Htn, resp rate changes</td>
<td>Urine analysis, BUN, Cr</td>
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<tr>
<td>GI</td>
<td>Liver disease</td>
<td>Hepatosplenomegaly</td>
<td>LFTs</td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preinduction/Induction/Maintenance**
- Preop management and treatment of underlying cause of hemolytic anemia
- The test obtained periop depends on the etiology, severity, and chronicity of the hemolytic anemia.
- Avoidance of hypoxia, hypercarbia, acidosis, low-flow conditions, and hypothermia
- Optimize CV status with adequate hydration; consider IV fluid treatment the day before surgery if hypovolemic
- RBC transfusion may be considered to improve O2 carrying capacity depending on etiology (most common in patients with sickle cell disease)

**Monitoring**
- Standard monitors and urine output, CV status, O2 saturation (pulse oximetry), and temp regulation (avoiding hypothermia)

**General Anesthesia**
- Choice of anesthetic technique can vary, but all approaches should have the goal of avoiding hypoxia, hypercarbia, acidosis, stasis, low-flow conditions, and hypothermia
- Avoidance of hypoventilation

**Regional Anesthesia**
- Goals for regional anesthesia are the same as for general anesthesia. No specific contraindications.

**Postoperative Period**
- Supplemental O2 therapy
- Adequate hydration
- Early ambulation
- Continued temp regulation
- Active pulm toilet

**Anticipated Problems/Concerns**
- Acute periop hemolysis; may warrant transfusion
- Periop sickling event due to hypoxia, acidosis, hypothermia, or low flow. Sickling can be decreased by increasing arterial oxygen tension.
- Hypothermia-induced cold agglutinin hemolysis; decreased by maintaining normothermia
- Hypoxia and end-organ damage

Angina, Chronic Stable

Risk
- Incidence in USA: 3 million
- Annual rates per 1000 new episodes of angina for non-black men are 28.3 for ages 65–74, 36.3 for ages 75–84, and 33.0 for age 85 and older. For non-black women in the same age groups, the rates are 14.1, 20.0 and 22.9, respectively. For black men, the rates are 22.4, 33.8 and 39.5, and for black women, the rates are 15.3, 23.6 and 35.9, respectively
- African Americans have highest death rates

Perioperative Risks
- Increased risk of periop MI and death varies, depending on study (3–12%)
- Risk of LV dysfunction, hypotension, MI

Worry About
- Increasing frequency of symptoms
- Signs of LV dysfunction with ischemia
- Silent myocardial ischemia

Overview
- Chronic stable angina identifies pts at risk for developing myocardial ischemia and MI
- Angina is present in <25% of episodes of myocardial ischemia
- Symptoms should be stable for previous 60 d for “stable” diagnosis
- Can result from:
  - Inadequacy of myocardial O₂ supply in pts with critical coronary artery stenosis
  - Coronary vasospasm
  - Inadequacy of myocardial O₂ supply 2° to increased demand from ventricular hypertrophy
  - Endothelial cell-mediated vasoconstriction

Perioperative Implications

Preoperative Preparation
- Continuation of chronic anti-anginal medications associated with a lower incidence of myocardial ischemia/infarction, especially beta blockers, statins, and antiplatelet agents

Monitoring
- ST-segment analysis
- PA catheter for large fluid shift operations or pts with signs of LV dysfunction, although RCT unable to document benefits of routine monitoring
- TEE most sensitive, but technical issues of real-time interpretation

Airway
- None

Preinduction/Induction
- May develop reduced CO and hypotension with ischemia
- Avoid tachycardia, hypotension

Maintenance
- Myocardial ischemia may manifest as
  - CV instability
  - Intraop myocardial ischemia
  - Reduced CO, increased PCWP
- No one agent or technique shown superior
- Maintain normothermia, adequate hematocrit (≥28%)

Extrusion
- Period at greatest risk for developing ischemia

Postoperative Period
- Pain management may be critical

Adjuvants
- β-adrenergic receptor antagonist, nitroglycerin, Ca²⁺-channel blockers

Anticipated Problems/Concerns
- Pts with angina who develop dyspnea on exertion are at greatest risk for developing periop cardiac complications
- Exercise tolerance may be the best predictor of periop risk. Pts with a good exercise tolerance may not require further evaluation for less-invasive procedures.
- Pts who develop periop MI are at increased risk of periop death and long-term morbidity/mortality. Elevated troponin also associated with worse long-term outcomes.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Myocardial ischemia</td>
<td>Angina Xs</td>
<td>Displaced posterior maximal impulse S₃</td>
<td>ECG</td>
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<tr>
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<td>LV dysfunction</td>
<td>Angina-equivalent Xs</td>
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<td>Exercise ECG</td>
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<td>Dyspnea</td>
<td>S₃</td>
<td>Exercise radionuclide scintigraphy</td>
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<td></td>
<td>Exercise tolerance</td>
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<td>Pharmacologic stress testing</td>
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<td>ECHO</td>
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<td>Coronary angiography</td>
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<td>Coronary CT</td>
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<tr>
<td>RESP</td>
<td>CHF</td>
<td>Dyspnea</td>
<td>Rales</td>
<td>CXR</td>
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<td></td>
<td></td>
<td>Nighttime cough</td>
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<td></td>
<td></td>
<td>Orthopnea</td>
<td>Wheezing</td>
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<td></td>
<td></td>
<td>Chest tightness</td>
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<tr>
<td>GI</td>
<td>Angina-equivalent Xs</td>
<td>Angina-equivalent Xs</td>
<td>See CV assessment</td>
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<tr>
<td></td>
<td>Renal perfusion</td>
<td>Upper OUGH pain</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Nausea, indigestion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>↓ Renal perfusion</td>
<td>↑ UO at night</td>
<td>Cr</td>
<td>Exercise stress test</td>
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<tr>
<td>CNS</td>
<td>Angina-equivalent Xs</td>
<td>Syncope</td>
<td>See CV assessment</td>
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</tr>
<tr>
<td></td>
<td>Arm pain/neck pain</td>
<td>Syncope with chest pain</td>
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</tbody>
</table>

Anhidrosis (Congenital Anhidrotic Ectodermal Dysplasia)  

**Risk**
- Rare, 1:125,000,000
- Clusters in Japan and Israel

**Perioperative Risks**
- Impaired thermoregulation (risk of hyperthermia in infants)
- Postop chest infections

**Worry About**
- Absence of sweat leads to impaired thermoregulation
- Insensitivity to superficial and deep painful stimuli with intact tactile perception. Still require considerable amounts of inhalational anesthetics to maintain hemodynamic stability.

**Overview**
- Innervation of the eccrine sweat glands is lacking; heat loss by evaporation is impaired
- Absent mucous glands from resp tract and esophagus; frequent resp infections
- Partial or complete absence of teeth
- Hypotrichosis (absent hair)
- Self-mutilating behavior and mental retardation
- Characteristic facies: Prominent supraorbital ridges, depressed bridge and root of nose, large deformed ears, thick lips, underdeveloped maxilla and mandible

**ICD-9-CM Code:** 705.0

**Assessment Points**

<table>
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<th>System</th>
<th>Effect</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Airway anomalies</td>
<td>Snoring</td>
<td>Difficult breathing</td>
</tr>
<tr>
<td>RESP</td>
<td>Decreased mucus</td>
<td>Repeated infections</td>
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</tr>
<tr>
<td>OPHTHAL</td>
<td>Decreased lacrimation</td>
<td>Dryness, ulceration</td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Hyperpyrexia</td>
<td>Record/monitor temp</td>
<td></td>
</tr>
</tbody>
</table>


**Preoperative Preparation**
- Avoid anticholinergic premedication; however atropine has been used to treat bradycardia

**Monitoring**
- Routine
- Temp

**Airway**
- Awkward mask fit

**Maintenance**
- Regional anesthesia may be preferable when possible
- Humidify anesthetic gases
- Controlled room temp to avoid hyperthermia

**Extubation**
- Vigorous postop chest physical therapy

**Adjuvants**
- Protect eyes with tape and ophthalmic ointment (lacrimation is reduced)

**Anticipated Problems/Concerns**
- Difficult airway (mask and/or intubation)
- Hyperthermia
- Postop chest infections
- High incidence of CV events (hypotension and bradycardia) reported

**Etiology**
- Sex-linked recessive disorder
- Human *TRKA (NTRK1)* encodes the receptor tyrosine kinases (RTKs) for nerve growth factor (NGF) and is the gene responsible
- Full expression only in males; carrier females may be mildly affected

**Usual Treatment**
- Protect from risks of hyperpyrexia due to infection, hot weather, vigorous exercise
Ankylosing Spondylitis

### Risk
- 1:2000 incidence in Caucasians, rare in non-Caucasians
- M:F: 10:1; more severe in males
- 18–50% incidence in Native Americans

### Perioperative Risks
- Difficult airway, atlantoaxial instability
- "Bamboo spine" with potential for fracture during airway manipulation
- Rigid chest with difficult ventilation
- Myocarditis, myocardial conduction defects
- Increased blood loss due to abnormal chest structure, mechanics

### Worry About
- Inability to intubate, spine fracture, arrhythmia, inability to ventilate, massive blood loss
- Airway edema after extubation

### Overview
- An arthritic process, seronegative for rheumatoid factor, that attacks ligamentous attachments of the spinal column
- Characterized by low back pain, sacroiliitis, multiplane rigidity of spine, chest stiffness, uveitis, and insidious onset at <40 y of age
- Autosomal dominant and strongly prevalent among first-degree relatives

### ICD-9–CM Code: 720.00

### Etymology
- Unknown
- Genetic transmission led to discovery of a genetic marker, HLA-B27. Also involved are the major histocompatibility complex, numerous HLA-B27 subtypes, and IL23R (also associated with ulcerative colitis) and ERAP-1.

### ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Uveitis</td>
<td>Visual disturbance</td>
<td>Funduscopic exam</td>
<td>Fiberoptic nasopharyngoscopy</td>
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<td></td>
<td>TMJ arthritis</td>
<td>Limited mouth opening, jaw pain, voice abnormality</td>
<td>Airway exam, indirect laryngoscopy</td>
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<tr>
<td></td>
<td>Articlenoid deviation</td>
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</tr>
<tr>
<td>CV</td>
<td>Cardiomyopathy, conduction defects</td>
<td>SOB, chest pain, palpitation</td>
<td>Distant heart sounds, rales, arrhythmia</td>
<td>ECG, CXR, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Pleuritic inflammation, chest rigidity</td>
<td>Chest pain, limited exercise tolerance</td>
<td>Decreased breath sounds, chest excursion</td>
<td>Pulm function tests, CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Irritable bowel syndrome</td>
<td>Abdominal pain, bowel dysfunction</td>
<td>Abdominal pain</td>
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<tr>
<td>GU</td>
<td>Chronic prostatitis</td>
<td>Pain with urination</td>
<td>Rectal exam</td>
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<tr>
<td>CNS</td>
<td>Atlantoaxial subluxation, occult spine fracture</td>
<td>Long tract signs, sphincter abnormality Sometimes no symptoms</td>
<td>Basic neurologic exam</td>
<td>Cervical spine x-ray with flexion-extension, MRI</td>
</tr>
<tr>
<td>PNS</td>
<td>Radiculopathy</td>
<td>Radiating pain in extremities</td>
<td>ROM of the extremity</td>
<td>EMG (medicolegal use)</td>
</tr>
<tr>
<td>MS</td>
<td>Back pain, sacroiliitis, joint ankylosis, kyphosis (&quot;chin on chest&quot;), &quot;bamboo spine,&quot; spondyloarthritis</td>
<td>Review of skeletal function</td>
<td>Spine, skeleton</td>
<td>Radiologic studies</td>
</tr>
</tbody>
</table>


### Perioperative Implications

#### Preoperative Preparation
- Airway evaluation, pulm function assessment; consider positioning difficulties
- Antisalagogue for awake intubation
- Review MRI of the spine

#### Monitoring
- ST-segment analysis; pulm artery catheter if severe myocardial dysfunction
- Arterial line, central venous access for extensive osteotomy secondary to blood loss

#### Airway
- Inability to intubate possible, owing to cervical spine fusion, distortion. Fiberoptic intubation may be necessary. Cervical spine instability possible. Spine fracture possible with airway manipulation. Occult spine fracture may already be present.
- Increasing role for videolaryngoscopy

#### Induction
- If general anesthesia, any approach acceptable. If limited cardiac reserves, avoid depressants of myocardial contractility.
- If regional, skeletal abnormality can make the block difficult to perform, and response to injection is unpredictable. In some cases, epidural space is obliterated and cannot be completely accessed. Strongly consider paramedian approach to central block. If local anesthetic toxicity, airway management can be difficult.

#### Maintenance
- With positive pressure ventilation, decrease tidal volume and increase rate
- High ventilating pressure may predict large blood loss

#### Exubation
- Awake is preferable
- Airway edema possible after extensive anterior osteotomy, decompression and/or fusion. Compression of the airway from retropharyngeal hematoma is possible. Consider leak test prior to extubation, or maintaining the pt intubated and sedation for 12–24 hr postop

#### Adjuncts
- Ischemic optic neuropathy with prolonged procedures in the prone position

#### Postoperative
- Comfortable position, pain control without airway embarrassment

### Usual Treatment
- Symptomatic, with exercise, NSAIDs, immunosuppression can be tried in severe cases
- Wedge osteotomy is a drastic surgical intervention
- Infliximab—monoclonal antibody specific for tumor necrosis factor (TNF)
- Enanercept—anti TNF protein
- Adalimumab—monoclonal antibody specific for tumor necrosis factor (TNF)

### Anticipated Problems/Concerns
- Airway control
- The extreme distortion of the spine, esp. the neck, may make intubating trachea and ventilating pt very difficult
- Any airway compromise or depression of ventilation can result in catastrophe
- Depression of ventilation with opiate analgesics can be dangerous
- Pulm function
- Because of abnormal mechanics of the thorax and neck, the ability to ensure normal oxygenation during surgery and in the postop period can be a potential problem
- Regional anesthesia
- Placement of spinal, epidural, or caudal block could be technically very difficult. Action of local anesthetics in the central axis could be unpredictable
- Prolonged postop intubation
- Substantial blood loss, fluid/blood product administration, and the prone position make airway edema likely, requiring extended postop intubation necessary. Pt should be informed preop to avoid postop panic.
Anomalous Pulmonary Venous Drainage

Risk
- 1% of all congenital heart defects
- Total anomalous pulmonary venous drainage (TAPVD), the severe form, or partial anomalous pulmonary venous drainage (PAPVD), the less severe form, exists when pulmonary veins drain into the venous circulation
- M/F 4:1 in infradiaphragmatic type

Perioperative Risks
- Rapid CV deterioration secondary to hypercapnia and resultant acidosis
- Sudden pulmonary HTN and RHF during hypoventilation
- Periop mortality: 2–20% depending on preop status

Worry About
- Air bubbles entering the venous circuit
- Endocarditis risk

Overview
- TAPVD incompatible with life unless an ASD allows adequate R→L shunting of blood. TAPVD pts with small ASDs are more critically ill and often require balloon septostomy as a bridge to surgery. Some cyanosis, usually with O₂ satura-
tions of 85–95%.
- Increased flow through pulmonary vascular beds, resulting in pulm HTN
- Four types of TAPVD:
  - Supracardiac: Pulm veins connect to the left inominate vein via an anomalous “vertical vein” or connect to right SVC via an anomalous “short connecting vein,” or connect to the left SVC (45%)
  - Cardiac: Pulm veins drain into coronary sinus or directly into the right atrium (23%)

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Hypoxemia</td>
<td>Decreased activity level</td>
<td>Rales</td>
<td>ECG—RVH, RAH</td>
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<tr>
<td>CV</td>
<td>CHF, Hypoxemia</td>
<td>Dyspnea</td>
<td>Cyanosis</td>
<td>ECHO; catherization, Cardiac consultation</td>
</tr>
<tr>
<td></td>
<td>Monitoring problems</td>
<td>Anomalous peripheral vessels</td>
<td>Pulses and blood pressures in all four extremities</td>
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<tr>
<td>RESP</td>
<td>Hypoxemia</td>
<td>Bronchospasm</td>
<td>Wheezing</td>
<td>CXR</td>
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<tr>
<td></td>
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<td>SOB, Pulmonary edema</td>
<td>Tachypnea</td>
<td>Granular lung fields</td>
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<td>Exertional cyanosis</td>
<td>Clubbing</td>
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<tr>
<td>HEME</td>
<td>Sludging, DIC</td>
<td>Polycythemia</td>
<td>Clubbing</td>
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<td>Bleeding or bruising</td>
<td>Bruises</td>
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<tr>
<td>CNS</td>
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<td>Previous stroke</td>
<td>Complete neurologic evaluation</td>
<td>CT scan if neurologic findings</td>
</tr>
<tr>
<td>MS</td>
<td></td>
<td>Feeding difficulty</td>
<td>Ht, wt, head circumference</td>
<td>Plot of growth curves</td>
</tr>
</tbody>
</table>


Preoperative Implications

Preoperative Preparation
- Desired hemodynamics: Preload—normal (CVP 10–12 mmHg), afterload—low; PVR—normal; HR—normal to high; contractility—normal
- Liberal oral fluids preop
- Avoid premedication causing hypoventilation
- Subacute bacterial endocarditis prophylaxis

Monitoring
- Absolute air bubble precaution
- Arterial catheter
- CVP catheter—know specific anatomy, incl SVC variations
- TEE
- Others as per ASA routine

Airway
- Associated congenital syndromes with airway anomalies
- Cricoid ring limiting diameter of airway
- Primary need to maintain airway and avoid increased Paco₂
- PEEP, with pulm edema or elevated pulm blood flow

Induction
- If IV in place use fentanyl or ketamine with pancuronium or vecuronium.
- If no IV
- If unstable, ketamine IM
- If stable, slow inhalational induction with sevoflurane (avoid high sevoflurane levels until IV placed)
- Actively avoid hypoventilation and agents that produce myocardial depression

Maintenance
- Use fluids judiciously to avoid RV overload
- Positive pressure ventilation usually improves oxygenation
- Use narcotics in conjunction with inhalational agents as tolerated
- Avoid nitrous oxide
- Use high FiO₂
- Capnographic ET CO₂ will not accurately reflect Paco₂
- Prepare for hypothermic cardiac arrest during TAPVR repair
- Avoid hypothermia before and after bypass

Exubation
- Do not attempt deep or early extubation
- Prior to extubation assess adequacy of ventilation with insp pressures of at least -20 mmHg and adequate tidal volumes

Postoperative Period
- Close monitoring of ventilation and pulse oximetry
- Active warming with avoidance of shivering
- Be prepared for immediate reintubation

Adjuncts
- Inotropic support with dopamine or dobutamine

Anticipated Problems/Concerns
- If pulm hypertensive crisis occurs
  - Hyperventilate
  - 100% inspired O₂
  - Consider prostaglandin E₃, tolazoline, amrinone, isoproterenol, or nitric oxide

Etiology
- Embryologic atresia or malformation of the common pulm venous system resulting in persistence of abnormal connections

Usual Treatment
- Severe TAPVD with little systemic shunt needs immediate cardiac correction after birth. Most children with TAPVD require cardiac correction before 1 y of age.
- Cardiac correction of PAPVD may be postponed into childhood.

ICD-9–CM Code: 747.41

<table>
<thead>
<tr>
<th>Risk</th>
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<tbody>
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<td>Pulmonary edema</td>
<td>Clubbing</td>
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<tr>
<td></td>
<td></td>
<td>Exertional cyanosis</td>
<td>Bruses</td>
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<td>Plot of growth curves</td>
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### Overview
- **Anorexia nervosa**
  - Obsessive fear of obesity, obsessive pursuit of thinness
  - Refusal to maintain weight above 85% IBW
  - Distorted body image
  - Amenorrhea for >3 mo
  - Radical restriction of caloric intake
  - Appears cachectic
  - Risk of death high if wt loss >40% of IBW
  - 40–50% recover with treatment, 20–30% improve with treatment
- **Bulimia**
  - Means “ox hunger” or voracious appetite
  - Obsessive fear of obesity, over-concern with body shape and weight
  - Appears well nourished
  - Averages two binge-eating episodes each week for at least 3 mo
  - Irresistible urge to overeat, loss of control in desire to eat
  - Wt control by self-induced vomiting, diuretic and laxative use, strict dieting/fasting, vigorous exercise

### Risk
- Primarily in white adolescent females from middle- or upper-class families, 4–10% are males
- More common in models, ballet students, professions demanding high achievement
- Bimodal peak age of onset: 14 and 18 years

### Perioperative Risks
- Predisposing conditions incl:
  - 0.4–1.5/100,000 population
  - Averages two binge-eating episodes each week for at least 3 mo
  - Irresistible urge to overeat, loss of control in desire to eat

### Worry About
- Degree and duration of malnutrition (excess protein depletion = impaired cellular function)
- Degree of organ dysfunction
- Greater weight loss = greater risk

### ASSESSMENT POINTS

#### System | Effect | Assessment by Hx | PE | Test |
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<tbody>
<tr>
<td>CV</td>
<td>↓ Response to SNS, Hypovolemia</td>
<td>Bradycardia, Hypotension (&lt;70 mmHg systolic)</td>
<td>ECG</td>
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<td>LV dysfunction (myocardial atrophy), ↓ LV wall thickness, ↓ LV cavity size</td>
<td>CHF symptoms</td>
<td>CHF, CXR</td>
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<td>MV prolapse, Cardiomyopathy 2° ipecac Conduction abnormalities</td>
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<td>Murmur, ECHO</td>
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<td>Hypercholesterolemia, Anemia, Thrombocytopenia, Hypofibrinogenemia</td>
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<td>Cholesterol, triglycerides, Hct</td>
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<td>Aspiration pneumonia, Respiratory insufficiency</td>
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<td>GI</td>
<td>Delayed gastric emptying, ↓ motility, Esophagitis, esophageal/gastric rupture</td>
<td>Early satiety, abdominal pain, Vomiting with bulimia, Pneumomediastinum</td>
<td>CXR, Abdominal x-rays</td>
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<td>RENAL</td>
<td>Prerenal azotemia 2° to ↓ volume, Renal insufficiency, Renal calculi, Polyuria</td>
<td>Starvation, Dehydration, vomiting ↓ GFR, XS caffeine and water ingestion, Vomiting, Acid-base abnormalities (metabolic acidosis / alkalosis)</td>
<td>BUN 60–70 mg/dl, Electrolytes (K, Na, P, Mg)</td>
<td>Serum creatinine</td>
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<td>ABGs, Electrolytes</td>
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<td>(&lt;3 grams/dl is evidence of severe protein malnutrition)</td>
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<td>CPK, LDH, aldolase</td>
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<table>
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<tr>
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<th>PE</th>
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<tbody>
<tr>
<td>ENDO</td>
<td>↓ BMR</td>
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<tr>
<td></td>
<td>Hypothermia (&lt;96.6°F rectally)</td>
<td>Amennorhea</td>
<td>Vasoconstriction</td>
<td>↓ WBC (leukopenia)</td>
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<td>Estrogen deficiency</td>
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<td></td>
<td>Depressed immune function</td>
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<td>Hypophosphatemia</td>
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<td>Hypoglycemia</td>
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<td>Euthyroid sick syndrome</td>
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<tr>
<td>CNS</td>
<td>Brain atrophy with dilated ventricles</td>
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<td>Starvation Illicit drug, alcohol use</td>
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<td></td>
<td>Depression</td>
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<td>PNS</td>
<td>Peripheral neuropathy</td>
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<td>EMG changes</td>
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<td>MS</td>
<td>Osteoporosis</td>
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<td>Estrogen, IGF-I deficiency</td>
<td>Vertebral compression fractures Stress fractures</td>
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<td>Cachexia (if anorexic)</td>
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<td>Dieting</td>
<td>X-rays of back, extremities</td>
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<tr>
<td></td>
<td>Myopathy</td>
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</tbody>
</table>


### Perioperative Implications

**Preoperative Preparation**
- Evaluate degree and duration of malnutrition
- Assess degree of organ damage (esp. cardiac, pulm, renal, hepatic)
- Severely malnourished for emergency surgery have significant increased morbidity and/or mortality
  - Delay elective surgery until pt is medically stable and nutritional status is improved
  - Optimize hemodynamics, volume status, acid-base status, electrolytes (Na, K, P, Mg) and glucose
  - Treat severe anemia if present
  - Consider metoclopramide to promote gastric emptying

**Monitoring**
- ABGs, lytes
- A-line, CVP, PA catheters may be indicated

**Airway**
- Induction
  - Consider rapid-sequence induction (decreased GE sphincter tone, decreased gastric emptying)
  - Cautious dosing because of possible LV dysfunction and hypovolemia
  - Antibiotics

**Maintenance**
- Aggressively avoid hypothermia
- Cautious use of potent inhalation agents to avoid hemodynamic depression
- Excess fluids may precipitate pulm edema, CHF

**Extubation**
- Consider awake extubation

**Adjuvants**
- Cautious use of muscle relaxants (decreased muscle mass, electrolyte and acid-base abnormalities)

**Anticipated Problems/Concerns**
- Temp control
- Hemodynamic stability
- Acid-base and electrolyte management
- Metabolic reserve adequate to accommodate intraop and postop surgical stress and/or demands of wound healing and combating infection?
Anticoagulation, Preoperative

Risk
- Pts with mechanical heart valves, atrial fibrillation, pulmonary embolism, recent venous thrombosis
- Oral anticoagulant therapy (warfarin, oral Xa inhibitor, dabigatran) and use of LMW heparin, pentasaccharide may increase potential risks in elective or emergency surgery
- Other populations are pts who receive heparin IV before vascular or cardiac surgery and pts undergoing cardiac surgery with extracorporeal circulation

Perioperative Risks
- Balance between risk of bleeding versus thromboembolic complication is major periop risk
- Risk increases with major and emergency versus elective surgery

Worry About
- Excessive allogeneic transfusions, either to correct effects of anticoagulation or for risk of excessive bleeding
- In pts with valvular heart disease, concomitant hepatic dysfunction due to HF may produce abnormal PT and/or thrombocytopenia
- Heparin-induced thrombocytopenia can be associated with heparin therapy due to acute administration or prolonged use (~5 d)

Overview

Heparin (Standard Unfractionated)
- For preventive therapy and acute management, binds to antithrombin III and factor X to inhibit their effects
- Variability in response to heparin depends on
  - Prep of heparin administered
  - Individual characteristics of pts
  - Duration of therapy (due to decreased antithrombin III levels)
  - Duration of action depends on dose and method of administration
    - 100U/kg: T½ 56 min
    - IV: 60 min
    - 400U/kg: T½ tripled
    - SQ: 3 hr
- Depolymerized in endothelial cells
- Eliminated in urine
- Heparin resistance (many proteins neutralize anticoagulant therapy; prolonged therapy can lower antithrombin III levels)
- Monitoring of the anticoagulant effect: PTT

Heparin (LMW)
- T½ 4–7 hr
- Higher and more predictable bioavailability: 100%
- Removed by renal filtration
- Not reversed with protamine, no current reversal therapy except time

ASSESSMENT POINTS

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<tr>
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<tbody>
<tr>
<td>ENDO</td>
<td>Risk of protamine reactions is 10- to 30-fold higher in diabetics receiving protamine-containing insulin</td>
<td>Hx of insulin use</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Elective surgery/warfarin therapy
  - Stop warfarin 5 d before surgery
  - Replace with heparin in checking INR, PTT, platelet count
  - Stop heparin 60–90 min before surgery
  - Reversal for emergency surgery

- Warfarin therapy can be acutely reversed with PCC, and heparin therapy can be reversed with protamine
- Consider avoiding regional anesthesia
- Approach anticoagulation reversal cautiously in the anticoagulated patient

Postoperative Period
- Restart heparin therapy immediately after surgery (PTT, plt count, blood cell count, bleeding)

Heparin Reversal Treatment
- Protamine reversal according to the ratio heparin:protamine 1:1.3 (or start with 50–100 mg and check the ACT)
- Monitoring: ACT in cardiac surgery

Warfarin
- Oral anticoagulant
- Member of the coumarin family
- Vitamin K antagonist causing inactivation of factors II, VII, IX, X and anticoagulants C, S
- Used for thromboembolic complication prevention
- Peak plasma concentration reached 1–4 hr after ingestion
  - T½: 36–42 hr
- International normalized ratio (INR) required: 2–3
- Stop for surgery and replace with heparin

Warfarin Reversal Treatment
- Vitamin K: 10–20 mg PO, IM, or IV, but takes several days for normalization of INR
- Fresh frozen plasma starting with 2U but higher doses required
- Purified protein concentrates of II, VII, IX, X with protein C and AT III (Beriplex and Octaplex) are used outside of USA and under investigation here.

Novel Agents Approved in Other Countries not yet Available in the United States
- Rivaroxaban and apixaban are oral Xa inhibitors
- Dabigatran is an oral thrombin inhibitor
- These agents studied in periop DVT prophylaxis and AF treatment; no current reversal therapy except time

Anticipated Problems/Concerns
- Introduction of epidural or spinal anesthesia requires minimum 60–120 min between stopping and restarting heparinization; consider removing catheter at least 120 min after stopping heparinization and complete restoration of normal clotting time. Longer times are required with other longer-acting anticoagulation agents.
## Antithrombin III Deficiency

### Risk
- Incidence in USA: 1 in 2000–5000 (may be higher)
- Men and women equally affected, no racial or ethnic difference

### Perioperative Risks
- Risk of postop thromboembolic phenomena; 40–70%, most common (in descending order): DVT, pulm embolus, mesenteric thrombosis, cerebral venous and retinal thrombosis, highest risk in those with antithrombin III (AT III) levels <50% of normal
- Risk of pregnancy-related venous thromboembolism may be >50% in untreated pts
- Heparin resistance is common

### Worry About
- Hypercoagulable state periop
- Thrombus formation on indwelling catheters
- Pulm emboli or DVT with immobility

### Overview
- AT III is an α₂-globulin and a serine protease inhibitor, capable of inactivation of thrombin and factor Xa in blood
- It has anti-inflammatory properties via interactions with the endothelium
- AT III deficiency results in an unusual susceptibility to thromboembolic disease
- Heparin resistance may be problematic during surgery
- Massive thromboembolism can occur periop with AT III levels <50

### ICD-9-CM Code: 286.5

### Key Reference:

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<td>CV</td>
<td>CAD</td>
<td>Angina, dyspnea</td>
<td>ECG, CXR, angiography</td>
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<tr>
<td>PERIPHERAL VASC</td>
<td>DVT Arterial occlusion</td>
<td>Dyspnea Exercise tolerance decreased</td>
<td>SOB</td>
<td>CXR V/Q scan</td>
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<tr>
<td>RESP</td>
<td>Pulm embolus</td>
<td>Abdominal pain</td>
<td>Serum albumin, AT III level</td>
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<td>Decreased AT III</td>
<td>Rectal bleeding, jaundice, hepatomegaly</td>
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<td>DIC</td>
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<td>Bleeding and thrombosis</td>
<td>Nephrotic syndrome, proteinuria</td>
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<td>Urinalysis, serum albumin</td>
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<td>GU</td>
<td>Decreased albumin and AT III levels</td>
<td>Seizure, loss of vision, loss of motor function</td>
<td>CT scan, angiogram</td>
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<td>CNS</td>
<td>CVA</td>
<td>Sudden onset, Hx of other embolic disease</td>
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### Perioperative Implications

**Preinduction/Induction/Maintenance**
- Assess whether congenital or acquired; if acquired, treat primary disease if possible
- Stop oral anticoagulation and substitute FFP or AT III concentrate to bring AT III level to 80–120% normal
- Heparin to provide PTT of >1.5 times control
- Provide mechanical and pharmacological thromboprophylaxis

**Monitoring**
- Careful attention to temp
- Volume status, resp variables
- PTT, AT III levels

**General Anesthesia**
- No special concerns with airway, induction, or adjuvant drugs
- Maintain normothermia to avoid hyperviscosity
- Mesenteric, inferior vena cava, or CNS thrombosis
- Withdrawal of warfarin sodium preop, as pts may be heparin-resistant
- Timing of neuraxial anesthesia in anticoagulated pts

**Regional Anesthesia**
- Neuraxial techniques require meticulous attention to the timing of
  - Neuraxial anesthesia in relation to the last dose of anticoagulant
  - First postop dose of anticoagulant in relation to the placement of neuraxial block and/or removal of indwelling catheter
- Plexus and peripheral blocks risks in anticoagulated patients remain undefined

**Postoperative Period**
- Consider ICU for monitoring
- Continue anticoagulation
- Early mobilization
- Remove indwelling catheters as soon as possible
- Oral anticoagulation might be reintroduced ASAP

**Anticipated Problems/Concerns**
- Embolic phenomena can occur intraop
- Monitoring lines may be foci for thrombus formation
- Periop thromboembolic events major concern; continuous anticoagulation is required, as is operative prophylaxis with, AT III concentrate (plasma derived or recombinant), FFP, and heparin
Aortic Regurgitation

**Risk**
- 100,000 aortic valve operations/y
- 20–30% of aortic valve replacements have AR
- 12–30% of aortic valve replacements have combined AR and stenosis
- M:F ratio: 3:1
- Racial predominance: None known

**Perioperative Risks**
- LVF
- RVF
- Subendocardial ischemia
- Splanchnic ischemia

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<tr>
<td>CV</td>
<td>Aortic valve dysfunction</td>
<td>High-pitched, early diastolic, decrescendo blowing murmur</td>
<td>MR</td>
<td>CXR, ECHO</td>
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<td>Mid-diastolic low-pitched murmur (Austin Flint)</td>
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<td>Widened arterial pulse pressure (water-hammer)</td>
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<td>To and fro bobbing of head (de Musset’s sign)</td>
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<td>LV</td>
<td>Dysfunction</td>
<td>Dyspnea with exercise</td>
<td>Displaced posterior MI</td>
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<td>Splanchnic ischemia</td>
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<td>Distended abdomen</td>
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</table>

**Perioperative Implications**

**Preoperative Preparation**
- Consider optimizing LV performance with vasodilators, inotropes, and diuretic
- Avoid reduction in aortic diastolic pressure
- Emergent procedures (acute AR): Full-stomach precautions

**Monitoring**
- Arterial catheter
- ECG leads II/V5 and ST-segment analysis
- Consider PA catheter or TEE

**Preinduction/Induction**
- Elective: Consider narcotic induction with inhalation supplement (0.25–50% MAC); nondepolarizing muscle relaxant devoid of bradycardic effects
- Emergency (acute AR with aortic dissection): Consider rapid-sequence technique with ketamine, etomidate, or low-dose narcotic plus amnestic agent
- Decreased aortic diastolic pressure and decreased coronary perfusion pressure and may lead to subendocardial ischemia
- Bradycardia and Htn increased regurgitant fraction and decreased cardiac output

**Maintenance**
- During period until institution of cardiopulmonary bypass, consider maintaining LV function with minimum of anesthetic interventions
- PCWP may underestimate LVEDP due to premature closure of mitral valve
- PCWP may overestimate LVEDP in pts with combined AR and MR

**Exubation**
- Consider extubation for pts undergoing valve replacement in ICU after respiratory and hemodynamic criteria are met

**Postoperative Period**
- Consider augmenting preload to maintain and preserve filling volume of still-dilated, hypertrophic LV
- Inotropic support may be required to maintain CO if inadequate intraop myocardial preservation
- Evaluation for neurologic injuries 2° to embolism during valve replacement

**Worry About**
- Aspiration pneumonitis (acute AR)
- Avoid htn, which increases AR and decreases cardiac output
- Avoid bradycardia, which increases AR and decreases cardiac output

**Etiology**
- Damage to leaflets
- Aortic root dilatation
- Loss of commissural support

**Treatment**
- Medical: Vasodilator, calcium channel blockers, ACE inhibitors, diuretic, digoxin
- Surgical: Prosthetic valve

**Overview**
- Long latency period between onset of hemodynamic changes and symptoms (~20-30 y)
- Myocardial ischemia uncommon
- Abdominal pain manifestation of splanchic ischemia

**ICD-9-CM Code:** 424.1

**Perioperative Implications**

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- Consider optimizing LV performance with vasodilators, inotropes, and diuretic
- Avoid reduction in aortic diastolic pressure
- Emergent procedures (acute AR): Full-stomach precautions

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**Anticipated Problems/Concerns**
- Prolonged Trendelenburg position poorly tolerated during PAC insertion
- Intra-aortic balloon counterpulsation contraindicated before valve replacement
- Atrial fibrillation or other supraventricular tachycardias poorly tolerated and require aggressive treatment
- Retrograde cardioplegia (not anterograde) may be required for myocardial protection
- Associated diseases may present difficult intubation, e.g., rheumatoid arthritis, Marfan’s syndrome, trauma (acute aortic dissection)

Aortic Stenosis

Risk
- Incidence: In persons older than 65 years, 25% have calcific aortic valve disease
- 1%–2% of the population has a bicuspid aortic valve; 5%–15% may become stenotic

Perioperative Risks
- Hypotension from impaired ability to augment cardiac output, (stenosed valve creates fixed cardiac output), in response to stress or hypovolemia
- Increased risk for myocardial ischemia due to LVH, high intraventricular pressures, and decreased diastolic time
- Increased risk of infective endocarditis when undergoing noncardiac surgical procedures

Worry About
- HD instability due to
  - Decreased SVR; decreases coronary perfusion causing hypotension-induced ischemia, subsequent ventricular dysfunction, and worsening hypotension
  - Decreased preload and subsequent stroke volume

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Progression of stenosis</td>
<td>Angina, dyspnea, syncope</td>
<td>Systolic murmur</td>
<td>ECHO</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
<td>Angina</td>
<td>Rales, edema, wheeze</td>
<td>ECG, coronary angiography</td>
<td></td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
<td>Dyspnea</td>
<td>CXR, ECHO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>Palpitations, syncope</td>
<td>ECG, Holter</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CNS Syncope Syncope ECG, Holter, ECHO


Preoperative Preparation
- Premedication is indicated to avoid anxiety-induced tachycardia
- Replace any preop fluid deficit, ensure adequate ventricular preload
- Pts with severe symptomatic AS may benefit from postponement of elective surgery until AVR is performed

Monitoring
- ECG for ST segment analysis
- Invasive arterial pressure monitoring
- Consider pulm artery catheter
- Transesophageal ECHO when blood loss or volume shifts are anticipated

Airway
- None

Preinduction/Induction
- Phenytoinephrine or norepinephrine prepared, since hypotension can cause myocardial ischemia
- Tachycardia; decreases diastolic filling time and increases myocardial oxygen demand
- Myocardial ischemia
- Diastolic dysfunction
- Atrial fibrillation; loss of atrial kick, can precipitate acute HD instability

Overview
- Stenosis of the aortic valve creates an obstruction to LV ejection
- Intraventricular systolic pressure increases to preserve forward flow
- Chronic pressure overload results in concentric LV hypertrophy
- Hypertrophy decreases LV compliance and diastolic dysfunction may ensue
- Normal contraction is often critical for maintaining adequate LV filling and stroke volume
- The ability to increase CO in response to a drop in SVR is impaired
- Decreased aortic root pressure decreases myocardial perfusion gradient
- Angina, dyspnea, and syncope are common presenting symptoms

Tachycardia; decreases diastolic filling time and increases myocardial oxygen demand
- Myocardial ischemia
- Diastolic dysfunction
- Atrial fibrillation; loss of atrial kick, can precipitate acute HD instability

Diagnosis is made echocardiographically
- AS is graded as mild for a valve area greater than 1.5 cm², moderate for areas between 1.0 and 1.5 cm², and severe for a valve area less than 1.0 cm²
- Mean and peak pressure gradients across the valve also are used to classify severity

ICD-9-CM Code: 424.1

Etiology
- Congenital bicuspid aortic valve
- Rheumatic aortic stenosis
- Calcific degenerative disease

Usual Treatment
- Surgical aortic valve replacement (AVR) is the definitive treatment
- Percutaneous aortic valve replacement is a new and evolving technology
- Balloon valvuloplasty as a bridge to surgical repair

Preoperative Preparation
- Premedication is indicated to avoid anxiety-induced tachycardia
- Laryngoscopy only after sufficient sympathetic attenuation

Maintenance
- Volatile agents may improve diastolic relaxation due to their intrinsic myocardial depression
- Consider beta blockade for tachycardia-induced ischemia
- Caution with agents that decreased preload and afterload (e.g., nitroglycerin, nitroprusside), or any agent with significant histamine release
- Caution with agents that directly or indirectly increased HR (e.g., pancuronium, atropine)
- Consider pharmacologic rate manipulation or artificial pacing for severe bradycardia
- Hypotension treated with
  - Volume expansion
  - Alpha agonists
  - Blood loss replaced expeditiously

Consider early electrical cardioversion for atrial fibrillation
- Neuraxial anesthesia associated hypotension from sympathetic may precipitate HD instability

Extubation
- Minimize sympathetic stimulation

Postoperative Period
- Aggressive pain control

Anticipated Problems/Concerns
- Myocardial ischemia
- Diastolic dysfunction
- Dysrhythmias

- Consider early electrical cardioversion for atrial fibrillation
- Neuraxial anesthesia associated hypotension from sympathetic may precipitate HD instability

Exstribution
- Minimize sympathetic stimulation

Postoperative Period
- Aggressive pain control

Anticipated Problems/Concerns
- Myocardial ischemia
- Diastolic dysfunction
- Dysrhythmias
### Apnea of the Newborn

**Risk**
- Full-term infants with neurologic disorders
- Premature infants, with or without neurologic disorders

**Perioperative Risks**
- More prone to apnea during local or epidural anesthesia
- More prone to apnea postop

**Worry About**
- Unexpected apnea in recovery room
- Unexpected apnea in hours after outpatient procedures
- Unexpected apnea on ward hours after inpatient procedures

**Overview**
- Apnea in term infant never physiologic
- Apnea in preterm infants may signal central nervous system disorder or developmental immaturity
- Sudden onset of apnea in any infant may also reflect sepsis or hypoglycemia
- Relationship to subsequent SIDS unclear
- Utility of pneumogram screening controversial
- Indications for home apnea monitoring controversial

**ICD-9-CM Code:** 770.8

**Etiology**
- Term or preterm infants:
  - CNS disorders (seizures, bleeds, structural changes)
- Systemic disorders (hypoglycemia, sepsis, GE reflux)
- Preterm infants:
  - Same as term infants
  - If full evaluation is negative, physiologic apnea of prematurity diagnosed

**Usual Treatment**
- Theophylline or caffeine
- \(O\_2\)
- Transfusion
- CPAP

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
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<th>Assessment By Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Congenital heart disease leads to desaturation PDA may cause CHF</td>
<td>CHD, PGE, treatment</td>
<td>Murmur; cyanosis</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Children with bronchopulmonary dysplasia may be prone to apnea</td>
<td>Hx of hyaline membrane disease or other parenchymal lung disorder</td>
<td>Abnormal pulmonary compliance or (O_2) requirement</td>
<td>CXR, ABGs; (O_2) sat</td>
</tr>
<tr>
<td>GI</td>
<td>GE reflux may cause vagal overload</td>
<td>Hx of reflux</td>
<td>None obvious</td>
<td>pH study; barium swallow</td>
</tr>
<tr>
<td>CNS</td>
<td>Seizures may cause apnea; structural abnormalities may create ineffective respiratory drive</td>
<td>Hx of seizures or change in neurologic development</td>
<td>Exam for seizures or neurologic change</td>
<td>EEG, head ultrasound; CT; MRI</td>
</tr>
</tbody>
</table>

**Key Reference:** Henderson-Smart DJ, Steer P. Postoperative caffeine for presenting apnea in preterm infants. *Cochrane Database Syst Rev.* 2000;CD000048.

**Perioperative Implications**

**Monitoring**
- Routine

**Airway**
- Not usually a problem; obstructive apnea may occur but is rare
- Bronchospasm may occur in infants with bronchopulmonary dysplasia

**Maintenance**
- Usually no problem during procedure; vigilance required postop

**Exubation**
- Watch for intermittent inadequate resp effort for hours

**Adjuvants**
- No special concerns

**Anticipated Problems/Concerns**
- Periop not complex; vigilance regarding care and assessment in postop period
## Appendicitis, Acute

**Risk**
- Life time risk: Men 8.6%, women 6.7%
- Peak incidence 2nd and 3rd decades, but all age can be affected
- >250,000 cases/y Dx in the USA
- More challenging diagnosis in the young and elderly as well as pregnant women

**Perioperative Risks**
- Mortality: <1% for nonperforated, = 3% for perforated, = 15% elderly patients, 4% pregnant women with perforation
- Fetal mortality: 3–5% nonperforated, 20–35% perforated
- Increased morbidity and mortality due to delay in diagnosis and treatment for young children, pregnant women, and the elderly
- Risks increase with perforation: Peritonitis, sepsis, and other complications
- Clinical diagnosis, approxi 15% negative appendectomies, improved accuracy with imaging studies (US, CT, MRI)

**Worry About**
- Aspiration (full stomach, delayed gastric emptying, oral contrast for CT scan)
- Antibiotic coverage
- Sepsis
- Carcinoid of the appendix
- Pregnancy: Pregnant women tend to have more advanced illness as symptoms and signs often overlap with those of pregnancy, causing a delay in diagnosis and treatment.
- Must consider the fetus
- Increased complication of pregnancy if appendectomy in first and second trimesters
- Incorrect diagnosis—possible conversion to more extensive procedure

### Overview
- One of the most common causes of surgical acute abdomen
- The most common reason for non-obstetric surgery in pregnant women
- Presentation varies depending on anatomic location of the appendix and stage of disease
- Pts often present with abdominal pain, anorexia, and N/V
- Mild dehydration and fever are common

**ICD-9-CM Code: 540.9 (Acute appendicitis without perforation)**

### Etiology
- Appendiceal obstruction: Fecalith (majority), hypertrophied lymphoid tissue (esp children), tumors, stones, infection, and parasites
- Obstruction causes mucus accumulation and distention. This in turn results in elevation of luminal and intramural pressure ultimately leading to thrombosis/occlusion of vessels and lymphatic stasis (schemia). There is inflammation as well as bacterial proliferation and neutrophilic infiltration of the wall of the appendix. Ultimately there may be necrosis, gangrene, and perforation.
- Perforation results in a local abscess or diffuse peritonitis

### Perioperative Implications
- Replace fluid deficits and correct electrolyte abnormalities (ideally prior to surgery)
- All women of childbearing age should have a pregnancy test
- Antibiotic coverage (gram negative and anaerobic coverage: A beta-lactam/beta-lactamase inhibitor, third generation cephalosporin plus metronidazole, fluoroquinolone plus metronidazole, or a carbapenem)
- Aspiration prophylaxis: Nonparticulate antacid and H2 blocker
- Avoid metoclopramide if bowel obstruction

### Monitoring
- Routine, unless septic

#### Airway
- Assume full stomach: Rapid-sequence induction versus awake intubation
- Secure airway with cuffed ETT

#### Induction
- Intravenous, rapid-sequence induction
- Anticipate hemodynamic instability if not sufficiently resuscitated or sepsis
- Consider nasogastric/orogastric tube and Foley catheter (esp if laparoscopic)
- Neuraxial anesthetic possible if non-septic, appropriately resuscitated, cooperative pt and limited likelihood of high abdominal exploration

#### Maintenance
- Balanced technique
- Requires muscle relaxation for dissection, but quick closure (consider intermediate duration non-depolarizing neuromuscular blocker)
- Laparoscopic procedure: Time depends on surgeon experience, skill, and intraop findings

#### Extubation
- Exhale when pt is fully awake
- Vomiting common, use antiemetic prophylaxis

### Postoperative Period
- Pain control with local anesthetic infiltration (SQ and deeper) by surgeon, opioids, and NSAIDs
- Laparoscopic procedures tend to be less painful, result in shorter length of stay, but may be associated with higher complications and readmission rate than open procedures

#### Adjuvants
- Antibiotic interaction with nondepolarizers
- Fever in postop period (postop sepsis versus malignant hyperthermia)

#### Anticipated Problems/Concerns
- Concern for aspiration
- Complications of appendicitis: Wound infections, abscesses, bowel obstruction, fistulae, pyoclechlitis, and portal venous thrombosis

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<tr>
<td>CV</td>
<td>Tachycardia</td>
<td>Fever, dehydration enesis, infection/sepsis</td>
<td>Resting pulse, Orthostatic signs</td>
<td>Pulse oximetry, RR</td>
</tr>
<tr>
<td>RESP</td>
<td>V/Q mismatch</td>
<td>Dyspnea, tachypnea</td>
<td>Splinting, Observation</td>
<td>Electrolytes (if protracted) Imaging studies Higher WBC and temp, more likely perforation</td>
</tr>
<tr>
<td>GI</td>
<td>Ileus</td>
<td>Anorexia, vomiting</td>
<td>Abdominal auscultation</td>
<td>Urine specific gravity, BUN/Cr (rarely needed) U/A</td>
</tr>
<tr>
<td></td>
<td>Perforation</td>
<td>Oliguria, Pyuria, hematuria</td>
<td>Skin turgor, Orthostatic signs</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Dehydration</td>
<td>Oliguria, Pyuria, hematuria</td>
<td>Skin turgor, Orthostatic signs</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Somnolence/confusion</td>
<td>Rule out sepsis, side effect of narcotics</td>
<td>Mental status exam</td>
<td>WBC</td>
</tr>
<tr>
<td>Pain</td>
<td>Visceral afferents T8-T10 with obstruction and distention of appendix Somatic nerve stimulation with peritoneal irritation</td>
<td>Classic periumbilical pain</td>
<td>Peritoneal signs: Rebound tenderness, guarding</td>
<td></td>
</tr>
</tbody>
</table>

Aspiration, Perioperative: Prevention and Management

Risk
- Risk of aspiration: ~3 per 10,000 anesthetics; ~11 per 10,000 emergency and/or afterhours cases
- Loss of protective reflexes and sphincter function
- Obstructed or abnormal GI motility
- Increased GI contents, decreased pH
- Trauma, emergency/night surgery, pregnancy, difficult airway, ASA status > 2

Perioperative Risks
- Mortality after aspiration: 5%; higher if ASA > 2
- Worry About
  - 20% of pts who aspirated had no risk factor; of these, 66% had difficult intubation
  - Rapid-sequence induction may have deleterious effects on heart rate, blood pressure
  - Difficult intubation

Overview
- Prevention of aspiration best, as there is no definitive treatment
- Vast majority of pts with risk factor(s) do not aspirate
- Consider aspiration in differential diagnosis of bronchospasm with hypoxemia

ICD-9-CM Codes: 997.3 (Aspiration pneumonia after procedure); 668.0 (Aspiration, peripartum)

Perioperative Implications

Preoperative Preparation
- NPO status
  - Generally, no solids for 6 hr, clear liquids allowed up to 2 hr preop
- Prophylaxis in selected patients:
  - Increase Gastric pH: Nonparticulate antacid, H2, blockers, proton pump inhibition
  - Decrease GI contents: Prokinetics, NG suction

Monitoring
- Routine
- Airway
  - Protect airway with ETT or maintain protective reflexes
  - Awake intubation in difficult airway
  - LMA not protective against aspiration

Preinduction/Induction
- Regional associated with aspiration if seizures or hypotension decrease alertness
- GA: Risk at induction, extubation

- Denitrogenation with 100% O2
- Check optimal pt position, table height, drugs and tools available, suction at hand
- Rapid-sequence induction; cricoid pressure until ET your placement assured by ETCO2

Maintenance
- Care with level of sedation during sedation/regional cases

Extubation
- Return of muscular strength/coordination/consciousness adequate to protect airway if emesis occurs
- If emesis, head-down or right-side tilt, thoroughly suction oropharynx and trachea

Postoperative Period
- If no symptoms in 2 hr, significant aspiration extremely unlikely
- If pneumonia occurs, initial postop CXR may be normal, proceeding to white-out in a few to 24 hr
- PEEP redistributes lung water, improves oxygenation; higher PEEP may decrease cardiac output and ventilation
- Maintaining low filling pressures may limit lung fluid accumulation, but may worsen negative effects of PEEP

Adjuvants
- Muscle relaxants must be dependably rapid-acting
- Regional drugs—avoid oversedation, hypotension
- Drug interactions between anesthetic drugs and 1 or 2 doses of aspiration prophylaxis not significant

Anticipated Problems/Concerns
- Must balance concern for aspiration risk against airway quality, cardiopulmonary reserve, and feasibility of regional techniques
Asthma, Acute

Risk
• Incidence in USA: 10 million, incidence nearly 5% for persons age 5–34 y
• Greatest incidence of new cases in persons less than 5 y old
• Increased prevalence and severity in African Americans, adult females, and atopic individuals

Perioperative Risks
• Risk related to preop control
• Symptomatic pts: Morbidity due to bronchospasm and laryngospasm

Worry About
• Bronchospasm due to mechanical stimulation of hyperreactive airways
• Resp resistance following tracheal intubation/extubation
• Medication side effects (e.g., β-agonists causing tachycardia and hypokalemia)
• Adrenal insufficiency (chronic corticosteroid use)

Overview
• Characterized by bronchial wall inflammation, reversible expiratory airflow obstruction, airway hyperreactivity, wheezing, dyspnea, and cough
• Types: Allergen-induced, exercise-induced, nocturnal, aspirin-induced, occupational, and infectious
• Airway obstruction from airway inflammation, intraluminal mucus, and bronchoconstriction
• Reversibility of obstruction is characteristic
• Severe airway obstruction may lead to dynamic hyperinflation
• Intubation frequently increases airway resistance

ICD-9-CM Code: 493.9 (Asthma, unspecified)

Etiology
• Allergen-induced immunologic: Repeated antigen exposure causes specific IgE antibodies, thus release of inflammatory mediators

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>CV</td>
<td>Tacharythmias, possible pulmonary HTN</td>
<td>Palpitations, HR</td>
<td>Tachycardia, irregular rhythm, loud P</td>
<td>EKG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Airflow obstruction decreased lung elastance, hyperinflation, hypoxemia, hypercapnia, variations in peak flow</td>
<td>Dyspnea, cough, wheeze, chest tightness, nighttime awakenings, symptoms induced by exercise, allergens, etc.</td>
<td>Prolonged I:E, decreased breath sounds, wheezing, pulsus paradoxus</td>
<td>PFT, CXR, ABG</td>
</tr>
<tr>
<td>ENDO</td>
<td>Steroid-induced hyperglycemia, adrenal insufficiency (prior &lt;1 y steroid users)</td>
<td>Polyuria, polydipsia, weakness</td>
<td>Hypotension in adrenal insufficiency</td>
<td>Glucose, electrolytes, cortisol, ACTH, stimulation test</td>
</tr>
<tr>
<td>MS</td>
<td>Steroid myopathy, steroid-paralytic myopathy</td>
<td>Difficulty climbing stairs or rising from chair, difficulty weaning mechanical ventilation</td>
<td>Proximal muscle weakness in steroid myopathy, possible quadriplegia in steroid-paralytic myopathy</td>
<td>Measurement of inspiratory muscle force, CPK, EMG, muscle biopsy</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preinduction/Induction/Maintenance
• Assess severity and characteristics of disease, review PFTs (reversibility with bronchodilators), blood eosinophil count, chest auscultation, CXR, ABG
• Consider chest physiotherapy, systemic hydration, antibiotics, and bronchodilators preop
• Goal of maintenance: Depress airway reflexes
• Consider alternatives to ETT (e.g., LMA, regional)
• Volatile anesthetics, propofol, ketamine cause bronchodilation

Monitoring
• Airway peak-to-plateau gradient (as determined by an inspiratory pause) is a useful measure of airway resistance at a constant flow for time-to-time comparison
• Plateau pressure (Pplat) serves as a measure of lung hyperinflation and may be best predictor of complications of hypotension and barotrauma. Peak pressure does not predict complications
• Aim for Pplat <30 cm H2O by prolonging expiratory time (e.g., decreased minute ventilation and/or increased inspiratory flow; use square flow waveform)

General Anesthesia
• Postintubation hypotension may result from lung hyperinflation, hypovolemia, and sedation. Significant lung hyperinflation mimics tension pneumothorax. A trial of hypoventilation improves cardiopulmonary status within 30–60 sec in former. Volume challenge is indicated for hypotensive pts.
• Rising peak-to-plateau pressure gradient suggests increased airway resistance
• Rising Pplat suggests worsening lung hyperinflation
• Consider keeping Pplat <30 cm H2O by prolonging expiratory time. May need to accept hypercapnia
• Exudation may precipitate exacerbation. Inhaled β-agonists may be needed more frequently post extubation.
• Muscle relaxants in addition to systemic corticosteroids may cause acute myopathy
• Volatile anesthetics, propofol, and ketamine are bronchodilators

Regional Anesthesia
• Excellent alternative to avoid airway instrumentation

• Abnormal autonomic nervous system regulation of airway function: Imbalance between excitatory and inhibitory neural input, thus responsive to β-agonist

Usual Treatment
• Bronchodilator drugs inhaled β-agonists albuterol, anticholinergics ipratropium
• Antiinflammatory drugs (corticosteroids, cyclosporin, and leukotriene inhibitors)
• Treatment of status asthmaticus: Supplemental oxygen, repeated administration of inhaled β-agonists, IV corticosteroids, SQ epinephrine, terbutaline in pregnancy
• Mechanical ventilation indicated for arrest, obtundation, impending ventilatory failure; high peak airway pressures, prolonged expiratory phase, lower PEEP, and permissive hypercapnia

Anticipated Problems/Concerns
• Hypokalemia from β2-agonist administration
• Hypotension or pneumothorax from lung hyperinflation
• Increased risk of tension pneumothorax. Clinical features of lung hyperinflation mimic tension pneumothorax. If trial of hypoventilation does not quickly achieve hemodynamic stability, consider chest tube placement.
Atherosclerotic Disease

Jacqueline M. Leung

Risk
- Incidence in USA: 2 million
- 56,000,000 persons have some form of CV disease

Perioperative Risks
- CAD increases the risk of developing postop myocardial ischemia
- Presence of CAD in vascular surgical pts increases operative and long-term mortality

Worry About
- Increased risk of periop myocardial ischemia and periop cardiac complications
- Increased risk of CVA not evident after nonvascular surgery
- Aortic dissection in cases of aneurysm requiring emergency surgery
- Co-existing diseases such as DM, tobacco smoking, and Htn

Overview
- Thickening and hardening of the medium and large arteries accounts for large proportion of heart attacks and cases of IHD
- Also leads to strokes, PVD, and aneurysm of lower abdominal aorta
- Blood vessels affected incl coronary, carotid, basilar, and vertebral arteries, as well as aorta and iliac arteries

ICD-9-CM Code: 414.0 (Atherosclerotic heart disease)

Etiology
- Multifactorial
- Risk factors: Hyperlipidemia, Htn, cigarette smoking, male sex, DM

ASSESSMENT POINTS

System
\| Effect \| Assessment by Hx \| PE \| Test
\|-----------------\|-----------------\|-----------------\|-----------------
CV
- Htn
- Coronary artery stenoses
- MI
- Usually asymptomatic
- Angina, may be asymptomatic
- Normal if treated
- S1 and/or S2
- Cardiomegaly
- Vital signs
- ECG exercise treadmill
- Pharmacologic stress test, coronary angiography, ECHO, radionuclide studies

Ventricular dysfunction (systolic and/or diastolic)
- Exercise intolerance
- Sx of heart failure
- S1 and/or S2
- Cardiomegaly
- LV ejection fraction and function by ECHO, radionuclide studies, diastolic function by Doppler ECHO (if indicated)

RESP
- COPD (many are smokers)
- Dyspnea on exertion
- Decreased breath sounds, prolonged expiration, wheezes
- ABCGs
- PFTs (if indicated)

CNS
- Cerebrovascular insufficiency
- Cerebral infarct
- TIAs
- Syncope
- Strokes
- Carotid bruits
- Focal neurologic deficits
- Doppler or angiogram (if indicated)

Peripheral arteries
- Occlusive lesions
- Abdominal aortic aneurysm
- Claudication
- Abdominal pain, may be asymptomatic
- Decreased pulses
- Pulsatile abdominal mass
- Angiogram (if indicated)
- Aortogram (if indicated)
- MRI (if indicated)

GI
- Intestinal ischemia
- Abdominal pain
- Occult blood in stool or gastric contents
- Leukocytosis
- Serum amylase may be elevated
- Abdominal exam may be paradoxically normal
- Mesenteric angiography


Perioperative Implications

Preoperative Preparation
- Stabilize cardiac sx medically
- Continue antiangular Rx (B-blockade, aspirin and statins)
- Attention to and stabilization of co-existent diseases
- Consider periop B-blockade to ↓ myocardial ischemia

Monitoring
- Cardiovascular
  - ECG with appropriate lead placement, ST trending
  - Consider CVP catheterization to monitor preload, esp in pts with Hx of CHF
  - Consider TTE, esp in pts with uninterpretable ECG (e.g., ventricular pacemaker or BBBB)
  - Cerebrovascular
  - In carotid endarterectomy, measurement of stump pressure, EEG, and SEPs have been used
  - CSF pressure monitoring and drainage in thoracoabdominal aneurysmectomy

Airway
- None

Preinduction/Induction
- Preventing tachycardia (use of short-acting B-blockers desirable)
- Treat BP changes aggressively

Maintenance
- No one anesthetic agent or technique superior; maintaining HR at low level and hemodynamic stability more important
- For peripheral vascular surgery, regional anesthesia in combination with postop epidural analgesia may decrease incidence of graft thrombosis (see also Peripheral Vascular Disease)
- For carotid endarterectomy, maintaining cerebral perfusion pressure important goal
- For abdominal aortic surgery, optimizing loading conditions, detecting and treating myocardial ischemia and ventricular dysfunction are important, particularly during and after aortic clamping

Extravation
- Same concerns as during induction

Adjuvants
- Rapid awakening to allow neurologic assessment after carotid endarterectomy

Anticipated Problems/Concerns
- Primary prevention incl modification of risk factors, esp in high-risk individuals, and prophylaxis with aspirin and statins
- Atherosclerotic heart disease: Antiangular Rx is employed for symptomatic persons; other treatment incl angioplasty and CABG surgery
- Carotid artery disease: Carotid endarterectomy or carotid stenting
- Other cerebrovascular insufficiency: Extracranial-intracranial bypass sometimes performed
- Peripheral vascular insufficiency: Angioplasty or revascularization of lower extremities
- AAA: Abdominal aortic aneurysmectomy or endovascular stent
Atrial Fibrillation

**Risk**
- Affects >1% of those >60 y
- 0.4% of adult population overall
- In the postcardiac surgical population, the incidence is as high as 27–40%
- Racial predominance: None
- Increased prevalence with older age
- In pts presenting for cardiac surgery, the incidence increases with increasing left atrial size as well as in the presence of valvular abnormalities

**Perioperative Risks**
- Rapid ventricular response in CHF
- May be a sign of impending or ongoing myocardial ischemia
- Embolization if persists beyond 48 hr without anticoagulation

**Worry About**
- Decreased cardiac output due to loss of atrial kick esp in the presence of left ventricular hypertrophy, aortic stenosis, or diastolic dysfunction
- Myocardial ischemia secondary to increased myocardial O2 demand
- Embolization risk increases with increased duration.

**Overview**
- Develops over 2 decades in 2% of pts >60 y
- Related to left atrial size, underlying heart disease, and abnormal electrophysiology
- Incidence increases with age
- Most affected people have underlying cardiac disease
- Common after cardiac surgery, particularly valve surgery

**ICD-9-CM Code:** 427.31

**Etiology**
- CAD
- RHD
- Cardiomyopathy, heart failure
- Mitral stenosis, mitral regurgitation esp with left atrial enlargement
- Htn and associated left ventricular hypertrophy
- Pericarditis
- Resp insufficiency incl hypoxia and hypercarbia
- Hypercatecholamine states such as hyperthyroidism
- Subarachnoid hemorrhage
- Sarcoidosis/amyloidosis
- Idiopathic

**Perioperative Implications**

**Preoperative Preparation**
- Search for precipitating causes—new onset may signify acute disease process, which may delay surgery
- Control ventricular response or perform synchronized cardioversion to normal sinus rhythm if unstable

**Monitoring**
- ECG with ST-segment analysis
- Additional monitoring such as use of arterial line or pulse artery catheter should be predicated on type of surgery, additional co-morbidities, or hemodynamic instability.

**Airway**
- None, consider intubation if shock present

**Preinduction/Induction**
- Avoid excessive sympathetic stimulation
- Maintain oxygenation/ventilation

**Maintenance**
- Monitor oxygenation, maintain normocarbia, correct electrolyte imbalances
- Control ventricular response

**Exubation**
- Avoid excessive sympathetic stimulation

**Adjuvants**
- Digitalis has little effect on anesthetic agents
- Ca2+ antagonists can decrease AV conduction; can increase NM blockade

- β-blocker agents can cause decreased AV conduction
- Quinidine (with digitalis) can increase NM blockade

**Postoperative Period**
- Maintain adequate analgesia
- New onset may require prompt treatment

**Anticipated Problems/Concerns**
- Rapid ventricular response may result in significant fall in cardiac output.
- Direct current (DC) synchronized cardioversion establishes sinus rhythm in >90%.
- Pretreatment with amiodarone increases chances of remaining in sinus rhythm.

Atrial Flutter

Risks
- Uncommon in children and young adults
- More common in the elderly
- Usually occurs in pts with structural heart disease (those with left ventricular dysfunction, right ventricular dysfunction, pulm vascular disease, RHD, and CHD)
- Occurs relatively frequently after cardiac surgery but seldom after noncardiac surgery

Perioperative Risk
- Circulatory insufficiency or myocardial ischemia from extremes of heart rate esp in pts with CHD
- Cerebral, coronary, or systemic embolism from left atrial thrombus
- Associated disease, esp adequacy of CV and pulm function
- Accelerated ventricular rates

Worry About
- Increased proarrhythmia risk with drugs for pharmacologic cardioversion particularly in high risk groups such as those with CAD, impaired left ventricular function, left ventricular hypertrophy, acquired or congenital long QT-syndromes, or history of proarrhythmia

Overview
- Mechanism is atrial re-entry, usually in right atrium
- Type I or typical atrial flutter: most common form characterized by regular atrial rates 240–340 bpm with fixed (often 2:1) atrioventricular (AV) conduction
- Type II or atypical atrial flutter: Less common presents with variable atrial rates 340–450 bpm with variable or fixed atrioventricular (AV) conduction that may result in irregular, irregular QRS complex and pulse

ICD9-CM: 427.32

Etiology

Usual Treatment
- Goals incl control of ventricular rate, restoring normal sinus rhythm, maintenance of sinus rhythm after cardioversion, and anticoagulation to prevent systemic embolization if sinus rhythm not restored.
- Cardioversion can be accomplished with direct current cardioversion, pharmacologically (amiodarone, ibutilide, procainamide, sotalol), or with overdrive atrial pacing (for type I flutter).
- Consider early cardioversion if pt is hemodynamically unstable.
- Drugs for ventricular rate control incl β-blockers and Ca2+-channel blockers such as diltiazem and verapamil.
- Anticoagulation should be considered for atrial flutter lasting more than 48 hrs or sooner if low cardiac output.
- Choice of anticoagulant determined by perceived embolic risk (aspirin for low risk or coumadin for higher risk)
- Prophylactic amiodarone and β-blockers lowers the risk for atrial flutter after cardiac surgery.
- For type 1 atrial flutter atrial pacing at 10% above the flutter rate (rate <350 bpm) for 15–30 sec using atrial or esophageal atrial pacing leads, frequently converts atrial flutter to sinus rhythm
- Radiofrequency ablation of the atrial flutter re-entrant pathway can prevent recurrence but this therapy is for chronic atrial flutter and it is not an acute treatment.

Perioperative Implications

Preoperative Preparation
- Adequate ventricular rate control (80–100 bpm) with β-blockers or Ca2+-channel blockers with AV conduction slowing properties
- Treat CHF if present; otherwise, optimize cardiopulmonary function
- If acute onset (<48 hr), consider cardioversion
- When atrial flutter is of >48 hr duration, intra cardiac thrombus must be excluded before cardioversion or the pt should receive course of anticoagulation before and after cardioversion

Monitoring
- ECG with ST–T trending and strip-chart recorder for documentation of new arrhythmias or myocardial ischemia
- Consider direct arterial and pulm artery catheter monitoring in the presence of concomitant left ventricular dysfunction depending on the type of procedure

Anesthesia Induction
- Left ventricular dysfunction and atrial flutter increase risk for hypotension during induction with agents such as thiopental or propofol.
- Desflurane, ketamine, and pancuronium may accelerate ventricular rate.

Maintenance
- Expect increased circulatory instability and less tolerance of large fluid shifts or blood loss.
- No anesthetic are esp contraindicated; caution should be used with drugs that speed conduction.

Tracheal Extubation
- Possibly at increased risk for thromboembolism with hyperdynamic circulatory state
- Sympathomimetic or antimuscarinic drugs may accelerate ventricular rate.

ASSESSMENT POINTS

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<tbody>
<tr>
<td>CARDIAC</td>
<td>Atrial flutter, left ventricular function, coronary disease severity</td>
<td>Palpitations, dizziness, weakness, lethargy, orthopnea, cough dyspnea, exercise intolerance, symptoms of angina</td>
<td>Irregular pulse/pulse deficit, $S_1–S_2$ intensity rales, wheezes</td>
<td>ECG, Holter monitoring, EP studies, ECHO, exercise ECG, MRI, cardiac catheter, stress ECG, dipyridamole scintigraphy, angiography</td>
</tr>
<tr>
<td>RESP</td>
<td>CHF</td>
<td>Dyspnea, orthopnea, cough</td>
<td>S_2, rales wheezes</td>
<td>CXR, pulm function testing</td>
</tr>
<tr>
<td>GI</td>
<td>↓ Perfusion</td>
<td>GI distress, diarrhea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>↓ Perfusion</td>
<td>Polypnea (nocturnal)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEURO</td>
<td>Ischemia or stroke</td>
<td>Syncope, mental changes, paresis/paralysis, dementia</td>
<td>Mental deficits Neurologic exam</td>
<td>See CV assessment</td>
</tr>
</tbody>
</table>

Atrial Septal Defect, Ostium Primum

Overview
- Failure of inferior atrial septum to close at the level of tricuspid and mitral valves
- Symptoms present earlier and are more severe than in secundum pts. These incl dyspnea, fatigue, recurrent resp infections and failure to thrive.
- Left to right shunt increases pulm blood flow
- Late in course: CHF, also more common than in secundum pts and shunt reversal
- Frequently associated with mitral regurgitation with a cleft in the anterior mitral leaflet present in 50% of the patients and/or tricuspid regurgitation
- Diagnosis is by ECHO, appearing as an absence of the lower atrial septum.
- Cardiac catheterization may be required to assess PVR and pulm Htn in large shunts

ICD-9-CM Code: 745.61

Etiology
- Failure of septum primum to fuse with endocardial cushion to close ostium primum

Usual Treatment
- Asymptomatic pts require no medications. Diuretics are used for CHF.
- ACE inhibitors may be used for afterload reduction in the presence of mitral regurgitation
- Antiarrhythmics are occasionally needed for atrial dysrhythmias
- Percutaneous closure is not possible as it is in secundum defects because there is inadequate rim of inferior atrial tissue to prevent the device from impinging on the valves
- Surgery is the definitive management, usually between 2–5 y. May be earlier if there is mitral regurgitation, CHF or failure to thrive. Incision is median sternotomy or right thoracotomy.
- Endocarditis prophylaxis is indicated for 6 mo after repair. Persistent AV valve abnormalities may require long-term prophylaxis.

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<tr>
<td>HEENT</td>
<td>Difficult intubation</td>
<td>Down syndrome</td>
<td>Down syndrome facies</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Atrial dysrhythmias, right-sided heart failure</td>
<td>Pulitations</td>
<td>Irregular rate and rhythm Right heart enlargement</td>
<td>ECG (RBBB + RVH)</td>
</tr>
<tr>
<td></td>
<td>L→R shunting, hypertrophic RA and RV Mitral regurgitation</td>
<td>SOB, frequent fatigue</td>
<td>Normal S1, fixed splitting of S2, and crescendo-decrescendo systolic murmur</td>
<td>CXR—cardiomegaly</td>
</tr>
<tr>
<td>RESP</td>
<td>↑ Pulmonary blood flow</td>
<td>SOB, frequent URIs</td>
<td>Rales, wheezing</td>
<td>ECHO—standard angiography</td>
</tr>
<tr>
<td></td>
<td>↑ PVR</td>
<td></td>
<td></td>
<td>Dye dilution study</td>
</tr>
<tr>
<td>GI</td>
<td>Hepatic dysfunction if severe CHF</td>
<td>Jaundice</td>
<td>Hepatomegaly</td>
<td>LFTs, PT</td>
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<tr>
<td>CNS</td>
<td>Embolic stroke from chronic AFIB</td>
<td>Various neurologic changes</td>
<td></td>
<td>Head CT, cardiac ECHO if emboli suspected</td>
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<tr>
<td>MS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>RENAL</td>
<td>Renal dysfunction if severe CHF</td>
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<td></td>
<td>Cr, BUN</td>
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Perioperative Implications

Preoperative Medications
- Midazolam 0.5–0.7 mg/kg po 30 min before the procedure
- Antibiotic prophylaxis

Monitoring
- Routine monitors, arterial line, CVP, TEE to assess anatomy before CPB, AV valve regurgitation and function, ventricular function and to check for air and residual shunt after CPB; central and peripheral temp monitoring

Induction
- IV induction theoretically slowed by left to right shunt because of increased pulm blood flow; inhalational induction not significantly affected
- May place an epidural with loss of resistance to saline technique to avoid air embolism. Must be placed 1 hr prior to heparinization.

Maintenance
- Avoid nitrous oxide to minimize size of air bubbles; any other techniques appropriate; watch for shunt reversal with hypoxemia, hypercarbia, and hypothermia
- Control BP with milrinone, nitroprusside or nitroglycerin.
- Keep mechanically ventilated if the repair has been complex or arrhythmias are present.

Adjuvants
- Watch for supraventricular dysrhythmias and AV conduction defects, must have pacing wires.

Postoperative Period
- Adequate analgesia for sternotomy or thoracotomy pain; pacemakers available for transient heart block

Anticipated Problems/Concerns
- Air emboli with vascular access
- Dysrhythmias: SA node or AV node dysfunction
- Heart failure
- Third degree AV block with repair of low lying defects
- Residual pulm Htn which can lead to tricuspid regurgitation and RV failure
- Residual mitral valve insufficiency may remain or worsen
- Endocarditis esp with a residual cleft mitral valve
- Discrete subaortic stenosis may be present which can progress after operation
Atrial Septal Defect, Ostium Secundum

**Risk**
- Incidence in USA: 140,000 with ostium secundum ASD (70–80% of ASDs)
- Accounts for 7% of all congenital cardiac defects; 30–40% of congenital cardiac defects in pts over 40 y
- Gender prevalence: Female > male, 2:1 in isolated ASDs
- Familial incidence: Significant if associated with P-R prolongation or forearm and hand abnormalities (Holt-Oram syndrome)
- Increased incidence in high altitude

**Worry About**
- Risk of infectious endocarditis and paradoxical air embolization with IV access

**Overview**
- Failure of closure of midseptal fossa ovalis
- Usually asymptomatic early in life
- 15% incidence of associated noncardiac anomalies
  - Associated with mitral valve prolapse (10–20%) [1,5].
- Left to right shunt increases pulm blood flow (shunt fraction is proportional to ASD size)
- Late in course: Pulmonary Htn, right heart failure with possible shunt reversal; supraventricular arrhythmias
- Uncorrected defect carries a mortality rate of 6% per year over the age of 40
- Diagnosis by echocardiography and Doppler color flow echocardiography
  - 80% spontaneous closure in the first year of life for small defects

**ICD-9-CM Code**: 745.5

**Perioperative Implications**

**Preoperative Medications**
- Narcotics and anticholinergics
- Antibiotic prophylaxis
- Continue digoxin if used for rate control

**Monitoring**
- Routine monitors, arterial line, CVP; TEE indicated for assessing anatomy before CPB; evaluating for air and residual shunting after CPB; central and peripheral temp monitoring

**Induction**
- IV induction theoretically slowed by left to right shunt; inhalational induction not significantly affected

**Perioperative Risks**
- Periop mortality rate: 1%
- Late in course, associated with atrial dysrhythmias, pulm hypertension and right heart failure
- Increased risk of atrial dysrhythmias, heart block (rare), and air embolus with surgical repair

**Postoperative Period**
- Adequate analgesia for sternotomy or thoracotomy pain

**Anticipated Problems/Concerns**
- Paradoxical air emboli with vascular access
- Dysrhythmia (5–10% if no prerepair dysrhythmia)
- Heart failure
- Heart block after CPB (rare)
- Sternal infection (rare)
- Endocarditis (rare)

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<td>Irregular rate and rhythm, right heart enlargement, loud S1, fixed S2, and crescendo-decrescendo systolic murmur</td>
<td>TEE w/ color Doppler flow, four chamber view, bicaval view</td>
</tr>
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<td>Right-sided heart failure L→R shunting</td>
<td></td>
<td></td>
<td>Angiography</td>
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<td>↑ Pulm blood flow</td>
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<td>Holt-Oram syndrome</td>
<td>Large left costal cartilage</td>
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Autoimmune Diseases, Cold

Joseph L. Seltzer

**Overview**
- In two circumstances antibodies will react in the cold to produce hemolysis:
  - IgG antibodies associated with mononucleosis, *Mycoplasma pneumoniae*
  - IgM antibodies are found in the idiopathic form of the disease and in lympho-proliferative disease.
- Hemolysis usually occurs at temp below 31°C.

**ICD-9-CM Code**: 283.0

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<tr>
<td>HEME</td>
<td>Mild to moderate anemia</td>
<td>Hgb, blood bank antiglobulin tests</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Hemoglobinuria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Dyspnea on exertion if anemia is severe</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SKIN</td>
<td>Agglutination of red cells in cold</td>
<td>Acrocyanosis</td>
<td></td>
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**Risk**
- Rare
- Autoimmune hemolytic anemias occur in 1 of 80,000 persons; of these, 17.3% are due to cold antibodies

**Perioperative Risks**
- Acute hemolysis due to cold
- Hemoglobinemia
- Hemoglobinuria
- Rarely, vascular occlusion

**Worry About**
- Cooling to 28–31°C will cause hemolysis

**Perioperative Implications**

**Perioperative Preparation**
- Plasmapheresis—may be used (no more than two d before surgery)

**Monitoring**
- Temp
- Urine output

**Maintenance**
- Keep warm, incl extremities
- Consider forced air warming
- Warm all fluids
- Normothermic cardiopulmonary bypass
- No preferred agent or technique
- Consider hemodilutional autologous transfusion or other techniques to avoid homologous transfusion and formation of new antibody.

**Anticipated Problems/Concerns**
- Hemolysis if temp falls
- Renal dysfunction due to hemoglobinuria
  - May see molting or cyanosis of the skin

**Etiology**
- Idiopathic
- Lymphoid malignancy
- Infections: *Mycoplasma pneumoniae*, mononucleosis, cytomegalovirus, varicella

**Usual Treatment**
- Keep warm, folic acid
- For severe cases, chlorambucil or cyclophosphamide
- Plasmapheresis
  - Rituximab
  - Prednisone

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Autonomic Dysreflexia (AD)

**Risk**
- AD esp occurs in pts with SCI at T6 or above.
- The higher the injury level, the greater clinical manifestations of CV dysfunction
- Risk of AD greater with complete (91%) vs. incomplete (27%) cord transections.
- AD occurs more often in chronic SCI; some clinical evidence seen in first days-weeks.

**Perioperative Risks**
- AD most commonly triggered by irritation and/or manipulation of urinary bladder, colon and in labor
- Severe increased BP and increased or decreased HR associated with stimulation below level of transaction
- Objectively, increased SBP >20–30 mmHg considered a dysreflexic episode. However, be aware usual resting ABP in these pts is 15–20 mmHg less than non SCI subjects.
- Awake pts may complain of HA, anxiety, sweating, piloerection and flushing above and dry, pale skin below the injury level. In anesthetized pts, SBP rising to up to 300 mmHg heralds the onset of severe, life-threatening AD.

**Overview**
- Physiologically, AD is caused by a massive sympathetic discharge triggered by a noxious or nonnoxious stimulus originating below the level of the SCI.
- Specifically, destruction of the vasomotor pathways results in a loss of inhibitory and excitatory supraspinal input to the sympathetic preganglionic neurons thus causing labile BP.
- Also, changes in spinal sympathetic neurons and primary afferents underlie abnormal CV Δs.
- Symptoms are usually short-lived because of treatment or self-limiting nature of episode.

**ICD-9-CM Code: 337.3**

**Etiology**
- Most common cause is traumatic interruption of the spinal cord
- Can also occur due to infectious or oncologic processes causing destructive spinal lesions

**Worry About**
- Untreated episodes can lead to intracranial hemorrhage, retinal detachment, seizures, and death.

**Usual Treatment**
- STOP initiating stimulus when possible.
- Can decrease or prevent AD by use of neuraxial blockade (spinal >> epidural)
- When signs of AD are evident, administer ganglionic blockers (trimethaphan), direct vasodilators (nitropusside) or α-antagonists (phentolamine), GA or spinal anesthesia
- Level 1 evidence that intrasp欣cıtıc anal block with lidocaine limits the AD response in pts undergoing anorectal procedures. Level 1 evidence that topical lidocaine does not.
- Level 1 evidence that prazosin is superior to placebo in prophylactic management of AD
- Level 2 evidence that nifedipine can prevent BPA’s during cysto in SCI pts with AD
- Level 4 evidence that epidural anesthesia may be effective in pts with AD during labor and delivery.
- Centrally acting hypotensive agents (e.g., clonidine) are not effective in treating AD
- Treat tachyarrhythmias with β-blockers in combination with antihypertensives
- Complete bladder deafferentation does not abolish AD during bladder urodynamic studies.

**Perioperative Implications**

**Preoperative Preparation**
- Nifedipine can be used for prophylaxis, given 30 min prior to procedure likely to trigger AD
- Attention to CV and pulm function, volume status, and airway exam.

**Monitoring**
- Consider pre-induction invasive monitoring (arterial, CVP/PA catheters) if volume changes are expected and in setting of poor cardiac reserve (high lesions) and renal insufficiency

**Airway**
- Be prepared for fiberoptic intubation

**Induction**
- Use nondepolarizing muscle blockers when relaxation is necessary.

- Succinylcholine can cause severe K+ release and hyperkalemia in chronic lesions.
- Consider nitropusside prior to induction.

**Maintenance**
- GA with volatile agent superior to nitrous-narcotic technique for prevention/treatment of AD

**Regional Anesthesia**
- Anesthetic technique of choice when possible
- Spinal anesthesia highly effective in preventing AD precipitated by surgery
- Ensure careful assessment of level of spinal blockade in SCI pts due to sensory deficits below injury—avoid unnecessarily high or inadequate blocks

- Epidural anesthesia effective in preventing AD in laboring pts

**Exubation**
- May be difficult due to resp insufficiency in pts with high level spinal lesions

**Adjutants**
- Muscle relaxants required in abdominal surgery due to diffuse increase in muscle tone

**Postoperative Period**
- AD can occur postop in setting of unrecognized or untreated distended bladder or rectum.
- Consider intracerebral hemorrhage protocol in the setting of unexplained delayed emergence with increased BP.

**AV and Bifascicular Heart Block**

### Risk
- **Prevalence:** First degree (0.65–1.6%); second degree (0.003% in young adults, higher in organic heart disease); third degree (overall 0.02%; congenital 1.200,000 live births); increases with age
- **Inferior MI:** Carries low mortality even if associated with high degree AV block
- **Anterior MI:** If high degree AV block results then mortality approaches 80%

### Perioperative Risks
- **Progression of benign heart block to second degree type II or third degree**
- **Heart failure, myocardial and global ischemia, shock, pacemaker failure**

### Worry About
- **Autonomic changes influencing the degree of blockade**
- **Pacemaker failure, electrocathery interferences**
- **Intracardiac wire or PA catheter placement leading to third degree block**
- **Beta blockers, calcium channel blockers, digoxin, and anticholinergic influencing degree of heart block**

### Overview
- **AV blocks:** First degree: PR interval >0.20 sec. Block site = AV node. Usually benign. If pt has structural heart disease this block type becomes more significant. Associated with anterior MI, digitalis, certain neuromuscular diseases
- **Second degree type I (Mobitz I or Wenckebach):** Increasingly prolonged PR interval until QRS dropped. Block site = AV node (normal QRS). Usually benign. Elderly pts and those with structural heart disease make this block type more significant. Usually does not progress over time to second degree type II or third degree. May progress acutely with anesthesia, autonomic influences, or intracardiac catheters/lines.
- **Second degree type II (Mobitz II):** Fixed PR interval with occasional dropped QRS. Block site = usually infranodal (wide QRS). Permanent. The more infranodal block site yields a slower ventricular rate and symptoms. High mortality. Common progression to third degree.
- **Bifascicular block:** Three ‘fascicles’/bundles of nerves conduct via the ventricles. Right bundle branch, left anterior fascicle, left posterior fascicle. When 2 of 3 are blocked it is termed ‘bifascicular.’ When third fascicle is blocked, the pt is in third degree heart block.
- **Third degree:** Atria and ventricles separate pacemakers. Any atrial rhythm (afl/fib/flutter, etc). Ventricular rate/rhythm depends on site of blockade. More infranodal block yields slower ventricular rate. If only upper AV node blocked pt may have junctional rhythm (normal QRS) and be more stable. If entire AV node blocked then ventricular rate 40–40 bpm and perfusion is compromised.

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by SX</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Heart failure</td>
<td>Syncope, SOB, DOE, ‘skipped beats’, last pacemaker battery replacement, fatigue</td>
<td>Bradycardia, JVD</td>
<td>EKG, ECHO, BNP</td>
</tr>
<tr>
<td>PULM</td>
<td>Pulm edema, hypoxia</td>
<td>Cough, pink sputum, orthopnea</td>
<td>Rales, tachypnea, wheezing, cough</td>
<td>CXR, pulse ox</td>
</tr>
<tr>
<td>RENAL</td>
<td>Pre-renal failure, fluid retention</td>
<td>Oliguria, edema, fatigue, N/V</td>
<td>Edema, impaired mentation</td>
<td>BUN, Cr, FeNa, lyes</td>
</tr>
<tr>
<td>NEURO</td>
<td>Poor cerebral perfusion</td>
<td>Lightheadedness, N/V</td>
<td>Impaired mentation</td>
<td>CT head</td>
</tr>
</tbody>
</table>

### Perioperative Implications

#### Preinduction/Induction/Maintenance
- **Ascertain indication for and type of pacemaker as well as functionality.**
- **Consider changing pacemaker to asynchronous mode if electrocatery to be used.**
- **Have external and/or intravenous pacemaker and magnet available.**
- **Consider preinduction arterial catheter.**
- **Anticipate medication influences on autonomic nervous system balance (i.e., vagolyis from pancuronium, glycopyrrolate, etc.).**
- **Avoid intracardiac placement of central line wire.**
- **Consider using bipolar electrocatery; ensure proper electrocatery return pad placement away from pacer.**

#### Monitoring
- **Low SaO2, and high peak airway pressures can signify pulm edema.**
- **Low ETCO1 may indicate low cardiac output.**
- **Arterial waveform: Diminished rate of rise may indicate poor cardiac output.**

#### General Anesthesia
- **Anticipate the effects of laryngoscopy, intubation, TEE placement.**
- **Avoid rapid increases in volatile anesthetic concentration.**
- **Avoid high-dose opiates.**
- **Use beta-blockers or calcium channel blockers carefully; use short acting agents.**
- **Retraction or insufflation of vagal mediated structures can worsen bradycardia.**
- **Surgeon may need to stop offending maneuver until pt stabilized.**
- **Monitor and maintain normal serum electrolyte concentration.**

### Regional Anesthesia
- **High thoracic spinal block will result in brady- cardia even without pre-existing heart block.**
- **Pre-existing heart block may worsen after sympathetic**
- **Atropine ineffective if heart block is below the AV node; use direct acting agents.**
- **Utilize epinephrine without delay.**

- **Verify or induce euvolemia.**

### Postoperative Period
- **Obtain EKG to verify preop baseline, cardiology consult.**
- **Pacemaker interrogation by electrophysiology, return to previous mode.**
- **Perform physical exam looking for signs of heart failure.**

### Anticipated Problems/Concerns
- **If heart block is at AV node then:**
  - **AV conduction is worsened by:** Increased vagal input, peritoneal insufflation, esophageal manipulation (intubation, TEE, esphagoscopy); beta blockers, calcium channel blockers, high dose opiates, anticholinesterases.
  - **AV conduction is improved by:** Vagolyis (atiumcarinics), exercise, isoproterenol.
- **If heart block is infranodal then automatic influences are opposite of above.**
- **Development of slow ventricular response rate <40–50 bpm.**
- **Transcutaneous and/or transvenous pacemaker availability and practitioner knowledge.**
- **Have direct-acting sympathomimetics available.**
Beckwith-Wiedemann Syndrome

**Risk**
- 1:13,700
- No gender predilection although with monozygotic twins seen more with females than males

**Perioperative Risks**
- Acute airway obstruction, difficult mask ventilation and intubation secondary to macroglossia
- Hypoglycemia due to islet cell hyperplasia and hyperinsulinemia
- Cardiac malformations

**Worry About**
- Persistent hypoglycemia, which may cause CNS damage and therefore necessitates intraop infusion of glucose containing solution and frequent glucose checks
- Difficult airway management

**Overview**
- Commonly known for EMG triad (exomphalos, macroglossia, gigantism)
- Other clinical features incl anterior earlobe creases, posterior helical pits, facial nevus flammeus, hemihyperplasia, renal anomalies, embryonal tumors, cardiac malformations and hypoglycemia
- 7.5% estimated risk for embryonal tumor development, which occurs in the first 10 y of life. Most common tumors are Wilms tumor and hepatoblastoma but may also incl rhabdomyosarcoma, adenocortical carcinoma and neuroblastoma
- Cardiac involvement often limited to mild cardiomegaly although other cardiac defects have been reported (atrial and ventricular septal defects, tetralogy of Fallot, hypoplastic left ventricle, cardiomyopathy, cardiac tumors, and valvular disease) • Hypoglycemia due to islet cell hyperplasia and hyperinsulinemia occurs in 50% of BWS pts, is often responsive to medical therapy and usually regresses during the first 4 mo of life. Persistent hypoglycemia refractory to medical management may require pancreatectomy

**Etiology**
- Clinically and genetically heterogeneous
- May be genetically transmitted (15%) or occur sporadically (85%)
- Variety of mutations in chromosome 11p15.5 region
- Mutation near gene for IGF-II

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Macroglossia</td>
<td>Hx of difficult mask ventilation and intubation</td>
<td>Determine extent by physical inspection and oral palpation, previous anesthesia Hx</td>
<td>No testing</td>
</tr>
<tr>
<td>CV</td>
<td>VSD, ASD, TOF, valvular disease, hypoplastic LV, cardiac tumor and cardiomegaly (most common) possible</td>
<td>SOB, DOE</td>
<td>Cardiac exam for murmurs</td>
<td>ECHO</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypoglycemia, Hypothyroidism</td>
<td>Shaking, lethargy</td>
<td></td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal medullary dysplasia, Nephrosis</td>
<td>Hx of renal tumors/previous resections; chronic UTIs</td>
<td>Palpate for masses</td>
<td>US</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preparation**
- Coordinated care with endocrinology and ENT to assist in the management of hypoglycemia and difficult airway
- Discussion with ENT for planned tracheoscopy if significant airway edema and swelling is anticipated following glossal resection
- Review of lab results (hypothyroidism, polycythemia, hypocalcemia and hyperlipidemia have been reported in pts with BWS in addition to hypoglycemia)
- Review cardiac work-up if available
- Pretreatment with antisialogogue (glycopyrrolate or atropine) if intubation is planned

**Monitoring**
- Standard monitoring appropriate for surgical procedure
- Frequent glucose checks

**Induction**
- Inhaled induction with sevoflurane versus awake intubation with sedation/topicalization
- Clinicians should be aware that administration of IV anesthetics and muscle relaxants may cause tongue to fall backward causing acute airway obstruction

**Airway**
- Assume difficult mask ventilation due to macroglossia
- Nasal > oral intubation may be more performed more easily in pts with significant macroglossia. Pre-treat with a nasal decongestant and dilate with nasal trumpets if nasal intubation is considered.
- Assistance with glossal manipulation if direct laryngoscopy is performed
- Backup airway devices (e.g., fiberoptic, glidescope, LMA) and surgical support (ENT) if conventional laryngoscopy fails
- Age-appropriate ET/TT

**Postoperative Period**
- After meeting strict extubation criteria, pts should be monitored in ICU or recovery area with immediate backup for management of airway issues and hypoglycemia

**Anticipated Problems/Concerns**
- Difficult airway
- Hypoglycemia

Bilirubinemia of the Newborn

Overview
- Bilirubin is derived from the catabolism of proteins that contain heme; usually from the breakdown of hemoglobin from RBCs.

- Heme is oxidized to biliverdin and then reduced to bilirubin which is unconjugated, nonpolar, and lipid-soluble (indirect-reacting).

- Unconjugated bilirubin circulates bound to albumin in equilibrium with its unbound fraction that readily crosses the blood brain barrier and can cause neurotoxicity.

- Bilirubin is converted in the liver cell microsome by the enzyme (UDP)-glucuronon transferase to form the polar, water-soluble glucuronide of bilirubin (direct-reacting).

- Most of the conjugated bilirubin is excreted as bile which is metabolized by intestinal flora and excreted in the feces.

- The danger of unconjugated hyperbilirubinemia is kernicterus (yellow staining of brain affecting the basal ganglion, hippocampus and cerebral and bulbar nuclei).

- Bilirubinemia peaks in term infants between 3–5 d; preterm infants 5–6 d.

- Clinical features of hyperbilirubinemia are lethargy, anorexia, nausea, vomiting, icteric skin and sclera.

- Clinical features of kernicterus (very rare): Acute: Opisthotonic posturing, muscle rigidity, seizure, oculogyric crisis. Chronic: Clinical tetard of chooreothetoid cerebral palsy, high-frequency central neural hearing loss, palsy of vertical gaze, and dental enamel hypoplasia as the result of bilirubin-induced cell toxicity.

- The ability of anesthetic agents to displace bilirubin from albumin has not been well studied.

- Goal of therapy is to prevent indirect-reacting bilirubin related neurotoxicity while not causing undo harm.

- Phototherapy and, if unsuccessful, exchange transfusion remain the primary treatment modalities used to keep the maximal total serum bilirubin below the pathologic levels.

- Phototherapy works by bypassing the hepatic system and produces photoisomers of bilirubin that are more water soluble and can be cleared directly in bile or urine without conjugation in the liver.

- Exchange transfusion removes infants’ sensitized and destroyed RBCs and circulating antibodies; double-volume exchange replaces 85% of circulating RBC volume and decreases bilirubin level by 50% and corrects anemia.

- AAP guidelines for healthy term infants: Phototherapy when serum bilirubin >12–15 mg/dL; Exchange transfusion >20–25; premature or ill term infants have lower threshold for starting therapy.

- Several factors are important when determining the bilirubin level above which kernicterus is possible (gestational age, degree of illness, evidence of hemolysis, rate of rise, albumin level and physiologic stress).

Persioperative Risks
- Must consider pathophysiologic conditions present in premature or LBW and ill-term infants (e.g., RDS, sepsis, hemolysis, hypoxemia and acidosis).

- Increased risk of CNS injury with elevated levels of unconjugated bilirubin and compromised blood-brain barrier. Neurotoxic effects are directly related to permeability of BBB and nerve cell membranes (all are adversely influenced by asphyxia, prematurity, hyperosmolality, and infection).

Worry About
- Factors that increase blood-brain barrier permeability to unconjugated bilirubin: Hypoxia, hypercarbia, acidosis, hyperosmolality, Htn, seizure activity, sepsis.

- Drugs (e.g., sulfonamides, ceftriaxone, ampicillin, salicylates, furosemide, and contrast dye) and physiologic states (dehydration, hypercarbia, and acidosis) that displace bilirubin from albumin can increase free fraction of unconjugated bilirubin in the blood.

- Binding of some drugs to albumin may be altered in the presence of hyperbilirubinemia in the neonatal period.

- Surgically induced increases in heme degradation (e.g., hematoma absorption).

- Liver dysfunction.

- Hemolytic anemia.

Risk
- Common and mostly benign problem in neonates.
- Observed during first week of life in 60% of term and 80% of preterm infants.
- Clinical, epidemiologic, and genetic risk factors associated with significant hyperbilirubinemia incl preterm gestational age, exclusive breastfeeding, glucose-6-phosphate dehydrogenase deficiency, Rh/ABO incompatibility, East Asian or Native American ethnicity, any jaundice observed in the first 24 hr of life (hemolysis until proven otherwise), cephalohematoma or significant bruising after delivery, and Hx of a previous sibling treated with phototherapy.

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>DERM</td>
<td>Jaundice resulting from accumulation of unconjugated, nonpolar, lipid-soluble bilirubin pigment in the skin</td>
<td>Jaundice progresses in cephalocaudal direction (face –5 mg/dL; abdomen –15 mg/dL)</td>
<td>PE</td>
</tr>
<tr>
<td>RESP</td>
<td>Pleural effusion, pulm edema</td>
<td>Maternal prenatal history</td>
<td>Resp distress</td>
</tr>
<tr>
<td>HEME</td>
<td>Hemolysis</td>
<td>Rh/ABO maternal-fetal incompatibility</td>
<td>Anemia, bruising, cephalohematomas hepatosplenomegaly, jaundice</td>
</tr>
<tr>
<td>CNS</td>
<td>Bilirubin toxic to CNS cells</td>
<td>High levels of bilirubin</td>
<td>Abnormal posture, toxicity, and reflexes</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Assess and correct hydration status.
- Active efforts to lower bilirubin levels.
- Address co-existing disease states.
- Consider atropine 0.1 mg IV to help prevent bradycardia.

Monitoring
- Arterial blood sampling may be indicated.
- Neonatal airway concerns.

Induction
- Maintain normal hemodynamics.

Maintenance
- No one agent or technique preferred.
- Few data reflecting effects of anesthetic agents on bilirubin levels.
- Adjust for FIO2 to SaO2 90–95%.
- Supplemental glucose and calcium.
- Maintain normothermia.

Extubation
- Maintain intubation if infant ill or premature or for extensive surgical procedure.

Adjuvants
- Chloral hydrate and pancuronium associated with hyperbilirubinemia.
- Maternal epidural bupivacaine associated with neonatal jaundice.

Postoperative Period
- Apnea/bradycardia risks.
- Monitor bilirubin levels.

Anticipated Problems/Concerns
- Ultimate goal of therapy and management is to prevent kernicterus.
- Decreased removal of bilirubin through gut (decreased meconium evacuation—increased enterohepatic recirculation, decreased bile flow due to liver disease or cholestasis).
- Sepsis and/or viral infection.
- Breastfeeding jaundice (occurs in first wk after birth and implies inadequate hydration or caloric intake).
- Breast-milk jaundice (unidentified factors in normal mature human milk that cause increased reabsorption of U/B from gut) can last for 3–4 w up to 3 mo.
Blebs and Bullae

**Risk**
- Prevalence of blebs as high as 6% of young, healthy adults, although spontaneous rupture occurs only in 7.4–18 per 100,000 patients.
- Incidence of ruptured bulla is 26 per 100,000 patients.
- Increased incidence of primary disease in young males.
- Increased prevalence with smoking Hx—inc alcohol and illicit substances, COPD, chronic bronchitis, cystic fibrosis, lung cancer, staphylococcal pneumonia, tuberculosis, Marfan’s syndrome, Ehlers-Danlos syndrome, alpha-1 antitrypsin deficiency, sarcoidosis, fiberglass pneumo-

**Perioperative Risks**
- Pneumothorax
- Bronchopleural fistulae
- Cava
collapse of non-ruptured giant bulla
- Pulm Htn and RV failure
- COPD

**Overview**
- “Bleb” usually refers to a collection of air caused by rupture of a single bulla or rupture of a large bulla
- Bulla are >1 cm in size and arise from various causes, which cause destruction of lung parenchyma
- Nitrous oxide is contraindicated and positive pressure ventilation should be avoided if possible
- Nitrous oxide is 35 times more soluble than nitrogen in blood. Because of this, nitrous oxide readily diffuses into any gas-filled cavity much more rapidly than nitrogen is absorbed, which leads to rapid expansion of pneumothoraces.
- In spontaneous ventilation, bullae are more compliant than normal lung tissue and preferentially fill. At higher pressures and volumes bullae are much less compliant than normal lung and therefore have much higher peak pressures than normal tissue and are prone to rupture.

**ICD-9-CM Code: 492.0**

**Perioperative Implications**

**Preinduction/Induction/Maintenance**
- Optimize oxygenation and deliver bronchodila-
tors if necessary.
- Regional or neuraxial anesthesia is preferential to general endotracheal anesthesia.
- Some associated conditions may have significant mucus plugging; fiberoptic bronchoscope with suction and irrigating capabilities may be useful.
- Careful attention to hemodynamic monitors and ventilator peak pressures and volumes is essential.
- Should have surgical team available during induction as this is most common time for pneumothorax to occur.
- Recent chest x-ray examination for severity of disease and progression is also essential.

**Monitoring**
- Routine
- Consider arterial line to more quickly recognize signs of CV collapse from pneumothorax or caval compression

**General Anesthesia**
- Maintaining spontaneous ventilation through induction can minimize complications. Avoid the use of paralytics or consider mask induction or awake fiberoptic intubation techniques.
- Inability to adequately ventilate due to bronchopleural fistula
- Inadequate venous return from caval compression
- Expansion of bulla leading to compressive effects or rupture

**Postoperative Period**
- Beware of CO2 narcosis in those who retain CO2.
- Spontaneous rupture can occur at any time. Continue adequate monitoring and watch for sudden dyspnea, desaturation, and loss of unilateral breath sounds.

**Anticipated Problems/Concerns**
- Rupture of bleb or bulla will cause a pneumothorax, which may rapidly progress to tension.
- Treatment of choice for tension pneumothorax is needle thoracostomy in second to third intercostal space in midclavicular line (in line with the nipple of a male pt). Most failures of needle thoracostomy occur from placement of needle too medial into the mediastinum.
- Obstructive pulmonary pathology incl bronchoconstriction and accessory muscle use even in the spontaneously breathing pt.
- Positive pressure ventilation is to be avoided and nitrous oxide is absolutely contraindicated.

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</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>CAD, pulm Htn, RV failure</td>
<td>Angina, DOE</td>
<td>Signs of RV failure (palpable PA, peripheral edema)</td>
<td>ECG, stress test, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Expiratory obstruction, air trapping V/Q mismatch</td>
<td>Hypoxia, hypercarbia</td>
<td>Exercise tolerance Cough</td>
<td>Pursed-lip breathing, tachypnea</td>
</tr>
<tr>
<td>ENDO</td>
<td>Possible steroid use</td>
<td></td>
<td></td>
<td>Glucose</td>
</tr>
<tr>
<td>MS</td>
<td>Barrel-chested</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Key Reference:** Kim H, Kim HK, Choi YH, Lim SH. Thoracoscopic bleb resection using two-lung ventilation anesthe-

**Etiology**
- Primary: Unknown but may be genetic. More common in young males
- Secondary: Emphysema, smoking, lung cancer, cystic fibrosis, pneumonia, tuberculosis

**Usual Treatment**
- No treatment for asymptomatic, incidental blebs
- First time rupture of a bleb is treated conserva-
tively depending on size of pneumothorax. Varies from 100% O2 to chest tube placement.
- Surgical treatment indicated for ruptured blebs in those in high-risk occupations which involve frequent changes in barometric pressure or recur-
rent spontaneous pneumothorax
- Surgical treatment of bullae done for increasing SOB or recurrent pneumothorax
- Surgical approach usually is VATS, but may require thoracotomy or median sternotomy. Laser ablation and mechanical pleurodesis may be utilized.
Bleomycin Sulfate Toxicity

**Risk**
- Pts with a Hx of germ cell tumors, squamous cell carcinomas, lymphomas, treated with bleomy- cin (BLM)
- Incidence of BLM lung toxicity (BLT) is 10–40%. The mortality is 1–2%.
- Risk of BLM increases with total dose >400 units; Creatinine clearance <50% or prior or concurrent chest radiation therapy
- Age older than 70 y
- O_{2} exposure, smoking

**Perioperative Risks**
- Exposure to high FIO_{2} can cause pneumonitis and potentially lethal ARDS.
- Pre-existing pulm fibrosis in combination with low FIO_{2} can lead to intraop hypoxia.
- Risk higher if pulm injury on PFTs, BLM exposure within 2 mo
- Pulm adverse events rarely related to the intrapleural administration of BLM for pleurodesis

**Perioperative Management**

**Preoperative Preparation**
- In pts with a Hx of testicular, squamous cell cancer, or lymphoma, inquire about exposure to BLM, dose and time of the last dose.
- Any pt with abnormal pulm studies or who is clinically symptomatic should be considered high risk for development of ARDS.
- Exposure to BLM within 2 mo should be considered high risk for postop ARDS, although even with a longer interval between the last BLM dose and exposure to hyperoxia, pts still can develop ARDS.

**Intraoperative Management**
- Maintain FIO_{2} at concentrations close to that of room air (30%) during surgery and the postop period. Accept SaO_{2} above 90% if appropriate. Consider use of PEEP to reduce FIO_{2}.
- Carefully monitor fluid replacement, focusing more on colloid administration rather than crystalloid. Consider using intravascular monitoring when large fluid shifts are expected. Treat hypotension with vasopressors and decreasing the anesthetic concentration rather than fluid boluses if appropriate.
- In high-risk pts pretreatment with corticosteroids (1 mg/kg prednisone) may be helpful in limiting postop ARDS.

**Postoperative Period**
- Provide adequate oxygenation with the lowest possible inspired FIO_{2}.
- Observe carefully for 3–5 d after surgery for signs of dyspnea, hypoxia, cough, or rales.
- Obtain daily X-ray for 3–5 d after surgery.
- Use PEEP or CPAP to treat postop hypoxia.
- Add methylprednisolone up to 1 mg/kg d if developing ARDS, diuretics in the presence of excessive lung water.

**Anticipated Problems/Concerns**
- Pts who had previously received BLM and needed O_{2} support during surgery were susceptible to development of lung toxicity and ARDS, even with low inspired FIO_{2}. O_{2}-free radicals may inactivate antioxidant enzymes leading to genetic injury, cell death, and resulting in alveolar injury.

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Pulm fibrosis ARDS with O_{2} exposure</td>
<td>Dyspnea, dry cough</td>
<td>Frequently normal Earliest sign is fine rales</td>
<td>CXR- nonspecific patchy opacities Decreased O_{2} sat PFTs—decrease in total lung volume and a decrease in vital capacity and DLCO</td>
</tr>
<tr>
<td>MUCOCUT</td>
<td>Inflammation, dermal fibrosis</td>
<td>Itching, burning, skin tenderness</td>
<td>Stomatitis, alopecia, scleroderma-like skin changes</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Minimal bone marrow toxicity</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**ICD-9-CM Code:** E930.7 (Bleomycin-therapeutic)

**Etiology**
- The toxicity is unpredictable. In the presence of Fe^{2+} and O_{2}, BLM causes DNA damage. Injury to double-stranded DNA is thought to be the major source of cytotoxicity.
- The event sequence to lung injury is: (1) endothelial and interstitial capillary edema; (2) pneumocytes type II proliferation and necrosis with surfactant release; (3) surfactant phagocytosis by alveolar macrophages with mediator release and stimulus to fibroblast production.
- Pts who had previously received BLM and needed O_{2} support during surgery were susceptible to development of lung toxicity and ARDS, even with low inspired FIO_{2}. O_{2}-free radicals may inactivate antioxidant enzymes leading to genetic injury, cell death, and resulting in alveolar injury.

**Overview**
- Antitumor antibiotic from a family of natural glycopeptides isolated from fungus *Streptomyces verticillus* used predominantly in treatment of germ cell testicular cancers, Hodgkin’s and non-Hodgkin’s lymphoma, squamous cell carcinoma of the head and neck.
- BLM is effective as a sclerosing agent for the treatment of malignant and recurrent pleural effusions.
- BLM is inactivated by the enzyme bleomycin hydrolase and thus are the predominant sites of injury.

**Worry About**
- Periop exposure to FIO_{2} >30%
- Periop hypoxia
- Carefully monitor fluid replacement, focusing on colloid rather than crystalloid.
- Intrapleural administration of BLM has been associated with local pain and hypotension requiring symptomatic treatment.

**Tests**
- Cleared by renal excretion. Elimination half time (T_{1/2}) 4 hr

Zoulfira Nisnevitch
Blindness

Risk
- Eye injuries represent 4% of claims analyzed in the American Society of Anesthesiologists (ASA) Closed Claims Project.
- Majority of entries in the ASA Postoperative Visual Loss (POVL) Registry are associated with cardiac and spine cases, with a reported incidence as high as 4.5% and 0.2%, respectively. Other surgical procedures with POVL reported incl head and neck, liver transplants, thoracoabdominal aneurysm resections, peripheral vascular procedures and prostatectomies.
- In the Registry, POVL is most often associated with ischemic optic neuropathy (ION) 89% of the time and central retinal artery occlusion (CRAO) 11% of the time.
- Blindness can result from injury to the eye, its surrounding structures (eyelid and conjunctiva), blood supply and optic nerve.
- Blindness may be transient (glycine absorption), prolonged or permanent (ischemic optic neuropathy, central retinal artery occlusion, traumatic, central ischemic events).

Perioperative Risks
- ION: Bilateral blindness in spine procedures in the prone position, cardiopulmonary bypass, head and neck dissections, where there is significant facial swelling and venous hemodynamics may be altered.
- CRAO: Periocular trauma and rarely bilateral blindness.
  * Procedure dependent factors: Anemia, blood loss greater than 1L, systemic hypotension and procedure duration greater than 6 hours.
  * Intraocular procedures, procedures around the eye, prone position with padding around the face and eyes, exophthalmos or ophthalmalic nerve blocks.
- 1.5% glycine irrigation during TURP, transurethral bladder procedures and hysteroscopic procedures in women.

Worry About
- Pressure on the globe or contact with eye by foreign objects or solutions
- Positioning of pt, esp prone
- Low blood flow states: Systemic hypotension, anemia, venous drainage impairment of the head and neck
- Operations in physical proximity to the eyes
- During ophthalmalic surgery:
  * Movement of pt under either MAC or GA during intraocular surgery
  * Trauma to optic nerve, retinal artery, or vein during orbital or sinus surgery
- Coughing or substantial Valsalva maneuvers by pt following intraocular surgery
- During ophthalmalic nerve block:
  * Perforation of globe
  * Trauma to the optic nerve, retinal artery, and vein

Overview
- Unless associated with glycine irrigating solution, blindness is often an irreversible complication following anesthesia and surgery.
- Blindness is most often associated with injury to the eye, its surrounding structures (eyelid and conjunctive), blood supply, and optic nerve.

ICD-9-CM Codes: 362.3 (Retinal vascular occlusion), 362.84 (Retinal ischemia), 368.12 (Transient), 369.00 (Acquired); 367.41 (Ischemic optic neuropathy), 950.9 (Due to nerve injury)

Perioperative Implications

Preinduction/Induction/Maintenance
- Proper positioning essential
- If prone, adequate padding so no pressure is transmitted to either globe or nasal bridge
- When the face is completely draped, consider use of a metallic Fox shield to protect eye from inadvertent pressure.

Monitoring
- Eye checks frequently during the procedure to ensure no pressure on the globe
- Ensure adequate venous drainage without increased venous pressure or increased intracranial pressure, particularly when venous outflow may be compromised by position or procedure.

General Anesthesia
- Anesthetic masks may injure eye, either through inadequate drying and application of cleaning solution to eye or through direct pressure.
- Hypotension and hypoxemia implicated in cases of CRAO.
- Hypotension, anesthesia, and prolonged procedures are implicated in ION.

Regional Anesthesia
- In ophthalmalic nerve blocks, needle does not enter globe or retinal artery, vein, or nerve. Avoid excessive volume of local anesthetic, which increases IOP and may compromise vascular supply of globe.

Postoperative Period
- When recovered in prone position, ensure that there is no pressure on orbit or globe.

Anticipated Problems/Concerns
- Absorption of glycine from 1.5% glycine irrigation fluid may be significant.
- ION almost always occurs without any other evidence of vascular injury.
- Optic nerve may be very vulnerable to hemodynamic changes in the prone position.
Botulism

Risks
• Infant botulism
• Wound botulism
• Foodborne botulism
• Adult intestinal toxemia
• Injection botulism
• Biological warfare/inhalational botulism (Category A biological threat)

Perioperative Risks
• Dx late, incorrect or missed
• Differential Dx: For adults, myasthenia gravis, Eaton-Lambert, Guillain-Barre, CVA, organophosphate exposure, tick paralysis. For infants, sepsis, failure to thrive, dehydration, encephalitis, metabolic disease
  • Non-specific history and physical findings
  • Laboratory result takes days-weeks and should be used only as confirmation; treat before confirmation
  • Triad: Bulbar symptoms, resp compromise and diluted pupils
  • Prolonged weakness requiring prolonged support
  • Enteral nutrition desired but problematic due to gastroparesis and bowel paralysis
  • Aspiration risk
  • Elevated potassium if immobile in ICU

Worry About
• Arrhythmias
• Hyperkalemia, arrhythmias, then cardiac arrest
• Prolonged weakness necessitating prolonged intubation and leading to nosocomial infection

Overview
• Botulism is a rare but serious neuropahtic illness caused by a nerve toxin (BoNT) produced by the rod-shaped bacterium Clostridium botulinum, commonly found in soil. C. botulinum grows best in low oxygen conditions; spores survive in dormant state until exposed to conditions that support growth. Seven types of toxins (A-G), but only A, B, E, F cause illness in humans; three different intracellular protein targets; different durations.
  • Infant: Between 2 w-1 y old; ingestion of spores which grow in intestine and release toxin, usually by honey ingestion; parent who works with soil; rural areas
  • Wound: IV/in skin popping drug users or organisms contaminating any traumatized tissue cause local infection and absorption of produced toxin—increased incidence over last several years in IV drug users (black tar heroin), esp in California
  • Foodborne: Improperly preserved or cooked food allows germination and toxin production by contaminating spores; consumption of food with preformed toxin results in absorption of potent neurotoxin; with education and control of food industries, now uncommon in USA; ingestion of infected inadequately cooked wildlife poses at least potential risk.
  • Intestinal: Spore colonization—possible in adults as well
  • Injection: Cosmetic (Black market toxin, Botox® overdose or spread beyond injection site), cerebral palsy (Botox® overdose or spread beyond injection site)
  • Inhalational: Genetically engineered toxin, development of biological warfare (at-risk locations). Concern is inadequate stocking of antidotes worldwide, inadequate preparation and medical support. Biological warfare in Iraq has led to organization of task forces such as Scorpio at the national/regional level to stockpile antidotes.

ICD-9-CM Code: 005.1: Botulism food poisoning, 040.01: Infant Botulism, 040.02: Wound Botulism

Etiology
• Botulinin toxin binds irreversibly to synaptic membrane of cholinergic nerves and prevents release of acetylcholine.

Usual Treatment
• Supportive, may be on ventilator for weeks, intense medical and nursing care
• Nutritional support; enteral preferred (basic maintenance plus need to keep bowels moving to eliminate spores), but parenteral also required
• Early antitoxin treatment shows better outcome, antitoxin blocks action of circulating toxin, prevents pts from worsening but recovery still takes many weeks
• Equine-derived antitoxin for adults (risk of serum sickness/anaphylaxis); skin testing and desensitization instructions provided with antitoxin; more broad spectrum antitoxins associated with increase in hypersensitivity
• Presently trivalent antitoxin preparation is available for adults (10 mL vial with 7500 IU type A, 5300 IU type B, 8500 IU type E)
• Baby BIG (human botulism immune globulin) used for infant botulism came out in 1990, more in use since 2003
• Botulism reportable to CDC or state health department, requires report to obtain antitoxin
• Antibiotics for secondary infections
• Avoid aminoglycosides and clindamycin, which may potentiate or exacerbate neuromuscular blockade
• Guanidine increases the release of acetylcholine from nerve terminals, appears to be useful in mild cases
• Modern clinical practice and early antitoxin treatment: mortality reduced from 60% to ≤10%

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Pharyngeal constrictor and genioglossal hypotonia, paralysis of resp musculature, Infection, Atelectasis</td>
<td>Drooling, Poor feeding, Decreased resp effort, Increased secretions, Tracheal secretions, Poor resp effort, Poor color</td>
<td>Poor head control, Absent gag, Weak cough, Fever, Rhonchi, Rales, Cyanosis</td>
<td>Diagnosis of elimination; electrophysiology studies are fastest diagnostic tool to rule out other causes; EEG and neuroimaging are normal as long as there is no hypoxic insult; edrophonium test to rule out myasthenia gravis shows no improvement</td>
</tr>
<tr>
<td>GI</td>
<td>Constipation</td>
<td>No bowel movement, Irritability</td>
<td>Palpable stool, Abdominal distention</td>
<td>Stool samples are difficult to collect due to constipation, can use sterile water enema</td>
</tr>
<tr>
<td>RENAL</td>
<td>UTI</td>
<td>Foul-smelling urine</td>
<td></td>
<td>Serum testing possible if stool unobtainable but low sensitivity compared to stool testing (negative serum test does not exclude possibility of infant botulism) Samples injected into mice, look for signs of botulism</td>
</tr>
<tr>
<td>CNS</td>
<td>SIADH, Seizures, Cranial neuropathies</td>
<td>Infrequent urination, Twitching, Altered consciousness, Ptsis, Expressionless face, Feeble cry</td>
<td>Diminished urine flow, Seizure activity, Fixed and diluted pupils, Facial palsy, Poor cough and gag</td>
<td>Laboratory result takes days-weeks and is used as confirmation Tests only performed at some state health department labs and CDC; samples must be collected sterilely, refrigerated and shipped with cold packs</td>
</tr>
<tr>
<td>PNS</td>
<td>Spinal neuropathies</td>
<td>Limp limbs, Hypotonia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Implications**
- Early diagnosis, treatment, and optimization
- Continue supportive resp care
- Sepsis from secondary infections
- Avoid resp depressants, paralytics
- Aspiration risk
- Pts may require feeding tube and/or parenteral nutrition
- Likely to OR for wound debridement
- If possible, avoid airway manipulation, unnecessary medications and those that are resp depressants

**Preoperative Preparation**
- Recommend pt receives antitoxin prior to wound debridement so additional toxin release does not cause further paralysis
- Low threshold for treatment if suspecting botulism
- Manage preop electrolytes
- Continue antibiotics
- CXR to help assess status
- Aspiration prophylaxis
- If pregnant, parturient can safely be given antitoxin (intrapleurally in severe cases); consider early tracheostomy to avoid sequelae of resp depression; botulism not known to cause direct fetal risks, only those associated with mother’s ventilatory compromise, since molecule is too large to pass through placental barrier

**Monitoring**
- Standard ASA monitors
- If unstable in ICU, consider arterial cannulation for management of autonomic dysfunction (infants may see motor function return before autonomic system)

**Airway**
- Aspiration risk
- May already be intubated

**Induction**
- Avoid succinylcholine
- May not require paralytic

**Maintenance**
- May not require paralytic throughout case

**Extubation**
- Likely unable to extubate
- Continue supportive care postop

**Adjuncts**
- Avoid resp depressants if possible
- Consider regional procedures rather than narcotics for pain control in wounds

**Postoperative Period**
- Continued supportive care
- Manage electrolytes

**Associated Problems/Concerns**
- Aspiration pneumonia
- Sepsis from wound
- Missed diagnosis
- Malnutrition
- Biological warfare: Limited information on effectiveness of antitoxin success with inhalational botulism, amount of neutralizing antibody in presently available formulation may not be enough for treatment of genetically engineered toxin.
Brain Death

Risk
- Shortage of organs for transplant persist despite utilization of expanded donor pool and living unrelated donors
- Inadequate donor organ function limits organ supply
- Optimizing donor management increases donor yield

Perioperative Risks
- CV instability
- Endocrine dysfunction
- Metabolic imbalance
- Coagulopathy
- Hypothermia

Worry About
- Organ loss secondary to CV collapse

Overview
- Brain death secondary to cerebral herniation from increased ICP
- Associated with autonomic storm, vasomotor instability and hormone deficiencies resulting in hypotension
- Criteria: Irreversible coma (no response to painful stimuli), absent brainstem reflexes, apnea and no confounding conditions (hypothermia <35° C, metabolic disturbances, intoxication incl neur muscular blockade)

ICD-9-Cm Code: 348.8

Etiology
- Traumatic brain injury and SDH

Usual Treatment
- Critical pathways promoted to enhance organs transplanted per donor

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Neurogenic pulm edema</td>
<td></td>
<td></td>
<td>ABGs, CXR</td>
</tr>
<tr>
<td></td>
<td>ALI/ARDS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Hypovolemia</td>
<td></td>
<td></td>
<td>BP</td>
</tr>
<tr>
<td></td>
<td>Ventricular dysfunction</td>
<td></td>
<td></td>
<td>Cardiac catheterization</td>
</tr>
<tr>
<td></td>
<td>Depletion of myocardial substrates</td>
<td></td>
<td></td>
<td>TEE</td>
</tr>
<tr>
<td></td>
<td>Unstable vasomotor center</td>
<td></td>
<td></td>
<td>Atropine resistance (2 mg)</td>
</tr>
<tr>
<td></td>
<td>Nonfunctional sympathetic system</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Arrhythmias</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulopathy</td>
<td></td>
<td></td>
<td>Coagulation studies</td>
</tr>
<tr>
<td></td>
<td>Anemia</td>
<td></td>
<td></td>
<td>Hct</td>
</tr>
<tr>
<td>GU</td>
<td>Diabetes insipidus</td>
<td></td>
<td></td>
<td>UO &gt;3 mL/kg/hr</td>
</tr>
<tr>
<td></td>
<td>Osmotic diuresis</td>
<td></td>
<td></td>
<td>Lytes</td>
</tr>
<tr>
<td></td>
<td>Hypernatremia</td>
<td></td>
<td></td>
<td>Urine SG &lt; 1.005</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Serum osmolality &gt;310 mOsm</td>
</tr>
<tr>
<td>CNS</td>
<td>Lack of cerebral and brainstem function</td>
<td></td>
<td></td>
<td>CT, EEG, cerebral angiography</td>
</tr>
<tr>
<td></td>
<td>Poikilothermic</td>
<td></td>
<td></td>
<td>Midsized pupils</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Absent brainstem reflexes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Positive apnea test</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Temp monitor</td>
</tr>
</tbody>
</table>


Perioperative Implications

Monitoring
- Temp
- A-line
- CVP or PA catheter
- UO
- ABGs

Airway
- Low FIO, and PEEP (5 cm H2O)
- Tidal volumes to keep peak pressure <30 mmHg

Maintenance
- Correct metabolic derangements (avoid acidosis, hypoxemia, and hypercarbia) and monitor for, and nudge toward correction of, electrolyte abnormalities (hypernatremia and hyperglycemia)

- Treat anemia to Hct >30% and keep coagulation studies normal (values <1.5X control)
- Dopamine or dobutamine for MAP >60 mmHg
- Hormonal resuscitation with insulin, steroids, vasopressin and thyroid hormone (Papworth cocktail) and pulm artery catheter placement for EF <45%
- Fluids and hemodynamic medications to minimize use of alpha-agonists
- Lung expansion ventilatory techniques, judicious colloid fluid resuscitation and steroids to preserve lung function
- Maintain UOP >100 mL/hr and treat DI with DDAVP or low dose AVP
- Keep normothermic

Extubation
- Not done
- Ventilation discontinued when cross-clamp:
  - Ascending aorta for heart-lung donors
  - Descending aorta for liver-kidney donors above SMA and celiac artery

Adjuvants
- Drugs per procurement team, e.g., heparin, chlorpromazine

Anticipated Problems/Concerns
- Anticipate increase in BP and HR with incision—does not obviate criteria for brain death
- Knowledge of sequelae of brain death
Bronchiectasis

Risk
- Incidence in USA: <1:10,000 hospital admissions
- Gender prevalence: None
- Socioeconomic or ethnic prevalence: Inbreeding and primitive health care, particularly lack of immunization and poor treatment of childhood bronchitides, increase the prevalence. Ciliary deformities have been shown in a Polynesian population.
- Occasionally seen in children:
  - Bronchial cartilage deficiency (Williams-Campbell syndrome)
  - Tracheobronchomegaly (Mounier-Kuhn's syndrome)
- Inherited immunoglobulin deficiencies, impaired phagocytosis, complement deficiency
- α1-Antitrypsin deficiency
- Occasionally seen in adults with acquired γ-globulin deficiency:
  - Cystic fibrosis
  - RA
  - Pulm ciliary dyskinesias (Kartagener's syndrome)

Perioperative Risks
- Spillage of infected secretions from bronchietatic regions to normal lung leads to pneumonia, retention of secretions

Worry About
- Exacerbation of asthma
- Amount of sputum produced and its nature
- Fever, hemoptysis: Acute pulm infection
- Right heart function
- Check frequency of cough and daily sputum volume; culture and smear for composition; check body temp and WBC count for acute infection
- Exercise tolerance will indicate associated impairment or disability. Right heart function may need assessment.

Overview
- Abnormal widening or dilatation of one or more branches of the bronchial tree. Widened segments commonly filled with purulent secretions; mucosa is swollen and inflamed and may be ulcerated with granulation tissue exposed. Extensive collateral flow occurs in these chronically inflamed bronchi (3–12% of CO).

ICD-9-CM Codes: 494; 748.61 (Congenital); 011.5 (Tuberculous)

ASSESSMENT POINTS

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<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Sinusitis</td>
<td>Postnasal drip</td>
<td>Translucency</td>
<td>X-ray, ultrasound, bright lights</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stuffed, headache</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Clubbing, cyanosis</td>
<td>Exercise tolerance</td>
<td></td>
<td>ABGs</td>
</tr>
<tr>
<td></td>
<td>CHF (cor pulmonale)</td>
<td>Pulm Htn, edema</td>
<td></td>
<td>Loud P</td>
</tr>
<tr>
<td></td>
<td>Kartagener's syndrome</td>
<td>Chronic sinusitis</td>
<td></td>
<td>Right heart studies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Situs inversus</td>
<td></td>
<td>Immotile spermatozoa</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchiectasis</td>
<td>Cough, sputum</td>
<td>Rhonchi</td>
<td>Smear, culture, high-resolution CT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hemoptysis</td>
<td>CXR—93% tram lines, 7% normal</td>
<td>Bronchoscopy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Wheezing</td>
<td></td>
<td>Bronchoscopy</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>PFTs</td>
</tr>
<tr>
<td>HEME</td>
<td>Immunodeficiency</td>
<td></td>
<td></td>
<td>IgG, IgA, WBC</td>
</tr>
<tr>
<td></td>
<td>Infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Brain abscess</td>
<td></td>
<td></td>
<td>CT/MRI</td>
</tr>
</tbody>
</table>


Perioperative Implications

Monitoring
- Routine: Consider PA catheter for cor pulmonale or CHF

Airway
- Careful frequent suctioning and humidification of inspired gases

Induction
- Avoid asthma exacerbation
- Consider regional anesthesia when possible

Maintenance
- Routine

Extubation
- Depends upon degree of pulm and cardiac dysfunction

Adjuvants
- Routine

Postoperative Period
- Use stir-up regimen; monitor for retained secretions and resp failure
- Check for platypnea, orthodeoxia if right atrial pressures become elevated

Etiology/Pathogenesis
- Exact etiology for acquired form remains unclear but often involves necrotizing infection in tracheobronchial wall. Five mechanisms may predispose:
  - Bacterial, viral, or fungal bronchopulmonary infections, incl TB, pertussis, and measles
  - Bronchial obstruction
  - Immunodeficiency states, incl IgG deficiency, IgA deficiency, and leukocyte dysfunction
  - Hereditary defects in ciliary-mucosal clearance, incl Kartagener's syndrome, α1-antitrypsin deficiency, and cystic fibrosis
  - Miscellaneous disorders, incl recurrent aspiration, inhaled irritants, Young's syndrome, and bronchiolitis obliterans following heart-lung transplantation

Usual Treatment
- Medical therapy: Postural drainage, deep breathing and assisted coughing, antibiotics, bronchodilators, and fluids/humidity. Drainage of sinuses.
- Surgical therapy: Resection indicated for uncontrolled hemoptysis; or lobar closely confined disease, age > 20 y. Bronchopulmonary lavage under GA with divided airway (double-lumen tube).

Anticipated Problems/Concerns
- Retained secretions, secondary resp failure
- Right heart decompensation if hypoxemia persists
- Bacteremia from airway manipulations

DISEASES
Bronchiolitis Obliterans Syndrome

Roy C. Levitt
Yiliam F. Rodriguez-Blanco

DISEASES

Risk
- Incidence in USA: 1:40,000
- Racial predilection: None
- Occurs primarily after lung and bone marrow transplantation
- Industrial workers who have presented with bronchiolitis obliterans syndrome (BOS): Nylon-flock, battery workers, manufacturer of flavorings (diacetyl butter-like flavoring)

Perioperative Risks
- Hypoxemia and severe periop airway obstruction
- Pulm infection, sepsis, pulm edema posttransplant
- Injury to tracheal anastomosis due to ETT placement
- Prolonged intubation (increased sensitivity to medications incl muscle relaxants, pulm functions, renal impairment and pulm edema)
- Complications of immunosuppression (infection, hemorrhage, renal impairment)
- Preop focus must differentiate between active invasive pulm infection and ongoing chronic rejection with colonization; and maximizing medical condition and stratifying risk.

Worry About
- Pulm functions
- Differentiating BOS from untreated invasive pulm infection and other disorders
- Side effects of immunosuppression incl infection with invasive techniques, hemorrhage, and renal failure with cyclosporine
- Allograft denervation (physiologic and pharmacologic side effects)
- Other effects of etiologic agents

Overview
- Persistent airflow obstruction often associated with chronic inflammation and scarring that obliterates small airways resulting in progressive obstructive lung disease.
- Because bronchiolitis obliterans (BO) is difficult to confirm histologically (transbronchial biopsy of larger airways with sporic involvement often provides insufficient samples and has a high false-negative diagnostic rate), the International Society for Heart and Lung Transplantation proposed a staged clinical definition of BOS termed bronchiolitis obliterans syndrome (BOS; stages 0-3 defined by changes in pulm functions rather than histology).
- BOS clinical staging is important to the clinician because it indicates allograft function.

ICD-9-CM Code: 491.8

Etiology
- The mechanism involved in the etiology of BO remains poorly understood.
- Two forms of BOS with inflammation and fibrosis: Rejection-related and non-rejection related.
- After transplant, the syndrome reflects small airway obliterations due to "chronic rejection."
- Several risk factors incl: Transplantation, ischemia-reperfusion injury; allomunity, Hx of acute graft rejection, mismatches at human leukocyte antigen (HLA) loci, development of antibodies to class I HLA, GE reflux with resultant aspiration; loss of cough reflex due to denervation, complication of prematurity (bronchopulmonary dysplasia); toxic inhalation ("Popcorn lung"); exposure to infectious agents (bacterial, viral, and some atypical organisms incl mycoplasma, chlamydia and fungi) (BO with organizing pneumonia or BOOP).
- BOS is described after lung, heart-lung, bone marrow, renal, pancreas, liver and hematopoietic stem cell transplantation; BOS remains the leading causes of death after lung transplantation.

Usual Treatment
- Varies depending on whether or not BOS is rejection-related
- Rejection-related BOS is mainly treated with additional immunosuppression and supportive care incl O2, bronchodilators, and chest physical therapy
- Non-rejection related BOS is treated with supportive care, anti-inflammatory agents, and may respond to steroids (esp toxic fumes and other environmental exposures).
- Newer treatments for rejection-related: Azithromycin, aerosolized cyclosporine, augmentation of immunosuppression agents, statins, (IL-1 receptors antagonist, IL-2 receptor antagonists, and adenosine A<sub>2a</sub> receptor agonists in post-transplant pts have been proposed recently)
- Severe cases often require lung transplant and even retransplant.

Portooperative Implications

Preoperative Preparation
- PFTs for BOS staging and resp status, bronchoscopy for biopsy and culture
- Treat active infections aggressively
- Evaluate renal functions, adjust periop medications where appropriate
- Continue anti-infective and immunosuppressive therapy during the periop period and adjust dosing to keep within the indicated therapeutic range
- Strict aseptic techniques due to immunosuppression
- Premedication useful due to excessive secretions, but avoid excessive resp depression
- Corticosteroids supplementation esp for long, invasive, stressful procedures
- Watch for: Increased sensitivity to opioids, hypercarbia, resp acidosis, bronchial hyperresponsiveness (bronchoconstriction), hyperkalemia and hypomagnesemia
- Most common side effect of immunosuppressive drugs: Cyclosporine and tacrolimus (HTN, diabetes, neuromyotoxicity, renal failure), OKT3 (leukopenia, fever, anaphylaxis), azathioprine (anemia, thrombocytopenia)

Monitoring
- Routine
- Consider arterial line placement if hypoxic, acidic or O2 saturation inadequate—invasive monitoring must be carefully weighed against possible infection from intravascular catheters
- TEE may be helpful in monitoring cardiac functioning in post heart-lung transplant pts, and when there is evidence of pulm edema and pulm Htn
- CVP insertion recommended (when necessary) on side of native lung (one lung transplant)

Airway
- ETT cuff placement should avoid tracheal anastomosis
- Oral intubation is preferred over nasal intubation (infection, thrombocytopenia)
- Increase FIO2
- Use aseptic tracheal suction technique

Induction
- Short acting agents are preferred and adjust doses to pt status and to avoid prolonged CV depression

Maintenance
- Avoid fluid overload (disruption of lymphatic drainage in posttransplant cases can lead to pulm edema with fluid overload)
- Significant reductions of cyclosporine or tacrolimus blood levels can be caused by dilution with IV fluids

ASSESSMENT POINTS

Use previous classification to determine possible cause of BOS including post-transplantation or environmental exposure(s).

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENERAL</td>
<td>Active infection</td>
<td>Fever, non-relation change in status</td>
<td>↑ Temp, tachycardia with infection</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Loss of lung functions (% FEV&lt;sub&gt;1&lt;/sub&gt;)</td>
<td>Recent change in functional capacity; invasive lung infections, meds, lung colonization (resistant bacteria), risk factors, BOS staging; environmental exposure (e.g., diacetyl production)</td>
<td>↑ Tachypnea, wheezes, cough, fever, cyanosis, pulm edema</td>
<td>CXR, PFTs (↑ FEV&lt;sub&gt;1&lt;/sub&gt;, ↓ O2 saturation, hypoxia) brochoscopy for endobronchial biopsy, culture, bronchoalveolar lavage, lung biopsy (diagnosis)</td>
</tr>
<tr>
<td>RENAL</td>
<td>Loss of function due to immunosuppression</td>
<td>Change in status, dialysis</td>
<td>A-V fistula (avoidance for procedures), fluid overload, pulm edema, ↑ weight</td>
<td>↓ Renal functions, tachycardia, peripheral edema, SOB</td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenia due to medications</td>
<td>Prolonged bleeding</td>
<td>Bruising</td>
<td>CBC with ↓ platelets, ↑ bleeding time</td>
</tr>
</tbody>
</table>

• Adjust neuromuscular blocking dosage due to interactions with immunosuppressive agents and adjust dosage if renal impairment. (Cyclosporine enhances the effect of muscle relaxants producing a prolonged block.)
• Prevent additional mechanical obstruction (ventilator-induced disease and excessive tidal volumes).
• Hyperventilation during mechanical ventilation should be avoided because seizure threshold in pts taking immunosuppressive agents may be lowered.
• Use shorter-acting agents to avoid prolonged CNS, CV, and resp depression to facilitate a swift recovery of functions and timely extubation.

Extubation
• Delay until adequate ventilation assured (sustained tetanus on monitoring).
• The lack of cough reflex below the tracheal anastomosis makes pts unable to clear secretions, unless they are awake, increasing the risk of silent aspiration.

Adjuvants
• Consider regional technique for anesthesia/periop analgesia.

Postoperative Period
• Monitor for and aggressively treat resp depression, infection, and fluid overload.

Anticipated Problems/Concerns
• Many pts with resting hypoxia and marginal compensated lung functions come to OR for diagnostic lung biopsy. A thorascopic technique may be impossible owing to adhesions post heart/lung transplantation or inability to tolerate one-lung ventilation.
• Anticipate further perioperative resp decompensation after open-lung biopsy.
• Arrange postop disposition (monitored bed and ventilator support) depending on preop functional status and the potential for periop complications.
# Bronchitis, Chronic

## Risk
- Incidence in USA: 14 million
- Race with highest prevalence: Caucasian
- M:F ratio: 1:2
- Smoking, second-hand smoke, occupational exposure to pulm toxic substances (radon, coal, silicates, asbestos)

## Perioperative Risks
- Bronchospasm

## Worry About
- Airway stimulation at light levels of anesthesia
- Laryngospasm (due to secretions and hyperreactivity)
- Hypoxia
- Hypercarbia

## Overview
- Chronic productive cough with periodic exacerbations (most d for at least 3 mo and for at least 2 consecutive y)
- Enlargement of the mucus-secreting glands in the airways with excessive sputum production
- Expiratory airways obstruction
- Derangement in V/Q relationships
- Chronic hypoxia with right heart failure
- Exacerbations with intercurrent bacterial or viral infections

## ICD-9-CM Code: 491.9

## Etiology
- Acquired, usually due to smoking
- May also be due to asthma or frequent childhood resp infections

## Usual Treatment
- Avoidance of environmental irritants such as cigarette smoke (preferably >8-10 wk prior to elective surgery)
- Antibiotics for acute exacerbations; ineffica-
cious for prophylactic treatment
- Oral glucocorticoids appropriate for acute exacerbations but not for maintenance therapy
- Short-acting bronchodilators, such as beta agonists or anticholinergics, for acute exacerbations; long-acting beta agonist bronchodilators plus inhaled steroids for long-term maintenance therapy

## Assessment Points

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
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<th>Test</th>
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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Right heart failure</td>
<td>Exercise tolerance</td>
<td>RV heave</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>Pulm Htn</td>
<td></td>
<td>Dependent edema</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Airways obstruction</td>
<td>Smoking Hx (current, recent, remote)</td>
<td>Cyanosis</td>
<td>PFT, DLco, ABGs</td>
</tr>
<tr>
<td>MS</td>
<td>Airways obstruction</td>
<td>Clubbing of fingers</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## Key Reference

## Perioperative Implications

### Preoperative Preparation
- Smoking cessation
- Antibiotics to decrease sputum production
- Resp conditioning

### Monitoring
- Consider arterial line to monitor blood gases
- Consider pulm artery catheter for large fluid shift operations

### Airway
- Often, truncal obesity (esp with corticosteroids); may have redundant soft tissue in airway or short, fat neck

### Preinduction/Induction
- Avoid stimulating the airway while in light levels of anesthesia; may precipitate bronchospasm (although less likely than with asthma)
- Regional anesthesia may be preferable

### Maintenance
- Frequent suctioning of ETT
- Limit narcotic administration (danger of periop CO, retention)
- Adjuvant regional anesthesia for postop pain management in procedures that affect resp mechanics (e.g., intercostal nerve blocks, epidural analgesia)

### Extubation
- Administer intratracheal bronchodilator in responsive pts prior to extubation
- Consider IV lidocaine prior to extubation

### Anticipated Problems/Concerns
- Postop resp complications (secretions, mucus plugging, atelectasis, pneumonia, prolonged requirement for mechanical ventilation)
Bronchiopulmonary Dysplasia

Risk
- The at-risk group is largely comprised of infants born at 24–28 wks gestation who have very low birth weight (VLBW).
- Incidence of bronchiopulmonary dysplasia (BPD) is inversely proportional to gestational age.
- Classic BPD occurs in infants with severe RDS requiring prolonged mechanical ventilation and O₂ therapy.
- Other VLBW infants require mechanical ventilation for apnea/poor resp effort likely due to immaturity of central respiratory control.

Perioperative Implications

Preoperative Preparation
- Optimal medical management achieved with bronchodilators and diuretics
- Electrolytes and Hg WNL for age
- GERD controlled with medication
- NICU parameters are: Pao, 50–70 mmHg, Paco₂, and pH about 7.3. Goals intraop are to avoid hyperoxia and hypocarbia without predisposing to hypoxemia. GERD common in VLBW infants, which improves with growth.
- Retinopathy of prematurity (ROP) in same at risk infants.
- Postop apnea following general anesthesia or sedation in infants <54 wks PCA.
- Occult RAD in formerly premature infants who otherwise appear well.

Perioperative Risks
- Adequate oxygenation and ventilation during intraop period and transport.
- NICU parameters are: Pao, 50–70 mmHg, Paco₂, and pH about 7.3. Goals intraop are to avoid hyperoxia and hypocarbia without predisposing to hypoxemia. GERD common in VLBW infants, which improves with growth.
- Preoperative Preparation
- Deep extubation as in pt with asthma may be appropriate in older child

Adjuvants
- Spinal anesthesia acceptable in suitable procedures, particularly premature infants

Anticipated Problems/Concerns
- Tenuous pulm status that is challenged during anesthesia and surgery
- Some infants may not fit extubation criteria and require additional mechanical ventilation postop
- Older child with Hx of BPD at risk for RAD.

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>CV</td>
<td>Cor pulmonale</td>
<td>Inability to wean from ventilator</td>
<td>Tachypnea</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>LV failure</td>
<td>Poor feeding and weight gain</td>
<td>Sternal retractions</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>↑ Airway reactivity</td>
<td>Inability to wean from supplemental</td>
<td>Tachypnea</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>↑ FRC</td>
<td>O₂ or ventilator</td>
<td>Sternal retractions</td>
<td>ABGs</td>
</tr>
<tr>
<td></td>
<td>↑ Paco₂</td>
<td>Poor feeding and weight gain</td>
<td>Nasal flaring</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Paco₂</td>
<td>Frequent URI associated with bronchospasm</td>
<td>Nasal flaring</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Wheezing/rhonchi</td>
<td>Wheezing/rhonchi</td>
<td></td>
</tr>
</tbody>
</table>


Airway
- Expect reactive airway
- Bronchodilators: Albuterol and/or ipratropium bromide
- Nutritional support for growth and additional work of breathing
- Diuretics and fluid restriction
- Methylxanthines to increase resp drive and decrease apnea

Expiratory System
- Cor pulmonale
- LV failure
- Inability to wean from ventilator
- Poor feeding and weight gain
- Dyspnea in extubated infant
- Tachypnea
- Sternal retractions
- Nasal flaring
- Cardiomegaly
- Pulmonary rales
- Oxygen exposure
- Early extubation and use of nasal CPAP
- Bronchodilators: Albuterol and/or ipratropium bromide
- Nutritional support for growth and additional work of breathing
- Diuretics and fluid restriction
- Methylxanthines to increase resp drive and decrease apnea

Preinduction/Induction
- Ensure adequate anesthetic depth prior to instrumentation of the airway (once the cycle of bronchospasm and desaturation has been initiated, it is difficult to recover).
- Usually inhalational with sevoflurane
- Judicious use of narcotics

Maintenance
- Awake with regular resp pattern during infancy
- Postop apnea monitoring

Anticipated Problems/Concerns
- Prolonged mechanical ventilation, pulm Htn, cor pulmonale, and prolonged O₂ dependence are poor prognostic indicators.
- Rehospitalization for pulm disease is most common in the first 2 y of life with gradual decrease in symptom frequency through childhood.

ICD-9-CM Code: 770.7

Mary A. Keyes
Buerger’s Disease: Thromboangiitis Obliterans

Jeremy Hansen
James Duke

Risk
- Current or recent chronic tobacco/nicotine exposure
- Ashkenazi Jewish ethnicity, prevalence much greater in Eastern Europe, Southeast Asia, Japan
- Age < 45, male gender (M:F ratio: 10–100:1)
- Incidence in the USA: Progressively decreasing in association with decreasing smoking prevalence: < 8–10/100,000

Perioperative Risks
- Similar to any pt with chronic tobacco exposure
- Risks to already compromised perfusion of distal extremities

Worry About
- Co-existing pulm disease as pts are tobacco smokers
- Abnormal Allen test result in young (<45 yr) male smoker with leg ulcerations (classic clinical scenario for Buerger’s)
- All extremities as TAO is never confined to a single limb

Overview
- Inflammatory vasculitis of small and medium arteries and veins in extremities
- Classic distribution is infrapopliteal or distal to the brachial artery

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Coronary lesions c/w ischemia</td>
<td>Angina, MI, CHF</td>
<td>Third heart sound, regular rhythm</td>
<td>ECG, coronary angiography if high</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>or no, rales</td>
<td>suspicion for coronary lesions</td>
</tr>
<tr>
<td>RESP</td>
<td>c/w Chronic tobacco exposure</td>
<td>SOB, cough, increased sputum</td>
<td>Findings c/w chronic smoker</td>
<td>PFT results c/w obstructive pattern</td>
</tr>
<tr>
<td>HEME</td>
<td>Carboxyhemoglobin</td>
<td>Smoking Hx</td>
<td>N/A</td>
<td>Blood gases with co-oxymetry</td>
</tr>
<tr>
<td>CNS</td>
<td>Vascular lesions -&gt; cerebral ischemia</td>
<td>Syncopal episodes, TIA, CVA</td>
<td>Carotid bruit</td>
<td>Carotid US, CT angiogram</td>
</tr>
<tr>
<td>GI</td>
<td>Mesenteric ischemia</td>
<td>“Intestinal angina”</td>
<td>Abdominal bruit</td>
<td>Mesenteric angiography</td>
</tr>
<tr>
<td>EXTREMITIES</td>
<td>Distal ischemia; gangrene</td>
<td>Claudication, rest pain, non-healing lesions, prior amputations</td>
<td>Cool extremities, poor capillary refill, hair loss, thrombosis migrans, ulcerations/gangrene</td>
<td>Allen test, Doppler ultrasound, angiography with c/o ‘corkscrew collateral’ revascularization</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preinduction/Induction/Maintenance
- Carefully document location/extent of distal extremity ulcerations and thrombosis migrans.
- Optimize pre-induction pulmonary status.
- Pay special attention to padding and protection of distal extremities.
- Prevent hypothermia in the entire periop phase by keeping extremities warmed and covered.

Monitoring
- Consider risks vs. benefits of distal arterial, e.g., radial arterial catheterization.

- Femoral arterial line would be a viable option for invasive monitoring.
- Pulse oximetry may be more accurate in a proximal location, such as the ear lobe.

General Anesthesia
- OR ambient temperature should be increased.
- Maintain intravascular volume and avoid alpha agonists if possible.
- Regional anesthesia can be performed safely
- Avoid epinephrine in local anesthetic solutions to limit risk of vasospasm.

Postoperative Period
- Keep distal extremities warm; 40% of pts have concurrent Raynaud’s phenomenon

Anticipated Problems/Concerns
- Excellent opportunity to reiterate importance of smoking cessation
- If no critical limb ischemia, smoking cessation will prevent amputation
- Long-term prognosis for major amputation: 11% at 5 y; 21% at 10 y; 23% at 20 y
Bulimia

Risk
- Affects 5–18% of adolescent girls and young women
- Bulimic symptoms can be part of anorexia nervosa syndrome

Perioperative Risks
- Increased risks (which have not been quantified) of hypotension, cardiac arrhythmias, hypothermia, aspiration of gastric contents, and metabolic abnormalities and their consequences

Worry About
- Reduced cardiac muscle mass with decrease in chamber size, impaired myocardial contractility with decreased cardiac output, and relative hypotension
- Mitral valve prolapse and its arrhythmogenic effects
- Starvation, dehydration and electrolyte abnormalities (hyponatremia, hypokalemia, hypoalbuminemia, hypomagnesemia, hypocalcemia, hypophosphatemia)
- Alterations (hypofunction) in autonomic nervous system function and a hypervagal state
- Abnormal temp regulation
- Decreased gastric emptying, gastric dilatation, diminished GE sphincter tone, aspiration of gastric contents, gastric rupture and accompanying peritonitis
- Mallory-Weiss tear or esophageal rupture leading to acute mediastinitis

Overview
- Eating disorder characterized by binge-eating episodes followed by self-induced vomiting, fasting, and abuse of diuretics or laxatives
- Greatest periop risks are associated with low cardiac output and cardiac arrhythmias
- Hx is characterized by denial and is often unreliable. Ps may report exercise intolerance, cold intolerance, weight fluctuation, syncope

ICD-9-CM Codes: 783.6; 307.51

Etiology
- Unknown; thought to be largely emotional

Usual Treatment
- SSRIs, such as fluoxetine (Prozac), have been found most effective pharmacotherapy. Second line of pharmacologic treatment is with tricyclic antidepressants.
- Cognitive behavioral therapy
- K+ supplements

Assessment Points

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<th>Test</th>
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<tbody>
<tr>
<td>CV</td>
<td>Cardiomyopathy, mitral valve prolapse, arrhythmia, specia cardiomyopathy</td>
<td>Exercise intolerance, syncope</td>
<td>Heart sounds, BP, pulse</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>GI</td>
<td>Gastric dilatation, diarrhea, Gastric rupture/peritonitis, Hepatic dysfunction, Inanition</td>
<td>Usually unreliable</td>
<td>Projectile vomiting, Skin turgor, pulse, BP, abdomen</td>
<td>Lytes, CT scan, ABG, CBC, Hepatic enzymes, Serum glucose</td>
</tr>
<tr>
<td>ENDO</td>
<td>Amenorrhea, “euthyroid sick,” ↓ norepinephrine, ↓ vasopressin secretion, abn temp regulation</td>
<td>Cold intolerance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Pancytopenia</td>
<td>Bruising, infections</td>
<td>Skin</td>
<td>CBC, plt</td>
</tr>
<tr>
<td>RENAL</td>
<td>↓ GFR on basis of dehydration</td>
<td>Marked weight fluctuation</td>
<td>Thin</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Depression, ↓ CSF norepinephrine</td>
<td>Marked weight fluctuation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Muscle mass</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Perioperative Preparation
- Assess cardiac, lyte, hepatic enzymes, volume status, UPT
- Consider urine toxicology screen to rule out co-morbid substance abuse

Monitoring
- Routine
- Arrhythmia, volume status, myocardial function
- Temp monitoring important

Airway
- May have increased risk of aspiration of gastric contents

Induction
- Hypovolemia, myocardial dysfunction, ANS dysfunction may make for CV instability.

Maintenance
- CV instability, volume and lyte status, temp should dictate anesthetic regimen.

Extubation
- Awake due to GI motility dysfunction
- Autonomic hypofunction may lead to sudden postop collapse.

Adjuvants
- Vary if lyte, renal, or hepatic dysfunction exists.

Anticipated Problems/Concerns
- Gastric volume changes may increase risk of aspiration.
- Volume status, lyte, CV, and ANS changes increase risk of hypotension, arrhythmia, and sudden postop collapse
- Habitus and metabolic changes may predispose to hypothermia.
- Menstrual irregularities. UPT advised.
Burn Injury, Chemical

Risk
• 3% of all reported burn injuries from 1999–2008.
• Risk increases with age; 1% of burn injuries from birth to age 16; 3.7% from 20–30; and 5% from 30–50 according to the National Burn Repository Data on Data from 1999–2008.
• Majority of chemical exposures are occupational, occurring in men working age. Assaults with caustic chemicals are more likely to occur against women.
• American Association of Poison Control Centers reports approximately 130,000 exposures to caustic substances in 2007.

Perioperative Risks
• Morbidity varies by exposure type and substance. Surface burns may be regarded like thermal burns after decontamination.
• Caustic ingestion may result in perforation and/or bleeding and resp compromise from upper airway edema.

Worry About
• Identify injury setting, chemical involved, areas of exposure, and duration before decontamination.

Assessment by Hx and frequent sampling of arterial blood.
• Consider arterial line placement for extensive debriement/procedures to allow beat-to-beat monitoring and frequent sampling of arterial blood.
• Presence of an arterial line should not preclude placement of an NIBP cuff (backup if arterial line fails during procedure). Negotiate with surgeon best location for NIBP cuff.

Overview
• A large number of chemicals can potentially cause injury incl acids, bases, organic, and inorganic compounds.
• Acid burns generally produce coagulative necrosis; depth may be limited by formation of coagulated proteins at base of burn.
• Bases typically generate liquefactive necrosis; depth often much deeper than acid burns.
• Organic compounds cause direct heat production and chemical reactions that disrupt skin.
• Inorganic compounds bind directly to the skin and create salts that damage skin integrity.
• Severity of the burn is related to a variety of factors incl the pH, concentration, volume, physical form, and contact time duration of the offending agent.

ASSESSMENT POINTS

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<tbody>
<tr>
<td>HEENT</td>
<td>Face and airway burns, eye injuries</td>
<td>Dysphonia, odynophagia, dysphagia Visual changes</td>
<td>Denuded or inflamed oral mucosa, conjunctivitis</td>
<td>Endoscopy; ophthalmologic evaluation</td>
</tr>
<tr>
<td>PULM</td>
<td>Chemical pneumonitis, ARDS</td>
<td>Dyspnea</td>
<td>Hypoxemia, possible rales or evidence of edema, auscultation may be normal</td>
<td>CXR, ABG</td>
</tr>
<tr>
<td>CV</td>
<td>Arrhythmias, hypovolemic shock</td>
<td>Palpitations, chest pain, dyspnea</td>
<td>Tachycardia or irregular rhythms</td>
<td>ECG, CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Esophagogastritis, perforated viscus</td>
<td>Odynophagia/dysphagia; hematemesis; epigastric pain</td>
<td>Abdominal tenderness/ guarding</td>
<td>Endoscopy; contrast CT scan; x-ray.</td>
</tr>
<tr>
<td>RENAL</td>
<td>Electrolyte disturbances, ARF, acute tubular necrosis</td>
<td>Deep or large surface area burns, associated crush injuries</td>
<td>Myoglobinuria, oliguria</td>
<td>Basic metabolic profile (BUN/Cr), urine myoglobin</td>
</tr>
<tr>
<td>MS</td>
<td>Rhabdomyolysis, compartment syndromes</td>
<td>Deep or large surface area burns, associated crush injuries</td>
<td>Evolving loss of motor/senory function</td>
<td>Serum myoglobin, compartment pressure monitoring</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preinduction/Induction/Maintenance
• Review Hx of the current injury, incl the amount of associated TBSA burn, and elapsed time since injury.
• Reliable vascular access is essential for adequate fluid resuscitation.
• Normalization of electrolytes, if possible.
• Preop medication should be avoided to alleviate anxiety and reduce pain, and to facilitate pt comfort during transfer and transport.

Monitoring
• Adequate intraop monitoring is essential due to the potential for extensive blood loss, frequent changes of position, and duration of surgery.
• Placement of surface monitors can be difficult due to location of burns.
• Try to place invasive lines away from injury, not through damaged skin.
• Consider arterial line placement for extensive debriement/procedures to allow beat-to-beat monitoring and frequent sampling of arterial blood.
• Presence of an arterial line should not preclude placement of an NIBP cuff (backup if arterial line fails during procedure). Negotiate with surgeon best location for NIBP cuff.

General Anesthesia
• Most surface chemical burns that proceed to OR are extensive enough to be treated as thermal injuries.
• Choices for induction and maintenance of general anesthesia depend on associated hemodynamic instability and airway status.
• Muscle relaxants: Avoid succinylcholine after acute phase (first 24 hr), resistance to nondepolarizers may evolve after acute phase.
• Narcotic tolerance may be higher in the chronic phase.
• Transfusions may be required in extensive debried procedures.
• Epinephrine-soaked pads may be applied by surgeon to decrease bleeding. This may result in tachycardia and a falsely stable BP that deteriorates after removal of pads.
• Thermoregulation is impaired. Warm OR as much as possible. Apply forced-air heating blankets. Administer warmed fluids and blood products.
• Extubation in the acute phase should be carefully considered if there is suspicion of airway edema or difficult reintubation.

Regional Anesthesia
• No contraindication in small or peripheral injuries.
• Preferable to place block through intact skin.
• Excision and grafting procedures may be accompanied by large fluid shifts and blood loss, in which case the loss of sympathetic tone resulting from a neuraxial block may be undesirable.

Postoperative Period
• Acute, extensive injury may require ICU care.
• Pain management can be challenging in chronic phase.

Anticipated Problems/Concerns
• Early, goal-directed resuscitation and correction of electrolyte abnormalities.
• Careful monitoring of airway and early airway intervention, if needed.
• Maintain normothermia.

ICD-9-CM Code 940-949; (Burns of internal organs from ingested chemicals: 947)

Etiology
• Surface burns: Most commonly work-related injury, accidental. Upper limbs more commonly injured as these substances are usually handled or carried. Injuries to the lower limbs and face can occur through splashing.
• Ingestions: Pediatric most commonly accidental; adult most frequently suicidal gesture.

Usual Treatment
• Remove contaminated clothes.
• Early decontamination with water or saline irrigation for surface exposures; elemental metals (K⁺, lithium) should not be exposed to moisture due to strong exothermic reaction.
• Prevent contaminated irrigation solution from running onto unaffected skin.
• After initial decontamination pt is treated as a typical burn pt.
• Ensure adequate fluid resuscitation for large BSA burns.
• Take measures to prevent complications (e.g., hypothermia, infection, rhabdomyolysis).
**Burn Injury, Electrical**

**Risk**
- 3–5% of all burns are electrical. Low-voltage burns (less than 1000 volts) commonly occur in children at the home.
- High-voltage burns (1000 volts or greater) are more common in adults and characteristically occur in outdoor environments near power sources and lines.
- Lightning electrical burns carry the highest rate of mortality and usually have energy greater than 30 million volts.

**Perioperative Risks**
- Pts with an acute burn or a Hx of burns may present an additional challenge to securing the airway. Fluid resuscitation in acutely burned pts may cause severe airway edema; pts with a history of burns, esp facial, may have limited mobility of mouth opening and neck extension.
- Difcult IV access is a common problem. Two large bore IVs are commonly needed for major burn surgery, however, depending on length of stay and surface area burn, central access and intra-arterial monitoring of BP may be necessary.
- Worry About
  - Arrhythmias and cardiac arrest.
  - Resp failure and edematous airway. Resp failure may occur due to tetany of resp muscles or cerebral injury.
  - Blunt injuries, fractures, and dislocations if pts were jolted from electrical shock or fell from high places.
  
  - Compartment syndrome: Delayed exploration and decompression may result in increased amputation rates along with increased organ failure and mortality.
  - Rhabdomyolysis and myoglobinuria from muscle injury leading to obstructive nephropathy and renal failure.

**Overview**
- Severity of electrical burn depends on current, route taken by the current, and the duration of contact with the electrical source.
- Entry wounds occur often in the hands with a leathery, charred appearance. Exit wounds are often explosive.
- Extent of injury may be misleading as visibly burned area is often small. Large amounts of destroyed tissue may be present under normal appearing skin leading to under resuscitation.
- Signs of electrical injury incl loss of consciousness, extremity mummification, loss of pulses in an extremity, myoglobinuria, elevated serum creatinine kinase, and cardiac arrest.
- The electrical current in most households is between 110–220V, which may produce a low-voltage burn and dysrhythmias. High-voltage burns often cause immediate cardiac arrest and/or resp paralysis.
- Direct lightning strikes are rarely survivable.

**ICD-9-CM Code:** 948 (Burns classified according to extent of body surface involved); 994.8 (Electrocution and nonfatal effects of electric current); 994.0 (Effects of lightning)

**Perioperative Implications**

**Preoperative Implications**

**Induction**
- Recommended for cases in which large blood loss anticipated.
- For pts with increased metabolic rate, NPO time should be kept to a safe minimum.
- Surgeon to anticipate extent of surgery and amount blood loss.
- Careful airway evaluation as an increased risk for airway edema and skin or muscle rigidity due to burns.
- Labs checked incl blood gases, K⁺, and type and cross.
- Large bore IV access may be needed in cases of complex debridement/grafting.

**General Anesthesia**
- Selected for most large skin graft procedures.
- Many pts already receiving ventilation support.
- Recommended for cases in which large blood loss anticipated.

**Monitoring**
- Standard ASA monitors. May be difficult to place monitors on burned surfaces. Use of staples and/or sutures to secure ECG leads or catheters may be required.
- Arterial monitoring may be necessary for large procedures or for pts receiving prolonged ventilatory support. Ultrasound can facilitate arterial cannulation.
- Maintaining normothermia is a major challenge. Ambient temp in OR must be raised, fluid warmers used, and sterile forced-air warmers may be needed.

**Induction**
- Many pts are catecholamine depleted. Induction agents such as ketamine may be useful in pts that are not already receiving ventilatory support.
- In burn pts succinylcholine is contraindicated after 24 hr from their injury. In addition, larger doses of non-depolarizing muscle relaxants may be required for adequate muscle relaxation.
- Larger doses of narcotics may also be needed since burn pts often develop tolerance to the narcotics. The analgies of ketamine make it a good choice for induction.

**Maintenance**
- Choice of inhaled anesthetic does not alter outcome.
- Judicious use of crystalloids, RBCs, and fresh frozen plasma to maintain normal blood volume and composition, and to avoid worsening edema.

Regional Anesthesia
• Can be used for analgesia after determining cause and extent of any neurologic sequelae and excl possibility of a compartment syndrome.
• May be used for anesthesia during minor procedures.

Postoperative Period
• Standard extubation criteria should be followed paying special attention to total fluids given and the possibility of airway edema.

• Increased analgesic demands. Consider physical ability to activate pt-controlled anagelsia (PCA) before instituting it.
• Careful monitoring during transport, esp in critically ill pts.

Anticipated Problems/Concerns
• Minimize the possibility of renal failure by maintaining adequate urine output and alkalizing the urine.

• Monitor edema during surgery as the ETT tape may become a facial tourniquet or the tube may migrate outside glottis.
• Pts that develop sepsis or multiorgan failure have worse outcome.
• Burn pts have an increased incidence of infection. Therefore, meticulous aseptic care during line placement, intubation, and all invasive procedures is essential.
Burn Injury, Flame

Effect
ABG, co-oximetry
Acute renal failure, ATN, Loss of consciousness, Electrolyte profile (BUN/Cr),

Perioperative Risks
• Major predictors of mortality: BSA >40%, age > 60, presence of inhalation injury.
• Predicted mortality is 0.3%, 3%, 33%, or 90%, depending on whether zero, one, two, or three of the above risk factors are present.
• Up to ½ of pts with inhalation injury will develop acute airway obstruction.
• Other incidental traumatic injuries may be present.

Worry About
• Airway protection and ventilation.
• Hypovolemia: Early goal-directed volume resuscitation is the single-most important therapeutic intervention.
• Hypothermia.

Overview
• Direct thermal energy produces direct cellular destruction and coagulative necrosis.
• Systemic microvascular integrity is lost in massive inflammatory response; proteins are lost into interstitial space.
• Significant shift of fluids, electrolytes, and proteins into the interstitium with rapid equilibrium of intravascular and interstitial compartments.
• Changes reflected by massive edema formation and loss of circulating plasma volume, hemocoencentration, decreased urine output, and depressed CV function.
• Cardiac output is reduced due to hypovolemia, decreased contractility, and increased afterload.
• Most of the edema occurs at the burn site and is maximal at 24 hr after the injury. Edema results in tissue hypoxia and increased tissue pressure with circumferential injuries.

ICD-9-CM Codes: 940–949 (948–Burn classified by percent body affected)

Etiology
• American Burn Association stratifies thermal injury etiologies as: Fire, hot liquids, contact with hot objects, electrical sources. Flame burns are the most lethal of all thermal injuries.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Face and airway burns</td>
<td>Dysphonia, dysphagia Reports of fumes or extraction from enclosed space</td>
<td>Singed face or nose hair, carbonaceous sputum, facial burns</td>
<td>Oral inspection, laryngoscopy, bronchoscopy</td>
</tr>
<tr>
<td>CV</td>
<td>Arrhythmias, hypovolemic shock, myocardial depression</td>
<td>Palpitations, dyspnea Loss of consciousness, depressed mental status</td>
<td>Tachycardia or irregular rhythms, hypotension</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumonitis, ARDS, restrictive disease from eschar, carboxyhemoglobinemia</td>
<td>Cough, dyspnea, stridor</td>
<td>Hypoxemia, circumferential chest eschar</td>
<td>ABG, co-oximetry, chest radiograph</td>
</tr>
<tr>
<td>RENAL</td>
<td>Acute renal failure, ATN, electrolyte disturbances</td>
<td>Large BSA burns, crush injuries</td>
<td>Myoglobinuria, oliguria</td>
<td>Electrolyte profile (BUN/Cr), urine myoglobin, urinalysis</td>
</tr>
<tr>
<td>CNS</td>
<td>Hypoxemia</td>
<td>Loss of consciousness, confusion</td>
<td>Focused neurologic exam</td>
<td>ABG, co-oximetry</td>
</tr>
<tr>
<td>MS</td>
<td>Tissue destruction, rhabdomyolysis, compartment syndrome</td>
<td>Large BSA burns, over-administration of fluids</td>
<td>Evolving loss of motor and/or sensory function</td>
<td>Serum myoglobin, compartment or bladder pressure monitoring</td>
</tr>
</tbody>
</table>


Preoperative Implications

Preoperative Preparation
• Thermoregulation is impaired. Warm OR as much as possible before pt arrives. Use forced-air warming blankets and fluid warmers intraop.
• Anesthesia services may be requested for bedside debridement and other procedures; adds the challenges of off-site care.
• Assess location and adequacy of venous access.
• Document presence of other invasive devices (arterial catheter, ET or tracheostomy tubes, feeding tubes, etc.) and ventilatory settings.

Monitoring
• Standard monitors may be difficult to apply to extensive burns.
• Arterial line is advisable for extensive grafting procedures that can be long and involve significant blood loss.
• Central venous access may be necessary if peripheral access sites are burned. Lines should preferentially be placed through intact skin.

Airway
• Intubate with largest feasible ETT to aid palatal toilet, minimize mucus plugging, and decrease work of breathing. Need for postop mechanical ventilation is common.

Preinduction/Induction
• Succinylcholine should be avoided after acute phase (first 24 hr after injury).
• Gastroparis and high residual gastric volumes are common after injury; use aspiration precautions.
• Induction agent doses should be adjusted in the context of hypovolemic shock.

Maintenance
• Requirements for neuromuscular blockers usually increased. Attributed to increased binding sites at extrajunctional receptors.
• Pts may need significantly increased levels of narcotics.
• Keep the OR room temp at ≥ 85°F to minimize heat loss and decrease metabolic rate.
• Communicate decreases in core body temp to surgeons; case may be shortened to prevent severe hypothermia.

Usual Treatment
• Most important points of initial phase are assessment of current (and prediction of subsequent) airway patency and documentation of the presence or absence of inhalation injury.
• Early intubation likely if face/inhalation injury or if BSA injured requires aggressive fluid resuscitation.
• Provide supplemental O₂ and monitor O₂ saturation in all burn pts with significant injury. Most pts with large burns will require prompt ET intubation and mechanical ventilatory support.
• Prompt establishment of large-bore IV access and rapid initiation of fluid resuscitation.
• Parkland or “Universal” formula is most commonly used (4 ml/kg/BSA% over 24 hr, first half given over first 8 hr).
• Insert urinary catheter early to monitor urine output as guide for volume status.
• Evaluate all extremities and chest wall for potential compartment requiring fasciectomy or escharotomy for urgent release.
• Multiple skin grafting procedures may be necessary during admission.
• Early debridement of eschar is performed to minimize infection; dead tissue readily supports bacterial growth.

Explantation
• Cautiously consider extubation in early stages of management. Emergent reintubation may be very difficult due to edema.

Anticipated Problems/Concerns
• Most common complications: Pneumonia, UTI, resp failure, cellulitis, and sepsis.
• Ventilator-associated pneumonia may develop in 70% of pts with inhalation injury.
• Pain management is usually challenging. Opioid doses often significantly exceed recommended standard dosing guidelines. Autograft donor sites are very painful; regional analgesia may be useful.
• Abdominal compartment syndrome (ACS) is a life-threatening complication caused by high-volume resuscitation. Extremity compartment syndromes can also result from extensive edema formation.
• Incidence of DVT in burn pts is increased (1% to 23%). Therefore, DVT chemoprophylaxis is routinely used.
Calcium Deficiency/Hypocalcemia

**Risk**
- Common in critically ill pts.
- Reported to range from 26% in hospitalized, non-ICU pts to 88% in critically ill ICU pts.

**Perioperative Risks**
- Neuromuscular instability leading to seizure, laryngospasm, bronchospasm, or resp arrest
- Impaired cardiac function: Heart failure, hypotension, and dysrhythmias

**Worry About**
- Symptomatic hypocalcemia

**Overview**
- Normal serum calcium content: 8.5–10.5 mg/dL.
- 40–50% bound to plasma proteins (albumin)
- 45–50% ionized (physiologically active)
- 10–15% non-ionized, bound to inorganic anions such as as phosphate, citrate, and sulfate
- Total calcium level can also be affected by albumin level, acid-base status.
- Ionized calcium level is the preferred measurement (normal: 4.75–5.3 mg/dL [1.19–1.33 mmol/L])
- Physiologic role of calcium:
  - Muscle contraction
  - Exocrine/endocrine/neurocrine hormone secretion
  - Cell growth
  - Transport and/or secretion of fluids

**ASSESSMENT POINTS**

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<tr>
<td>CV</td>
<td>Calcium involved in generation of cardiac pacemaker activity and generation of cardiac action potential</td>
<td>Hx of dysrhythmia SOB (or other symptoms of heart failure)</td>
<td>Prolonged QT Hypotension Pulm vascular congestion</td>
<td>EKG Continuous cardiac monitoring Chest x-ray</td>
</tr>
<tr>
<td>HEME</td>
<td>Citrate in stored blood products chelates calcium</td>
<td>Massive transfusion of citrated blood products (&gt;1.5 mL/kg/min)</td>
<td></td>
<td>Ionized calcium level</td>
</tr>
<tr>
<td>GASTRO</td>
<td>GI smooth muscle spasm</td>
<td>Abdominal cramping</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Resp smooth muscle contraction/tetany</td>
<td>SOB Laryngospasm Bronchospasm</td>
<td>Hypoxia Stridor Wheezing Resp arrest</td>
<td>Pulse oximetry</td>
</tr>
<tr>
<td>NEURO</td>
<td>Calcium is essential for all muscular movement. Calcium is involved in the muscular excitation/contraction coupling.</td>
<td>Muscle spasm Seizure Depression Psychosis Irritability Circumoral numbness Tingling in fingers/toes</td>
<td>Facial grimacing Seizure Papilledema (secondary to increased intracranial pressure) Irritability</td>
<td>Chvostek's sign (twitch of circumoral muscles with tapping of the facial nerve anterior to the ear) Trouseau's sign: Carpal spasm induced by inflation of a BP cuff to 20 mmHg above systolic BP for 3 min</td>
</tr>
<tr>
<td>INTEG</td>
<td></td>
<td>Dry scaly skin Brittle nails</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preinduction/Induction/Maintenance**
- Correct symptomatic hypocalcemia preop.
- Goal of treatment is to eliminate symptoms, not necessarily return calcium levels to normal range.

**Monitoring**
- Serial ionized calcium measurements.
- Continuous EKG monitoring.

**General Anesthesia**
- Negative inotropic effects of anesthetic medications may become more pronounced.

**Regional Anesthesia**
- Hypocalcemia results in increased neuronal membrane irritability/tetany.
- Parasthesia is a common finding.
- Thorough Hx and physical exam is essential.

**Postoperative Period**
- Acute hypocalcemia may develop after thyroidectomy/parathyroidectomy.

**Anticipated Problems/Concerns**
- Risk of hypocalcemia with massive transfusion of citrated blood products (>1.5 mL/kg/min).
- Alkalosis increases Ca²⁺ binding to proteins, therefore decreasing ionized calcium.

**Etiology**
- Acute
  - Severe, acute hyperphosphatemia (tumor lysis syndrome, acute renal failure, rhabdomyolysis)
  - Acute critical illness (sepsis, burns, pancreatitis, fat embolism)
  - Large-volume transfusion with citrated blood (chelation) or albumin
  - Medications: Protamine, heparin, glucagon
  - Acute hypoparathyroidism after thyroidectomy/parathyroidectomy
  - Alkalosis (metabolic/respiratory): increased calcium binding to proteins
- Chronic
  - Hereditary/acquired hypoparathyroidism
  - Hypomagnesemia
  - Chronic renal failure
  - Hyperphosphatemia
  - Vitamin D deficiency

**Usual Treatment**
- No need to treat low total calcium if ionized calcium level is normal.
- Asymptomatic hypocalcemia rarely requires treatment.
- Symptomatic hypocalcemia requires emergent treatment.
- 1–5 mL 10% calcium chloride (27.2 mg Ca²⁺/mL) or 10–20 mL 10% calcium gluconate (9.5 mg Ca²⁺/mL) over 10 min.
- Follow with 0.3–2 mg/kg/hr elemental calcium if continuous replacement is needed.
- Administer slowly as venous irritation can occur. Central venous administration is preferred as calcium chloride can cause tissue necrosis if extravasated from a peripheral vein.
- Must rule out hypomagnesemia/hyperphosphatemia. Treat as needed.
Cancer, Bladder

Risk
- Primary risk factor is smoking (smokers are more than twice as likely to get bladder cancer as nonsmokers)
- Incidence: Males 37 per 100,000; females 9 per 100,000
- No associate increased risk with alcohol or caffeine consumption
- Median age of diagnosis: 73 y
- Caucasian > African-Americans
- Quitting smoking decreases risk over time (baseline in 5–8 y)
- Incidence on a decline since 1999

Perioperative Risks
- Risks vary based on surgical procedure and co-existing disease
- Chemotherapy: Pulm fibrosis, renal and cardiac dysfunction
- Fatty infiltration of liver in those with poor nutritional status
- Protein–calorie malnutrition due to the cancer, metabolism and anorexia: anemia, hypoalbuminemia; dehydration

Overview
- Transitional cell cancer generally systemic disease at time of Dx—60% will die of metastatic complications

ASSESSMENT POINTS

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<tbody>
<tr>
<td>CV</td>
<td>Doxorubicin (Adriamycin) toxicity; cardiomyopathy</td>
<td>&gt;550 mg/m², prior or concurrent mediastinal radiation therapy</td>
<td>Signs of CHF</td>
<td>Endomyocardial biopsy, serial ECHO; radionuclide angiography, DLCO, ECG</td>
</tr>
<tr>
<td></td>
<td>5-Fluorouracil Myocardial ischemia (rare)</td>
<td>Angina</td>
<td>ECG</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cyclophosphamide Pericarditis with effusion</td>
<td>CHF</td>
<td>Signs of CHF</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Smoking-related injury</td>
<td>Cough, sputum, infections</td>
<td>Wheezes, rhonchi, barrel chest</td>
<td>CXR, PFT</td>
</tr>
<tr>
<td></td>
<td>Bleomycin or cyclophosphamide toxicity: pulm fibrosis</td>
<td>&gt;500 mg (bleo), cough, dyspnea</td>
<td>Rales, fever</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>Methotrexate: Inflammation</td>
<td>Pulm edema, effusions, infiltrates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Cisplatin: ATN</td>
<td>Occurs 3–5 d after course</td>
<td>BUN, Cr, proteinuria, hyperuricemia</td>
<td></td>
</tr>
<tr>
<td>HEPATIC</td>
<td>Methotrexate: Renal failure</td>
<td></td>
<td>Hematuria, proteinuria</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Methotrexate: Encephalopathy</td>
<td>Confusion, somnolence, ataxia, tremors, focal signs</td>
<td>SGPT</td>
<td></td>
</tr>
</tbody>
</table>


Preoperative Implications

Preoperative Preparation
- Consider rehydration after bowel preparation
- 2 large bore IVs or one peripheral IV plus a central line

Monitoring
- Considering arterial catheterization
- Renal perfusion difficult to judge after division of ureters: Consider CVP or PAC or TEE
- Anesthesia technique
- Consider combined general-epidural anesthesia to treat postop incisional pain, reduce blood loss and fluid requirements for cystectomy as well as less risk of postop ileus
- Epidural placement ideally T9, T11

Induction
- Watch for hypotension due to volume depletion from prep and/or decreased systolic function from cardiotoxic chemotherapeutic agents

Maintenance
- Avoid high concentrations of O₂ in pulm fibrosis
- Consider avoiding N₂O (bowl surgery)
- Maximize efforts to prevent hypothermia

Postoperative Considerations
- Consider overnight ventilation if long procedure, significant blood loss/fluid resuscitation. Epidural catheter can optimize pulm toilet and recovery.
- Fluids shifts occur during first 48hr
- EBL: TURBT about 200 mL; cystectomy between 500–1000 mL
- Pain score: 7–9 (cystectomy)
### Cancer, Breast

**Risk**
- 100 times more common in women than men
- 1 in 8 women develop breast cancer. Besides skin cancer, most common cancer in USA for women
- The chance of getting breast cancer goes up as women get older. About 2 out of 3 women with invasive breast cancer are 55 or older when the cancer is found
- Racial predilection: Caucasians > African-Americans > Asians, Hispanics, and Native Americans
- African-Americans are more likely to die of breast cancer because their cancers tend to be more aggressive.
- 10% of breast cancer cases are directly due to inherited mutations of the BRCA1 and BRCA2 gene
- Increased risk: Family Hx among close blood relatives, personal Hx increases the risk of developing a new cancer in the other breast.
- 70% of breast cancers are diagnosed in women with no family Hx
- Associated increased risk: Obesity, high-fat diets, aging, high alcohol consumption, and estrogen exposure

**Perioperative Risks**
- Mortality very rare
- Lymphedema of arm following axillary node dissection
- Ipsilateral brachial plexus injury from extensive abduction of the arm, or iatrogenic
- Injury to long thoracic and/or thoracodorsal n. during surgical dissection of axilla
- Rare incidence of unrecognized pneumothorax
- Breast surgery is associated with postop N/V, incidence as high as 60%

**Worry About**
- Systemic or regional impact of metastasis to lung, brain, or bones
- High incidence of postop N/V
- NMB and identification of major n.
- Access to an upper extremity may be restricted or limited
- Potential adverse effects of chemotherapeutic drugs and chest radiation therapy

**Overview**
- Abnormal growth of adenomatous tissue that results in systemic symptoms and metastasizes to liver, bone, lung, and brain
- Early detection of breast cancer increases time of survival.
- There is controversy over the role of mammography in detection of breast cancer.
- Physical exam and mammography are complementary.
- Needle biopsies provide histological Dx.
- Presurgical needle localization may be necessary for nonpalpable lesions.
- Most breast biopsies yield benign diagnosis.

**ICD-9-CM Code:** 174

### ASSESSMENT POINTS

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<tbody>
<tr>
<td>CHEST</td>
<td>Lung lesions</td>
<td>Nipple discharge, chest pain or discomfort</td>
<td>Breast asymmetry, nipple discharge, erythema, crusting, or erosion</td>
<td>Physical exam, Mammography, Fine-needle aspiration biopsy, CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Liver metastasis</td>
<td>Fatigue, abdominal pain</td>
<td>Enlarged or nodular liver</td>
<td>Liver US or CT scan</td>
</tr>
<tr>
<td>HEME</td>
<td>Bone metastasis</td>
<td>Lethargy, SOB</td>
<td>Anemia, pancytopenia</td>
<td>CBC</td>
</tr>
<tr>
<td>CNS</td>
<td>Brain metastasis</td>
<td>Change in mental status, seizures</td>
<td>Neurologic exam</td>
<td>Head CT</td>
</tr>
<tr>
<td>MS</td>
<td>Bone metastasis Pathologic fractures</td>
<td>Severe pain, immobility, arm swelling</td>
<td>Deformities, pain on palpation, axillary adenopathy</td>
<td>Bone scan, X-rays, Physical exam</td>
</tr>
</tbody>
</table>


### Perioperative Implications

**Preoperative Preparation**
- Optimal preop preparation, in response to associated anxiety, which can be achieved through both pharmacologic and nonpharmacologic means
- Monitoring
  - Routine with attention to placement of ECG leads
  - IV site and BP cuff on contralateral arm
- Airway
  - Table arrangements may warrant a secure airway
  - Nasal O₂ or LMA may be appropriate
- Induction
  - Thoracic epidurals, intercostal nerve blocks, and local infiltration have successfully been administered as primary anesthetics and adjuvants to GA

**Maintenance**
- Consideration for the high incidence of postop N/V
- Incision over operative breast that can also incl axilla
- Dissection can incl breast areolar tissue, muscle down to chest wall, and extension into axilla
- Identification of thoracodorsal and long thoracic n. often requires stimulation that contraindicates presence of NM blocking agents
- Surgical field will be in view and allow for monitoring of active blood loss
- Surgical team leaning on chest can affect ventilatory performance

**Postoperative Considerations**
- Pain score: 2–6
- Pain adequately managed with Toradol, narcotic PCA, or regional block
- Communicate with PACU that no venous sticks or BP measurements should be performed on arm of operative side when axillary lymph node dissection is involved

**Anticipated Problems/Concerns**
- Anxiety associated with the fear of breast cancer and altered body image can be quite significant
Cancer, Bronchial

Risk
- Incidence in USA: 160,000 cases/y
- Race: No difference among ethnic groups when >30 cigarettes/d smoked, but higher in people of Asian ancestry, African Americans, and Native Hawaiians when < 30 cigarettes/d smoked
- Tobacco cigarette consumption is the major risk factor; males slightly > females

Perioperative Risks
- Resp and cardiac complications: Atelectasis, pneumonia, pulm edema, resp insufficiency, right ventricular dysfunction, arrhythmias, and ischemia

Worry About
- Endobronchial obstruction, obstructive pneumonitis, consolidation, atelectasis, localized air trapping, and resp insufficiency

Perioperative Implications

Preoperative Preparation
- Adequate hydration, correct electrolyte abnormalities, bronchodilators, antibiotics, steroid coverage for adrenal insufficiency, and PFTs
- Incentive spirometry instruction

Monitoring
- Arterial line for lung resection and OLV

Airway
- Determine need for left- or right-sided double lumen ETT

Preinduction/Induction
- Bronchodilators
- Antibiotics
- Arrhythmia drugs if indicated

Perioperative Period

Maintenance
- No one agent or technique is superior
- Volatile agents decrease bronchomotor tone and HPV minimally, but permit high FIO₂
- CPAP and PEEP as required, esp during OLV
- Consider thoracic epidural

Extubation
- Change to single-lumen ETT if pt will remain intubated.

Adjuvants
- Consider bronchodilators and anti-arrhythmia medications

Postoperative Period
- Consider thoracic epidural, intrapleural catheters, paravertebral nerve blocks or cryoanalgesia for pain management.

Anticipated Problems/Concerns
- Potentially life-threatening problems: Bronchial disruptions, cardiac herniation, tension pneumothorax, cardiac dysrhythmias, and resp insufficiency
- Adequate analgesia is esp beneficial for pts with COPD

Etiology
- 85% related to cigarette smoking; other risk factors are passive smoking, ionizing radiation, asbestos, heavy metal exposure (arsenic, chrome), halo ethers, polycyclic aromatic hydrocarbons, vinyl chloride, formaldehyde, and genetic factors

Usual Treatment
- Surgical resection for localized non–small cell carcinoma
- Chemotherapy, radiation therapy for small-cell carcinoma
- Unresectable endobronchial or endotracheal tumors treated with external beam radiation and/or bronchoscopic laser resection

Overview

• Histology: Squamous, adeno-, large cell, and small-cell carcinomas
• Leading cause of cancer deaths in USA for both men and women; 20% 1-y and 8% 5-y survival
• Severe COPD may limit lung resection and affects periop management
• Paraneoplastic syndromes occasionally affect management

ICD-9-CM Code 162.9

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<td>HEENT</td>
<td>Tracheal fixation or obstruction</td>
<td>Dyspnea, cough, stridor</td>
<td>Rhonchi</td>
<td>CXR, CT, PFT, FOB</td>
</tr>
<tr>
<td>CV</td>
<td>Pericardial effusion</td>
<td>Dyspnea, cough</td>
<td>Dilated neck veins</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>SVC syndrome</td>
<td>Dyspnea, cough</td>
<td>Facial edema</td>
<td>CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Lung mass, consolidation, atelectasis, pleural effusion</td>
<td>Dyspnea, cough, fever</td>
<td>Rhonchi, fever, percussion dullness</td>
<td>CXR, ABG</td>
</tr>
<tr>
<td>GI</td>
<td>Rarely pancreatitis from ↑ Ca++</td>
<td>Anorexia, constipation</td>
<td></td>
<td>Serum, Ca++</td>
</tr>
<tr>
<td>CNS</td>
<td>Brain metastasis, paraneoplastic syndrome (optic neuritis, neuropathy, cerebellar degeneration, LEMS)</td>
<td>Headache, visual changes, unsteady gait, sensory or motor symptoms</td>
<td>Fundoscopic or neurologic findings</td>
<td>Head CT scan</td>
</tr>
<tr>
<td>MS</td>
<td>Bone metastasis</td>
<td>Bone pain</td>
<td>Bone tenderness</td>
<td>↑ Alk phosphatase, PET/CT, ↑ CK, EMG</td>
</tr>
</tbody>
</table>

Cancer, Esophageal

Risk
• Incidence in USA: 7.7 in 100,000 white men, 2.0 in 100,000 white women, 12.7 in 100,000 in black men, 4.2 in 100,000 black women
• Incidence of adenocarcinoma has increased in white men, while incidence of squamous cell carcinoma is highest in black men
• Overall mortality rate: 8.8%

Perioperative Risks
• Reflux as a risk for aspiration
• Malnutrition with dehydration due to swallowing dysfunction
• Periop arrhythmias occur 20–60% of esophagectomies
• Anastomotic leak most frequent surgical complication

Worry About
• Pulm compromise due to lung injury from preop chemotherapy/radiation therapy, chronic aspiration, extensive tobacco Hx, and inflammatory response to mechanical ventilation
• Hydration status
• Airway protection at time of anesthesia induction and postop
• Alcohol withdrawal syndromes
• Arrhythmias

Overview
• Primarily either squamous cell from esophageal squamous epithelium or adenocarcinomas of gastric origin
• Usually 55–65 y, with a long-standing Hx of tobacco and alcohol intake
• Dysphagia and wt loss are initial symptoms, often present for 3–4 mo
• Characterized by extensive local growth and lymphatic involvement before becoming widely disseminated

ICD-9-CM Code: 150

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>Pe</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Alcohol abuse–induced cardiomyopathy and arrhythmias</td>
<td>DOE</td>
<td>Exercise tolerance</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Tobacco abuse, Chronic aspiration, Radiation/chemotherapy</td>
<td>Pneumonia; RV Fhm</td>
<td>Cough, dyspnea</td>
<td>RV heave</td>
</tr>
<tr>
<td>GI</td>
<td>Obstruction, Reflux, Malnutrition</td>
<td>Difficulty swallowing, unable to sleep flat, wt loss</td>
<td>Debilitated</td>
<td>ECHO, stress test</td>
</tr>
<tr>
<td>CNS</td>
<td>Alcohol abuse, Delirium tremens</td>
<td>Last alcohol ingestion and amount</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Weakness</td>
<td>Poor nutrition</td>
<td>Muscle wasting</td>
<td>Serum albumin</td>
</tr>
<tr>
<td>RENAL</td>
<td>Dehydration</td>
<td>Limited intake</td>
<td>Lytes, Cr, BUN</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
• Premedication not to obtund a pt at risk for aspiration
• Antisialagogue (atropine 0.4 mg or glycopyrrolate 0.2 mg)
• Premedication with H, blocker for acid aspiration prophylaxis plus metoclopramide to promote gastric emptying
• Steroids given if recently used
• Consider β-blockade for prophylaxis
• Placement of thoracic epidural for postop pain control

Monitoring
• Central venous or PA catheter placement for volume assessment and replacement, and for vol-ume loading prior to surgical compression of the mediastinal structures optional
• Arterial line for BP monitoring and ABGs

Airway
• Rapid-sequence induction or awake fiberoptic intubation
• The surgical need for one-lung ventilation if thoracoabdominal approach requires a double-lumen ETT, a bronchial blocker, or a Univent tube and proper positioning

Induction
• Hypovolemia often results in BP fluctuation
• Aspiration risk during intubation

Maintenance
• No one agent or technique shown superior
• Volume requirements due to mediastinal compression, blood loss, and initial dehydration status
• Oxygenation concerns during one-lung ventilation, the use of 100% O2 and chemotherapy Hx (bleomycin, mitomycin), prior pulm compromise due to tobacco history, volutrauma during mechanical ventilation
• Lung-protection advocated during mechanical ventilation, lower tidal volumes 5–6 mL/kg recommended with/without peep, using either volume or pressure modes of ventilation to maintain adequate oxygenation with peak inspiratory pressures <30–35 cm of water
• Hypothermia is concern in long procedures
• Placement of NG tube with surgical guidance

Extubation
• Continuing risk of aspiration
• Aim for early extubation in the OR or within a few hours of surgery. Less need for postop sedation leading to less fluid requirements. Requires presence of functioning epidural.
• Caution with obese and sleep apnea pts
•Pts with double-lumen ETT in place should be reintubated or bronchial blockers pulled back (Univent) or removed if postop ventilation is required.

• Reintubation difficult because of edema and fluid shifts. With solid paralysis and pharyngeal suctioning, and a tube exchanger (Cook airway exchange catheter) is recommended. Double-lumen tube is withdrawn over the tube exchange and a single lumen tube is threaded over the exchanger with a laryngoscope used to help with soft tissue that may impede placement.

Adjutants
• Pts who have received chemotherapy (mitomycin or bleomycin) might be administered an O2 concentration of 28% or as low as possible (see Bleomycin in Drugs section)

Postoperative Period
• Epidural analgesia is beneficial
• Increased risk for supraventricular tachycardia and atrial fibrillation. Rate control recommended by the AHA initially with IV diltiazem and/or β-blockers if BP can tolerate

Anticipated Problems/Concerns
• Airway management: Aspiration risk, reintubation problems, extubation criteria
• Volume status in a dehydrated pt undergoing a lengthy surgical procedure with mediastinal compression and a thoracic epidural
• Arrhythmias in the postop period, use of prophylatic β-blockade
Cancer, Lung Parenchyma

Roger A. Moore

Risk
- Lung cancer is primary cause of cancer death
- Asbestos exposure increases risk 5-fold
- Smoking increases risk 15-fold
- Radon exposure increases risk 2-fold

Perioperative Risks
- Associated CAD
- Pulm insufficiency following lung tissue resection

Worry About
- Optimization of preop pulmonary status
- Issues secondary to metastatic spread, such as superior vena caval syndrome
- Myasthenic syndrome (Eaton-Lambert) with oat cell carcinoma
- Massive hemoptysis with cancer invasion of bronchial arteries
- Active pneumonia in pulm parenchyma distal to obstructed bronchioles

Overview
- Four primary types of lung cancers: Squamous cell or bronchogenic; adenocarcinoma (most common); large cell carcinoma; small cell carcinoma
- 70% with COPD need extra postop pulm care
- Pts often nutritionally depleted
- Many have alcohol abuse history
- Preop pulm state may limit option of lobectomy
- Hormonal imbalances common due to hormone secreting tumors
- 3% of pts are cushingoid
- 70% of bronchogenic carcinomas have increased ACTH or pro-ACTH
- Up to 60% with lung cancer have inappropriate ADH
- Myasthenic syndrome occurs owing to decreased release of nerve-ending acetylcholine leading to increased sensitivity to all muscle relaxants

ICD-9-CM Code: 162

Perioperative Implications

Preoperative Preparation
- Resp optimization with bronchodilatation, antibiotics, pulm hygiene, and smoking cessation
- Correction of lyte imbalances

Monitoring and operative care
- Routine monitors
- Intra-arterial line and possible pulm catheter, but if a PA catheter is used, be alert to it being caught in the surgical pulm incision
- Neuromuscular blockade monitor
- Thoracic epidural

Airway
- Double-lumen tube or bronchial blocker needed—usually left-sided
- Fiberoptic bronchoscope should be available for positioning of endobronchial tube

Induction
- Anesthetic choice dependent on associated medical problems
- Light or no premedication to decrease CO₂ retention

- When right-sided double-lumen tube used, ensure right upper lobe ventilation (easiest with fiberoptic bronchoscope)

Maintenance
- Nerve damage with lateral position
- Use axillary roll
- Brachial plexus injury with arm hyperextension
- Pad all pressure points
- Substantiate pulse oximetric and capnographic readings with ARBs
- If O₂ saturation falls during one-lung ventilation, PEEP on dependent lung may help. If not, CPAP on nondependent lung may help.
- Intraop fluid restriction, incl use of blood and blood products, can significantly decrease postop resp failure

Extravasation
- If postop ventilation required and double lumen tube has been used, it needs to be switched to single-lumen tube
- Extravasation should be determined by adequacy of resp variables

Adjuvants
- Bronchodilators for intraop use, inotropes for myocardial depression, antiarrhythmics for post lobectomy-pneumonectomy arrhythmias (some advocate prophylactic digoxin—but conflicting reported results)

Postoperative Period
- If pneumonectomy performed, there is a significant risk for postop ARDS
- Adequate pain management usual for recovery of pulm function
- PCA or use of intercostal blocks can be effective
- Thoracic epidural most efficacious
- Be watchful for DTs, inappropriate ADH, and decreased neuromuscular strength

Anticipated Problems/Concerns
- Intensive pulm toilet postop
- Careful suctioning of bronchial stump because of possibility of rupture
- Bronchopleural fistula or tension pneumothorax should be anticipated

Cancer, Prostate

Risk
- The second leading cause of cancer death in men after lung cancer
- Estimated 192,280 new cases and 27,360 deaths from prostate cancer in the USA in 2009
- Screening with prostate-specific antigen (PSA) and digital rectal examination led to an increase in earlier-stage detection
- The incidence increases with age; more than 75% of cancers diagnosed are in men older than age 65
- Men who undergo prostatectomy have a very high chance of surviving at least 15 years

Perioperative Risks
- Perioperative mortality for open radical prostatectomy (ORP) is approximately 0.3%.
- Risks of ORP incl excessive blood loss, rectal laceration, ureteral injury, wound infection, DVT, puls embolus, anastomotic leak, MI, and later lymphocele, incontinence, impotence, and anastomotic stricture.
- Laparoscopic radical prostatectomy (LRP)—less morbidity, less blood loss
- Robotic-assisted laparoscopic prostatectomy (RALP)—less blood loss and rare blood transfusion, a trend for symptomatic ileus to be more prevalent

Worry About
- Increased prevalence of age-related, multiple concomitant diseases and a decline in basic organ function in these elderly men

Overview
- Early Dx by the triad of elevated serum PSA, an abnormal digital rectal exam, and transrectal ultrasound (TRUS)-guided prostate biopsies
- Localized disease—rarely causes symptoms
- Locally advanced or metastatic disease—obstructive voiding and irritative voiding symptoms, bone pain
- TNM system is the most widely used clinical staging system. Briefly, in TNM system, T1 and T2 tumors are confined to the gland, whereas T3 and T4 tumors have local extension. The most prominent histologic grading system is the Gleason Scoring System.
- Heterogeneous tumors (usually acinar adenocarcinomas) composed of hormone-sensitive and hormone-insensitive cells

ICD-9-CM Code: 185

Etiology
- Genetic predisposition, hormonal influences, dietary and environmental carcinogenic influences, infectious agents

Usual Treatment
- Quality-of-life issues and co-morbid condition help to guide treatment choices
- Watchful waiting—esp if other diseases present or age >70 y with moderately differentiated, low-volume cancer, and a life expectancy of fewer than 10 y
- Open radical prostatectomy (ORP)—(retropubically or perineally) in selected pts with clinically confined prostate cancer, usually for those <70 y.
- Laparoscopic radical prostatectomy (LRP)—offers decreased blood loss and transfusion rate, and shorter length of hospital stay
- Robotic-assisted laparoscopic prostatectomy (RALP)—has the following advantages: Robotics provides three-dimensional visualization, magnification, tremor filtration, expanded degrees of freedom and wristed instrumentation, less blood loss and postop pain, and shorter hospital stay. Demand for this procedure is increasing worldwide—more than halfe of all prostatectomy surgeries performed in the USA are robotic
- Radiation therapy and brachytherapy—external beam radiotherapy or interstitial radioactive seed implantation, proton therapy
- Hormonal therapy—for locally advanced or metastatic cancer: Estrogens, bilateral orchiectomy, LH-RH agonists, anti-androgens, and combined androgen blockade
- Others—Cytotoxic chemotherapy; cryosurgery; high-intensity focused ultrasound; radiofrequency interstitial tumor ablation

ASSESSMENT POINTS

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<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Age-related changes</td>
<td>COPD, smoking Hx</td>
<td>HTN, CAD, exercise tolerance</td>
<td>Anemia in extensive metastases</td>
</tr>
<tr>
<td>CV</td>
<td>Age-related changes</td>
<td>HTN, CAD, exercise tolerance</td>
<td>Rock-hard nodule on digital rectal exam</td>
<td>Renal function test</td>
</tr>
<tr>
<td>HEME</td>
<td>In advanced stages: anemia, azotemia, uremia</td>
<td>Pathologic finding in prostate tissue; renal function impairment</td>
<td>Blood work, T &amp; Screen or T &amp; C</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Early—asymptomatic; late stages—hesitancy, intermittency, urgency, frequency, retention, infection, impotence, hematospermia</td>
<td>Hx of CVA, Alzheimer's</td>
<td>Blood work, T &amp; Screen or T &amp; C</td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Increased incidence of DM; malnutrition</td>
<td>Malaise</td>
<td>Blood work, T &amp; Screen or T &amp; C</td>
<td></td>
</tr>
<tr>
<td>CNS and eye</td>
<td>Age related changes, glaucoma, metastasis</td>
<td>Hx of CVA, Alzheimer's</td>
<td>Blood work, T &amp; Screen or T &amp; C</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Arthritis</td>
<td>Bone pain (commonly in lumbarosacral area)</td>
<td>Documenting neurologic changes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Metastases to spine</td>
<td>Pathologic fracture (uncommon)</td>
<td>Radionuclide bone scans if bone metastases present</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Assess CV and pulsus status
- Leg pumps or compression stockings or low-dose coumadin to reduce DVT
- Appropriate bowel preparation

Anesthetic Technique
- ORP: General, may use epidural, or spinal for some cases
- RALP and LRP: General
- Consider pt's concomitant diseases, position on the operating table, intraop blood loss, and possible thromboembolic events in choosing anesthetic technique

Monitoring
- Consider arterial line depending on surgery
- Consider CVP and/or PA catheter for expected excessive blood loss and/or severe co-existing disease

Anticancer therapeautcs
- Hormonal therapy causes abnormal liver metabolism

Surgical Stages
- In RALP: Access to the pt is limited as soon as the robot is docked, place invasive monitors preop in high-risk pts; cushioned stirrups are used in modified lithotomy position; pt is well strapped to operating table to prevent pt sliding off table; arms and legs are properly positioned and adequately padded; decompression of stomach; fluid restriction minimizes facial edema and excessive urine output, complete muscle relaxation is essential; pneumoperitoneum with CO2 insufflation is associated with adverse hemodynamic and resp effects, esp in pt with limited cardiac reserve or impaired resp function; occult blood loss may occur
- In ORP—higher intraop blood loss in retro pubic group; higher risk for rectal injury in the perineal group

Anticipated Problems/Concerns
- Air embolism from prostatic fossa during surgery in Trendelenburg position
- Intraop hemorrhage—esp with ORP retropubic approach
- In open radical prostatectomy—injury to obturator nerve, ureter, or rectum; immediate postop DVT and pul embolism; symptomatic pelvic lymphocele; wound or UTIs; periop main CV complications—MI and postop arrhythmias; long-term surgical complications—incontinence and impotence
- In LRP and RALP—possible complications resulted from steep Trendelenburg position (25–45 degrees head down): post-extration resp distress secondary to laryngeal edema, brachial plexus injury, serious ocular consequences secondary to increased intraocular pressure
- For nonprostate surgery—worry about effects of chemotherapeutic agents, hormones, or radiation on hematologic, liver, renal, and vascular systems

Wen-Shin Liu
Candidiasis

**Risk**
- Ps with suppressed immune system from diseases like AIDS; chemotherapy drugs; extended steroid therapy
- Current and recent broad spectrum antibiotic therapy
- Diabetes, leukemia, and neutropenia
- IV hyperalimentation and prolonged ICU stay
- Breaches of protective epithelial barrier: Surgical trauma, burn injury, long-term indwelling IV or bladder catheters
- Even in healthy individuals, candida can be cultured from the oral cavity in a third to more than half; this increases with chronic illness and duration of hospitalization.
- As systemic bacterial infections have declined with aggressive antibiotic use, systemic fungal infections have correspondingly increased.
- Candida is fourth most common organism recovered from blood cultures.

**Perioperative Risk**
- Candidemia with septic shock is infrequent in non immunocompromised pts but has a very high mortality rate, ~30% higher than bacteremic septic shock, and a high likelihood of MOF along with delayed recovery from this organ failure
- Ps more likely to have compromised renal function at baseline

**Worry About**
- Disseminated candidemia and associated organ dysfunction
- Candidemic septic shock
- Side effects of -azole, nystatin, or amphotericin-B therapy

**Overview**
- Candidemia in 30 cases per 100,000 admissions (in USA), associated with ~14.5% increase in mortality, 10-day increase in hospital stay and ~$40,000 increase of charges

**Perioperative Implications**

**Preoperative Preparation**
- Choose drugs based on septic signs and symptoms
- Worry about hypotension and hypoxemia at induction

**Maintenance**
- Choose drugs based on hemodynamic status
- Choose ventilatory modes based on presence of ARDS

**Exubation**
- May have to be delayed if ARDS or septic state require hemodynamic support

**Pre induction /Induction**
- Choose drugs based on septic signs and symptoms
- Worry about hypotension and hypoxemia at induction

**Adjuvants**
- In the presence of compromised renal or hepatic function modify anesthetic drugs accordingly

**Anticipated Problems/Concerns**
- Candidemia presents with a diverse clinical picture, from low-grade fever to fulminating septic shock. There is higher periop mortality in this group of pts.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Thrush</td>
<td>Dysphagia</td>
<td>White oral plaques</td>
<td>Bleed on scraping</td>
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<tr>
<td></td>
<td>Endophthalmitis</td>
<td>Visual changes</td>
<td>Ophthalmic lesions</td>
<td>Fundoscopic and field of vision</td>
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<tr>
<td>CVS</td>
<td>Endocarditis</td>
<td>SOB</td>
<td>Cardiac murmurs</td>
<td>Auscultation</td>
</tr>
<tr>
<td></td>
<td>Septic shock</td>
<td>Refractory hypotension</td>
<td>Fever</td>
<td>CVP, CO, PCWP</td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumonia</td>
<td>SOB, cough, tachypnea, dec exercise tolerance</td>
<td>Rapid shallow breathing, hypoxemia, consolidation</td>
<td>PFT, ABG, CXR</td>
</tr>
<tr>
<td>CNS</td>
<td>Meningitis</td>
<td>Altered mental status, signs of increased ICP, nausea, vomiting, headache, seizures, loss of appetite</td>
<td>Mental status exam, neck stiffness, photophobia, confusion</td>
<td>CT, MRI, blood cultures, CSF cultures</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal abscess</td>
<td>Dysuria, polyuria, low back pain, hematuria</td>
<td>Costovertebral tenderness on affected side</td>
<td>Urine culture, cystoscopy, CT</td>
</tr>
<tr>
<td>SKELETAL</td>
<td>Fungal osteomyelitis</td>
<td>Tenderness over bone, skin breakdown over infected bone</td>
<td>Moderate to severe bone pain, limited range of motion</td>
<td>X-ray, culture and sensitivity, bone scan</td>
</tr>
<tr>
<td>GI</td>
<td>Inflammation through GI tract, intra-abdominal abscess</td>
<td>Dysphagia, abdominal pain, diarrhea</td>
<td>Abdominal tenderness, signs of peritoneal irritation, hepatomegaly, splenomegaly</td>
<td>CT or MRI, endoscopy, abdominal ultrasound</td>
</tr>
</tbody>
</table>

Carbon Monoxide (CO) Poisoning

Risk
- Most frequent toxic gas in smoke (COHb can reach 10% in smokers)
- Major cause of death
- CO produced by all internal combustion engines, incomplete oxidative combustion (e.g., house fires, charcoal and gas grills, malfunctioning butane/propane stoves), and endogenous sources (e.g., by liver from exogenous exposure to paint stripper)
- No odor, taste, color, or irritation
- Toxicity potentiated by low inspired O₂ concentration (e.g., smoke inhalation)
- To minimize CO in circle circuit carbon dioxide absorbers: Use fresh soda lime, use sevoflurane, and minimize drying (lower FGF and stop FGF during non-use)
- During GA, use semiclosed circuits, esp when machine has not been used for 2–3 d (e.g. Monday morning)

Perioperative Risks
- Main target organs: Heart and brain
- Heart: Can resemble ischemia; potentiated by CAD
- Brain: Acute loss of consciousness; after initial improvement, up to 30% risk of secondary syndrome: chronic psychiatric dysfunction and cerebral and cerebellar syndromes

Overview
- CO, a colorless, nonirritating, and odorless gas, is a natural byproduct of combustion
- CO binds avidly to Hgb (≥200 × O₂) to form carbonyhemoglobin (COHb), which carries no O₂, and causes left shift in oxyhemoglobin dissociation curve (decreases O₂ off-loading to tissues)
- CO binds to intracellular hemoproteins such as myoglobin and cytochrome a₉ (esp cardiac) to inhibit O₂ uptake and metabolism
- “Classic” cherry-red complexion rarely observed (need COHb >40%; may be obscured by co-existent hypoxia and cyanosis)
- COHb level correlates poorly with clinical condition
- Treatment should be guided by symptoms and signs, not by blood COHb concentration

ICD-9-CM Code: 986

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Thermal/toxic upper airway injury</td>
<td>Fire exposure/smoke inhalation</td>
<td>Perioral burns</td>
<td>Airway edema</td>
</tr>
<tr>
<td>RESP</td>
<td>CO diffuses rapidly into blood → COHb</td>
<td>Dyspnea, tachypnea</td>
<td>Bronchoconstriction and pulm edema</td>
<td>Co-oximetric COHb: Po₂ usually normal</td>
</tr>
<tr>
<td>CV</td>
<td>↓Blood O₂ content and ↓Tissue O₂ unloading</td>
<td>Possibly angina or evidence of heart failure; tachycardia</td>
<td>Cardiac failure</td>
<td>ECG: Ischemic ST-T changes; CXR</td>
</tr>
<tr>
<td>METAB</td>
<td>Tissue hypoxia → acidosis</td>
<td>Temporal headache, N/V, restlessness</td>
<td>Muscle weakness, altered mental status</td>
<td>Abnormal neuropsychometric testing Can occur after initial recovery</td>
</tr>
<tr>
<td>CNS</td>
<td>Coma, cerebral edema Neurpsychiatric syndrome</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Continuous 100% O₂
- Document CNS status
- Consider hyperbaric O₂ if mental status altered or pt has myocardial ischemia or is pregnant

Monitoring
- Routine monitors
- Standard pulse oximetry (SpO₂) does not distinguish between O₂Hb and COHb. Thus, SpO₂ overestimates O₂Hb during CO poisoning
- Newer SpO₂ monitors (Masino Corp., Irvine, CA) can discriminate between O₂Hb and COHb (and metHb)
- Mixed venous oximeter catheters overestimate O₂Hb in presence of COHb
- Arterial cannula for frequent blood sampling
- Venous and arterial COHb levels are almost identical

Airway
- Airway injury and edema often occur during smoke inhalation, which may require emergent airway management

Induction
- Avoid cardiac depressant agents

Maintenance
- 100% O₂ (no N₂O)
- Assess muscle weakness to guide muscle relaxant dosage

Extubation
- Ensure CNS status permits natural airway maintenance and protection

Adjuvants
- Consider treatment for concomitant cyanide poisoning (see under Cyanide Poisoning in Diseases section)

Postoperative Period
- Maintain 100% O₂
- Consider hyperbaric O₂

Anticipated Problems/Concerns
- Heart and brain affected most
- Follow CNS function carefully
- Seek concomitant smoke inhalation injury and cyanide toxicity
- CO toxic in trace quantities (breathing 0.1% inspired CO for 1 hr results in significant toxicity, with COHb ~ 30%); CO not detectable with conventional gas analysis instruments (e.g., capnographs, mass spectrometers)
- Standard pulse oximeters do not specifically measure COHb, and SpO₂ measurements are only minimally affected, even by severe CO poisoning

Etiology
- CO produced by incomplete oxidative combustion (e.g., house fires, malfunctioning butane/propane stoves, home heaters, and all internal combustion engines)
- Suicide attempts

Usual Treatment
- Normobaric O₂: T₁/₂ of COHb decreases from 3.5 hr (air-breathing) to 0.75 hr (O₂-breathing)
- Treat clinical symptoms, not just increased COHb
- General supportive care, esp for other aspects of smoke inhalation injury
- Hyperbaric O₂ (2.5 atm) decreases COHb T₁/₂ to 20 min and has been shown to decrease probability of development of delayed neurologic complication; for pts with significant exposure (e.g., COHb >25%), hyperbaric O₂ is recommended if feasible, within 6–8 hr of exposure
Carcinoid Syndrome

Stanley H. Rosenbaum

Risk
- Most common GI endocrine tumor
- 15 cases/1 million population per year

Perioperative Risks
- Associated with pt's ability to tolerate abrupt hemodynamic change and/or bronchospasm

Worry About
- Abrupt Htn or hypotension with stress
- Right-sided valvular heart disease
- Bronchospasm

Overview
- Endocrinologically active tumor from GI mucosa
- May release histamine-like substances leading to hypotension and bronchospasm, or may release serotonin leading to hypertensive reactions (and hypovolemia)
- Commonly in ileum (esp appendix) or rectum, less so in pancreas and lung
- Systemically active when metastatic to liver, or when released substances avoid metabolism by liver (carcinoid syndrome)

ICD-9-CM Codes: 209.7 (Tumor); 259.2 (Syndrome)

Etiology
- Acquired disease
- May be associated with other ectopic humoral tumors, such as MEN 1 syndrome

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Cutaneous flushing, lacrimation</td>
<td>Episodic flushing induced by stress, eating, alcohol consumption</td>
<td>Hyperkeratosis, hyperpigmentation</td>
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<tr>
<td>CV</td>
<td>Histamine-induced hypotension</td>
<td>Sx of right-sided CHF</td>
<td>Murrums of pulmonic stenosis, tricuspid regurgitation, ascites, edema</td>
<td>ECHO Cardiac catheterization</td>
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<td>RESP</td>
<td>Bronchospasm</td>
<td>Episodic asthma poorly responsive to medication</td>
<td>Wheezing associated with episodes of flushing</td>
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<td>GI</td>
<td>Diarrhea</td>
<td>Episodic watery diarrhea</td>
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<td>ENDO</td>
<td>Serotonin secretion</td>
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<td>Dehydration from chronic vasoconstriction or diarrhea</td>
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<td>CNS</td>
<td>Hemodynamic instability, vasodilation</td>
<td>Hypertensive headache</td>
<td>Syncope with flushing</td>
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<td>MS</td>
<td>Cutaneous flushing, lacrimation</td>
<td>Episodic flushing, induced by stress, eating, alcohol consumption</td>
<td>Hyperkeratosis, hyperpigmentation</td>
<td></td>
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</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Assess adequacy of fluid balance.
- Assess right-sided valvular status.
- Somatostatin analogue (octreotide) available; its use has dramatically decreased hazards of anesthesia for pts with carcinoid syndrome.

Monitoring
- Expect rapid fluctuation of BP.
- Central venous pressures may not correlate well with fluid volumes.

Airway
- Risk of stress-induced wheezing (Rx: somatostatin analogue)

Induction
- Chronic vasoconstriction and diarrhea may cause hemodynamic instability.

Maintenance
- Volume assessments complicated by changing vascular tone
- Cardiac function limited by right-sided valvular lesions

Extubation
- Possible stress-induced hemodynamic instability (Rx: somatostatin analogue)

Postoperative Period
- Hemorul effects of hemodynamically active metastatic carcinoid usually not eliminated by surgery

Adjuvants
- Caution! Catecholamines may increase hemorul release and worsen symptoms.
- Somatostatin analogue for hypo- or hypertension or bronchospasm has dramatically decreased anesthesia risk for pts with carcinoid syndrome
Cardiomyopathy, Alcoholic

**Risk**
- Incidence in USA: 15 to 20 million chronic heavy ethanol users
- As much as 50% of dilated cardiomyopathy may be ethanol related
- Population at risk: Unclear; likely incl chronic ethanol users with at least 90g of daily ETOH for at least 5y (1 standard drink = 12 grams ETOH)
- Gender: Male predominance

**Perioperative Risks**
- Alcohol withdrawal
- CHF
- Dysrhythmias common: AFIB, PAC, PVC

**Worry About**
- Myocardial ischemia: supply < demand (CAD rare)
- Abnormal systolic and diastolic function
- Chronic alcohol use alters myocardial response to inotropes
- Alcohol withdrawal symptoms

**Overview**
- Insidious onset; Sx uncommon unless severely stressed until late in course
- Dilated cardiomyopathy: ventricular hypertrophy early, chamber dilation later
- Low-output cardiac failure (as compared with high-output failure in cirrhosis and beriberi)
- Malnutrition often co-exists

**ICD-9-CM Code:** 425.5

**ASSESSMENT POINTS**

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<tr>
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<td>Plethora, reflux, esophageal varices, friable mucosa</td>
<td>Reflux Sx Hematemesis</td>
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<td>LV dysfunction</td>
<td>Fatigue, orthopnea</td>
<td>Narrow pulse pressure</td>
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<td>CHF</td>
<td>PND</td>
<td>Cardiomegaly</td>
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<td>Myocardial ischemia</td>
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<td>Dysrhythmia</td>
<td>Palpitations</td>
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<td>RENAL</td>
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<td>CNS</td>
<td>Poor perfusion</td>
<td>Confusion</td>
<td>Abn mental status</td>
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<tr>
<td>MS</td>
<td>Proximal muscle weakness</td>
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<td>Proximal limb weakness and muscle atrophy</td>
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</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Pharmacologic management of CHF

**Monitoring**
- ECG with ST-segment analysis
- Consider arterial pressure catheter, pulm artery catheter, TEE depending on surgery and ventricular function

**Airway**
- None

**Preinduction/Induction**
- May have intravascular volume depletion

**Maintenance**
- Avoid tachycardia, increased sympathetic activity
- Avoid depression of myocardial contractility

**Extubation**
- Routine

**Postoperative Period**
- Consider monitoring in critical care unit
- Observe for ethanol withdrawal

**Etiology**
- Direct myocardial damage by ethanol and its metabolites
- Progressive chamber dilation and ventricular hypertrophy; microscopic fibrinoid deposition
- Possible intracellular calcium dysregulation
- Possible muscle excitation-contraction impairment

**Usual Treatment**
- Abstinence: Ventricular function improves markedly after abstinence
- Pharmacologic management: Digitalis, diuretics, beta-blockers, ACE inhibitors
- Address nutritional deficits: Thiamine, folate, multivitamin

**Adjuvants**
- Multivitamins, thiamine, B12, folate continued
- Consider benzodiazepines, a2 agonists for prophylaxis against withdrawal symptoms
- Volume of distribution may be increased; consider adjusting drug dosages

**Anticipated Problems/Concerns**
- Postop ventricular dysfunction and CHF can occur.
- Alcohol withdrawal symptoms can develop.
Cardiomyopathy, Hypertrophic (HCM)

Risk
• Incidence: 0.2% (1/500) of general population; equally affects males and females, no racial group predominance; median age of 35 y (but can manifest in any age)
• Heterogeneous clinical presentation: Frequent cause of sudden cardiac death (SCD) in young athletes; elderly pts tend to be less symptomatic, reflecting diverse genetic background.
• May be asymptomatic (20–25%) or undiagnosed at the time a pt presents for general surgery (anesthesia may “unmask” HCM)

Perioperative Risks
• Dynamic left ventricular outflow obstruction (either at rest or provoked) present in approx 50% of pts with HCM
• Diastolic dysfunction, risk for heart failure from impaired relaxation of a hypertrophic and non-compliant LV
• Risk for myocardial ischemia even in the absence of obstructive CAD, due to increased myocardial O₂ demand (LVH, high intraventricular pressures) and limited supply (abnormal impaired coronary reserve, microvascular and subendocardial ischemia, intramyocardial coronary arteries)
• Supraventricular (atrial fibrillation) and ventricular dysrythmias

Worry About
• Factors that can aggravate dynamic outflow obstruction and lead to hypotension and hemodynamic compromise: Decreased preload (hypovolemia), decreased afterload (vasodilation), increased sympathetic activation (from pain, surgical stimulation), increased LV contractility
• Myocardial ischemia (even in a background of a “normal” coronary angiogram or thallium)
• Diastolic dysfunction; heart failure difficult to control with traditional diuresis (caution with volume depletion)
• Supraventricular (atrial fibrillation increases risk of embolic stroke) and ventricular dysrythmias

Overview
• Definition: Hypertrophied, nondilated LV in absence of other cardiac or systemic causes for LVH.
• Variable clinical spectrum (from asymptomatic to severely symptomatic to sudden cardiac death)
• Although left ventricular outflow tract (LVOT) obstruction, clinical symptoms, family Hx or documented genetic mutation may be present, none of the above are considered mandatory criteria for the diagnosis of HCM.
• Myocardial disarray on histopathology (95%)
• The interventricular septum is usually (~70%) disproportionately “thicker” (> 15 mm). Less common variants (20–30%) involving the apex (often in Asian pts) or LV free wall have been described.
• Vigorous LV contractility and hypertrophied myocardium may physically obstruct left ventricular outflow tract (LVOT) during systole, resulting in dynamic outflow tract gradient.
• The gradient may be absent or minimal at rest in about 20–30% of pts, but increases with dynamic “provocation” maneuvers (Valsalva, post-PVC, anything that increases contractility or promotes vasodilation).
• Associated mitral regurgitation (MR) is often present.
• Systolic anterior motion (SAM) of the mitral valve leaflet is considered the predominant mechanism of MR in HCM. The anterior leaflet of mitral valve may be “drawn” into the LVOT by Venturi effect during systole, contact the septum and contribute to dynamic obstruction.
• Primary structural abnormalities of the mitral valve apparatus and/or papillary muscles have been described and may be present in at least 1/3 of HCM pts, independent of SAM.
• Clinical manifestations of HCM may relate to diastolic heart failure (dyspnea, fatigue), ischemia (angina, often in the absence of obstructive CAD) arrhythmias (palpitations, syncope, even sudden death) or hypotension associated with obstruction (dizziness, syncope).
• Rarely (~10%) advanced “burn out” stage with LV dilatation resembling dilated cardiomyopathy.
• Diagnostic modalities incl:
  • 12-lead ECG (LVH voltage criteria, arrhythmias, characteristic deep T-wave pattern in apical HCM)
  • ECHO (LVH in non-dilated LV, LVEF > 60%, LVOT gradient at rest or with Valsalva, SAM, or other mitral valve abnormalities).
  • Cardiac catheterization is not mandatory but frequently performed to exclude CAD and confirm the diagnosis or “localize” the gradient (differentiate from AS) when ECHO images are suboptimal.
• Cardiac MRI with gadolinium hyperenhancement demonstrates a characteristic pattern of HCM-related microvascular ischemia and can be helpful to plan surgical management.
• Other tests (HOlTER, exercise stress ECHO, biopsy) are rarely necessary.

ICD-9-CM Code: 425.1 (Hypertrophic obstructive cardiomyopathy)

Etiology
• Genetically heterogeneous: At least 400 mutations in 12 genes that encode for cardiac sarcomeric proteins (beta myosin heavy chain, myosin binding protein C, troponins T and I, troponymosin) identified; the diversity in mutations and variability in penetrance account for the very wide spectrum in diagnosis (from asymptomatic to sudden death). Certain mutations carry worse prognosis, esp regarding risk of SCD. Genetic testing is now widely available and offered to pts and their first degree relatives.

Treatment
• Medical: Negative inotropes (β-blockers, non-dihydropyridine Ca²⁺-channel blockers, i.e., verapamil or diltizem, the class Ia antiarrhythmic disopyramide)
• ICD in pts at high risk of sudden death based on clinical risk factors or family Hx or “high-risk” genetic mutations (DDD pacemaker alone rarely used)
• ”Septal reduction” therapies to reduce LVOT gradient:
  • Surgical septal myectomy (produces LBBB, mitral valve repair or replacement if warranted
• Percutaneous intervention: Septal ablation by alcohol injection (produces RBBB, risk of high-grade AV block)


ASSESSMENT POINTS

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<tr>
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<th>Test</th>
</tr>
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<tbody>
<tr>
<td>CV</td>
<td>Myocardial ischemia</td>
<td>Angina</td>
<td>Worse with nitrates (avoid)</td>
<td>ECG, exercise tests, coronary angiography (may be “normal”), cardiac MRI</td>
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<td>LVOT obstruction</td>
<td>Dyspnea, syncope, dizziness</td>
<td>Systolic murmur accentuated by Valsalva</td>
<td>ECHO</td>
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<td>Mitral regurgitation</td>
<td>Dyspnea</td>
<td>Holosystolic murmur</td>
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<td>Dysrhythmias</td>
<td>Syncope, sudden death, palpitations</td>
<td>ECG, Holter</td>
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<td>Diastolic dysfunction</td>
<td>Dyspnea</td>
<td>Rales, wheeze, edema</td>
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<td>RESP</td>
<td>Pulm congestion</td>
<td>Dyspnea, orthopnea</td>
<td>Rales, wheeze</td>
<td>CXR</td>
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<td>Secondary pulm hypertension</td>
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<td>Right heart catheterization</td>
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<tr>
<td>CNS</td>
<td>Syncope</td>
<td>Syncope, presyncope</td>
<td></td>
<td>Negative CNS work-up</td>
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</tbody>
</table>

Perioperative Implications

Preoperative Preparation
• Avoid physiologic changes that reduce LV cavity size (maintain preload and afterload, avoid tachycardia)
• Ensure adequate preload, replace any preoperative volume depletion
• Optimize pre- and periop β-blocker or Ca²⁺-channel blocker therapy
• Note: HCM pts often are on high doses of β-blocker preop (caution for withdrawal)

• Also note disopyramide (used preop in severe HCM) has anticholinergic activities
• Sedate adequately to prevent anxiety-induced sympathetic stimulation
• β-blocker preop recommended for HCM pts with severe MR or septal myectomy
• ICD/pacemaker evaluation (rarely pt may be pacer-dependant, esp postseptal ablation)

Monitoring
• Depending on nature of surgical procedure, consider invasive arterial pressure monitoring and/or pulm artery catheter
• Transesophageal ECHO can be invaluable, esp if major blood loss, volume shifts, or sympathetic stimulation are anticipated.

Airway
• None

Preinduction/Induction
• Avoid drug-induced vasodilation or sympathetic activation. When choosing an induction agent, etomidate may be advantageous over ketamine or propofol.
• Phenylephrine infusion (alternatives: vasopressin, norepinephrine) should be immediately
available, as worsening dynamic outflow tract gradient may be anticipated with any drop in SVR or sympathetic stimulation.

- Avoid prolonged laryngoscopy, as it may induce sympathetic stimulation.
- Insertion of CVP/PAC may induce atrial or ventricular dysrhythmias.

**Maintenance**

- Volatile agents that decrease LV contractility without dramatic vasodilation are desirable. Halothane is the classic example. Likewise, sevoflurane is preferable over isoflurane or desflurane.
- Avoid agents that decrease preload and afterload (e.g., nitroglycerin, nitroprusside) or increase contractility (inotropes) as well as agents associated with significant histamine release.
- Avoid agents that directly or indirectly increase HR and contractility (e.g., pancuronium, atropine, epinephrine, ephedrine).
- Hypotension treated with
  - Volume expansion (avoid anemia; promptly replete blood loss).
- Pure α-adrenergic agonist (e.g., phenylephrine).
- Spinal anesthesia may be associated with hypotension. Epidural analgesia may be considered to avoid sympathetic stimulation from pain, but with caution to avoid significant afterload reduction and hypotension.
- Consider early electrical cardioversion for atrial fibrillation. Defibrillator available in OR.
- Consider β-blockade or Ca²⁺-channel blockade to prevent tachycardia, LVOT obstruction or ischemia.

**Extubation**

- Avoid sympathetic stimulation. Assure adequate analgesia.
- Anticipate subendocardial ischemia. Utilize β-blockade or Ca²⁺-channel blockade.
- Pulmonary edema from diastolic dysfunction and MR is difficult to treat (use diuretics very judiciously if at all).
- Secondary pulmonary HTN from HCM and related MR worsens with “conventional” pulm vasodilators (increase LVOT obstruction).
- Advisable to maintain minute ventilation by using higher rates, lower tidal volumes (higher tidal volumes with lower rates will decrease venous return).

**Postoperative Period**

- Aggressive postop pain management: If neuraxial analgesia is considered, administration of intrathecal or epidural narcotics only is the preferred and better tolerated approach.

**Anticipated Problems/Concerns**

- Myocardial ischemia (usually subendocardial if conditions favor high O₂ demand)
- Profound hypotension in settings of hypovolemia, decreased preload/afterload, or increased contractility
- Dysrhythmias (ventricular, supraventricular, bradyarrhythmias in the setting of prior septal reduction procedures)
- Diastolic dysfunction with possibility of pulm edema (esp with prolonged mechanical ventilation) and secondary pulm HTN (exacerbated by significant MR)
Cardiomyopathy, Ischemic

Risk
- Approximately 1:1000 incidence per year
- M:F ratio 2:1

Perioperative Risks
- Most important periop risk factor for cardiac morbidity and mortality
- Risk of CHF, hypotension, pulm edema, myocardial ischemia and infarction, renal insufficiency, arrhythmias
- Left ventricular ejection fraction (LVEF) important for prognosis, periop complications. LVEF may not correlate with symptoms or exercise tolerance

Worry About
- CHF exacerbation, pulm edema, hypotension, myocardial ischemia and infarction, renal insufficiency, inability to tolerate fluid shifts associated with major surgery, arrhythmias

Overview
- Severe impairment of LVF leading to CHF; that arising from myocardial ischemia and infarction has extremely poor prognosis, with 30–50% 2-y mortality.
- Pts may benefit from intensive medical therapy for underlying ischemia (nitrates, β-blockers, calcium antagonists, aspirin), CHF (ACE inhibitors, hydralazine, digoxin, diuretics, aldosterone antagonists, angiotension-II receptor blockers), prevention of cardiac thrombus formation (warfarin), and HR control for atrial fibrillation (digoxin, β-blockers).
- An implantable cardioverter-defibrillator (ICD) for secondary prevention of sudden cardiac death (SCD) will be present in many pts.
- Cardiac resynchronization therapy (CRT) with biventricular pacing improves symptoms in pts in prolonged QRS duration with low EF.
- Associated mitral regurgitation, left ventricular aneurysm, and ventricular arrhythmias may have specific periop considerations.

ICD-9-CM Code: 414.8

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<tr>
<td>CV</td>
<td>Myocardial ischemia</td>
<td>Angina, Dyspnea, PND palpitations</td>
<td>S1, S2, loud P2</td>
<td>ECG</td>
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<td>Arrhythmias CHF</td>
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Perioperative Implications

Preoperative Preparation
- Pharmacologic control of myocardial ischemia and CHF

Monitoring
- ECG (V1 or multilead) with ST-segment analysis
- Arterial catheter (close BP monitoring, ABGs)
- Consider PA catheter or TEE for major operations and/or poor medical condition

Airway
- None

Preinduction/Induction
- Avoid tachycardia and increased afterload to prevent ischemia and reduced cardiac output.

- Hypovolemia may result from diuretic therapy.

Maintenance
- Limited ability to increase cardiac output in response to stress
- Attention to fluid balance, monitor PAWP to avoid pulm edema or low cardiac output
- High doses of inhaled anesthetics may be poorly tolerated because of myocardial depression superimposed on cardiomyopathy

Extubation
- May be time of greatest stress for developing myocardial ischemia or LV dysfunction.
- Consider postop mechanical ventilation if a large fluid resuscitation was required intraop.

Adjuvants
- Extensive preop medical therapy may have circulatory consequences.
- Preop anticoagulation may preclude regional anesthesia.

Postoperative Period
- Epidural pain management techniques may limit stress if operation was major (beware of warfarin therapy).
- Intensive care and hemodynamic monitoring may prevent complications if operation was major.

Anticipated Problems/Concerns
- Periop myocardial ischemia and CHF remain paramount concerns.
Carnitine Deficiency

Risk
- Rare (1:40,000 in Japan)

Perioperative Risks
- Hypoglycemia triggered by fasting.
- Massive rhabdomyolysis and cardiac arrest described following GA and succinylcholine. The response may be confused with malignant hyperthermia.

Worry About
- Periop hypoglycemia: Avoid prolonged fast; IV glucose should be administered.
- Neurologic and cardiopulmonary status: Determine if a cardiomyopathy is present.

Overview
- Carnitine is essential co-factor in enzymatic transport of long-chain fatty acids into mitochondria, in which they are oxidized.
- When carnitine is deficient, peripheral tissues cannot use fatty acids for energy production and the liver cannot adequately make ketone bodies as an alternative substrate.
- The tissues become glucose dependent, and their metabolism exceeds liver’s capacity for glucose production.
- This glucose dependency can lead to severe liver failure (↑ hepatic enzymes, lactic acidosis, hypoketotic encephalopathy) and hypoglycemia.

ICD-9-CM Code: 277.8

Etiology
- Mutations in the SLC22A5 gene lead to the production of defective OCTN2 carnitine transporters.
- Differentiate from carnitine palmityl transferase (CPT) deficiency, which results in impaired transfer of fatty acids into mitochondria.
- CPT deficiency associated with rhabdomyolysis and higher incidence of renal insufficiency.

Usual Treatment
- Dietary supplementation with l-carnitine and high-carbohydrate diet to prevent hypoglycemia.

ASSESSMENT POINTS

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<td>Cardiomyopathy</td>
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<td>with fatty infiltration</td>
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<td>Coagulopathy</td>
<td>Bleeding</td>
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<td>CNS</td>
<td>Encephalopathy</td>
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<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Recurrent myoglobinuria</td>
<td>BUN/Cr</td>
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</table>


Perioperative Implications

Preoperative Preparation
- Continue daily carnitine therapy
- Glucose infusion preop
- Avoid protracted preop fasting
- For emergency surgery while in metabolic crisis, rehydrate; correct glucose, acid-base, and electrolyte imbalances, use IV carnitine if necessary, treat hypoprothrombinemia with FFP

Monitoring
- Routine

Airway
- Best to avoid succinylcholine for intubation

Maintenance
- IV glucose infusion, frequent monitoring of serum glucose level
- Muscle weakness may be present and requires careful titration of muscle relaxant dosing

Extubation
- No unusual concerns

Adjuvants
- Consider antiemetic prophylaxis to speed resumption of oral intake

Anticipated Problems/Concerns
- Periop hypoglycemia, metabolic acidosis/decompensation
- In the presence of carnitine deficiency, propofol may theoretically result in mitochondrial dysfunc- tion and cellular hypoxia
Carotid Sinus Syndrome

Risk
• Male > female
• 9% of pts with recurrent syncope
• Incidence increases with age
• Peripheral vascular disease/previous carotid endarterectomy
• Head and neck cancer

Perioperative Risks
• Presence of carotid sinus syndrome (CSS) does not increase rate of mortality, sudden death, or stroke when compared to pts with similar age and risk factors.
• CSS does increase morbidity, secondary to injuries sustained during syncopal episodes.

Worry About
• Presence of co-morbid conditions: Coronary artery disease, carotid stenosis, neck tumor
• Severity of CSS and frequency of syncopal episodes
• Hemodynamic compromise: Bradycardia and/or hypotension

Overview
• The carotid sinus reflex occurs with changes in transmural pressure of the baroreceptors at the carotid sinus.
• Reflex
  • Afferent signals are sent via glossopharyngeal and vagus nerves to the nucleus tractus solitarius.
  • Efferent signaling occurs through sympathetic and vagus nerves to the heart and blood vessels.
• Carotid sinus hypersensitivity (CSH) is defined as an exaggerated response to baroreceptor stimulation.
• Carotid sinus syndrome (CSS) occurs in pts with CSH when direct carotid sinus massage (CSM) or accidental neck stimulation produces symptoms such as dizziness/syncope or bradycardia and/or hypotension.
• There are 3 types of CSS
  • Cardioinhibitory type comprises 70–75% of cases due to vagal stimulation of SA and AV nodes, resulting in sinus bradycardia and may be treated with atropine.
  • Vasodepressor type represents 5–10% of cases, resulting in hypotension due to inhibition of vasomotor sympathetic tone; differentiated with cardioinhibitory type by not responding to atropine treatment.
  • Mixed type occurs in 20–25% of cases and results in bradycardia and loss of vasomotor tone.
• Diagnosis: Perform carotid sinus massage (CSM) in supine position and massage each carotid individually for 3 sec. Test is positive if any of the 3 are true: asystole greater than three sec (cardioinhibitory type); decrease in systolic BP greater than 50 mmHg (vasodepressor type); and combination of previous (mixed type)

ICD-9-CM Code: 337.0

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Syncopel</td>
<td>Dementia, DLB</td>
<td>CSM</td>
<td>MRI brain</td>
</tr>
<tr>
<td>AIRWAY / HEENT</td>
<td>Potential difficult intubation/ventilation Bradycardia and/or hypotension</td>
<td>Neck mass, neck surgery, symptoms with neck movement</td>
<td>Airway exam, tracheal deviation, Carotid bruit</td>
<td>CSM with EKG monitoring and A-line Carotid duplex CT/MRI neck</td>
</tr>
<tr>
<td>CV</td>
<td>Bradycardia and/or hypotension</td>
<td>Syncopel with head turning or neck stimulation, CAD, PVD</td>
<td>CSM</td>
<td>CSM with EKG monitoring, A-line</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative
• EKG, increased workup if advanced CAD
• CXR and/or CT scan to rule out tracheal compression if neck mass present
• Interrogate pacemaker, convert to DOO mode if unipolar cautery is to be used.

Monitoring / Lines
• Consider arterial line for symptomatic pts
• If no pacemaker is present, must have external pacer readily available.

Airway
• Minimize neck extension during laryngoscopy, inline immobilization may be used.
• Asleep fiberoptic if pt has frequent symptoms with neck movement.
• Awake fiberoptic if mediastinal mass may cause tracheal compression with loss of spontaneous ventilation.

Positioning
• Avoid turning of pts neck.
• Ensure that instruments or personnel are not causing pressure to pts neck.

General Anesthesia
• Emergency drugs may be required based on type of CSS.
• General anesthesia may be preferred since inhalational agents have been shown to attenuate baroreceptor reflexes.
• Avoid hypotension on induction if coronary or carotid disease is present.
• Avoid long-acting beta blockers or antihypertensive drugs.

Regional Anesthesia
• Glossopharyngeal nerve block for CSS treatment may be performed.
• As an adjuvant to general anesthesia, local anesthetic may also be injected around carotid sinus prior to ipsilateral neck dissection to attenuate the baroreceptor response.

Etiology
• Unclear, several proposed theories
• Mechanical deformation of the carotid sinus from neck tumors or previous carotid endarterectomy may lead to an exaggerated response.
• Degenerative process of the nucleus tractus solitarius that occurs with age
• Possible association with dementia, esp dementia with Lewy body disease (DLB)

Usual Treatment
• Medication
  • Atropine or vasopressors for acute, symptomatic pt
  • Serotonin reuptake inhibitors and alpha antagonists (midodrine) have been used with moderate success.
• Permanent dual chamber cardiac pacing is effective for cardioinhibitory and mixed types of CSS in pts who are symptomatic (pacing is of no benefit in vasodepressor type)
• Surgical denervation of carotid sinus may be attempted to treat vasodepressor type or pts who remain symptomatic despite pacing.
• Blocking the afferent limb (glossopharyngeal nerve) of the reflex with ethanol ablation is controversial due to a high complication rate.
• Volume expansion and increased salt intake is beneficial in vasodepressor type.
• Surgical removal of neck mass causing carotid sinus compression

Postoperative Period
• Strict postop orders in PACU outlining no head turning or neck compression.
• If intraop asystole has occurred, surgeon may have to place a temporary transvenous pacer.

Anticipated Problems/Concerns
• Potential difficult intubation
• Must assess pacemaker function if present
• Pt may undergo profound hypotension or asystole at any time in the peripd setting; emergency drugs should be readily available.
• Goal: Avoid neck stimulation and maintain hemodynamic stability.
Central Neurogenic Hyperventilation

Roy F. Cucchiara
Perry S. Bechtle

Incidences
• True central neurogenic hyperventilation (CNH) is exceedingly rare; exact incidence unknown
• In pts with neurologic injury, it is not rare and most often associated with pulm dysfunction or shunting (aspiration, pneumonia, pulm edema, baseline disease)
• No association with age or gender

Overview
• A diagnosis of exclusion in neurologic disorders and hyperventilation; life-threatening causes of hyperventilation (hypoxemia, ischemic bowel, acidosis) must be sought and ruled out
• Primary diagnostic criteria are hyperventilation that persists during sleep; low PaCO₂, high PaO₂, and absence of drug or metastatic causes

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Tachypnea</td>
<td>Tachypnea that persists during sleep and is unpleasant to conscious pt</td>
<td>Resp rate Normal inspiratory and expiratory excursion</td>
<td>ABGs (all must be present to diagnose): Pco₂ (low) pH (alkalotic) Pao₂ (increased for age) Decreased bicarbonate Alveolar-to-arterial gradient not larger than normal</td>
</tr>
<tr>
<td>CNS</td>
<td>Pt cannot volitionally inhibit hyperventilation Focal or nonfocal CNS findings</td>
<td>CSF pH may be normal CT/MRI</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Differential Diagnosis for Hyperventilation
• Metabolic acidosis
• Bowel ischemia with acidosis
• Pulm pathology with hypoxemia (pneumonia, pulm embolus, pulm edema, restrictive or obstructive lung disease)
• Drug toxicity (saliclylates, theophylline, cyanide, topiramate)
• Sepsis
• Encephalopathy/CNS lesions (glioblastoma, encephalitis, multiple sclerosis, brainstem lympha, brainstem glioma, liver dysfunction)
• Anxiety
• Psychogenic
• Cardiac (CHF, valvular disease)
• High altitudes
• Hyperthyroidism
• Pregnancy
• Must exclude other etiologies for resp alkalosis with appropriate lab/Dx testing

Adverse Effects
• Resp alkalosis shifts oxyhemoglobin curve to left
• Hypocapnia is a potent cerebral vasoconstrictor, subsequently decreasing cerebral blood flow and volume
• Hypocapnia in injured brains may result in ischemic insults
• Effect of severe hypocapnia in normal brains is less clear and may produce ischemia when combined with Bohr effect

Treatment
• No completely effective or consistent treatment
• Narcotics may attenuate resp rate and improve blood gases but will not correct rate or alkalosis.
• Increasing dead space ventilation, administration of supplemental oxygen and benzodiazepines are not effective.

Etiology
• Exact etiology and level of brainstem dysfunction not known
• Probable etiology
• Uninhibited stimulation of inspir and expir centers in the medulla and/or loss of descending inhibitory control of ventilation by cerebral cortex with brainstem lesion

Outcome
• Death from progressive neurologic deterioration or other complications (aspiration, pneumonia) likely
• Improvement with treatment of tumor or long-term narcotics

Outcome

CT/MRI

• Primary diagnostic criteria are hyperventilation that persists during sleep; low PaCO₂, high PaO₂, and absence of drug or metastatic causes

Differential Diagnosis for Hyperventilation

- Associated primarily with brainstem tumors with inconsistent involvement of midbrain, pons, and/or medulla
- CNS lymphomas and astrocytomas are the most common tumor types with gliomas, lymphomatoid granulomatosis, medulloblastoma, metastatic tumors also reported
- Effects of GA unknown

ICD9-CM: 786.01 (Hyperventilation)

Etiology

- Exact etiology and level of brainstem dysfunction not known
- Probable etiology
- Uninhibited stimulation of inspir and expir centers in the medulla and/or loss of descending inhibitory control of ventilation by cerebral cortex with brainstem lesion

Outcome

- Death from progressive neurologic deterioration or other complications (aspiration, pneumonia) likely
- Improvement with treatment of tumor or long-term narcotics

Outcome

- Primary diagnostic criteria are hyperventilation that persists during sleep; low PaCO₂, high PaO₂, and absence of drug or metastatic causes
Cephalopelvic Disproportion

Darren Cousin

Risk
- 1% to 3% of pregnant population

Perioperative Risks
- Increased maternal and fetal morbidity and mortality
- Protracted labor
- Arrested labor
- Uterine rupture
- Increased rate of cesarean section
- Increased rate of forceps or assisted delivery

Worry About
- Increased need for surgical delivery

Overview
- Leads to abnormal labor pattern with subsequent high incidence of operative delivery
- Operative delivery associated with higher incidence of morbidity and mortality to mother and fetus
- Anesthesia necessities: Complete system exam inc airway for possible emergency C-section and landmarks for regional anesthesia

ICD-9-CM Code: 653.4

Etiology
- Maternal causes incl abnormalities of the pelvis, Hx of scoliosis, previous pelvic trauma, Hx of poliomyelitis
- The primary fetal cause is macrosomia often secondary to gestational diabetes

Usual Treatment
- Obstetric: Proper evaluation prior to and during labor
- Anesthesia: Regional for pain relief during labor or operative delivery

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</tr>
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<tbody>
<tr>
<td>GYN</td>
<td>CPD</td>
<td>Failure to adequately respond to oxytocin</td>
<td>Pelvic exam</td>
<td>Radiographic cephalopelvimetry</td>
</tr>
</tbody>
</table>


Perioperative Implications
- **Labor** is usually more prolonged and painful in pts with CPD.
- Epidural or combined spinal epidural is adequate to cover pain without prolonging the course of labor.

Anesthetic Technique
- Epidural analgesia: Low concentration of bupivacaine .25% or ropivacaine .20% supplemented with an opioid of your preference
- Combined spinal and epidural analgesia. In **early labor**: 25mcg fentanyl combined with 1 mL of .20% ropivacaine or .25% bupivacaine intrathecally followed by continuous epidural at 8 to 10 mL per hr. In **late labor**: The initial intrathecal injection of local anesthetic and opioid is often sufficient for the remainder of the first and second stages of labor.
- **C-section** (in all cases): Anesthesia machine checked. Left uterine displacement to maximize uterine blood flow. Apply ASA standard monitors.
- **Elective C-section**: Spinal anesthesia in usual fashion. Prehydration with 15mL/kg, followed by 1.4 cc of .75% bupivacaine injected at L4. Maintain BP within normal limits using ephedrine when necessary.
- **Emergency C-section**: Following labor without fetal distress (failure to progress): If pt has a reliable epidural block, epidural anesthesia is extended using 3% chloroprocaine, 2% lidocaine, or .5% ropivacaine if time permits. If pt does not have an epidural catheter, then a spinal technique may be used.
- **Following labor with fetal distress**: If pt has an epidural catheter, dose with 3% chloroprocaine or 2% lidocaine if time permits. If pt doesn’t have a catheter, GA may be necessary.

Anticipated Problems/Concerns
- If there is no epidural catheter in place and a difficult airway is suspected, then use spinal anesthesia or perform an awake intubation. A fast track LMA, a fiberoptic scope, or a video-enhanced laryngoscope should be available.
Cerebral Arteriovenous Malformations (AVMs)

**Overview**
- Localized arteriovenous shunt comprised of a tangle or "nidus" of abnormally walled vessels which cause symptoms by rupture, ischemia, and diversion of flow or pressure on adjacent structures. Many are detected on routine scans.
- 70% are supratentorial, 4–10% are associated with aneurysms.
- AVMs usually present in the second to the fourth decade of life with a yearly risk of bleed of 3–4%. There is an increased risk of rebleed of 6% within the first year. The majority of AVMs will bleed at least once.
- Mortality from an initial hemorrhage is 10–30%.
- Vein of Galen malformations are rare congenital lesions with connections between the intracerebral vessels and the great vein of Galen. This disorder in neonates or infants may result in high output CHF or increased ICP from hydrocephalus.

**Etiology**
- Although congenital in origin, no specific genetic defect has been determined. Sometimes associated with hereditary hemorrhagic telangiectasia.

**Usual treatment**
- Evidence-based neurosurgical management is predicated on the Spetzler-Martin grading scale. AVMs are graded 1–5 based on their size, location, and pattern of venous drainage.
- Smaller and more superficial AVMs (grade 1–2) might be surgically resected. Higher grade AVMs may be treated with neuroembolization, radiosurgery or conservative management. Combinations of therapy are common, esp embolization prior to surgical resection.

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Airway protection</td>
<td>Aspiration</td>
<td>Active gag reflex</td>
<td></td>
</tr>
<tr>
<td>CVS</td>
<td>CHF in children with vein of Galen AVM</td>
<td></td>
<td>S3, CHF</td>
<td>CXR, ECHO, ECG</td>
</tr>
<tr>
<td>CNS</td>
<td>Seizures, focal deficits, CVA, raised ICP</td>
<td>Headaches, seizures, changes in mentation</td>
<td>Neurologic exam</td>
<td>MRI, MRA, CT, cerebral angiography</td>
</tr>
</tbody>
</table>


**Perioperative considerations**

**Preoperative preparation**
- Endovascular embolization procedures may require GA or MAC.
- Craniotomy for resection requires preop preparation similar to aneurysm clipping.
- Neurologic exam with attention to focal deficits, raised ICP.
- Prior embolization may have been performed.
- Careful assessment of size and location of AVM.

**Monitoring**
- Invasive BP monitoring.
- For craniotomy--preparation for extensive blood loss incl central assess if surgery will involve deep structures.
- Precordial Doppler for detection of air.
- Jugular bulb venous O₂ monitoring has been described.
- Intraop neuro monitoring may incl EEG, SSEPS.

**Airway**
- ETT for craniotomy, LMA or intubation for airway management for embolization.

**Induction**
- Careful management of BP to prevent Htn (increased ICP, hemorrhage) or hypotension (ischemia).

**Maintenance**
- Careful management of BP, ICP exp with intubation, pinning, and incision.
- Surgeons may request burst suppression with propofol or barbiturates for brain protection if temporary clips are used.
- Surgeons may request hypotension or hypercapnia.
- Blood glucose control
- Strict isotonc/hypertonic fluid management
- Angiography prior to emergence
- Plan for arousal and neurologic testing immediately postop.

**Extubation**
- Careful BP control—labetolol, additional opioid
- Expect request for neurologic exam
- May elect to remain intubated

**Adjuncts**
- Cell saver
- BP control with nicardipine, NTG, NTP, beta blockers
- Phenylephrine infusion
- Propofol infusion for TIVA or burst suppression
- Steroids, mannitol
- Antiepilepsy medications

**Postoperative period**
- Complete obliteration of a large AVM will lead to redistribution of the CBF and hyperemia or 'normal perfusion pressure breakthrough". Until auto-regulation returns, the pt may require lower BP and control of CO₂ by intubation and ventilation.
- Postop ICU neurologic monitoring will be required.
## Cerebral Palsy

### Risk
- Leading cause of childhood motor disability
- 1–2 per 1000 live births in developed countries
- Incidence has not decreased despite improved perinatal care because of increased survival in prematurely born

### Perioperative Risks
- Dehydration
- Electrolyte imbalance
- Hypothermia
- Delayed recovery

### Worry About
- Difficult intubation
- GE reflux and aspiration
- Associated resp impairment

### ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Tongue thrusting, Poor dentition, Salivary drooling</td>
<td>Difficulty swallowing</td>
<td>Dental malocclusion</td>
<td>Formal airway assessment usually difficult</td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive defect, Aspiration pneumonia, Recurrent chest infections</td>
<td>Cough, Dyspnea (difficult to detect if mobilization limited)</td>
<td>Often normal, Reduced air entry, Bronchial breathing, Wheeze</td>
<td>Pulm function tests ABGs, CXR</td>
</tr>
<tr>
<td>CV</td>
<td>Right-sided heart failure from restrictive lung disease</td>
<td>Often normal, Dyspnea</td>
<td>Tachycardia, S1 or S3, Distended JVP, Hepatomegaly</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>GI</td>
<td>GE reflux, Esophageal dysmotility</td>
<td>Poor swallowing, Night waking</td>
<td>Dehydration, Pallor, Malnutrition</td>
<td>CBC, Electrolytes, Endoscopy</td>
</tr>
<tr>
<td>MS</td>
<td>Spasticity, Dyskinesia, Ataxia</td>
<td>Muscle pain and spasms</td>
<td>Increased muscle tone, Contractures, Tremor</td>
<td>Gastrin analysis performed before major orthopedic surgery</td>
</tr>
<tr>
<td>CNS</td>
<td>Epilepsy (30%), Visual and hearing defects</td>
<td>Tonic-clonic and complex-partial seizures</td>
<td>Myopia, Visual field defects, Strabismus</td>
<td>Not usually relevant</td>
</tr>
<tr>
<td>HEME</td>
<td>Iron-deficiency anemia</td>
<td>Fatigue</td>
<td>Pallor</td>
<td>CBC, differential</td>
</tr>
<tr>
<td>METAB</td>
<td>Electrolyte imbalance</td>
<td>Laxative use, Fatigue</td>
<td>Dehydration, Malnutrition</td>
<td>UA, Albumin</td>
</tr>
</tbody>
</table>

### Drug interactions
- Latex allergy

### Overview
- Any nonprogressive central motor deficit dating to events in the pre-, peri-, or postnatal periods
- Wide spectrum of symptoms
  - Cognitive impairment
  - Seizures
  - Sensory loss (visual, hearing)
  - Communication, behavioral disturbances
  - Resp, GI, orthopedic problems
- Often have normal intellect (esp dyskinetic group)
- Classified as spastic (70%), dyskinetic (10%), ataxic (10%), and mixed (10%)

**ICD-9-CM Code:** 343.9

### Key Reference:

### Perioperative Implications

#### Preoperative Preparation
- Can have normal intellect
- Involve parents in management, as parents have good insight into periop care
- Avoid unfamiliar faces if possible
- Optimize resp status (bronchodilators, antibiotics, physical therapy)
- Optimize nutrition, fix lyte imbalance
- Continue medical Rx, esp anticonvulsants
- May need antireflux, antisialagogue, or sedative prescribed (cautious doses of sedatives)
- Topical local anesthetic for venipuncture
- Discuss periop analgesia (often regional technique for lower limb surgery)

#### Monitoring
- Core temp (susceptible to hypothermia)
- Neuromuscular blockade
- Airways pressures

#### Airway
- ETT is better sized to age, not wt
- Salivary secretions may make ventilation difficult
- Overbite may make intubation difficult

#### Induction
- Rapid-sequence may be required but often impractical
- IV access often difficult
- Inhalation sometimes favored (in semisitting position if concerns of reflux)

#### Maintenance
- Careful positioning, check frequently
- Consider antiepisodics
- IV fluids
  - MAC may be lower in cerebral palsy—as much as 20% and up to 30% if pt is on anticonvulsants
- Use warming devices
- Consider regional (epidural) techniques for lower limb surgery

#### Other Intraoperative Challenges
- Bleeding
  - Anecdotal: Pts with neuromuscular scoliosis bleed > idiopathic scoliosis
  - Poor nutritional/nonambulatory status
  - Borderline low platelet count and function due to anticonvulsants
  - Subnormal clotting factor level
  - Temp
    - Severe CP kids may be unable to regulate temp
    - Little subcutaneous fat
    - Some arrive to OR with temp <35°C
  - Warm room till pt draped, use warming blanket/gases/liquids

### Etiology
- Mostly unknown
- Antenatal cerebral events causing complications at time of delivery, e.g., periventricular hemorrhage and infection
- Postnatal events such as trauma and infection
- All causes result in damage to CNS during early brain growth.

### Usual Treatment
- Anticonvulsants, antispasmodies (benzodiazepines, baclofen, dantrolene), antidepressants, antireflux agents, laxatives, anticholinergics
- Often intramuscular botulinum toxin injections and orthopedic procedures (tendon releases and osteotomies), fundoplication for reflux, and dental extractions

### Exubation
- Awake if prone to reflux

### Drug considerations
- Baclofen should not be stopped abruptly but may cause postop bradycardia and hypotension
- Resistance to nondepolarizing NMB (probably not clinically significant)
- Ketamine and methohexital may be avoided in epileptic pts
- N2O and opiates may worsen nausea

### Postoperative Period
- Aggressive resp care
- Maintain normothermia
- Susceptible to N/V
- Avoidance/treatment of muscle spasm (IV diazepam, epidural clonidine)
- Early mobilization

### Anticipated Problems/Concerns
- Latex allergy
- Hypothermia
- Prolonged recovery time
- Postop NIV (worse with opiates)
- Postop muscle spasms
- Retention of secretions and postop chest infection
Cerebrovascular Transient Ischemic Attack (TIA)

**Risk**
- Overall risk in USA is approx 83/100,000
- Risk related to demographic factors of age, gender, race
- Age and gender: Estimated prevalence of TIA in men of 2.7% vs. 1.6% in women for 65–69 y old, 3.6% in men vs. 4.1% in women for 75–79 y of age. The overall prevalence is estimated to be 0.4% among adults 45–64 y of age.
- Race: Blacks and men are at highest risk

**Perioperative Risk**
- Pts with Hx of TIs at increased risk of postop stroke
- Increased risk of periop stroke in pts with medical Hx of cerebral vascular disease, peripheral vascular disease, Htn, diabetes, chronic renal insufficiency, COPD, and atrial fibrillation
- Pts with coronary artery disease for CAGB have a high incidence of carotid stenosis (50% with some, 20% with stenosis ≥50%)
- Likewise, pts with carotid stenosis have high incidence of coronary artery disease (over 50%).
- Increased risk of periop stroke in pts with planned surgery: CAGB (3%–6%), vascular (1%)

**Worry about**
- Crescendo TIs
- Duration of symptoms >1 hr

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<tbody>
<tr>
<td>HEENT</td>
<td>Neck trauma Compression</td>
<td>TIA Sx Previous stroke</td>
<td>Carotid bruit Bilat BP</td>
<td>Carotid Doppler Angio: Carotid, vertebral artery.</td>
</tr>
<tr>
<td>CNS</td>
<td>CV disease dz</td>
<td>CAD disease</td>
<td>Hx of MI, angina Arrhythmia Decreased exercise tolerance Risk factors for atherosclerosis</td>
<td>Murrum Irregular rate/rhythm EKG Stress test Holter TEE/TTE</td>
</tr>
<tr>
<td>CV</td>
<td>CAD disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gl</td>
<td>Transient focal neurologic deficit</td>
<td>N/V Vision changes, changes in language, weakness, sensory changes, ataxia Ischemic retina Often neuro exam normal CT/MRI CV imaging</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Preparation**
- Determine BP range that pt normally experiences
- Manage BP with both cerebral perfusion and CAD in mind
- Preop cardiac workup and medical stabilization if non-emergent surgery
- Preop neurologic exam
- Avoid excessive premedication (pt can be more sensitive)
- Avoid long-acting intraoperative agents that can obscure postop neuro exam

**Monitoring**
- EKG monitoring for ischemia and arrhythmia
- Consider arterial line and central line/PA catheter if extensive CV disease

**Airway**
- Avoid extreme neck manipulation and pressure on carotid artery during ventilation and intubation.

**Preinduction/Induction**
- Maintain pressure to allow for sufficient cerebral perfusion (rightward shift in cerebral auto-regulation in Htn)
- Titrate medication as pt requirements can be decreased

**Maintenance**
- Pts can be more sensitive to medications
- Avoid long-acting agents if neurologic exam to be performed postop

**Exubation**
- Ensure pt is awake, following command, and able to protect airway
- Ensure pt does not have large neurologic deficit which would lead to swelling and resp insufficiency postop.

**Postoperative Period**
- Period of greatest risk for stroke after general surgery
- Resume antiplatelet therapy and anticoagulation as soon as possible.
Cervical Spine Disease (Cervical Disk Disease)

**Risk**
- Incidence in the USA: 12,000 deaths/y; 70 million with cervical disk disease, spondylosis, or trauma
- Disk disease—a consequence of aging (third-fifth decades)
- Present in rheumatoid arthritis (RA), ankylosing spondylitis, other rheumatic disorders
- Trauma, esp motor vehicle accidents
- M/F ratio: 3:2

**Perioperative Risks**
- Mortality (acute) 1–5% (depending on associated injuries)
- Spinal cord damage with C-spine movement
- Difficulty intubating or reintubating postextubation
- After neck surgery, swelling or hematoma can cause obstruction of airway
- Steroid-induced complications

**Worry About**
- Airway management; C-spine movement during or after intubation

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<tbody>
<tr>
<td>HEENT</td>
<td>Numbness and pain in RA: Superior migration of odontoid, atlantoaxial subluxation, atlas-dens interval (ADD) increased (&gt; 4 mm unstable), subaxial subluxation, cricoarytenoid arthritis, airway abnormalities, trauma, swelling</td>
<td>Hoarseness, snoring</td>
<td>In RA: TMJ problems, hypoplastic mandible</td>
<td>In RA: Neck x-ray flexion and extension (measure ADD) Evaluate bones, ligament alignment, soft tissue swelling, motion</td>
</tr>
<tr>
<td>CV</td>
<td>Trauma: Possible cardiac contusion/ injury, spinal shock</td>
<td></td>
<td>Heart sounds distant</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Rheumatologic disorders: Fibrosis, honeycombing Ankylosing spondylitis: Restrictive pattern Trauma: Diaphragm function (C3–C5), pneumothorax, hemothorax, contusion, aspiration, rib fractures</td>
<td>SOB</td>
<td>In trauma: Dyspnea, paradoxical ventilation, flail chest, breath sounds absent with pneumothorax</td>
<td>CXR, ABGs</td>
</tr>
<tr>
<td>GI</td>
<td>Ulcers 2° to aspirin for RA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>RA: Anemia 2° to medications</td>
<td>Trauma: Look for signs of bleeding</td>
<td>Hgb</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Vertebral artery compression: Dizziness, vertigo, nausea, blurred vision</td>
<td></td>
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</tr>
</tbody>
</table>


**Perioperative Implications**
- Assess neck in disk disease, rheumatic diseases, trauma
- Consider intubation with neck stabilized by assistant to avoid flexion or extension or awake fiberoptic intubation.
- Consider intubating with fiberoptic intubation, Glidescope, AirTraq, laryngeal mask airway, light wand or other airway assistance device.
- Avoid medications (e.g., midazolam) incl muscle relaxants if they are used at all for initial intubation that might interfere with specialized spinal cord monitoring Somatosensory Evoked Potentials (SSEPs) or Transcranial Motor Evoked Potentials (TCMEPs)

**Monitoring**
- Acute spinal cord shock may require arterial and PA catheters or TEE to facilitate monitoring and treating hemodynamic disturbances
- When using intraop TCMEPs, protect the tongue and ETT from masseter and muscles of mastication contraction during stimulation. Remember, muscle relaxants cannot be used when TCMEPs are utilized.

**Induction**
- Consider not initiating irreversible steps (e.g., muscle relaxants) until airway is secured.

**Exubation**
- Consider not extubating until pt is able to maintain airway without threat of swelling or airway obstruction.

**Adjutants**
- Steroids reduce injury in acute traumatic spinal cord injury.

**Postoperative Period**
- Observe for neck swelling, hoarseness, airway obstruction
- Assess neurologic status

**Anticipated Problems/Concerns**
- Anticipate difficulty intubating pts due to abnormal anatomy or limitation of motion. Prepare pt for fiberoptic intubation.
- Associated traumatic injuries—cardiac, brain, lung, abdomen, bladder, long bones—and their consequences
- ARDS from aspiration in preop traumatic event
- Injury to tongue or ETT from biting down as a result of muscle contraction from TCMEP stimulation.
Chagas’ Disease

Overview
- Acute infection mostly in pediatric population, asymptomatic in ⅔ of pts, followed by chronic disease after latency of > second and third decades
- In endemic areas, mild forms of disease common with benign course
- Pathogenesis to chronic, progressive end-organ disease poorly understood; autoimmune, microvascular dysfunction, autonomic neuropathy implicated
- Cardiac involvement most serious end-organ manifestation; colon and esophagus also affected
- Mechanisms proposed for cardiac involvement unclear but include neurogenic mechanisms, parasite-dependent inflammation, microvascular disease and immune-mediated injury.
- In USA, Dx usually not considered; presentation as CAD or dilated cardiomyopathy, or with AV heart block, CHE, ECG conduction abnormalities, sustained VTach.
- Serologic test for Dx, based on hemagglutination, immunofluorescence, ELISA, PCR, is usually negative during first week so Dx dependent on detection of circulating parasites.
- Continues to cardiac involvement—decapillarization of the myocardium.
- Downregulation of the nicotinic Ach receptors and associated myasthenia gravis symptomatology.

ICD-9-CM Code: 086.0

Perioperative Risks
- Not defined
- Most important prognostic factor is degree of myocardial dysfunction
- Esophageal changes due to megaesophagus and reflux
- Associated with myasthenia gravis
- CNS symptoms—meningoencephalitis (particularly in immunocompromised pts)

Monitoring
- Assessment of conduction abnormalities,
- Prophylaxis against pulmonary aspiration.
- Thromboembolism, stroke

Etiology
- Protozoan infection: Trypanosoma cruzi
- Transmission to humans by reduviid bug, the “kissing bug”.
- Transmission by blood transfusion, organ transplantation, vector, laboratory accident, reactivation of chronic disease during immunosuppression. Recently oral chagasic infection via food contamination (sugar and acai juices) also possible with more severe clinical course.

Usual Treatment
- Nifurtimox (limited efficacy, poor oral bioavailability); for acute disease; usefulness for indeterminate phase or chronic disease not established.
- Benznidazole (similar efficacy as nifurtimox) second agent, not available in USA.
- Recent success with protriptyline in the acute and chronic form.
- Allopurinol for the cutaneous form
- No evidence trypanocide drug therapy cures disease.
- Other treatment related to symptomology: Amiodarone for arrhythmias related to LV dysfunction; sotalol. Invasive treatment modalities incl surgical excision, catheter ablation, aneurysmectomy, epicardial mapping.
- Pts at high risk for sudden cardiac death will have ICD placed.
- Heart transplant, bone marrow cell transplant uncertain.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Conduction abnormalities LV dysfunction and aneurysm</td>
<td>Syncope, DOE, orthopnea, fatigue Antypical Angina</td>
<td>JVD, edema, rales, cardiomegaly Murmurs TR, MR, wide split S2, prominent diffuse apical thrust Biventricular enlargement</td>
<td>ECG ECHO MUGA Cardiac catheter CXR: cardiomegaly Holter electrophysiologic study, TTE, TEE</td>
</tr>
<tr>
<td></td>
<td>Ventricular arrhythmias</td>
<td>Syncope, palpitations</td>
<td>Abdominal distention</td>
<td>Barium studies CXR, endoscopy</td>
</tr>
<tr>
<td>GI</td>
<td>Megaesophagus, megacolon</td>
<td>Dysphagia, GE reflux, constipation</td>
<td></td>
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</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- LV function optimization with diuretics, ACE inhibitors, consider β-blockers and Ca2+-channel blockers. Consider amiodarone in cases of V'Tach/VTIB.
- Prophylaxis against pulmonary aspiration.
- Assessment of conduction abnormalities, arrhythmias.

Monitoring
- Dictated by degree of LV dysfunction and proposed procedure; consider PA catheter or TEE. On TEE may see biventricular enlargement, thinning of ventricular walls, apical aneurysm, intramural thrombus.
- ECG during entire periop period. Often see long QT interval, AV block, bundle branch block. Can have V'Tach/VTIB.

Preinduction/Induction
- Consider temporary pacing if symptomatic AV block
- Caution with negative inotropic drugs
- Awake or rapid-sequence intubation
- Consider judicious use of muscle relaxants

Maintenance
- Technique dictated by preferences, procedure, degree of cardiac involvement
- Avoid hypoxemia (facilitates ischemic myocardial changes on capillary level, which can further progress to wall thinning and aneurysm formation)

Postoperative Period
- Continued monitoring depends on pre-existing LV dysfunction and operative procedure.
- ECG monitoring for ventricular arrhythmias and AV conduction block
Diseases

Cherubism

Risk
- >250 cases in world literature
- Cherubs have a 40% chance of a cherub offspring

Perioperative Risks
- Swelling of lower face causing airway obstruction
- Displacement of ocular orbit and lower eyelid causing visual changes
- Excessive blood loss from curettage of vascular lesions
- Association with Noonan syndrome

Worry About
- Pulm valve stenosis (Noonan syndrome)
- Undiagnosed hyperparathyroidism
- Convex, V-shaped hypertrophied hard palate
- Small mouth opening and mild trismus

Overview
- Progressive symmetric fullness of cheeks and jaw, with retraction of lower eyelids exposing an inferior rim of sclera
- Onset age: 2–12 y
- These round-faced, upwardly gazing infants look like Renaissance art cherubs
- Diagnostic biopsy of mandible shows multinucleated giant cells
- Associated problems with speaking, breathing, swallowing, chewing
- Pathognomonic x-ray of jaw demonstrates radiolucent lesions
- ICD-9-CM Code: 526.89

Etiology
- Mutations in the SH3BP2 gene cause cherubism
- Familial: Autosomal dominant

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Orbits shifted</td>
<td>Loss of binocular vision</td>
<td>Upward gaze</td>
<td>Jaw series</td>
</tr>
<tr>
<td></td>
<td>Enlargement</td>
<td>Photo review by age</td>
<td>Painless jaw swelling</td>
<td></td>
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<tr>
<td></td>
<td>Poor opening</td>
<td>Moderate trismus</td>
<td>Lymphadenopathy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Malocclusion</td>
<td>Absence of third molar</td>
<td>Soft tissue swelling</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>If associated with Noonan syndrome</td>
<td>Pulmonic valve disease</td>
<td>Loose teeth</td>
<td>X-ray</td>
</tr>
<tr>
<td>RESP</td>
<td>Generally unaffected</td>
<td>Obstructive airway</td>
<td>Pulm valve stenosis</td>
<td>Sleep study</td>
</tr>
<tr>
<td>ENDO</td>
<td>Rule out hyperparathyroidism</td>
<td>Onset at older age</td>
<td></td>
<td>Normal Ca++, K+</td>
</tr>
<tr>
<td>CNS</td>
<td>Midparental intelligence</td>
<td>No developmental delay, except with Noonan syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Long bone lesions</td>
<td>Humerus, anterior ribs, femoral neck</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Rule out parathyroid disease
- Available blood for curettage replacement

Monitoring
- Routine

Airway
- Difficult airway protocol
- Oral intubation using a laryngeal mask technique has been reported. Fiberscopic control of the exchange and the introduction of a Cook exchange catheter into the trachea through the oral tube before withdrawal permits oxygenation of the pt and acts as a guide for oral tube reintroduction if required.

Preinduction/Induction
- Spontaneous ventilation
- Laryngeal mask airway

Maintenance
- Consider hypotensive technique for minimizing blood loss

Exubation
- May require ICU admission for prolonged intubation

Adjuvants
- Routine

Postoperative Period
- Extubation awake with confirmation of no bleeding

Anticipated Problems/Concerns
- Nasal intubation for oral procedures may be problematic, similar to Pierre Robin, Goldenhar’s, and Treacher Collins syndromes. As mandibular rami approach midline, no space for visualization of airway.
Cigarette Smoking

Risk
- Incidence in the USA: Estimated 43.4 million (36.4%)
- Native Americans have highest rate of smoking (36.4%)
- More common in impoverished individuals
- M:F ratio: 4:3; young females fastest-growing group

Perioperative Risks
- Increased risk of CAD × 2.0 of nonsmokers of same age
- Postop pulm complications up to × 6 of nonsmoker
- Carboxyhemoglobin (COHb) increased (up to 15%)
- Hyperreactive airway
- Does not increase risk of pulm aspiration
- Reduces risk of postop N/V

Worry About
- CAD, COPD, PVD, productive cough, reactive airway
- Increases physiologic age by 8 y (30 pack-y) relative to nonsmoker
- Decreased tolerance to pain, requiring increased doses of analgesics

Overview
- Addictive habit. Cigarette smoke contains > 4000 identifiable constituents, many of which are pharmacologically active, toxic, or have tumorigenic effects. Acute effects relate to CO and nicotine.
- 90% of tobacco smoke is gaseous, consisting of nitrogen, O₂, carbon monoxide along with gaseous irritants and carbon monoxide. Particulate matter consists of nicotine, tar, and other volatile organics.
- Nicotine stimulates the sympathetic ganglia, causing release of catecholamines from the adrenal medulla and sympathetic nerve endings, increasing BP, HR, and SVR, that persists for 30 min after one cigarette.
- Associated with decreased MAO and increased dopamine levels in brain
- Inhaled CO produces up to 5–15% COHb compared to 0.3–1.6% in nonsmokers. Combined effects of nicotine and COHb put diseased myocardium at risk.
- An irritant to pulm system, increasing mucus production while decreasing ciliary activity and mucus flow, markedly impairing tracheobronchial secretion clearance
- Chronic use associated with CAD, HTN, COPD, peripheral vascular disease, numerous cancers
- Smoking also increases all blood cell lines, increases platelet reactivity, and fibrinogen.
- Cessation for 3–4 hr results in insignificant hemodynamic side effects of nicotine and improves myocardial O₂ supply/demand.

ASSESSMENT POINTS

<table>
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<tr>
<th>System</th>
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<th>Assessment by Hx</th>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Oral, pharyngeal, head and neck cancers</td>
<td>Exercise tolerance, angina</td>
<td>Two-flight walk</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(see Coronary Artery Disease in Diseases section)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>↑ Heart rate, SVR, coronary vascular resistance</td>
<td>Exercise tolerance, chronic productive cough, character of sputum</td>
<td>Auscultation</td>
<td>CXR if symptomatic Hct, sputum (see COPD)</td>
</tr>
<tr>
<td></td>
<td>→ Myocardial ischemia</td>
<td></td>
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<tr>
<td></td>
<td>→ PVR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>→ Blood viscosity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>↑ COHb, COPD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ FEV/FVC</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>↑ Secretion</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>↓ Clearence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ Airway reactivity</td>
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</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Cessation overnight will decrease COHb and nicotine.
- Cessation for 8 wk will decrease postop pulmonary complications.
- If chronic productive cough, consider preop antibiotic treatment.

Monitoring
- Routine
- SpO₂ monitoring, may read higher SpO₂ than actual if COHb present (SpO₂ = % HbO₂ + % COHb)
- Consider invasive monitoring if symptomatic pulm or cardiac disease.

Airway
- Potential laryngeal hyperreactivity

Premedication/Induction
- Consider deep induction if Hx of reactive airway disease.

Maintenance
- Routine unless symptomatic cardiac or pulm disease
- Avoid light anesthesia and desflurane to reduce potential bronchospasm.

Extubation
- Consider deep extubation if severe reactive airway disease but is easy to intubate and ventilate, with no asporization risk.

Adjuvants
- Routine; smoking increases metabolism of theophylline, decreases half-life from 265 to 180 min

Postoperative Period
- Epidural analgesia may be beneficial in decreasing complications of hypercoagulability, CAD, or COPD.

Etiology
- Habituation

Usual Treatment
- Nicotine patch and clonidine, varenicline, bupropion, Smokers Anonymous, or self-withdrawal

Treatment
- Cessation for a minimum of 12–24 hr. Decrease in COHb and nicotine.
- Cessation for ≥8 wk will reduce postop pulm complications
- Cessation for ≥2 y decreases risk of MI

Anticipated Problems/Concerns
- Long-standing Hx of smoking with symptomatic pulm disease leads to high risk of developing postop pulm complications (pneumonia) due to increased mucus production and decreased ciliary function. Cessation for 8 wk is recommended.
- Airway reactivity significantly increased in smokers; abstinence for 24 hr does not change this reactivity. Reactivity starts reducing after 24–48 hr and reduces to near level of nonsmokers after 10 d of cessation.
- Risk of MI decreases to that of nonsmokers after several years of cessation.
## Cigarette Smoking Cessation

### Risk
- Incidence in USA: Adults ~20% smoke tobacco, higher among lower socioeconomic classes
- Minorities are more likely to smoke and less likely to quit.
- Prevalence among adults and teens declining.

### Perioperative Risks
- Risk not well defined through controlled studies; 25 pack-year Hx increases physiologic age 8 y in those 40–65 y
- Increases perioperative morbidity and mortality related to smoking-associated diseases
- Increases risk of postop lung complications

### Worry About
- Undiagnosed or poorly treated smoking-related disease that may require modification of the anesthetic plan (e.g. CAD, cerebral vascular disease, COPD)
- Propensity for bronchospasm, coughing, and mucus plugging
- Decreased O₂ content 2° to high carboxyhemoglobin (COHb) levels
- Increased autonomic activity (increased heart rate and BP) 2° to nicotine in pts who have smoked just prior to anesthesia

### Perioperative Implications

#### Preoperative Preparation
- Advise smoking cessation for at least 12 h before operation (so that carboxyhemoglobin levels fall to near-normal)
- Advise that a much longer period of cessation (i.e., ~2 mo) is necessary to achieve a decrease in postop pulmonary morbidity; may rarely be worthwhile in true pulmonary cripples undergoing major procedures and very worthwhile for long-term motivation
- Suggest that now is an excellent time to quit smoking (reduce future disease risk, improve post-surgical wound healing, recovery, reduce smoking-related aging)
- Evidence suggests that both the anesthesiologist’s reinforcement and in-hospital tobacco cessation programs consisting of a brief education and counseling visit, self-help take-home materials, and a follow-up phone call are cost-effective in promoting cessation.

#### Monitoring
- Routine
- Most SpO₂ monitors do not distinguish between COHb and oxyhemoglobin. Significant levels of COHb may exist without decrease in SpO₂ reading (obtain ABG with co-oximetry if concern exists)

#### Airway
- Smokers vulnerable to bronchospasm or mucus plugging obstruction anytime
- Children with second-hand smoke exposure may be at increased risk of laryngospasm

#### Induction
- Avoid instrumentation of airway until deep level of anesthesia
- Provide complete preoxygenation since less tolerance of apnea

### Anticipated Problems/Concerns
- Propensity for bronchospasm
- Decreased O₂ content secondary to high carboxyhemoglobin levels

<table>
<thead>
<tr>
<th>ASSESSMENT POINTS</th>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Oral/laryngeal cancer</td>
<td>Hoarseness</td>
<td>Oral exam (and inspection during direct laryngoscopy)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>CAD (± altered LV function)</td>
<td>Exertional chest pain, dyspnea, poor exercise tolerance, orthopnea, paroxysmal nocturnal dyspnea</td>
<td>S, gallop, dysrhythmia</td>
<td>ECG, stress test, ECHO, angiography</td>
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<tr>
<td>RESP</td>
<td>COPD</td>
<td>Dyspnea, poor exercise tolerance</td>
<td>Tachypnea, rales, wheezing, pursed lip breathing</td>
<td>CXR, ABGs</td>
<td></td>
</tr>
<tr>
<td>OTHER</td>
<td>↑ Carboxyhemoglobin (with recent smoking)</td>
<td>Dyspnea</td>
<td>Tachycardia, tachypnea</td>
<td>ABGs with co-oximetry (measure CoHb%)</td>
<td></td>
</tr>
</tbody>
</table>

Risk
- ~1/800 live births
- Racial predominance: Caucasian
- Frequently associated with cleft lip
- Gender predominance: Cleft lip/palate more common in males (2:1); isolated cleft palate more common in females (3:1)

Perioperative Risks
- Morbidity and mortality extremely low; only five life-threatening cases of postop airway obstruction described in literature

Worry About
- Difficult airway when associated with syndromes such as Mohr, Shprintzen, 4P, or Pierre Robin
- Submental obstruction of airway during mask ventilation; tongue obstructs view on direct laryngoscopy
- Laryngospasm on anesthetic induction and airway obstruction due to chronic URI, chronic otitis media, and/or tongue becoming wedged in cleft

Overview
- Congenital condition occurs by 7th to 12th wk of intrauterine life and is multifactorial but can be associated with a single cause such as benzodiazepine usage
- Cleft palate repaired at 12–18 mo; cleft lip is closed at 3 mo, if also present; single to multiple stage methods employed dependent on type of defect(s)
- Usually not associated with severe blood loss

Perioperative Implications
Preoperative Preparation
- Recognize possibility of multiple future procedures and attempt to minimize stress during induction; consider oral premedication

Anesthetic Technique
- GA usually induced via mask using increasing concentrations of volatile agent in O₂
- Oral airway or gauze packing of cleft may help manual ventilation by preventing tongue from lodging in cleft

Monitoring
- Precordial stethoscope
- Maintain normocapnia if epinephrine injection and halothane inhalation

Postoperative Considerations
- Significant risk for airway obstruction due to edema
- Often obligate mouth breathers
- Transfusion usually not required for cleft palate repair
- Judicious use of opioids in a monitored setting; rectal acetaminophen frequently sufficient

Anticipated Problems/Concerns
- Airway difficulty during induction and intubation, esp when associated with other facial anomalies
- Postop airway obstruction due to forgotten pharyngeal pack, severe lingual edema, or obligate mouth breathing

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<tbody>
<tr>
<td>HEENT</td>
<td>Rino. media</td>
<td>Ear pain</td>
<td>Temporomandibular exam</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Clear rhinorrhea</td>
<td>Snore, grunt</td>
<td>Airway exam (micrognathia)</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Associated congenital heart disease</td>
<td>SOB, cyanosis, poor growth</td>
<td>CV exam, club foot</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>URI</td>
<td>Cough, fever</td>
<td>ECG, ECHO</td>
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<tr>
<td></td>
<td>Aspiration</td>
<td>Congestion</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>SOB, cyanosis</td>
<td>Chest exam</td>
<td></td>
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<tr>
<td>GI</td>
<td>Impaired deglutition Malnutrition</td>
<td>Nasal regurgitation Poor growth</td>
<td>CHEST</td>
<td></td>
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<td></td>
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<td></td>
<td>Exam</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td>Malnutrition Club feet</td>
<td>UA, Hgb/Hct</td>
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<td></td>
<td></td>
<td>Pallor</td>
<td></td>
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<tr>
<td>RENAL</td>
<td>Associated congenital defects</td>
<td>UTI</td>
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</tbody>
</table>

Coagulopathy, Factor IX Deficiency

Risk
- Incidence in USA: 3000–4000 (15% of all hemophiliacs). Incidence = 1:25,000–50,000 males
- Race with highest prevalence: None
- Gender with highest prevalence: Overwhelmingly male
- Acquired factor IX deficiency associated with liver disease
- Adult levels may not be reached in healthy newborns until 6 mo of age

Perioperative Risks
- Increased risk of hemorrhagic complications from any and all operations

Worry About
- Excessive and/or uncontrollable hemorrhage
- Tendency for recurrent hemorrhage after initial control
- Expansive deep and soft tissue hematomas

Overview
- Inherited disorder also called hemophilia B or Christmas disease
- Clinically indistinguishable from hemophilia A (classic hemophilia)
- Hemarthroses account for 75% of bleeding episodes; chronic debilitating arthritis is a common development
- Soft tissue hematomas and hematuria also common
- Intracranial hemorrhage is common fatal complication, accounting for death in 25%
- Severity of disease proportional to circulating factor IX activity (<1% normal activity = severe disease, >5% = generally mild disease)

ICD-9-CM Code: 286.1

Perioperative Preparation
- Collaboration with consulting hematologist.
- Schedule surgery early in wk to allow optimal laboratory support of postop assessment of hemostasis; if multiple procedures are contemplated in near future, schedule simultaneously.
- Assess preop factor IX activity; determine goal as guided by magnitude of hemostatic challenge (15–30% factor IX activity for minor lacerations/hematomas; 30–50% for hemarthroses or major hemorrhage, 50–75% for periop coverage or life-threatening bleeding).
- Units factor IX needed = (2)(wt in kg)/(plasma volume in mL/kg)(fractional increase in factor IX activity desired); once-daily dosing is sufficient for maintenance.

Monitoring
- Confirm expected increase in factor IX activity after preop dose but before incision.

Airway
- Laryngoscopy to avoid tissue trauma, consider mask ventilation
- Nasotracheal route best avoided

Maintenance
- Consider tourniquets and local cooling to minimize blood loss

Exubtation
- Avoid coughing on ETT
- Cautious oropharyngeal suction, best done under direct vision

Adjuvants
- Regional anesthesia not absolutely contraindicated but consider with caution; successful brachial plexus block at the axilla has been described.
- Postop factor IX activity requirement: 15–40%

Anticipated Problems/Concerns
- Excessive periop blood loss, hematoma formation
- Potential for delayed or recurrent bleeding after initial control
- Increased likelihood of infectious blood-borne disease (HIV, hepatitis)
Coarctation of the Aorta

**ASSESSMENT POINTS**

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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>Failure to thrive</td>
<td>Poor feeding</td>
<td>Poor growth</td>
<td>Growth chart</td>
</tr>
<tr>
<td>NEURO</td>
<td>Intracranial aneurysm (child and adult)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEENT</td>
<td>Upper body Htn (rare in neonate &lt;5 d old)</td>
<td>Epistaxis</td>
<td>Systolic pressure and pulse gradient between upper and lower extremities (may not be present with PDA)</td>
<td>Four extremity blood pressure measurement</td>
</tr>
<tr>
<td>CVS (General)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVS (neonate/ infant)</td>
<td>CHF</td>
<td>Poor feeding</td>
<td>Tachypnea, cyanosis, hepatomegaly, metabolic acidosis</td>
<td>ABG, CXR</td>
</tr>
<tr>
<td>CVS (child/adult)</td>
<td>Development of collateral circulation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PULM</td>
<td>CHF (neonate, infant)</td>
<td>Resp failure</td>
<td></td>
<td>CXR showing rib notching (a late finding)</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal failure secondary to poor perfusion (neonate, infant)</td>
<td></td>
<td></td>
<td>ABG, CXR</td>
</tr>
<tr>
<td>MS</td>
<td>Poor peripheral perfusion</td>
<td>Claudication, lower extremity pain, paresthesia, muscle weakness</td>
<td>Diminished or absent femoral pulses</td>
<td>Electrolytes, BUN, creatinine, urine output and analysis</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Preparation and Induction**
- Neonate/infant: Maintain PDA with PGE; PDA closure can lead to CHF, upper body Htn, lower body hypoperfusion and shock.
- The presence of a VSD leads to significant left-to-right shunting and a further steal of the systemic blood flow. Do not decrease PVR further by hyperventilation or the use of 100% O2.
- Right lateral decubitus position for left thoracotomy. Good padding important.
- Regular ETT for neonates and infants but consider bronchial blocker or double lumen ETT in older child and adult.

**Monitoring**
- Standard monitors, pulse oximeter x 2 (right upper and either lower extremity), urinary catheter.
- Right upper extremity arterial catheter (radial, ulnar, or axillary). Lower extremity arterial catheter if pressure gradient is high. Otherwise a combination of arterial and NIBP monitoring used in RUE and a lower extremity.
- Central venous access required for infusion of vasoactive medications.
- SSEPs may be used to motor spinal cord perfusion during aortic cross-clamping (particularly if aortic gradient is high or little collateral circulation).

**Maintenance**
- To prevent spinal cord ischemia: Passively cool to core temp 34–35 °C, maintain normocapnia and keep distal mean arterial pressure >40 mmHg.
- Control Htn with titratable agents: Inhalation agent, sodium nitroprusside, esmolol, nicardipine.
- If mean arterial pressure <40 mmHg or significant change in SSEP signal with aortic cross-clamp application, institute left heart bypass.
- Be prepared to treat a sudden drop in BP and acidosis following aortic cross-clamp release with fluids and sodium bicarbonate.

**Postoperative**
- Neonates and infants with CHF remain intubated and ventilated until condition improves.
- Children and adults may usually be extubated in the OR.
- Pain management: Opioids, dexmedetomidine, intercostal nerve block by surgeon, paravertebral catheter, epidural catheter (must consider risk of epidural hematoma).

**Anticipated Problems/Concerns**
- Paraplegia, likely secondary to spinal cord ischemia, particularly if clamp time >30 min
- Postcoarctectomy syndrome: Severe abdominal pain with tenderness, Htn, fever, vomiting, ileus, melena, leukocytosis (occurs 2–3 d postop)
- Pulm Htn in neonates and infants with CoA and VSD (Rx: NO, milrinone)
- Stridor/partial airway obstruction at extubation secondary to recurrent laryngeal nerve injury
- Ventilatory compromise at extubation secondary to phrenic nerve injury causing hemidiaphragmatic paralysis
- Postop bleeding
- Chylothorax from thoracic duct injury
- Recoarctation (late complication)


**Etiology**
- Several theories: Abnormal flow patterns in the developing fetal heart may cause decreased aortic flow resulting in aortic hypoplasia; ectopic ductal tissue in the aorta; or a combination of both.
- May be a component of trisomy 13, trisomy 18, deletion of chr 22q11, Turner syndrome, or Kabuki syndrome.

**Usual Treatment**
- Surgical repair for initial management using several techniques: Subclavian flap aortoplasty, resection and end-to-end anastomosis, prosthetic patch augmentation. Left thoracotomy common; but repair of associated defects may require sternotomy and CPB with or without DHCA.
- Transcatheter balloon angioplasty for initial management of native coarctation and for management of recoarctation. May incl endovascular stent placement.
Complement Deficiency

**Risk**
- C1 esterase inhibitor deficiency incidence 1:50,000 to 1:150,000 of general population
  - Symptoms onset and Dx approx at 20 y, by 30 y approx 98% of pts have symptoms.
- C2 deficiency incidence: <0.1% of general population
  - M/L/F ratio: 1:6
- Higher (6%) in pts with autoimmune disease (see Immune Suppression in Diseases section)
  - Pts with Hx of Neisseria meningitidis have incidence of 15%
- C3 and C5–C8 deficiencies are noted to have increased risk for infections.

**Perioperative Risks**
- Life threatening airway compromise possible
- Increases risk of postop infection, particularly if the deficiency affects the early complement components.
- Risk for inflammatory complications, e.g., glomerulonephritis, vasculitis

**Worry About**
- Acute airway edema resulting from laryngeal or mucous membrane swelling can result in definitive airway obstruction. Abdominal pain from intestinal edema may be an associated finding on exam.
- Increased risk of infection

**Overview**
- Hereditary angioneurotic edema is associated with a complement deficiency of the enzyme C1 esterase inhibitor. This is a rare genetic deficiency that can lead to uncontrolled production of C2, C3, and C5 complement resulting in acute non-inflammatory, painless, non-puritic, non-pitting edema. Initial inciting events are often the result of trauma, but may even be attributed to emotional stress.
- May affect any component of classical pathway, alternate pathway, or terminal common pathway
  - Virtually all deficiencies show some ↑ risk of infection and/or autoimmune disease
- Deficiencies in other complement components, C2 and C3, have also been associated with immunocompromise, resulting in recurrent life-threatening infections due to a variety of organisms.
- Increases risk of autoimmune diseases
- Deficiency in any of the terminal components C5–C8 show selective risk of recurrent Neisseria infections, usually not life-threatening

**ICD-9-CM Code:** 279.8

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>IMMUNO</td>
<td>Infectious risk for all systems</td>
<td>CH50 screening test for complement-mediated lysis of sheep erythrocytes; tests for specific complement components available at reference laboratories. Assess other specific organs as indicated by autoimmune disease (renal for SLE, etc.)</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- In C1 deficiency consideration to preop administration of 2 units of fresh frozen plasma or C1 concentrate should be considered with appropriate consideration to risks and benefits of this therapy.
- Sterile technique strictly observed

**Monitoring**
- Routine
- Coagulation profile
- Minimize invasive lines

**Airway**
- Airway management should minimize trauma. Tracheal intubation is acceptable, but preparations for an emergency tracheostomy should be made. Laryngeal mask airway use should be tempered by the concerns for upper airway edema and resulting ineffective ventilation. Regional anesthesia is an acceptable alternative to prevent airway manipulation.

**Induction**
- Routine

**Maintenance**
- Routine

**Extubation**
- Extubate and remove all lines at earliest opportunity

**Postoperative Period**
- Maintain sterile techniques

**Anticipated Problems/Concerns**
- If an emergency intubation is required, an otolaryngologist is recommended, or surgical personnel, to be present for a possible tracheostomy or cricothyroidotomy.
- Meticulous sterile technique to minimize risk of infection

**Etiology**
- C1 complement results from a heterozygous deficiency of C1 esterase inhibitor. The mediators of the angioedema response result from coagulation, complement, and the kinin pathway. C1 esterase inhibitor is key regulator for Haemagen factor, coagulation, plasmin, and plasma kallikrein. More than 100 mutations on the C1 esterase gene, for pts without hereditary angioedema, 20% of those new mutations with no prior Hx.
- All complement proteins inherited in autosomal fashion, with possible exception of properdin, which appears to be X-linked

**Usual Treatment**
- Stanazolol, danazol, methyltestosterone, oxymetholone, aminocaproic acid, tranexamic acid, and cinnerazine. Mechanism of action for therapeutics being increased synthesis of C1 esterase inhibitor, for the steroids, and inhibition of plasmin activation, for the antifibrinolytics.
- Acute preop prophylaxis consist of fresh frozen plasma and epinephrine. But caution because plasma provides substrates which may aggravate the scenario and worsen the edema. Purified concentrates of C1 esterase inhibitor given IV have been used outside of the USA.
- Antibiotic treatment dictated by specific infection
Congenital Pulmonary Cystic Lesions/Lobar Emphysema

Risk
- Cause of cardiopulmonary compromise
- 10–15% associated with CHD

Perioperative Risks
- May develop worsening of cardiopulmonary status
- Contamination of unaffected lung by infected material from cyst

Worry About
- Associated congenital anomalies
- Tension pneumothorax
- Cardiorespiratory compromise

Overview
- There are two types of congenital pulmonary cystic lesions.
  - Bronchogenic: Abnormal budding and branching of tracheobronchial tree

Perioperative Implications
Preoperative Preparation
- Assess the severity of cardiopulmonary compromise.
- Identify associated congenital anomalies.
- Optimize resp infection if pt is stable.
- Aspirate cyst prior to induction if there is cardiopulmonary compromise or airway obstruction.

Monitoring
- Arterial line for blood pressure monitoring and blood gas analysis.

Induction
- Avoid positive pressure ventilation until thorax is opened to avoid expansion of cyst or lobe.
- Avoid N₂O, which will expand the lobe or cyst.
- Inhalation induction with 100% O₂.
- Intubate without the use of muscle relaxants.

ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE | Test
--- | --- | --- | --- | ---
RESP | ↓ Lung volume | Cyanosis, dyspnea, grunting, coughing | Tachypnea, retractions, wheezing, ↓ BS, asymmetric chest expansion | CXR, CT scan
CV | Mediastinal shift, ↓ CO | Irritability, poor feeding | ↓ Heart sounds | CXR, ECG, ECHO
VSD, PDA | | | | |


Etiology
- Congenital pulmonary cystic lesions may be bronchogenic, alveolar, or a combination of both; anomalous development of bronchopulmonary system.
- Congenital lobar emphysema have extrinsic bronchial obstruction from abnormal vessels or enlarged lymph nodes; intrinsic bronchial obstruction from deficient bronchial cartilage, bronchial stenosis, or redundant bronchial mucosa

Usual Treatment
- Surgical removal

Extubation/Postoperative Period
- May be extubated after uncomplicated surgery and when cardiopulmonary function is adequate

Anticipated Problems/Concern
- Pts with altered cardiopulmonary reserve before surgery may require postop intubation and ventilation.
- If pneumonectomy performed; there will be overinflation of the remaining lung with a decrease in vital capacity. These children may have significant exercise intolerance for a prolonged period after surgery.
- To avoid postop atelectasis, coughing, and early ambulation or increase in activity, important
- Altered pulm mechanics (decreased forced vital capacity and delayed forced expiration) may be present throughout childhood.
Diseases

92

Bronwyn R. Rae

Congenital Methemoglobinemia

Risk
- Navajo Indians, Alaskan Indians, people of Puerto Rican and Cuban ancestry
- Normal life span (except for recessive congenital methemoglobinemia [RCM] type II)

Perioperative Risks
- Oxidizing agents may increase MetHB to dangerous levels
- Pregnancies not compromised

Worry About
- Measurement of SpO2
- Oxidant drugs, e.g., prilocaine, benzocaine, nitroglycerin, sulfonamides, phenacetin, nitric oxide, contraindicated
- Myocardial ischemia due to decreased O2 delivery
- Blood loss due to decreased O2 carrying capacity

Overview
- Enzyme deficiency. Shift of O2 dissociation curve to left leads to mild erythrocytosis. Normal RBC life span.
- Heterozygotes have increased susceptibility to metHb formation after exposure to oxidant drugs and chemicals.
- RCM type I defect restricted to red cell soluble cytochrome b5 reductase only. Cyanosis is sole clinical symptom.
- RCM type II: Defect in all tissues; involves both soluble and microsomal forms of cytochrome b5 reductase. Mental retardation, spasticity, opisthotonos, microcephaly, growth retardation. Death by 2–3 y.
- RCM type III: Nonlythroid enzyme deficiency, but CNS spared.

ICD-9-CM Code: 289.7

Etiology
- RCM types I, II and III: Autosomal recessive inheritance. Due to deficient reducing capacity of oxidized heme due to NADH cytochrome b5 reductase (diaphorase) deficiency.
- HbM variants—autosomal dominant inheritance. Due to structural abnormality in globin moiety: Amino acid substitutions create abnormal environment for heme residues, displacing the equilibrium toward the ferric state.

Usual Treatment
- RCM types I, II and III: Reducing agents, e.g., riboflavin 20–60 mg orally, methylene blue 1 mg/ kg IV. Effect lasts 10–14 d. Ascorbic acid used for chronic management.
- HbM variants: No chronic treatment available. In an emergency, hyperbaric O2 therapy and exchange transfusion may be used.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Look cyanosed but more “blue” than “sick”</td>
<td>15–30% MetHb</td>
</tr>
<tr>
<td>HEME</td>
<td>RCM types I and II: Mild erythrocytosis HbM variants: Mild hemolytic anemia</td>
<td>CBC</td>
</tr>
<tr>
<td>CVS</td>
<td>May be unable to meet increased metabolic demand</td>
<td>ECG</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Can give reducing agents to pts with RCM type I but no data on whether treatment is indicated prior to anesthesia

Monitoring
- Pulse oximeter overestimates at low SpO2 and underestimates at high SpO2. In practice reads between 80–85% regardless of true saturation.
- Use co-oximetry for SaO2 and MetHB levels.
- Monitor ECG for ischemic changes.

Airway
- None

Preinduction/Induction
- Adequate preoxygenation with 100% O2 as O2-carrying capacity is already decreased.

Maintenance
- Prilocaine, benzocaine, EMLA cream contraindicated. Literature contradictory on lidocaine—use with caution.
- Nitrous oxide, propofol, volatile agents OK

Adjuvants
- None

Postoperative Period
- Avoid acetanilids for pain relief; narcotics OK

Anticipated Problems/Concerns
- Avoid oxidant drugs in both homozygotes and heterozygotes
- Pulse oximetry is inaccurate; use ABGs with co-oximetry
- May require supplemental O2 postop
Congestive Heart Failure

Risk
- Heart failure is a syndrome, not a disease
- Incidence in USA: about 4.8 million; 400,000 new cases diagnosed annually. Primary discharge diagnosis in 1 million pts.
- 1-y and 5-y survival rates are 57% and 25% in men and 64% and 38% in women; Median survival after onset is 1.7 y in men and 3.2 y in women

Perioperative Risks
- Heart failure occurs in 1 to 6% of pts after major surgery; between 6 and 25%, in pts with existing cardiac conditions
- EF <35% associated with increased operative risk
- Single greatest risk factor for cardiac surgery. Use congestive heart failure score (CASS): Hx of CHF = 1; Rx digitalis = 1; Rales = 1; Overt symptoms after treatment = 1; Total 0–4; If score = 4, operative risk is 8x greater.

Worry About
- Ventricular dysfunction preop; associated with increased operative mortality
- Pt with diastolic dysfunction may be asymptomatic at rest, but sensitive to increases in heart rate, which may result in flash pulm edema
- Dyshrhythmias due to cardiac ischemia (sudden cardiac death)
- Associated acute or chronic mitral insufficiency
- Volume status
- Prolonged effect of ACE inhibitors

Overview
- Different types of failure (left versus right; acute versus chronic; systolic versus diastolic; low output versus high output)
- Reduced contractility, decreased stroke volume, increased heart rate, hypertrophy and ventricular dilatation
- Acute ischemia can lead to global diastolic dysfunction and CHF
- Papillary muscle ischemia may lead to severe mitral regurgitation and pulm congestion
- New York Heart Association classification: I: no limitation; II: slight limitation; III: marked limitation; IV: inability to carry out any physical activity. Overall 1-y mortality for classes III and IV: 34–58%

ICD-9-CM Code: 428.0

Etiology
- Acquired, acute or chronic: CHD, MI; cardiomyopathy (idiopathic, hypertrophic, hypertrophic obstructive, congestive, alcoholic). Valvular heart disease: Arrhythmias, severe hypertension
- Congenital: Congenital heart disease, left to right shunts; intracardiac (ASD, VSD, aorticoventricular canal), extracardiac (PDA, anomalous pulm venous connection). Obstructive (coarctation of the aorta, aortic stenosis). Complex (Elstein’s anomaly).
- Multiple precipitating causes: Noncompliance with medications (digitalis, diuretics), excessive Na+; excessive IV fluids; drugs (doxorubicin, corticosteroids, disopyramide, nortriptyline, NSAIDs, thiazolidinediones, metformin, cilostazol, PDE-5 inhibitors [sildenafil, vardenafil]) androgens and estrogens). Pulm embolism: High-output states (pregnancy, fever, hyperthyroidism, sepsis, AV fistula, anemia).

Usual Treatment
- Chronic
  - Physical activity encouraged
  - Restriction of sodium intake
- Chronic, well titrated β-blockade may lead to substantial clinical benefit (carvedilol, metoprolol)
- Inhibit renin-angiotensin-aldosterone system (RAAS) (ACE inhibitors, angiotensin receptor blockers, aldosterone inhibitors)
- Improvement in systolic heart failure (digitalis)
- Diuretics (hydrochlorothiazide, furosemide, spironolactone)
- Vasodilators
- Acute
  - Optimize pre- and afterload before starting inotropes and vasodilators
  - Inotropes (dobutamine, epinephrine, milrinone, amrinone)
  - Vasodilators (nitroglycerin, nitroprusside, and nesiritide)
  - Maintenance of beta-blocker therapy in acute exacerbation of systolic heart failure
  - Special measures
    - Stimulation therapy (biventricular pacing + ICD)
    - Surgical correction (CABG, CHD, valvular surgery, cardiomyoplasty, cardiac transplantation)
    - Assist devices (IABP, LV assist, artificial heart)

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Inadequate cardiac output, congestion</td>
<td>Tachycardia, arrhythmias</td>
<td>Peripheral edema</td>
<td>Exercise testing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Facial edema (infants/young children), cardiomegaly, pulus alternans, distended neck veins, Kussmaul’s sign, abdominojugular reflex</td>
<td>ECG, CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm congestion, decreased lung compliance, VC, TLC, pulm diffusion capacity</td>
<td>Breathlessness (exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea)</td>
<td>Rales and wheezes</td>
<td>PFT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Frequent resp infections</td>
<td>Pleural effusions</td>
<td>ABGs</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Expectoration: frothy blood-tined sputum</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Hepatic and intestinal congestion</td>
<td>Nausea, bloating, fullness</td>
<td>Congestive hepatomegaly, ascites, icterus, cachexia</td>
<td>Liver enzymes</td>
</tr>
<tr>
<td>RENAL</td>
<td>Decreased GFR, activation RAAS</td>
<td>Nocturia, oliguria</td>
<td>Ankle edema</td>
<td>BUN/Cr, K+, Na+, proteinuria, specific gravity</td>
</tr>
<tr>
<td>CNS</td>
<td>Hyoperfusion</td>
<td>Confusion, impairment of memory</td>
<td>Mental status exam</td>
<td></td>
</tr>
<tr>
<td>PNS</td>
<td>Increased sympathetic tone</td>
<td>Cool extremities</td>
<td>Peripheral vasoconstriction, pallor, diaphoresis, tachycardia, clubbing</td>
<td></td>
</tr>
</tbody>
</table>

**Maintenance**
- Maintain myocardial contractility, reduce afterload, and normalize PVR

**Extubation**
- May be delayed owing to CV and pulm insufficiencies

**Adjuvants**
- Rx inotropes; digitalis, diuretics
- May be less responsive to catecholamines

- Regional anesthesia debated and not recommended by some (sympathectomy, volume status) or preferred (reduce preload) by others

**Postoperative Period**
- Inotropic support and mechanical assistance may be needed
- Pulm edema develops in 2–16% of pts

**Anticipated Problems/Concerns**
- Pulm edema may necessitate prolonged ventilation with high FIO₂
- RV and/or LV failure in the postop period
Conn’s Syndrome

Risk
- Accounts for 0.05–1% of the population with Htn
- Twice as common in women as in men
- Peak incidence occurs in the third to sixth decades of life
- Morbidity and mortality are primarily related to Htn and hypokalemia

Perioperative risks
- Associated with hypokalemia and chronic Htn if not corrected preop
- Hypokalemic effects from kaliuresis (arrhythmias, muscle weakness, tetany, and alkalosis) and worsening hypokalemia from hyperventilation
- Potentiation of neuromuscular blocking agent effect from hypokalemia
- Longstanding hypertensive effects on the CV system (CAD, CHF)

Overview
- Characterized by increased aldosterone secretion from the adrenal glands, suppressed plasma renin activity (PRA), Htn, and hypokalemia.
- Aldosterone promotes active reabsorption of sodium and excretion of K⁺ through the renal tubules. Water is retained resulting in an increase in extracellular fluid volume of the order of 10–30%, and accounts for occasional refractory Htn. Increase in serum sodium concentration, despite a total body increase, is rarely more than 2–3%, because of the dilutional effect of the retained water. There is also tubular secretion of hydrogen ions and magnesium ions, resulting in a mild degree of metabolic alkalosis.
- Htn, esp if left untreated for many years, can lead to many complications, incl heart disease (e.g., CAD, CHF), and intracerebral hemorrhage (with very high blood pressure).
- Hypokalemia, esp if severe, causes cardiac arrhythmias, which can be fatal.

Etiology
- Present when there is excess secretion of aldosterone from a functional tumor (aldosteronoma) independent of a physical stimulus.
- Most common etiology of primary aldosteronism (50–60% of primary aldosteronism) cases.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Effect</th>
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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>ENDO Abnormal glucose tolerance (↑ Glucose)</td>
<td>Polyuria</td>
<td>PE</td>
<td>Electrolytes</td>
</tr>
<tr>
<td>RENAL ↓ Serum K⁺, total K⁺ body depletion</td>
<td>Nocturia, muscle weakness, and cramps</td>
<td>See MS</td>
<td>Serum and urine electrolytes, Suppressed renin level in untreated pts Aldosterone: Renin level, sodium challenge</td>
</tr>
<tr>
<td>CV Htn</td>
<td>Headache</td>
<td>S4 gallop</td>
<td>ECG, 2-D ECHO</td>
</tr>
<tr>
<td>RESP muscle weakness</td>
<td>Exercise tolerance</td>
<td>Tachypnea</td>
<td></td>
</tr>
<tr>
<td>MS Weakness</td>
<td>Fatigue, muscle weakness</td>
<td>Decreased or absent DTRs</td>
<td>K⁺ (serum electrolytes)</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Treatment of Htn (ideally at least 6 wk of antihypertensive treatment prior to surgery)
- Correction of electrolyte imbalance (specifically K⁺ and magnesium)
- Assessment of cardiac function (ECG, 2-D ECHO)

Intraoperative

Airway
- No change from normal

Induction
- Avoid etomidate, which causes adrenal suppression; hypokalemia may modify responses to nondepolarizing paralytics

Maintenance
- Avoid hyperventilation (this will decrease K⁺); Pts may be sensitive to rapid blood loss if hypovolemic (pre-op diuretic); with bilateral adrenalectomy, may need to replace mineralocorticoids (cortisol), follow electrolytes

Monitoring
- Excessive preop preparation (diuretic) may leave pt hypovolemic. A CVP or Swan-Ganz catheter may be useful in some cases to follow fluid status

Postoperative
- Should continue to monitor acid base status and plasma electrolytes postop

Anticipated Problems/Concerns
- Hypokalemia-induced arrhythmia, hypomagnesemia
- Adrenal insufficiency postop in bilateral adrenalectomy pts
Diseases

Constipation

Risk
- 12% of people worldwide, 17% in the Americas suffer from self-defined constipation
- Incidence in USA: 12–19% (depending on definitions and ascertainment methods)
- Prevalence in elderly 27–50% and as high as 74% in nursing home residents
- Occurs in 20–83% of ICU pts
- M:F ratio: 1:3

Perioperative Risks
- Increased risk of N/V, abd pain, headache
- Delayed weaning from mechanical ventilation in ICU pts
- Delayed discharge of ICU pts

Worry About
- Possible risk of pulm aspiration due to abd distension
- Risk of pseudo-obstruction of the intestine
- Increased PIP, decreases VC and decreased FRC if intra-abd pressure is significant

Overview
- Can cause N/V, abd pain and distension
- Excessive straining may effect cerebral and coronary circulation and lead to syncope
- Severe constipation may lead to fecal impaction, incontinence, and urinary retention
- No evidence that toxins from constipation harm the body
- Idiopathic form, if not complicated, does not usually affect life expectancy

ICD-9-CM Code: 564.00

Etiology
- Primary or idiopathic in most cases (types: functional, slow-transit, and outlet dysfunction)
- Secondary (or combined form): Due to underlying conditions; congenital (e.g., Hirschsprung’s disease) or acquired diseases (e.g., DM, MS, depression). Also, as a result of diet, lifestyle, and the use of certain medications (e.g., opioids, calcium channel blockers, beta-blockers, diuretics, antidepressants, anticonvulsants, antacids, anticholinergics)
- Imbalance of neurotransmitters (serotonin, somatostatin, peptide YY, and vasoactive intestinal peptide) may play a role in idiopathic form of constipation

Usual Treatment
- Lifestyle and diet modifications with increased water intake, which are not always sufficient
- Treatment depends on the etiology and underlying cause
- First line: Bulking agents, increase dietary fiber, increase physical activity, dietary adjustment
- Second line: Stimulant and osmotic laxatives, stool softeners, suppositories, and enemas
- Lactulose and polyethylene glycol are effective in critically ill pts
- Enteral naloxone: For the reversal of opiate-induced constipation in ICU pts
- Surgery is rare; outcomes of colectomy and ileorectostomy in elderly are uncertain

Perioperative Implications

Monitoring
- PIP and other ventilatory parameters if increased intra-abd and intrathoracic pressure
- Electrolytes and/or intravascular volume status, if vigorous bowel preparation preceded anesthesia

Airway
- Vital capacity and FRC may be decreased.

Induction
- Rapid-sequence induction may be indicated if intestinal obstruction is present.

Maintenance
- Avoid using nitrous oxide if there is an obstruction of the intestine.
- Consider regional anesthesia, when feasible, to reduce the use of opioids.

Extubation
- Extubate after the airway reflexes have recovered.

Adjuvants
- Opioids and other μ-opioid agonists (e.g., loperamide) delay GI transit.
- Aspirin and other NSAIDs may insignificantly contribute to constipation in elderly.
- Agents with anticholinergic effect (e.g., atropine, antispasmodics, antipsychotics, TCA) may cause slow transit constipation.

Anticipated Problems/Concerns
- Distention of gut and elevated diaphragm may be present.
- Mechanical ventilation might require higher PIP.
- GI transit may be delayed.
- Discharge from ICU may be delayed if constipation is not managed effectively.

Conversion Disorder

Robert I. Cohen

Risk
- Reported prevalence varies widely (11–500/100,000); may account for as much as 1–14% of general medical/surgical pts
- Reported to be more common in rural populations, developing areas, lower socioeconomic groups, those less medically sophisticated and following physical and sexual abuse

Perioperative Risks
- Hx of conversion disorder may not increase periop morbidity or mortality per se although risk may increase for failure to diagnose if new symptom complexes are too quickly attributed to conversion disorder
- Presence of undiagnosed cognitive, neurologic or general medical illnesses, drug or treatment adverse effect
- Periop appearance of conversion symptoms mimicking medical disturbances, drug effects, or anesthetic or surgically related complications
- Malingering, factitious disorder, dissociative disorder, addiction, pseudoad restitution and withdrawal

Overview
- DSM-IV TR (2000): In conversion disorder, a subclassification of somatiform disorder, a pt generates symptoms suggestive of a medical condition that is not present.
- Following anesthesia, seizures, generalized or focal weakness or sensory loss, trouble with speaking, swallowing, or voiding have serious implica tion that require workup though may also be the presentation of conversion disorder. The amount of medical knowledge held by the pt may predict how closely presenting symptoms mimic known medical conditions and how accurately the symptoms are reproduced on serial evaluation.
- Different from malingering and factitious disorder, the pt is not consciously generating false symptoms. In isolation, neither report of pain nor sexual dysfunction is sufficient to meet criteria.
- Most common in the second through fourth decades, with initial symptom onset lasting up to two wk, according to the DSM-IV-TR, loss of body movement, sight, or speech have better long-term outcome than symptoms of seizure or tremor.
- Coding: In effect at the time of this writing, both DSM-IV-TR (2000) and ICD-9-CM code “conversion disorder” as 300.11. However in DSM-V and ICD-10 drafts, the term conversion is broadened to become synonymous with the dissociative disorders group, of which it is currently a distinct subdivision.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Four subtypes:</td>
<td>Differential diagnosis incl almost any medical condition (e.g., myasthenia gravis, MS, porphyria, diabetic neuropathy, hyperparathyroidism, tumors, idiopathic or substance-abuse dystonias)</td>
<td>Findings may not conform to known anatomic pathways or physiologic mechanisms, symptoms may be inconsistent, (e.g., unacknowledged strength in antagonistic muscles; normal muscle tone, intact reflexes; equal difficulty swallowing solids and liquids; paralyzed extremity moves on own with dressing; arm held over patient’s head by examiner and dropped will not fall on head; stocking-glove anesthesia without proximal to distal gradient; equal loss of touch, temperature, and pain at sharply demarcated anatomic landmarks rather than dermatomes)</td>
<td>Absence of expected findings (including EEG, EMG, lumbar puncture, CT, MRI, SPECT scan, nerve conduction velocity, drug screen) suggest and confirm diagnosis</td>
</tr>
<tr>
<td>GENDER</td>
<td>Gender tendencies:</td>
<td>Men—antisocial personality, work-related or military injury</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Perioperative Preparation
- Careful Hx and PE, carefully documenting normal function as well as any pre-existing neurologic deficits
- Confer with treating providers, (e.g., internist, neurologist, psychiatrist/psychotherapist)
- Consider possibility that reason for surgery in pt with multiple procedures may involve conversion symptom

Premedication/Induction/Maintenance
- Attempt to treat reported pain in holding area prior to titrating anxiolytic
- Regional anesthesia not contraindicated

Extubation
- None

Adjuvants
- Pt to take usual dose of psychiatric medications preop

Postoperative Period
- Consider conversion disorder when thorough work-up of medical condition does not explain symptoms, esp if a prior trauma or unresolved neurotic stressors can be readily identified.

Etiology
- While unknown, symptoms may occur as an unconscious solution to trauma or unresolved neurotic conflict.
- More common in pts with prior medical and psychiatric diagnoses
- Possible genetic predisposition suggested in twin and familial studies

Treatment
- Confirm Dx with psychiatric consultant while excluding possible medical conditions
- Reassure pt and family members that symptoms do not appear to represent a life threatening condition and that investigation and treatment will continue.
- Optimize treatment of co-existing psychiatric (esp anxiety and depression) and medical conditions
- Conversion disorder may respond to behavioral, psychodynamic therapy, or psychoanalysis and may also respond to psychopharmacologic treatment of co-morbid anxiety and depression.
- There is no specific psychopharmacologic intervention for conversion disorder. ECT is not indicated unless used to treat a co-morbid condition.

Anticipated Problems/Concerns
- As conversion disorder is more common among pts with other psychiatric and medical diseases, clear documentation of these during the preop clinic may prove of immeasurable value to the treating anesthesiologist in the postop period when new symptom complexes are reported and conversion disorder is considered within the differential diagnosis.
Cor Pulmonale

Risk
- Third most common cardiac Dx after age 50 y
- 10–20% of all CHF admissions have some aspect of right heart failure
- Gender predominance: Male > female

Perioperative Risks
- Increased risk for resp failure, severe right heart failure (≥10% if cor pulmonale Dx made preop)
- Risk of prolonged postop ventilatory support

Worry About
- Increased pulm vascular resistance (PVR) may cause systemic hypotension
- Hypoxia, hypoxiaemia, hypercarbia, and acidosis intraop or in early postop period, which increases PVR
- Underlying CAD, LV dysfunction

Overview
- Alteration in RV structure (hypertrophy) and function

ASSESSMENT POINTS

<table>
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<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>RV failure ↑ PVR Tricuspid regurgitation</td>
<td>DOE Effort-related syncope Chest pain</td>
<td>Accented pulm S, Diastolic or systolic murmur Dependent edema</td>
<td>CXR ECHO Right heart catheterization</td>
</tr>
<tr>
<td>RESP</td>
<td>COPD</td>
<td>DOE Chronic cough, sputum</td>
<td>Hyperinflated lungs Wheezing, rhonchi</td>
<td>CXR PFTs</td>
</tr>
<tr>
<td>GI</td>
<td>Passive congestion of liver, spleen</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Impaired ability to excrete Na+, H2O</td>
<td>Edema</td>
<td>Edema</td>
<td>Urinary Osm</td>
</tr>
<tr>
<td>CNS</td>
<td>Stimulation of sympathetic nervous system 2° to hypoxia</td>
<td></td>
<td>Tachycardia</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Mortality risk in pts with primary pulm Htn and cor pulmonale high (7%)
- Treat underlying infections
- Maximize treatment of reversible airway disease
- Avoid preop medications that will depress ventilation
- Consider baseline ABG to assess PaO2, PaCO2

Monitoring
- Consider arterial line for beat-beat arterial pressure monitoring, noninvasive cardiac output measurement and ABG collection
- Consider intraop TEE to monitor RV function, RV dilatation
- Consider pulm arterial catheter to monitor PA pressures, CVP monitoring for evaluation of RV function for large fluid shift reoperations

Airway
- Potential for bronchospasm

Induction
- Try to increase SVR in face of fixed increased PVR

- Common causes: Pulm embolic events resulting in RV outflow obstruction and COPD resulting in increased PVR (2° to chronic hypoxia and structural changes)
- Any disease that increases PVR chronically can induce RV changes, incl idiopathic and toxin-induced pulm Htn, pulm fibrosis, severe obstructive sleep apnea, congenital heart disease (CHD) with chronic RV overload or RV outflow obstruction
- Prognosis: Favorable for those who can maintain a near-normal Pao2; unfavorable for those with structural changes

ICD-9-CM Code: 416.9

Ecology
- COPD: Smoking or severe asthma
- Longstanding untreated OSA
- Left ventricular heart failure
- Acute or chronic pulm embolus
- CHF with RV volume overload (L>R shunt, long-standing pulmonic insufficiency) or afterload increase (pulm outflow obstruction)
- Primary pulm Htn or severe pulm fibrosis

Usual Treatment
- Decreasing PVR toward normal levels by increasing Pao2 to 60 mmHg (beware of depression of hypoxin drive to breathe; may have desensitized hypercarbic drive to breathe 2° to chronic increasing Pao2), giving diuretics, digoxin to relieve symptoms of CHF (Caution: Diuretics may increase Hct by hemoconcentration; if Hct already increased 2° to decreasing Pao2, this may further increase the viscosity of blood, increasing risk for sludging and microemboli)
- Vasodilators (only ½ of pts improve); inhaled nitric oxide or iloprost (stable prostacyclin analogue); phosphodiesterase-5 inhibitors (such as sildenafil) have shown promise; other vasodilators, such as calcium-channel blockers, have been tried; use caution because may decrease SVR in face of fixed increasing PVR, causing severe systemic hypotension (unable to increase CO); antibiotics for prompt treatment of infection

Extubation
- Bronchospasm may occur during emergence
- Avoid hypoventilation and resultant hypercarbia

Adjuvants
- Regional anesthesia an option, but high level may decrease SVR in face of increased PVR leading to CV collapse
- Inhaled nitric oxide or iloprost increasing in use
- Preop phosphodiesterase-5 inhibitors may be used routinely in future; may ameliorate effects of intraop vasodilators

Postoperative Period
- Postop pain management with either low-dose epidural local anesthetics with low-dose opioids or low-dose intrathecal opioids can minimize resp depression

Anticipated Problems/Concerns
- Increased PVR and RV dysfunction from hypoxia/hypercarbia or hypothermia
Coronary Artery Disease (Left Main and Non-Left Main Disease)

Risk
- Incidence in USA: 16.8 million
- ~1.5 million pts per year with CAD will have an acute MI; one third of these will die
- CAD responsible for ~ one of every five deaths in USA
- Male predominance <55 y, M = F >55 y
- Risk factors: Htn, diabetes, smoking, familial incidence, hyperlipidemia, and high cholesterol

Perioperative Risks
- Presence of disease by coronary anatomy is good predictor of survival with CAD
- Presence of left main disease with high degree of stenosis is life-threatening
- Recent MI increases risk, but revascularization interventions protect pt
- Impaired ventricular function, unstable anginal pattern, major surgery, and emergency surgery increases risk
- Increased risk if reoperation for bypass surgery
- Presence of a bare metal stent or drug eluting stent places pt at risk for MI secondary to in stent thrombosis (esp <3 mo after bare metal stent and <12 mo after drug eluting stent)

Worry About
- Myocardial ischemia can lead to MI
- Postop MI carries very high mortality (>50%) in noncardiac surgical pts

Perioperative Implications
Preoperative Preparation
- Supportive prep interview to decrease stress and anxiety
- Consider analgesic (opioid) if pain or likelihood of pain prior to anesthesia
- Give morning cardiac medications, esp β-blockers, antiplatelets if pt has intracoronary stent

ASSESSMENT POINTS

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Noncardiac Surgery</td>
<td>Ischemia</td>
<td>Causes ventricular dysfunction and arrhythmias Can herald and/or cause MI</td>
<td>Angina Dyspnea on exertion</td>
<td>Holter monitor, ECG exercise radionuclide, treadmill stress ECHO</td>
</tr>
<tr>
<td>Infarction</td>
<td>Indicates severe CAD Causes death</td>
<td>Unstable angina</td>
<td>ECG, CK-MB and troponin enzyme release</td>
<td></td>
</tr>
<tr>
<td>Impaired function</td>
<td>Heart failure, shock</td>
<td>Activity Hx Stair climbing Orthopnea</td>
<td>Orthopnea gallop Rales Peripheral edema</td>
<td>Ejection fraction (cath, ECHO, radionuclide)</td>
</tr>
<tr>
<td>Stent thrombosis</td>
<td>Cardiogenic shock, death ↑ risk if bare metal stent implanted &lt;3 mo or drug eluting stent &lt;12 mo</td>
<td>Antiplatelet regimen Type of stent (bare metal vs. drug eluting) Stent(s) location and date implanted</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Cardiac Surgery
Cardiac function Best predictor of outcome Activity Hx, stair climbing Ventricular angiogram (EF >50% = good risk)
Coronary anatomy Extent of disease and overall long-term survival Coronary angiography
Renal function ↑ Risk if impaired Cr ≥1.4 mg/dL denotes ↑ risk
CNS ↑ Risk of stroke Aortic atherosomatic disease and prior stroke increase risk Hx of TIA Amaurosis fugax Carotid bruit Carotid Doppler study, epiaortic ultrasound

Overview
- Atherosclerosis of vessels supplying blood to heart results in ↓ blood flow by limitation of flow due to anatomy or due to vasoactive dysfunction (spasm, etc.)
- Single greatest cause of death in USA population (~500,000 deaths/y)
- Most prevalent form of CV disease: >16.8 million in USA population has CAD
- Leading cause of death in major noncardiac surgery

ICD-9-CM Code: 414.0
See also Angina, Chronic Stable, in Diseases section

Etiology
- Atherosclerosis and obstructive deposits in coronary artery
- Interaction of genetics, diet, and environment: Htn, cigarette smoking, and diabetes are three common predisposing factors
- Myocardial O₂ delivery does not meet myocardial O₂ demands: causes myocardial ischemia
- Myocardial O₂ supply does not reach myocardium after thrombosis of coronary artery: causes MI

Usual Treatment
- Medical: Nitroglycerin, β-blockers, calcium-channel blockers (low dose and in vasospastic component), diet, antihyperlipidemia drugs, antiplatelet therapy, exercise, wt loss, antioxidants
- Catheter-based interventional cardiology (indicated in ≤2-vessel CAD: PTCA has 30% 3-mo closure rate), intracoronary stent (has good angiographic result and lower closure rate, but event-free survival is little different from PTCA)
- CABG surgery (indicated in ≥2-vessel CAD, left main disease, diabetics)
- Coronary revascularization is indicated in pts with stable angina before noncardiac surgery in left main disease, 3-vessel disease and in pts with high-risk unstable angina
- If possible, delay surgery for 3 mo after bare metal stent implantation and 12 mo after drug eluting stent implantation. During the periop period, continue antiplatelet therapy if possible.
• Decrease HR, contractility, and wall tension (O2 consumption)
• No outcome difference demonstrated among general anesthetics
• Regional and conduction anesthesia with postop analgesia may be beneficial
• Transient periods of Htn are well tolerated; prolonged periods of hypotension, tachycardia, and anemia are not well tolerated

**Adjuvants**
• Nitroglycerin, sublingual or (preferably) by continuous infusion (0.5–2.0 μg/kg/min), can treat myocardial ischemia.
• β-blockers by bolus or infusion decrease HR and myocardial contractility and can prevent and treat ischemia
• RBCs to maintain Hct ≥ 28%

**Postoperative Period**
• Second and third postop days are most common time for MI in noncardiac surgical pts; ischemia intraop, designate as high risk in postop period
• Maintain good analgesia to decrease stress response
• Maintain cardiac medications (esp β-blockers)
• Consider use of aspirin or other medications to decrease coronary thrombosis in high-risk non-cardiac surgical pts (esp in pts with intracoronary stents)
Coronary Artery Spasm (CAS)

**Risk**
- Mostly disease of middle and old-aged men and post-menopausal women
- Gender difference: Higher incidence in women
- Periop CAS is prevalent in elderly male pts with coronary risk factors
- Teenagers and young adults with illicit substance abuse, primarily cocaine
- Occurs in 1% to 5% of percutaneous coronary interventions
- Ethnic differences: Higher frequency in eastern populations
- Type A behavior pattern, severe anxiety, and panic disorder
- Age, smoking, high sensitivity C-reactive protein (marker of inflammation)

**Perioperative Risks**
- Change of sympathetic activity may trigger CAS
- CAS can lead to myocardial ischemia
  - Chest pain, ischemic ST segment changes on ECG
  - May result or associated with myocardial infarction
  - Coronary thrombosis may trigger CAS, leading to acute MI, unstable angina, or ischemic sudden death

**Worry About**
- Cardiogenic shock: Decreased LV and RV compliance, decreased pump function

**Overview**
- Classical CAS (Prinzmetal, variant, or spastic angina)
  - Diagnosed by severe chest pain, usually at rest, with concurrent ST segment elevation on ECG
  - Characterized by spasm of normal coronary arteries on arteriography
- Other forms of CAS
  - Without chest pain silent angina, diagnosed with Holter monitoring
  - CAS can be associated with ischemic heart disease and myocardial infarction
  - Effort angina, unstable angina, microvascular angina (female prevalence)
  - ECG changes may incl either ST segment elevation, ST depression or T wave abnormalities
  - Coronary arteriography can demonstrate normal or diseased coronary arteries

**ICD9-CM: 414.0 (Coronary atherosclerosis)**

**ASSESSMENT POINTS**

<table>
<thead>
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<tbody>
<tr>
<td>GENERAL</td>
<td>Risk factor search: Smoking, illicit drug use, esp cocaine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Chest pain, myocardial ischemia, cardiogenic shock, ischemic sudden death, arrhythmias</td>
<td>Chest pain at rest or exertion, Hx of rapid heart rate, Hx of syncope</td>
<td>Palpitations, cold sweat, nausea, vomiting, syncope, hypotension</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ECG, ST segment analysis, Holter, exercise testing</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Coronary arteriography, TEE or TTE—wall motion abnormalities, cardiac biomarkers</td>
<td></td>
</tr>
</tbody>
</table>


**Etiology**
- The exact mechanism of coronary artery spasm is unknown. Several contributing factors thought to play a role:
  - Change in sympathetic activity
  - Vagal withdrawal
  - Coronary thrombosis
  - Endothelial dysfunction
  - Increased Ca\(^{2+}\) sensitivity
  - Reduced endothelial NO activity
  - eNOS gene polymorphism
  - Signs of chronic low grade inflammation
  - Oxidative stress

**Usual treatment**
- Cessation of smoking
- Calcium-channel blockers (primary)
- Long-acting nitrates (short when symptomatic)
- β-blockers (when associated with fixed lesions)
- Magnesium supplementation (may have a preventative effect)
- Statin therapy (improving endothelial function)
- Coronary angioplasty (medically intractable)
- Coronary artery bypass surgery (medically intractable)
- Automatic defibrillator implantation (life-threatening arrhythmias)

**Perioperative Implications**

**Preoperative Preparation**
- Continue treatment medication until the morning of surgery
- IV nitroglycerin, nicardipine, β-blockers available
- Have a plan for postop pain control
- Consider regional or neuraxial techniques

**Monitoring**
- 2 lead (II. and V5) ECG & ST segment analysis
- Consider arterial line

**Airway**
- Blunt intubation reflexes, avoid sympathetic surcharge on intubation

**Preinduction/Induction**
- Cardio-stable induction
- Avoidance of hypotension and tachycardia

**Maintenance**
- Heart rate and BP control (maintain adequate diastolic BP)
- Avoid hypothermia
- Maintain Hct
- Optimize supply/demand

**Exubation**
- Smooth opioid wake up and extubation
- Heart rate and BP control
- Avoidance of hypercapnia and hypoxemia

**Postoperative Period**
- Adequate pain control
- Heart rate and BP control
- Treatment of shivering

**Adjuvants**
- Careful ST segment monitoring throughout periop period

**Immediate recognition and treatment of coronary ischemia by optimizing supply and demand, special attention to adequate diastolic blood pressure**

**Anticipated Problems/Concerns**
- Anticipate potentially life-threatening arrhythmias
- Myocardial ischemia or infarction, LV and RV dysfunction
- Place defibrillator pads for high-risk pts
### Craniosynostosis

**Risk**
- May be simple (non-syndromic) or associated with a syndrome
- 1/3–5000 live births for simple craniosynostosis
- 80% nonsyndromic, 20% syndromic
- Sutural involvement (decreasing incidence): Sagittal, coronal, metopic, lambdoid
- Prenatal or perinatal in onset, rarely later
- Majority of mutations found in fibroblast growth factor receptor genes

**Perioperative Risk**
- Difficult airway
- Blood loss (>1/2 blood volume)
- Venous air embolism (micro emboli common)
- Increased intracranial pressure (ICP), (all pts should have eye exam for papilledema)
- Associated cardiac anomalies

**Worry About**
- Difficult mask ventilation and intubation
- Management of intraop blood loss

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</thead>
<tbody>
<tr>
<td>Airway</td>
<td>Diff mask vent and/or intubation</td>
<td>Hx or previous mask ventilation or intubation</td>
<td>Facial symmetry Size of mandible Neck range of motion</td>
<td>Neck films may be indicated (Apert may have cervical fusion)</td>
</tr>
<tr>
<td>RESP</td>
<td>OSA</td>
<td>Apnea during sleep, snoring</td>
<td>Noisy breathing from upper airway</td>
<td>Polysomnogram (apnea-hypopnea index) Overnight pulse oximetry Room air O₂ saturation</td>
</tr>
<tr>
<td>CARDIAC</td>
<td>CHD (ASD, VSD, tetralogy of Fallot)</td>
<td>Bottle feeds &gt; 30 min Diaphoresis with feeds Failure to thrive</td>
<td>Murmur</td>
<td>ECHO</td>
</tr>
<tr>
<td>MS</td>
<td>Diff IV access Diff a-line access</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Increased ICP</td>
<td>Irritable, vomiting, somnolence</td>
<td>Papilledema</td>
<td>Ophthalmology exam</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia (nadir at 3 mo of age)</td>
<td></td>
<td></td>
<td>Preop Hct, type and cross</td>
</tr>
</tbody>
</table>


### Perioperative implications

**Preoperative implications**
- Determine whether surgery for cosmetic reasons related to isolated defect or for elevated ICP
- Determine if there is an associated syndrome
- Prepare for possible difficult airway if syndromic
- Prepared for significant blood loss with IV access, type and cross, and preparation of blood products accordingly

**Monitoring**
- At least two large (20–22G) peripheral IVs should be placed. *Endoscopic* surgery for single suture may have less blood loss, but it can still be significant.
- Multiple suture repairs require an open approach. Expect significant (≥3–1 blood volume) blood loss. Will need 2–3 large bore IVs, arterial line for pressure monitoring and frequent lab draws. Central venous access may be needed for IV access and for CVP monitoring. Precordial Doppler monitoring should be considered in all pts.
- Airway
  - Have multiple airway devices (primarily LMA) available for multimodal airway management. May need to perform an asleep fiberoptic intubation.
  - Consider awake fiberoptic as a possible choice for intubation.
  - Depending on airway concerns and Hx, have surgeon available for possible tracheostomy.

**Positioning**
- May be supine or prone depending on suture involved
- If prone, be careful to avoid eye pressure (esp if proptosis is present).

**Induction**
- Standard inhalational induction is appropriate for most pts.
- An inhalation induction is often appropriate in the syndromic pt with potentially difficult airway because of lack of cooperation
- If increased ICP with symptoms, may need to do IV induction and avoid ketamine.

**Maintenance**
- General inhalational or IV maintenance with muscle relaxant
- Large fluid shifts can occur due to blood loss and massive transfusion.
- Need for frequent monitoring of blood gases, electrolytes, coagulation, and positioning
- Forced air warmer required due to length of case. IVF warmer should be used.
- Mannitol may be required prior to calvarial removal if increased ICP.
- Isotonic IVF should be used during the intraop period.

**Extubation**
- The majority of pts can be extubated at the end of the surgery.
- Place nasal pharyngeal airway prior to extubation.
- Have airway equipment available during extubation.

**Position**
- Difficult airway
- Massive blood loss and transfusion
- Extended length of surgery
- Possible postop intubation

**Etiology**
- Can be involved in more than 150 different syndromes
- Most often occurs by sporadic gene mutation (fibroblast growth factor receptor, TWIST), however can be inherited in autosomal recessive or dominant pattern

**Usual Treatment**
- Surgery is typically indicated for cosmetic reasons or increased ICP
- Results are best when surgery performed prior to 12 mo of age
- Important to consider associated syndromes prior to surgery
- Team usually incl plastic surgeon, neurosurgeon, and anesthesiologist
**CREST Syndrome**

**Risk**
- Pts with Hx of exposure to silica dust or PVC
- Usual age group is of 30 to 50 y
- 4- to 9-fold higher in women than men, seen in all races
- In USA, systemic sclerosis has an estimated incidence of 19 cases per million and prevalence of 240 cases per million population (range 138 to 286)

**Perioperative Risk**
- Pts more likely to have compromised renal function at baseline
- Hypoxia from pulm Htn and/or restrictive lung disease
- Difficult intubation from narrow mouth opening

**Worry About**
- Reflux and thereby aspiration, renal crises, restrictive lung disease, CHF, pulm Htn, difficult intubation due to small mouth opening, keeping pt warm to avoid Raynaud’s

**Overview**
- Calcinosi s, Raynaud’s phenomenon, Esophageal dysmotility, Sclerodactyly and Telangiectasia (CREST)
- Symptoms involved in CREST or limited cutaneous systemic sclerosis are associated with the generalized form of systemic sclerosis

**ICD-9-CM Code 710.1 (Scleroderma)**

**Etiology**
- Exact etiology of systemic sclerosis is unknown; following pathogenic factors are always present: endothelial cell injury, fibroblast activation, cellular and humoral immunologic derangement


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<tbody>
<tr>
<td>SKIN and HEENT</td>
<td>Sclerodactyly, few wrinkles or joint creases, decreased range of motion, hair loss, puritus telangiectases</td>
<td>Observation</td>
<td>Tightness, indurations, hyper or hypopigmentation</td>
<td>Airway examination</td>
</tr>
<tr>
<td>CVS</td>
<td>Pericardial effusion, CHF, myocardial fibrosis, misconduction</td>
<td>Dyspnea, palpitation, irregular heart rate, chest pain from vasospasm</td>
<td>Rales and murmurs on auscultation</td>
<td>EKG, Holter monitoring, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm Htn, aspiration pneumonia, dyspnea</td>
<td>SOB, cough, tachypnea, dec exercise tolerance</td>
<td>Dry rales</td>
<td>PFT, ABG, CXR, DLCO, HRCT</td>
</tr>
<tr>
<td>CNS</td>
<td>Carpal tunnel syndrome, trigeminal neuralgia (rare), entrapment neuropathies</td>
<td>Pain over wrist, other typical signs depending on nerve involved</td>
<td>Limited ROM</td>
<td>Conduction studies, CT</td>
</tr>
<tr>
<td>RENAL</td>
<td>Htn, oliguria</td>
<td>Headache, SOB, edema</td>
<td>Swelling of hands and feet</td>
<td>Check BP, UO &amp; monitor serum creatinine</td>
</tr>
<tr>
<td>MS</td>
<td>Raynaud’s phenomenon, arthralgias, myalgias, morning stiffness</td>
<td>Acroosteolysis, muscle weakness</td>
<td>Palpable tendon friction rubs, muscle wasting, flexion contractures</td>
<td>Increased serum CK and aldolase</td>
</tr>
<tr>
<td>GASTRO</td>
<td>GE reflux, esophagitis, esophageal strictures, watermelon stomach, primary biliary cirrhosis, colonic diverticula, anal sphincter incompetence</td>
<td>Bitter taste, dysphagia, retrosternal and abdominal pain, diarrhea, self-soiling</td>
<td>Abdominal tenderness, decreased rectal sphincter tone</td>
<td>Barium swallow CT or MRI, endoscopy Abdominal ultrasound, anti-mitochondrial antibodies for PBC</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Preparation**
- Continue PPI, consider FOI, evaluate for regional anesthetic techniques for pulm issues

**Monitoring**
- If co-morbidities dictate A-line, (try to avoid due to Raynaud’s, but difficult to get cuff pressure due to reduced flow, may need ABG)
- CVP ± PA catheter if pulm Htn, along with standard monitoring

**Airway**
- Airway may be a challenge due to small oral opening

**Preinduction/Induction**
- Worry about hypotension and hypoxemia at induction

**Maintenance**
- Choose drugs based on hemodynamic status
- Keep warm

**Exubation**
- May have to be delayed if significant pulm compromise

**Adjuvants**
- In the presence of compromised renal, cardiac, or pulm function modify anesthetic drugs accordingly

**Anticipated Problems/Concerns**
- Challenging airway, hypoxemia, CHF, renal function, and positioning challenges with contractures
# Cri Du Chat Syndrome (5p Syndrome)

## Risk
- 1:15,000 to 1: 50,000 births

## Perioperative Risks
- Difficult airway management
- CHD—30%
- Aspiration risk

## Worry About
- Difficult mask ventilation—airway obstruction secondary to hypotonia
- Difficult intubation
- Temp regulation
- Mental retardation

## ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Micrognathia</td>
<td>Resp distress in neonatal period; inspiratory stridor</td>
<td>Receding mandible</td>
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<tr>
<td>CV</td>
<td>ASD, VSD, PDA, PS</td>
<td>SOB, cyanosis</td>
<td>Murmur, gallop</td>
<td>ECHO</td>
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<tr>
<td>RESP/GI</td>
<td>Pneumonia, chronic aspiration</td>
<td>Dyspnea, rales, rhonchi, wheezing</td>
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<td>CXR</td>
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<tr>
<td>ORTHO</td>
<td>Scoliosis</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Mental retardation, seizures</td>
<td>Hypotonia in infancy, hypertonia later</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## Key Reference:

## Perioperative Implications

### Preoperative Preparation
- Difficult airway management

### Monitoring
- Routine
- Pay particular attention to temp and neuromuscular blockade

### Airway
- Laryngeal mask airway available
- Fiberoptic bronchoscope

### Overview
- Microcephaly with profound mental retardation and hypotonia
- Characteristic facies with micrognathia, low-set ears, facial asymmetry
- Characteristic high shrill cry may be due to laryngeal abnormality (narrow diamond-shaped larynx, long, floppy epiglottis) or neurogenic defect
- CHD, 30% ASD, VSD, PDA, or pulmonic stenosis

### Etiology
- Partial or total deletion of the short arm of chromosome no. 5
- Loss of the critical 5p15.2 region is responsible for most of the features.
- Most cases occur by spontaneous gene mutation (90%).
- 10% arise by unbalanced translocations.

### ICD-9-CM Code: 758.31
Crohn’s Disease

Overview
• Chronic inflammatory disease of the GI tract that can give rise to strictures, inflammatory masses, fistulas, abcesses, and hemorrhage.
• Pt may develop bowel obstruction and perforation.
• Pt may develop rectocutaneous fistulas, rectal fissures, and perirectal abcesses.
• Pt may have anemia from several causes, chronic disease, chronic blood loss, folate and vitamin B12 deficiency.
• Chronic malnutrition and weight loss.
• Extraintestinal manifestations occur in a minority of pts. These manifestations include uveitis and episcleritis, erythema nodosum and pyoderma gangrenosum, ankylosing spondylitis, and primary sclerosing cholangitis. When present, their symptoms can be more serious than the primary intestinal disease.

ICD-9-CM Code: 555.9 (Regional enteritis of unspecified site)

ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hypovolemia</td>
<td>Bowel prep, wt loss, diarrhea</td>
<td>Hypotension, tachycardia</td>
<td>Electrolytes, Hct</td>
</tr>
<tr>
<td>GI</td>
<td>Bowel perforation</td>
<td>Abdominal pain</td>
<td>Abdominal tenderness, fever</td>
<td>WBC</td>
</tr>
<tr>
<td></td>
<td>Malabsorption</td>
<td>Diarrhea, wt loss</td>
<td>Cachexia</td>
<td>Albumin</td>
</tr>
<tr>
<td>MS</td>
<td>Ankylosing arthritis</td>
<td>Joint mobility</td>
<td>Decreased ROM of joints</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
• Ensure volume status and electrolytes are normalized
• If pt is on hyperalimentation preop, continue it during the case; monitor glucose
• Assess current or recent steroid use and need for periop supplementation
• Discontinue methotrexate at least 2 wk before surgery as it has been shown to decrease wound healing
• Pts with significant anemia should be transfused preop

Monitoring
• Routine
• Consider CVL if pt has difficult IV access
• Consider arterial line if significant co-morbidities

Airway
• Aspiration risk if bowel obstruction present

Induction
• Rapid sequence induction in pts with gastric outlet or bowel obstruction
• Consider preinduction placement of NG tube to suction gastric contents

Maintenance
• Avoid nitrous oxide if bowel obstruction present
• Abdominal relaxation with non-depolarizing muscle relaxants usually needed. If liver disease present, avoid muscle relaxants dependent on hepatic metabolism
• Check glucose regularly if on hyperalimentation

Extubation
• Awake extubation

Postoperative Period
• Consider epidural analgesia or IV PCA for pain control
• Monitor fluid status carefully in the postop period

Anticipated Problems/Concerns
• May need aggressive fluid replacement due to hypovolemia and anemia worsened by third space losses
• May have severe nutritional deficiency, esp with short bowel syndrome from extensive resection

Risk
• Incidence of 4 cases per 100,000/y, prevalence of 80–150 per 100,000
• Race: Caucasians > African-Americans > Hispanics and Asians
• Three to four times more common in ethnic Jews than non-Jewish whites
• Peak occurrence between ages 15 and 25 y, with a second smaller peak between ages 60-80 y

Perioperative Risks
• Aspiration
• Arrhythmias due to electrolyte disorders

Worry About
• Intravascular fluid volume and electrolyte imbalances
• Chronic steroid use and need for periop supplementation
• Nutritional status, chronic weight loss, and malnutrition
• Difficult IV access due to chronic illness and frequent venipunctures
• Psychological mindset of the pt due to chronicity of the disease and relatively young age of the pts

Etiology
• Unknown
• Theories incl response to an infectious agent, defective mucosal barrier allowing exposure to antigens
• Smoking is a risk factor

Usual Treatment
• Pharmacologic: Aminosalicylates, steroids, immunomodulating agents such as azathioprine and cyclosporine, infliximab—a monoclonal anti-tumor necrosis factor-α antibody given in an effort to decrease inflammation
• Surgical: Indications for surgery are intractability, intestinal obstruction, intra-abdominal abscess, fistulas, fulminant colitis, toxic megacolon, massive hemorrhage, cancer and growth, retardation
• Both medical and surgical management of Crohn’s disease are aimed at providing long lasting symptomatic relief while avoiding excessive morbidity
Croup (Laryngotracheoobronchitis)

**Diseases**

Maurice S. Zwass

**Overview**

- Common childhood ailment with prodromal illness accompanied by a characteristic cough (often sounds like seal barking)
- Sx and resp compromise from progressive swelling of subglottic region tracheal mucosa
- Frequently present when inspiratory stridor and resp distress develop
- Radiographs of neck often demonstrate gradual progressive tracheal narrowing, most narrow just below level of vocal cords (referred to as steeple sign); upper glottis on lateral neck radiograph is normal
- When obtained, evaluation of CBC is consistent with viral illness

**ICD-9-CM Codes:** 464.4 (Croup); 464.2 (Laryngotracheitis)

**Etiology**

- Viral agents are usual etiologies and include parainfluenza viruses (most common); adenoviruses, influenza virus, resp syncytial virus (RSV), and measles virus also associated

**Usual Treatment**

- Cool mist often greatly improves Sx; supplemental \( \text{O}_2 \)
- If symptoms more severe, aerosolized racemic epinephrine can dramatically reduce airway swelling (rebound tracheal edema risk several hours after administration necessitates observation in hospital)
- Steroid administration controversial; may decrease severity of disease and decrease need for tracheal intubation or hasten improvement in first 24 hr of illness
- Small percentage with this disease needs tracheal intubation
- Parenteral steroids (dexamethasone) and inhaled (budesonide) have been used
- Breathing helium-oxygen mixtures has been reported as helpful in some cases (lower density and viscosity)

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<thead>
<tr>
<th>ASSESSMENT POINTS</th>
<th>CROUP</th>
<th>EPIGLOTTITIS</th>
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</thead>
<tbody>
<tr>
<td>AGE</td>
<td>3 mo–3 y</td>
<td>1–7 y</td>
</tr>
<tr>
<td>ONSET</td>
<td>Gradual</td>
<td>More rapid (usually &lt;24 hr)</td>
</tr>
<tr>
<td>FEVER</td>
<td>Low grade</td>
<td>High</td>
</tr>
<tr>
<td>COUGH</td>
<td>Characteristic barking</td>
<td>None</td>
</tr>
<tr>
<td>SORE THROAT</td>
<td>Occasional</td>
<td>Frequently severe</td>
</tr>
<tr>
<td>POSTURE</td>
<td>Any</td>
<td>Frequently sitting forward, mouth open, drooling</td>
</tr>
<tr>
<td>AIRWAY SOUND</td>
<td>Inspiratory stridor</td>
<td>Inspiratory stridor</td>
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<td>SEASONALITY</td>
<td>Peak winter, epidemic</td>
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**Perioperative Implications**

**Induction**

- Induction common with IV access already obtained

**Anticipated Problems/Concerns**

- Symptomatic pts who require intubation of trachea need tube 0.5–1.0 mm smaller in diameter than equivalent in children without croup.
- Pt who requires tracheal intubation usually requires sedative management to tolerate ventilation; often followed for development of leak around tracheal tube as a sign of improvement of edema; most pts improve within 2–4 d; when leak is present at 20–25 cm \( \text{H}_2\text{O} \) of pressure, extubation can be considered; complicated cases and pts with prolonged courses may benefit from examination of airway in operating room at time of extubation.
- Although a viral illness, some pts may acquire bacterial superinfection of airway and require antibiotic therapy.

**Risk**

- Children between 6 mo and 6 y are at risk, (6 mo–3 y at greatest risk)
- Children with underlying airway abnormalities (e.g., subglottic stenosis) or difficult intubations (e.g., micrognathia) and symptoms are at increased risk and require particular planning

**Perioperative Risks**

- Difficulty with intubation because of very narrowed subglottic region
- Obstruction of the small tracheal tube because of airway secretions

**Worry About**

- Risk of rebound tracheal edema several hours after racemic epinephrine treatment
- Cardiorespiratory crisis in progressive or severe Sx, agitation, younger pts, difficulties with oxygenation or ventilation, failure to oxygenate
- Bacterial superinfection of airway

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**AssesMenT POinTs**

**Differential points between croup (laryngotracheobronchitis) and epiglottitis**

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Cryptococcus Infection

**Risk**
- 0.4–1.3 cases per 100,000 in general population. AIDS pts, 2–7 cases per 1000.
- Underlying immunocompromised conditions and other risk factors: Acquired immunodeficiency syndrome (AIDS), systemic lupus erythematosus, prolonged treatment with corticosteroids, organ transplantation, advanced malignancy, diabetes, sarcoidosis, cirrhosis, idiopathic CD4 lymphocytopenia or use of immune-modifying monoclonal antibodies (alemtuzumab, infliximab, etanercept, or adalimumab).
- Only 20% of pts who have cryptococcosis without HIV infection have no apparent underlying disease or risk factor.

**Perioperative Risks**
- Resp insufficiency, severe ARDS
- Elevated intracranial pressure

**Worry About**
- Underlying immunocompromised conditions

**Overview**
- *C. neoformans* typically infects immunocompromised persons. 80–90% of infections occur in HIV pts in the USA
- Wide range of clinical presentations from asymptomatic resp colonization to dissemination of infection into any organ. In severely immunosuppressed pts, involvement of multiple body sites. Common sites for infection are the lungs and CNS.
- Pulm cryptococcosis: Multiple clinical presentations—asymptomatic nodules, lobular infiltrates, interstitial infiltrates, cavities, endobronchial colonization or masses, mediastinal adenopathy, hilar adenopathy, miliary pattern, or pleural effusions/empyema, pneumothorax, and life-threatening pneumonia with ARDS.
- Cryptococcal meningitis: Primary life-threatening infection. Mortality rate about 12%. Other CNS clinical manifestations: Cryptococcomas (abscesses) of brain, spinal cord granuloma, chronic demented (from hydrocephalus).

**ICD9-CM: 117.5 (Cryptococcosis)**

**Etiology**
- *Cryptococcus neoformans* is an encapsulated heterobasidiomycetous fungus
- Enters the host primarily through the lungs but special predilection for invading the CNS
- No human-to-human transmission, except in cases of contaminated transplant tissue

**Usual Treatment**
- Cryptococcal meningitis or CNS infection: IV amphotericin B deoxycholate 0.7–1 mg/kg/d (or liposomal amphotericin B (AmBisome) 3–6 mg/kg/d with less nephrotoxicity) in combination with IV fluconazole 100 mg/kg/d for 2 wk. Adding flucytosine to amphotericin B reduces the rates of failure and relapse compared with amphotericin B monotherapy. Then fluconazole 400–800 mg/d for 10 wk. In HIV pts, maintenance fluconazole 200–400 mg/d po therapy lifelong.
- Corticosteroids not recommended for the treatment of cryptoccocal meningitis.
- Control of increased intracranial pressures (external drainage or CSF shunt, or surgical drainage of abscesses).
- Antiretroviral therapy in HIV pts.
- Pulm disease in HIV-negative pts: fluconazole 200–400 mg/d for 6–12 mo.
- Pulm disease in HIV pts: Fluconazole 200–400 mg/d/lifelong.
- For more severe disease and immunocompromised hosts, treat like CNS disease.

**Perioperative Implications**

**Preoperative Preparation**
- Disposable anesthetic delivery circuits with bacterial filters
- Protect and maintain airways for altered mental status, seizures, focal neurologic signs, cranial nerve palsies
- Organ system effects of HIV infection or underlying immunocompromised conditions

**Monitoring**
- ARDS network low tidal volume protocol in severe ARDS pts
- Consider to monitor increased intracranial pressures

**Extubation**
- Consider if it adequately protects airway

**Induction/Maintenance**
- Anesthetic drugs associated with lower ICP and having neuroprotective qualities
- Possible interaction of antiretroviral drugs with the anesthetics and/or toxicity

Cushing’s Syndrome

Risk
• Onset generally occurs in the third and fourth decades
• Cushing’s disease is roughly three times more common in women than men
• 5-y mortality rate from adrenal carcinomas has been estimated to be >70%

Perioperative Risks
• Electrolyte abnormalities
• Consequences of untreated Htn
• Hyperglycemia

Worry About
• Challenges related to obesity, incl airway management and IV access
• Significant osteoporosis secondary to impaired calcium absorption, making positioning difficult
• Htn due to fluid retention
• Increased risk of infection as a result of corticosteroid’s immunosuppressive qualities
• Hypokalemia alkalosis, commonly seen in ectopic ACTH production

Overview
• The most common cause of Cushing’s syndrome is iatrogenic administration of exogenous glucocorticoids.

Perioperative Implications

Preinduction/Induction/Maintenance
• Prior to induction, normalize volume status, electrolytes, BP, and blood glucose levels. Spironolactone can be used to mobilize fluid and normalize potassium levels.
• Anxiety can cause increased secretion of cortisol. This response may be blunted by benzodiazepine.
• Make preparations to deal with a potentially difficult airway.
• Cortisol secretion is unlikely to be affected by the type of anesthesia used.
• Choice of anesthetic agents used for induction and maintenance of anesthesia are not affected by the presence of Cushing’s syndrome.
• Etomidate can be used at induction for its temporary suppression of the adrenal gland. However, this effect is likely overcome by the significant cortisol release with surgical stimulation.

Monitoring
• Intraop monitoring should be based on the pt’s current clinical state.
• An arterial catheter may be indicated in cases of poorly controlled systemic Htn.

• Spontaneous Cushing’s syndrome can result from adrenal gland hyperplasia secondary to increased ACTH production from a pituitary tumor, or an ectopic non-endocrine ACTH tumor.
• Other causes incl primary gland disorders such as adrenal adenoma or carcinoma.
• Symptoms incl primary gland disorders such as adrenal adenoma or carcinoma.
• Symptoms incl primary gland disorders such as adrenal adenoma or carcinoma.
• A 24-hr urine cortisol test can demonstrate elevated cortisol levels.

• Dexamethasone suppression test is used to aid in differentiating pituitary adenomas from adrenal tumors. Dexamethasone causes depression of cortisol and 17-hydroxycorticosteroid levels due to a negative feedback response, which is absent with ectopic ACTH or primary gland disease.
• ACTH plasma levels can also be tested directly.

• Radiologic evaluation incl: Abdominal CT scan to evaluate the adrenal glands, pituitary MRI scan with gadolinium contrast to evaluate the pituitary gland, and a chest CT scan when ectopic ACTH is the etiology.

ICD-9-CM Code: 255.0

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<td>CV</td>
<td>Hypertension</td>
<td>HA, visual disturbances</td>
<td>Decreased strength</td>
<td>Noninvasive BP</td>
</tr>
<tr>
<td>FEN</td>
<td>Hypokalemia</td>
<td>Weakness, constipation, nausea</td>
<td>Serum/urine osmolality</td>
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</tr>
<tr>
<td>RENAL</td>
<td>Fluid retention</td>
<td>Leg swelling</td>
<td>Peripheral edema</td>
<td>Fasting blood glucose</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperglycemia</td>
<td>Thirst, frequency</td>
<td>Difficulty rising from chair/climbing stairs</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Muscle wasting</td>
<td>Weakness</td>
<td>Thin extremities</td>
<td>Bone density scan</td>
</tr>
<tr>
<td></td>
<td>Impaired calcium absorption</td>
<td>Osteoporosis</td>
<td>Easy fracture</td>
<td></td>
</tr>
</tbody>
</table>


Etiology
• ACTH dependent (excessive ACTH secretion, stimulating adrenal production of cortisol)
• Pituitary microadenoma (70% of cases)
• ACTH production from a non-endocrine tumor (e.g., tumors of the lungs, pancreas, or thymus)
• ACTH independent (excessive cortisol production by adrenals and suppression of ACTH production)
• Adrenocortical adenoma or carcinoma
• Exogenous administration of glucocorticoids (e.g., treatment of asthma)
• These pts will likely need periop stress dose steroids.

Usual Treatment
• ACTH dependent Cushing’s disease
• Transsphenoidal resection of a pituitary microadenoma
• Radiation therapy
• ACTH independent Cushing’s disease
• Unilateral/bilateral adrenalectomy
• Medical adrenalectomy

Anticipated Problems/Concerns
• Meningitis following microadenomectomy
• Obese leading to a possible difficult airway
• Increased susceptibility to infection
• Hyperglycemia
• Increased risk of periop thromboembolic events
• Increased risk for intraop pneumothorax with open adrenal resection when compared to laparoscopic approach
Cyanide Poisoning

**Risk**
- Potent and rapid-onset toxin, esp inhalation of hydrogen cyanide (HCN, volatile liquid)
- CN ingestion results in slower onset
- Diffuses rapidly through body with high intra-cellular fixation to cytochrome aa3, in cellular mitochondria to paralyze aerobic metabolism

**Perioperative Risks**
- Main target organs: CNS and heart
- Animal experiments: Apnea precedes cardiac collapse

**Worry About**
- If CN toxicity resulted from fire or smoke exposure, consider also carbon monoxide (CO) and other toxins
- ⅔ of pts from domestic fires with CO toxicity also have increased CN
- Be alert for CN poisoning in donor for organ transplantation

**Overview**
- Major route of CN detoxification: Conversion to thiocyanate, which requires sulfane sulfur donor (e.g., thiosulfate) and enzyme (e.g., rhodanase); without renal excretion, thiocyanate can cause CNS abnormalities
- Minor route: Hydroxocobalamin (one form of vitamin B12) chelates CN to form cyanocobalamin
- Methemoglobin (metHb) ferric ion has high affinity for CN

**ICD-9-CM Code: 989.0**

**Etiology**
- Combustion product of natural and synthetic polymers
- Industrial chemistry (e.g., metals and plastics preparation)
- Plants: May contain cyanogenic glycosides
- Na nitroprusside: Overdose (0.5 mg/kg/hr within 24 hr)
- Abuse (e.g., suicide, Chicago CN-laced-Tylenol murders [1982], terrorism, chemical warfare)

**Usual Treatment**
- Rescue victim from exposure
- Intubation and ventilation with 100% O2 (hyperbaric O2, effective experimentally, is not practical)
- Gasetic decontamination (if necessary)
- Weigh risks and/or benefits of drug therapy since ⅔ of CN- is short (about 1 hr)
- Na thiosulfate (25%) 150 mg/kg IV (minimal side effects but thiocyanate requires renal excretion or hemodialysis)
- Hydroxocobalamin, 5–10 g IV, safe and rapid
- Methemoglobinemia induction (metHb, 30%) with 10% sodium nitrate (4 mg/kg IV) slow and unpredictable; can be hazardous in presence of carboxyhemoglobin (from CO toxicity) because neither metHb nor COHb carries O2; can be fatal in G6PD deficiency
- Dicobalt EDTA (ethylenediaminetetraacetate), 300 mg IV, followed by glucose infusion; potent and rapid but unsafe (esp arrhythmias, hypotension, and allergic reactions)

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<tbody>
<tr>
<td>HEENT</td>
<td>↓ CNS → ↓ Airway maintenance/protection</td>
<td>Concomitant smoke inhalation injury</td>
<td>↑ Cardiac output</td>
<td>Laryngoscopy/bronchoscopy</td>
</tr>
<tr>
<td>CV</td>
<td>Stimulation at low CN conc Depresssion at high CN conc</td>
<td>Htn, tachycardia Hypotension, bradycardia</td>
<td>↓ cardiac output, arrhythmias</td>
<td>ECG: Arrhythmias, esp ↓ conduction, V-Tach, VFib</td>
</tr>
<tr>
<td>RESP</td>
<td>Aerobic cellular respiration paralyzed Thermal/toxic airway and parenchymal injury</td>
<td>Concomitant smoke inhalation injury</td>
<td>Bronchoconstriction and pulm edema</td>
<td>↑ Blood PvO2 and ↑ SvO2 ↓ VO2 ↓ VCO2 ↓ PETCO2 Chest x-ray Bronchoscopy</td>
</tr>
<tr>
<td>METAB</td>
<td>Cellular aerobic metabolism disabled</td>
<td>Combination of ↑ SvO2 and lactic acidosis suggests CN</td>
<td>Lactic metabolic acidosis</td>
<td>Whole blood CN levels (not available in all labs)</td>
</tr>
<tr>
<td>CNS</td>
<td>Stimulation at low CN conc Depression at high CN conc</td>
<td>↑ Inhalatory CN intake Anxiety, dyspnea, headache Auditory/visual disturbances</td>
<td>↑ Resp rate Confusion</td>
<td>Funduscopy: Red retinal veins (↑ SvO2)</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Preparation**
- Continuous 100% O2

**Monitoring**
- SpO2 unreliable in presence of metHb (or COHb if co-existant CO poisoning)
- Mixed venous continuous SO2 (SvO2) or blood PO2 (PrO2)
- PETCO2
- Measure of VO2 and VCO2 helpful

**Airway**
- Protect and maintain airway

**Induction**
- Avoid CV depressant agents

**Maintenance**
- 100% O2 (no N2O)
- Ensure CNS status permits natural airway maintenance and protection

**Adjutants**
- Consider treatment for concomitant CO poisoning (see Carbon Monoxide Poisoning in Diseases section)

**Postoperative Period**
- Maintain 100% O2 breathing

**Anticipated Problems/Concerns**
- Heart and brain are target organs
- Prompt CPR (ventilation with O2) determines outcome
- Follow CNS function
- Seek concomitant smoke inhalation injury and CO toxicity

Cystic Fibrosis

**Risk**
- Prevalence ranges from 1:2500 births in Caucasians to 1:17,000 in African Americans
- Incidence ranges from 1:569 in Amish to 1:90,000 in Hawaiian Asians
- 2–5% of Caucasians are carriers

**Perioperative Risks**
- Pulmonary
  - Hypoxia and hypercarbia
  - Ventilation/perfusion (V/Q) mismatching
  - Pneumothorax
  - Airway obstruction with distal air trapping
  - Pancreatic
  - Glucose intolerance
  - Upper airway
  - Nasal polyps occlude nasal airways

**Overview**
- A disease of the exocrine glands which affects the lungs, pancreas, GI and hepatobiliary tracts
- Pulm exacerbations are caused by airway obstruction with thickened mucus.
- Pulm infections are common, esp *P. Aeruginosa*
- Pancreatic insufficiency can develop leading to malabsorption, incls vitamins A, D, E, and K, as well as glucose intolerance.

**ICD-9-CM Code: 277.0**

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effects</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Frequent nasal polyps</td>
<td>Nasal obstruction, Difficulty sleeping, Fever, headaches</td>
<td>Nasal polyps</td>
<td>Nasal endoscopy</td>
</tr>
<tr>
<td></td>
<td>Sinusitis</td>
<td></td>
<td>Sinus drainage</td>
<td>Sinus x-ray, culture</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cor pulmonale</td>
<td>Dyspnea, Orthopnea</td>
<td>Tachypnea, Rales, rhonchi, wheezing, Clubbing of fingers</td>
<td>ECG, CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchiectasis, atelectasis, pneumonitis, bronchospasm</td>
<td>Cough, Dyspnea, Poor exercise tolerance, Orthopnea</td>
<td>Hyperinflation of lungs, Poor ventilation, cyanosis, Clubbing</td>
<td>CXR, PFIs, A-a gradient</td>
</tr>
<tr>
<td>GI</td>
<td>Cholelithiasis, gallbladder dysfunction, Pancreatic insufficiency, Focal biliary cirrhosis, Intestinal obstruction</td>
<td>Abd pain (may be asymptomatic), Poor fat absorption, glucose intolerance, Abd pain</td>
<td>Jaundice, US, Cholangiography, Glucose, Liver function, Abd x-rays</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Poor muscle development</td>
<td>Hx poor nutrition, muscle weakness</td>
<td>Gachexia</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Hx and evaluation of baseline pulm status, exercise tolerance
- CXR: Hyperexpansion indicated by flattened diaphragm
- PFTs: Obstruction indicated by increased RV to TLC ratio and decreased FEF 25–75%
- ABG, electrolytes, blood glucose, LFTs
- Medications: Bronchodilators, antibiotics
- Chest physiotherapy

**Monitoring**
- Routine plus arterial pressure and/or CVP as pt's cardiopulmonary status and procedure dictates
- Blood glucose should be checked frequently

**Airway**
- Oropharyngeal airway for upper airway obstruction due to possibility of nasal polyps

**Induction**
- IV induction faster than inhalation due to larger FRC, smaller tidal volumes, and V/Q mismatching

**Maintenance**
- Volatile anesthetics useful as bronchodilators
- PPV may be necessary but should be used cautiously in light of pneumothorax risk.
- Warm and humidity gases
- Suctioning of airway mucus and bronchial lavage may help maintain oxygenation and ventilation.
- Muscle tone is important in maintaining patency of airways so muscle relaxants should be used only when needed.
- Opioids may be used as well, but pain control must be balanced with the risk of resp depression.

**Exubation**
- Carried out as soon as preop resp function resumes and after lung recruitment maneuvers

**Adjuvants**
- Bronchodilators, NSAIDs

**Postoperative Period**
- Pain key in encouraging coughing/deep breathing
- Chest physiotherapy and early activity

**Anticipated Problems/Concerns**
- Pneumothorax
- Postop resp insufficiency
- Cor pulmonale
- Electrolyte disturbances (Na⁺, Cl⁻)
- Regional anesthetics may be particularly beneficial in minimizing instrumentation of the airways while providing postop pain control.

**Usual Treatment**
- Goals: Control infection, promote mucus clearance, improve nutrition
- Pulm: Chest physiotherapy, mucolytics (short term), bronchodilators, humidification, antibiotics for infections
- Pancreatic: Pancreatic enzyme replacement
Cytomegalovirus Infection

Andrew D. Badley
Stacey A. Rizza

Incidences
- Incidence in USA: < 10 y: 25%; 10–25 y: 35%; 25–50 y: 50%; > 50 y: 50+
- Disease from CMV in HIV-positive pts 20–30% (increased risk with low CD4 count)
- Approx 1 in 150 children is born with congenital CMV

Overview
- Double-stranded DNA virus; member of herpes family of viruses. Vast majority of North American adults have had prior exposures and are CMV seropositive.

Assessment Points

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<tbody>
<tr>
<td>HEENT</td>
<td>Destruction of retina</td>
<td>Decreased visual acuity, blind stops</td>
<td>Fundoscopy; white and red lesion</td>
<td>Ophthalmology evaluation</td>
</tr>
<tr>
<td>CV</td>
<td>Myocarditis; LV dysfunction</td>
<td>CHF symptoms, palpitations</td>
<td>Irregular rhythm, displaced PMI S3</td>
<td>ECG, ECHO, heart biopsy</td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumonitis; impaired gas exchange</td>
<td>Dyspnea, nonproductive cough</td>
<td>Wheezes, crackles</td>
<td>CXR, ABGs, bronchoscopy ± biopsy</td>
</tr>
<tr>
<td>GI</td>
<td>Viral infection of organ</td>
<td>Hepatitis/cholangitis; -Right upper quadrant pain -Jaundice, itching, acholic stools -Esophagitis: dysphagia, odynophagia -Colitis: diarrhea, abdominal pain -Gastritis: pyrosis, anorexia</td>
<td>Signs of hepatic failure, fever, hepaticus, jaundice, bruising, painful liver, nonspecific abdominal pain</td>
<td>Liver function tests, ERCP, US, viral blood cultures ± biopsy</td>
</tr>
<tr>
<td>HEME</td>
<td>Bone marrow suppression</td>
<td>Rash, fatigue</td>
<td>Petechiae, pallor, tachycardia</td>
<td>CBC</td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalitis</td>
<td>Motor or sensory abnormalities, altered mental status</td>
<td>Motor weakness, sensory abnormality, cerebellar ataxia, abnormal tests of cortical function</td>
<td>CT MRI, lumbar puncture</td>
</tr>
</tbody>
</table>


Perioperative Implications

Perioperative Preparation
- Evaluate for signs of pulm, cardiac, hepatic, CNS, bone marrow, or adrenal dysfunction

Monitoring
- Routine

Airway
- May require high FIO2 and PEEP

Preinduction/Induction
- Avoid tachycardia/hypotension

Maintenance
- Follow CO, PCWP, SaO2, BP

Extravasation
- No special concerns

Postoperative Period
- Monitor for clinical signs of disease progression

Adjuncts
- No special concerns
Deep Vein Thrombosis

**Risk**
- Incidence in USA: 170,000–200,000 diagnosed new cases/y 90,000–100,000 recurrent cases/y.
- True incidence (underdiagnosis) closer to 1 million cases/y. Could be as high as 2 million.
- Race with highest prevalence: Unclear. African-Americans have a higher rate of PE being diagnosed than DVT compared to Caucasians. Asians seem to have the lowest rates of either.
- Smoking, obesity, being bedridden, and decreasing LVEF are predisposing factors for developing DVT
- Risk factors incl age >40, previous DVT or family Hx of DVT, paraplegia, spinal cord trauma, major orthopedic surgery, malignancy, hypercoagulable states.
- Decreased risk with regional anesthesia vs. general, esp in LE orthopedic surgery.

**Perioperative Risks**
- Without prophylaxis, DVT develops in close to 30% of general surgery cases
- Incidence of fatal pulmonary emboli: 0.1% (general)—5% (total knee replacement)

**Worry About**
- Pulm embolism
- Cardiac arrest, electromechanical dissociation
- Hypoxemia and increased dead space potentially leading to resp acidosis in pt with controlled ventilation

**Overview**
- Clinical findings (e.g., Homans sign) helpful less than 50% of the time.
- Ascending phlebography (venography) is standard for comparison, but has 2–3% incidence of inducing peripheral thrombosis.
- Impedance plethysmography (IP), which detects proximal veins, reasonable in symptomatic pts, but lacks sensitivity and specificity in asymptomatic pts.
- Compression ultrasonography with Doppler flow imaging better than IP (proximal veins), yet sensitivity falls off in asymptomatic pts. If IP or Doppler-supplemented ultrasonography negative, pt needs serial exams to detect potential progression of distal disease.
- CT and MRI are reliable, yet are cumbersome, costly, and not routinely available.
- D-Dimer has a high negative predictive value.

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<tbody>
<tr>
<td>CV</td>
<td>Pulm embolism</td>
<td>Chest pain</td>
<td>SVT RV strain</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Hemothysis</td>
<td>Tachypnea, wheezing possible</td>
<td>PT, APTT Pt count Hgb, d-dimer</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Calf pain</td>
<td>Venography, US</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Sequential compression devices may decrease incidence by activating fibrinolytic system.
- Anticoagulation needed for 6 mo after diagnosis and up to the time of procedure.
- Consider preop use of an IVC filter in high-risk pts.
  - If pt on anticoagulants or antiplatelets drugs preop ensure adequate products available in blood bank
  - When possible, administer SQ heparin preop

**Monitoring**
- Bleeding from residual anticoagulation or drug-induced thrombocytopenia
- Screening US for ICU pts done on a weekly basis has started to become the vogue. Cost-effective data supporting this approach are pending.

**Airway**
- None

**Preinduction/Induction**
- Regional anesthesia may reduce risk in some orthopedic and genitourinary procedures

**Adjutants**
- Depends on etiology: Examine specific etiology (e.g., Dilated Cardiomyopathy) in Diseases section
- Heparin, LMWH, warfarin tissue plasminogen activator, streptokinase/urokinase, anisoylated plasminogen-streptokinase activator complex all increase periop bleeding diathesis. Some effect of these agents on other drugs (verify specific drug effects in Drugs section).

**Postoperative Period**
- In high-risk pts consider full anticoagulation postop as prophylaxis
- If using SQ heparin, it should be administered every 8 hr if possible
- Continue sequential use of elastic stockings until pt is ambulatory, but do not start in pts suspected of having DVT

**Anticipated Problems/Concerns**
- Pulm embolism represents life-threatening complication of DVT
- Post-thrombotic syndrome with chronic venous stasis with skin and wound effects

**Etiology**
- Stasis
- Activation of coagulation cascade by tissue trauma
- Hypercoagulability related to congenital or acquired antithrombin III, protein C, or protein S deficiency, antiphospholipid syndromes
- Hypercoagulability related to malignancy, smoking, sedentary lifestyle, increasing physiologic age, decreasing LVEF
- Hyperviscosity states such as polycythemia vera

**Usual Treatment**
- Heparin administration prior to warfarin to avoid acute decreases in endogenous anticoagulant protein C
- LMW heparin may be used
- Thrombolitics
- Thrombectomy, catheter or open surgical...
Degenerative Disk Disease

Risk
Risk factors determined by spinal level
• Cervical spine: C3 and C4 most common, 10% of degenerative disk disease
• Thoracic: Uncommon, can be related to trauma, tumor, 0.2–1.8% of disk disease
• Lumbar: Very common, 85–90% of disk disease, third most-common cause of chronic pain in USA

Perioperative Risks
• Difficult airway
• Spinal cord injury from airway manipulation or positioning
• Positioning injury from prone position
• Ischemic optic neuropathy

Worry About
• Cervical spine instability or chronic subluxation
• Difficulty with intubation
• Injury to the spinal cord, nerve roots

Overview
• Pain from herniation of an intravertebral disk with nerve root compression is the third most-common chronic disease in the USA and the most common indication for elective spine surgery
• Incidence varies among spinal segments, being absent in sacral area, most common in lumbar area, next in cervical region, and uncommon in thoracic region

ICD-9-CM Codes: 722.40 (Cervical); 722.51 (Thoracic); 722.52 (Lumbar)

ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Difficult airway</td>
<td>Neck pain</td>
<td>Decreased ROM</td>
<td>Flexion/extension x-ray to detect instability</td>
</tr>
<tr>
<td></td>
<td>Visual acuity</td>
<td>Pt report</td>
<td>Pt report</td>
<td>Eye examination</td>
</tr>
<tr>
<td>RESP</td>
<td>Lung tumor can mimic symptoms of thoracic disk disease</td>
<td>Chest pain with chest excursion</td>
<td>Abnormal pulmonary auscultation</td>
<td>CXR, MRI</td>
</tr>
<tr>
<td>GI</td>
<td>GI malignancy can mimic symptoms of thoracic or lumbar disk disease</td>
<td>Truncal pain, abdominal pain</td>
<td>Abdominal mass</td>
<td>CT, MRI</td>
</tr>
<tr>
<td>RENAL</td>
<td>Pyelonephritis, cancer of prostate can mimic symptoms of lumbar disk disease</td>
<td>Lumbar pain, muscle spasm, fever/chills</td>
<td>Costovertebral angle tenderness to percussion</td>
<td>Urinalysis, prostate-specific antigen, lumbar spine x-ray, MRI</td>
</tr>
<tr>
<td>CNS</td>
<td>Myelopathy, anterior spinal cord syndrome</td>
<td>Radiating pain, incontinence, sexual dysfunction, paraplegia</td>
<td>Long tract signs, abnormal reflexes, pathologic, Babinski reflex</td>
<td>X-ray, MRI</td>
</tr>
<tr>
<td>PNS</td>
<td>Radiculopathy, absent deep tendon reflexes, peripheral nerve deficits</td>
<td>Sciatica, numbness, weakness of the extremities</td>
<td>Sciatic pain with ROM, motor deficits, patchy sensory deficits</td>
<td>EMG</td>
</tr>
<tr>
<td>MS</td>
<td>Pain, decreased ROM, calcification</td>
<td>Pain, night pain, disability from work</td>
<td>Decreased ROM in spine</td>
<td>Spine x-ray, MRI</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Assessment
• Evaluate coagulation if heavy aspirin or NSAID use or symptoms of bleeding
• Airway assessment. If signs of cervical instability or other indicators of difficult airway management, flexion-extension x-ray of cervical spine.
• Antisialagogue if awake intubation
• If spinal or epidural anesthesia planned, lumbar x-rays may be needed
• Planned regional anesthesia may reduce minor complications, such as pain and nausea; intraop bleeding may be reduced

Monitoring
• Potential for air embolism, greater with sitting position for posterior approach to cervical spine
• Consider multilumen right atrial catheter, pre-cordial Doppler if sitting position for cervical spine procedure
• If large blood loss estimated, arterial line becomes indicated

Airway
• If cervical spine not involved, then routine
  • Pressure injuries or ventilatory difficulty with the prone position
  • Optimum perfusion to the head. Ischemia, neck position, or venous congestion may contribute to ischemic optic neuropathy
  • Airway edema at the conclusion of surgery

Overview
• Pain from herniation of an intravertebral disk with nerve root compression is the third most-common chronic disease in the USA and the most common indication for elective spine surgery
• Incidence varies among spinal segments, being absent in sacral area, most common in lumbar area, next in cervical region, and uncommon in thoracic region

ICD-9-CM Codes: 722.40 (Cervical); 722.51 (Thoracic); 722.52 (Lumbar)
Delirium (Postanesthetic)

Risk
- Risk factors for development of postop delirium (POD) are either pt or procedure related. Pt related factors can increase the pt’s inherent risk for development of POD. Most important factors are:
  - Old age
  - Pre-existing cognitive dysfunction
  - Hx of stroke, depression, alcohol abuse, psychiatric diagnosis, diabetes, peripheral vascular disease, atrial fibrillation, and heart failure

- Procedure related factors that may trigger the development of delirium are:
  - Type of surgery: Cardiac, orthopedic, and vascular procedures are associated with the highest incidence of the syndrome
  - Emergent or urgent procedures
  - Anticholinergic medications
  - Poorly controlled postop pain
  - Benzodiazepines, polypharmacy, and meprobamate

- Factors with no effects on the pt’s risk for development of POD are length of surgery, type of anesthetic (general versus regional), and type of postop analgesia (regional techniques versus systemic opioids).

Perioperative Implications

Preoperative Preparation
- The team effort should start by identifying pts at risk for POD when they present for the preadmission screening process. The second step should focus on modifying the risk factors that are modifiable. Drugs are the most common reversible causes of delirium.
- Correct pre-existing electrolyte abnormalities
- Sensory impairments, visual and auditory should be addressed preop.
- Pro-active geriatric consultation and involvement of anesthesia team with an expertise in dealing with geriatric population can prove to be very effective in prophylaxis against POD.

Monitoring
- Standard monitors. Monitor blood glucose level, electrolytes, and volume status.
- Increased length of hospital stay and ICU days after development of POD
- Increased risk for falls, longer intubation/re-intubation, need for urinary catheters, urinary infections, pressure ulcers, aspiration pneumonia, periop myocardial ischemia, and wound infections
- Increased risk of subsequent functional loss and institutionalization
- Added cost to the health care system

Worry About
- Other causes of change in mental status (i.e., rule out any metabolic causes for the syndrome).
- Pt can present with a violent behavior or symptoms of withdrawal. The first category can be harmful to themselves and others.
- Drug drug interaction in the periop period may result in the change in mental status.

Overview
- Acute confusional state that is characterized by disorientation, disturbed sleep-wake cycle, memory impairment, perceptual disturbance, and altered psychomotor activity.
- Reported incidence ranges from 5.1% to 52.2%. The wide range is due to the disparate and subjective symptoms and the different tests used to establish the diagnosis.

ICD-9-CM Code: 292.81 (Drug induced)


Airway
- Maintain a patent airway, maintain adequate oxygenation and ventilation.

Preinduction/Induction
- Avoid centrally acting anticholinergics as premedications.

Maintenance
- As dictated by the type of surgery.
- Incl an effective plan for postop analgesia in your anesthetic regimen.

Extubation
- Standard criteria for extubation. Avoid hypoxia and hypercarbia.

Adjuvants
- Low-dose haloperidol 1–5 mg for symptoms control.
- If anticholinergic-induced delirium is suspected, use phystostigmine 0.5–2 mg IV.

Etiology
- The pathophysiology of POD is not clearly understood. Some of the causes cited are:
  - Disturbed neurotransmitter systems esp. cholinergic deficiency
  - Stress response to surgery and anesthesia
  - Global cerebral hypoperfusion
  - Drug–drug interaction
  - Alcohol and drug withdrawal in the periop period

Usual Treatment
- Supportive measures
  - Team approach is the best; physicians, nurses, and family members should be involved.
  - Anticholinergics, H2-blockers, benzodiazepines, opioids, and antipsychotic medications should be replaced with drugs that have no central effects whenever possible.
- Maintain adequate ventilation, oxygenation, cerebral perfusion, and normal electrolytes and acid-base balance.
- Maximize awareness of the pt with his environment and surroundings.
- Consider one-to-one companion rather than applying physical restraints unless the pt is posing a threat to themselves and others.
- Symptoms control
  - Low-dose haloperidol for symptoms control
  - Phystostigmine for anticholinergic induced delirium

Anticipated Problems/Concerns
- POD is a risk factor for increased functional loss and institutionalization. Confusion can be transient or can remain up to 6 mo after discharge. Quality of life measures are affected during and after an episode of delirium. Psychological stress to family members and caregivers should not be underestimated.
- Side effects of haloperidol incl extrapyramidal symptoms and neuropsychiatric malignant syndrome.

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<tbody>
<tr>
<td>CNS</td>
<td>POD</td>
<td>Preoperative: Mental status, assess baseline risk, drug therapy</td>
<td>Anxiety, agitation, violent behavior, impaired cognition, emotional lability, agitation, hallucinations, fluctuating states of consciousness</td>
<td>O2 saturation, arterial blood gases, electrolytes, and blood glucose levels</td>
</tr>
</tbody>
</table>

Comments: The pt’s preop mental status is assessed. The team should be aware of the pt’s baseline mental status. The pt’s mental status should be assessed periop and postop. The pt’s mental status should be reassessed after an episode of delirium.

Adjuvants
- Anticholinergics, H2-blockers, benzodiazepines, opioids, and antipsychotic medications should be replaced with drugs that have no central effects whenever possible.

Supportive measures
- Maintain adequate ventilation, oxygenation, cerebral perfusion, and normal electrolytes and acid-base balance.

Usual Treatment
- Maintain awareness of the pt with his environment and surroundings.

Anticipated Problems/Concerns
- POD is a risk factor for increased functional loss and institutionalization. Confusion can be transient or can remain up to 6 mo after discharge. Quality of life measures are affected during and after an episode of delirium. Psychological stress to family members and caregivers should not be underestimated.
- Side effects of haloperidol incl extrapyramidal symptoms and neuropsychiatric malignant syndrome.
Dementia

Risk
- Worldwide, approx 24.3 million individuals have dementia, and it is estimated that this number will increase to 81 million people by 2040. Incidence in the USA: More than 5 million.
- Affects approx 10% of adults 65 y or older and up to 50% of community-dwelling individuals age 85 or older.
- Age and genetic susceptibility are risk factors. Increasing evidence suggests that vascular risk factors (HTN, obesity, DM, atherosclerotic cardiovascular disease (ASCVD), cerebrovascular disease, hyperlipidemia, tobacco use, and diets high in saturated fat) as well as psychosocial factors (degree of education, social disengagement, and physical activity) may also play a role in disease development.

Perioperative Risks
- Risks related to cognitive impairment (e.g., impaired ability to cooperate with anesthetic care, aspiration risk, trauma risk from falls).
- Risks associated with concomitant neuropsychiatric symptoms (e.g., agitation, hallucinations, aggressive behavior, delusions), the medications used to control these symptoms, and the potential interactions between these medications and drugs commonly utilized during anesthesia.

Worry About
- Central neurotransmitters levels may be reduced (particularly acetylcholine), and drugs administered during anesthesia may further attenuate these levels, potentially contributing to further cognitive deterioration.
- The immediate potential for anesthetics to further impair cognitive function periop as well as to exacerbate any accompanying neuropsychiatric symptomatology.
- Inability of the pt to fully comprehend and cooperate with the anesthetic plan.

• Baseline impairment of cognitive function and its impact on potential anesthesia-related injuries (e.g., increased aspiration risk, positioning injuries, inadvertent removal of indwelling catheters or ETT).
• Potential side effects of neuropsychiatric medications used in the treatment of dementia.
• Consequences related to prolonged immobilization (e.g., limited exercise tolerance, risk of hyperkalemia with succinylcholine, presence of decubitus ulcers, development of DVT, and positioning injury risk).
• Obstacles to adequate postop pain relief incl the pt’s limited ability to communicate as well as the potential for centrally acting analgesics to exacerbate cognitive impairment and neuropsychiatric symptomatology.

Overview
- Dementia is a clinical syndrome characterized by memory impairment as well as one or more of the following disturbances: Language impairment (aphasia), perturbations in performing executive functions (e.g., organizing, planning, initiating, and abstract reasoning), the inability to recognize familiar objects or persons (agnosia), and the impaired ability to perform motor activities despite having intact motor function (apraxia).
- Although not part of the diagnostic criteria, dementia is commonly associated with neuropsychiatric symptoms incl agitation, personality changes, depression, delusions, hallucinations, aggressive behavior, repetitive vocalizations, and sleep disturbances.
- Alzheimer’s disease (50–70%), and vascular dementia (15–20%) account for the majority of the cases; however, mixed pathologies (i.e., AD and VaD) are quite common (10–20%).
• All other dementias account for <10% of the total number of cases.

Recent data suggest that the average survival time from the onset of dementia is 4.5 y.

ICD-9-CM Codes: 290.10 (Alzheimer’s type); 290.40 (Multi-infarct dementia)

Etiology
- In Alzheimer’s disease (AD), the two histopathological hallmarks incl senile neuritic plaques (comprised primarily of β-amyloid protein) and neurofibrillary tangles containing aggregated hyperphosphorylated tau protein. These pathological changes have been associated with a loss of cholinergic neurons and tracts, which are felt to correspond with the clinical cognitive impairment. Precise mechanisms of AD are still unknown.
- Extensive atrophy of cortical convolutions, esp in hippocampus and temporal lobes are commonly observed (much greater than normal aging).
- 10–30% of dementia cases can be potentially reversible if they are secondary to an underlying treatable cause incl structural or traumatic brain injury, medication toxicity, infectious causes, vitamin deficiencies, inflammatory diseases, as well as endocrine and metabolic disorders.

Usual Treatment
- Symptomatic treatment has incl the use of cholinesterase inhibitors: Donepezil, galantamine, rivastigmine, and tacrine.
- Neuropsychiatric symptoms are common in dementia and are often treated with the following classes of psychotherapeutic agents: Typical antipsychotics (haloperidol, perphenazine), atypical antipsychotics (risperidone, olanzapine), antidepressants (sertraline, citalopram, trazodone), mood stabilizers (carbamazepine, sodium valproate), cholinesterase inhibitors (donepezil, rivastigmine, galantamine), and NMDA antagonists (memantine).

Perioperative Implications

Preoperative Preparation
- Pt most likely cannot give consent or a reliable Hx; determine if guardian or surrogate is identified. Pre-existing medical records, nursing home medical documents, and the pt’s relatives may be of critical importance.
- If possible, establish a general preanesthetic baseline of cognitive performance and behavior incl degree of orientation.
- Agitation and anxiety may be pronounced during this period. Sedatives best avoided, particularly in severe cases with low Mini Mental State Examination scores. Benzodiazepines have been used to treat agitation in pts with mild cognitive decline, and should be performed in a monitored setting.
- Centrally acting anticholinergics (atropine, scopolamine) are best avoided; glycopyrrolate is acceptable, as it does not cross the blood brain barrier.

Monitoring
- Routine
- More invasive monitoring (e.g., A-line, CVP, PA catheter) may be necessary given that elderly pts with dementia may also demonstrate significant cardiac, pulmonary, or renal impairment.

Airway
- Cervical range of motion may be limited by arthritis.
- Check whether the pt has removable prosthetic dental appliances.

Preinduction/Induction
- Regional anesthesia (RA) may provide advantages incl reducing the cognitive and neurobehavioral side effects associated with the use of sedation. Furthermore, the need to manipulate the airway and to provide mechanical ventilatory support may be avoided. A reduction in the perip
development of DVT, particularly in pts with limited mobility, has also been observed.
- Monitored anesthetic care (MAC) with minimal sedation may also limit perturbations in cerebral functioning. However, the greater risk of aspiration in this pt population should be appreciated when considering this technique.
- Despite the theoretical advantages of RA and MAC, general anesthesia may still be necessary due to lack of pt cooperation.
- Propofol may offer most rapid recovery.
- DVT prophylaxis may be indicated (SQ heparin, intermittent pneumatic compression).

Maintenance
- No one technique or agent best.
- Avoid sedatives and narcotics with long half-lives.
- Consider using short-acting anesthetic agents (e.g., desflurane, sevoflurane, propofol, remifentanil).
- Consider the impact of potential renal or hepatic impairment on the metabolism of drugs administered during general anesthesia.
- The duration of action of succinylcholine may be prolonged by donepezil.
- The cholinesterase inhibitors used to treat dementia may antagonize non-depolarizing neuromuscular blockade.
- Bradycardia can be observed in pts treated with cholinesterase inhibitors; therefore, caution should be taken when using agents with known bradycardic effects.
- If bradycardia requires treatment with an anticholinergic medication, glycopyrrolate is preferred, as it does not cross the blood-brain barrier.
- The maintenance of normothermia as hypothermia may further depress pt’s mental status and may increase myocardial O₂ demand.

Extubation
- Exhale when awake; orientation postop may be further impaired by anesthetics.
- Impaired cognitive performance and co-morbid diseases may increase the need for postop ventilatory support and critical care resources.

Adjuvants
- Can see prolonged effects on mental status with sedatives, hypnotics, and narcotics.

Anticipated Problems/Concerns
- Disorientation and delirium are common postop—provide familiar person and written orientation material.
- Close observation incl 1:1 nursing care may be necessary.
- Temporary restraints may still be necessary and requires familiarization with hospital and TJC policy on their use in the PACU.
- May be poor candidates for catheter-based postop regional analgesia (e.g., epidural or brachial plexus catheters) or for PCA in the postop period. Whenever possible, regional analgesia using single-dose peripheral nerve blocks should be considered, as this reduces the need for opioid analgesia.
- Consider the physiologic and pharmacologic effects of pre-existing decreases in renal and hepatic function whenever the use of parenterally or orally administered analgesics and sedatives is indicated in elderly pts.
Depression, Unipolar

ICD9-CM: 296.2 (Major depressive disorder, single episode)

Etiology
• Unknown pathophysiology, but suspect abnormalities of amine neurotransmitter (serotonin, dopamine, and norepinephrine) pathway.
• Multifactorial. Familial pattern thought to exist.

Usual Treatment
• Selective serotonin reuptake inhibitor (SSRI): Works by blocking reuptake of serotonin at presynaptic membranes with little effect on adrenergic, cholinergic, histaminergic, or other neurochemical system. Associated with fewer side effects.

Perioperative Implications
• Serotonin syndrome
  • Potentially life-threatening drug reaction from interactions between SSRIs, atypical and cyclic antidepressants, MAOIs, opiates, and anti-biotics, (e.g., phenylzine and meperidin, phenylzine and SSRIs, lineagezole and citalopram).
  • Symptoms incl agitation, delirium, autonomic hyperactivity, hyperreflexia, clonus, and hyperthermia.
  • Treatment involves discontinuing the suspected agent(s), supportive measures, and control of autonomic instability, excess muscle activity, and hyperthermia.
  • In mild cases, lorazepam, propranol, or cyproheptadine (a 5-HT antagonist only available in PO form that binds to serotonin receptors) can be administered.
  • Fluoxetine
    • Potent hepatic cytochrome P-450 inhibitor, which increases plasma concentration of drugs that depends on P-450 for clearance
    • Fluoxetine may increase the concentration of tricyclic antidepressants by two-to-five-fold.
  • Some cardiac arrhythmic and beta-blockers may also be potentated as a result.
  • Tricyclics
    • Anticholinergic effect causes CV abnormalities such as orthostatic hypotension and cardiac dysrhythmias.
    • Due to increased availability of neurotransmitters in the CNS, anesthetic requirement may be increased. Likewise, increased availability of norepinephrine may cause exaggerated BP response in reaction to indirect-acting vasopressor such as epedrime.
    • Acute treatment with tricyclics (first 2–3 wk) is associated with potential significant Htn, whereas long-term treatment is associated with down-regulation of receptors.
  • Tachydysrhythmias have been observed following administration of pancerumion, ketamine, meperidine, or local anesthetics containing epinephrine to pts who are also on imipramine.
  • MAOIs
    • Anesthetic requirement may be increased due to increased concentration of norepinephrine in the CNS.
  • Serotonin syndrome from combining MAOI and meperidin has been noted.
  • Current belief is to continue MAOIs during the periop period despite previous thought of discontinuing MAOIs 14 d prior to elective surgery.
  • Benzodiazepene (midazolam) may be used to treat preop anxiety.
  • Ketamine, a sympathetic stimulant should be avoided.
  • Serum cholestearerase activity may be decreased in pts on phenelzine, so the dose of succinylcholine may need to be reduced.
  • The addition of epinephrine to local anesthetic solutions should probably be avoided.
  • If hypotension develops direct-acting drugs such as phenylephrine are preferred. The dose should also be decreased to minimize the likelihood of an exaggerated hypertensive response.
  • Anticipated concerns
    • In periop period, general rule is to try to continue antidepressant therapy.
    • Be aware of potential interactions between anesthetic agents and antidepressants.
    • Pts should be monitored for signs of seroton syndrome.
Diabetes, Type I (Insulin-Dependent)

**Risk**
- Incidence in USA: 1.2 million
- Races with highest prevalence: Hispanics and Native Americans

**Perioperative Risks**
- Increased risk of CABG 5–10x if end-stage renal, CHF, or autonomic neuropathy; without renal, CHF, or autonomic dysfunction, risk is 1–1.5 × normal

**Worry About**
- Autonomic neuropathy, gastroparesis, and sudden postop death
- Painless myocardial ischemia
- Atlanto-occipital joint immobility
- Tight glucose control might be indicated if pregnant, difficult weaning from bypass (ECC), or predictable global or focal CNS ischemia.

**Overview**
- Endocrinopathy assoc with end-stage renal, ophthalmic, myocardial, and neuropathic disease

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Possible atlanto-occipital dislocation 2° to abn collagen glycosylation</td>
<td>Pain</td>
<td>Neck ROM, prayer sign</td>
<td>Usually not needed, neck x-rays in extension</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Angiopathy LV dysfunction (4–10x with Htm) Ischemic PVD</td>
<td>Poor exercise tolerance Angina CHF symptoms</td>
<td>2-flight walk Chest exam for signs of CHF BP lying and standing</td>
<td>ECG, CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>↓ Lung elastance; ↓ FEV; ↓ FVC</td>
<td>Poor exercise tolerance</td>
<td>Generally not needed</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Gastroparesis</td>
<td>Early satiety</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Nephropathy, esp if hypertensive</td>
<td>N/V; impotence; orthostatic Sx Nonprotein foods</td>
<td>BUN/Cr</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>↓ Insulin from islets</td>
<td></td>
<td>FBS, electrolytes</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Autonomic dysfunction 2° to neuropathy</td>
<td>Early satiety, impotence, N/V, orthostatic symptoms</td>
<td>RR interval variation on ECG BP change on standing</td>
<td></td>
</tr>
<tr>
<td>PNS</td>
<td>Impaired joint mobility 2° to non-enzymatic glycosylation of collagen</td>
<td>PNS exam, esp. if regional planned</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Metoclopramide (10 mg/70 kg) in pts with gastroparesis
- Assess myocardial and volume status.

**Monitoring**
- Myocardial ischemia. Can have CHF if volume overload and LV dysfunction present.
- Blood sugar

**Airway**
- Atlanto-occipital dislocation possible—see HEENT. Do prayer sign test; may have gastroparesis.

**Induction**
- Osmotic diuresis can make hypovolemic; ANS and CV dysfunction make BP and HR fluctuate.

**Maintenance**
- CV instability; volume status key to avoid renal and myocardial dysfunction with operation; checking RR variation (HRV) to determine autonomic insufficiency likelihood still not widespread

**Exubation**
- CV and pulm drive insufficiencies common with neuropathies

**Adjuncts**
- Rx for tight control
- Regional: Diabetic nerves may be more prone to edema esp if epinephrine used. Reduce dose (e.g., lidocaine from 2.0% to 1.5%) for same effect.

**Postoperative Period**
- Sliding scale of insulin Rx based on q 1–3 hr blood glucose determinations

**Etiology**
- Genetic predisposition to autoimmune destruction of glucose transporter on islet cells leads to increased blood glucose, which affects proteins via nonenzymatic glycosylations
- Swells cells (sorbitol is oncrotically active)
- Increased viscous proteins (macroglobulins), which impede blood flow
- Increased substrate for anaerobic metabolism
- Deranges autoregulation of blood flow

**Usual Treatment**
- Insulin injections, lifestyle changes incl stress management, diet, and exercise
- Pancreas and islet transplant is option if renal disease is end-stage
- Control of BP

**Anticipated Problems/Concerns**
- Gastroparesis with presence of solid food 24 hr after last meal if ANS dysfunction present. Consider Rx with metoclopramide 10 mg IM 1½ hr prior to induction.
- ANS dysfunction assoc with sudden death postop; can keep in ICU/PACU overnight; vested adult who can measure blood glucose and call 911 if sent home postop
Diabetes, Type II (Noninsulin-Dependent)

Risk

- Incidence in USA: more than 25 million
- Highest prevalence: Hispanics and Native Americans
- Gender predominance: None

Perioperative Risks

- Increased risk 5–10x if end-stage renal, CV, CHF, or autonomic neuropathy; without renal, CV, or autonomic dysfunction, risk is 1–1.5x normal
- Metabolic abnormalities increased with periop insulin Rx
- Unclear if same risks as for type I diabetes

Worry About

- Autonomic neuropathy, gastroparesis, and sudden postop death
- Myocardial ischemia; CV instability
- Tight glucose control might be indicated in pregnancy (see under Diabetes, Type III), difficult weaning from bypass (ECC), predictable global or focal CNS ischemia
- Disordered autoregulation makes hypertensive BP fluctuations more dangerous
- Fluid and electrolyte imbalance

Overview

- Endocrinopathy that can cause same organ dysfunction as in diabetes type I: End-stage renal, myocardial, and neuropathic disease
- Associated with deranged blood flow autoregulation to CNS (at blood sugar 250 mg/dL), renal (at blood sugar 200 mg/dL), and cardiac (at blood sugar 100 mg/dL) vessels
- Ketosis is rare, since some endogenous insulin
- Primarily controlled by diet and/or oral agents, although insulin more frequently used
- Usually has high insulin levels for glucose level, but peripheral resistance to insulin effect. Can develop hyperosmolar nonketotic coma.
- Blood sugar control per se not associated with increased periop morbidity in absence of:
  - Hypoglycemia
  - Hyperosmolar coma
  - CNS ischemia
  - Pregnancy
  - Extracerebral circulation
- Preoperative hyperglycemia A1c levels ideally <7% indicate quality of recent blood sugar control. High levels correlate with chronic microvascular complications.

ICD-9-CM Codes: 250.00; 250.02 (Uncontrolled)

Perioperative Implications

Preoperative Preparation

- Metoclopramide (10 mg/70 kg) if gastroparesis
- Assess myocardial and autonomic function and volume status, half-life of hypoglycemic agent(s) taken chronically

Monitoring

- Blood sugar (questionable need for very tight control in type II diabetes) and metabolic abnormalities
- Painless myocardial ischemia can cause CHF if volume overload and LV dysfunction
- Peripheral vasculature and nerves vulnerable to pressure ischemia

Airway

- Atlanto-occipital dislocation possible: See HEENT, do prayer sign test

Induction

- Osmotic diuresis, autonomic nervous system, and CV dysfunction can make BP/HR fluctuate.

Maintenance

- CV instability: Volume status and avoidance of Htn key to avoiding renal and myocardial dysfunction periphr

Exubation

- CV and pulm drive insufficiencies common with neuropathies

Adjuvants

- Regional: Diabetic nerves may be more prone to edema, esp if epinephrine used. Reduce dose (e.g., lidocaine from 2.0% to 1.5%) for same effect.
- Oral hypoglycemics may ablate preconditioning.

Postoperative Period

- Debate as to whether control to tighter than 60–250 mg/dL is of value in absence of Htn

Anticipated Problems/Concerns

- Autonomic nervous system dysfunction associated with sudden death postop; can monitor for resp function in ICU/PACU overnight; presence of adult at home who can measure blood glucose and call 911
- Infections and end-organ risk substantially increased with blood sugar >250 mg/dL. Hypoglycemic symptoms hidden by autonomic nervous system dysfunction, effects of regional, sedative-narcotic, and β-adrenergic blocking agents.

**Diabetes, Type III (Gestational Diabetes Mellitus)**

**Risk**
- Incidence of gestational diabetes mellitus (GDM) is about 7% of all pregnancies
- Increased in African-American, Hispanic, Asian, Native American, or Pacific Islander women
- Risk factors are:
  - Maternal age > 25 y
  - Previous delivery of macroscopic infant
  - Glucosuria
  - History of polycystic ovarian syndrome
  - Previous unexplained fetal demise
  - Previous pregnancy with GDM
  - Strong immediate family history of NIDDM or GDM
  - Obesity
  - Fasting glucose >126 mg/dL or a glucose >140 mg/dL after a 50 g oral glucose tolerance test

**Perioperative Risks**
- Unlikely renal, ocular, cardiac, neurologic, or orthopedic complications in GDM
- Hypoglycemia if insulin is used

**ASSESSMENT POINTS**

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Possible facial/pharyngeal edema</td>
<td>Snoring</td>
<td>Neck ROM Mallampati exam</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>CV changes of pregnancy—possible worse hypovolemia from osmotic diuresis</td>
<td>BP/HR with orthostatic maneuvers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Resp changes of pregnancy, ↓ FRC, etc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Gastroparesis of pregnancy</td>
<td>Early satiety</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Neonatal hypoglycemia if maternal hyperglycemia, obesity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>No change, unless type I diabetes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>↓ Renal function</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>ANS dysfunction</td>
<td>Gastroparesis, early satiety</td>
<td>Orthostatic BP</td>
<td></td>
</tr>
<tr>
<td>PNS</td>
<td>Neuropathy not present unless type I diabetes</td>
<td></td>
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<td></td>
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</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Full-stomach precautions: Nonparticulate antibiotic administration usual

**Monitoring**
- Blood sugar in maternal and umbilical vein blood

**Airway**
- Examine for edema

**Induction**
- Regional anesthesia preferred to general anesthetic due to risks of aspiration and failed airway attainment if C-section is performed
- Fetal risk (if not controlled: polyhydramnios or macrosomia [6x normal])
- RDS (2–3x normal); preeclampsia, neonatal hypoglycemia, prematurity

**Worry About**

**Overview**
- GDM is defined as a carbohydrate intolerance that occurs (or is first recognized) during pregnancy.
- A glucose tolerance test is used to identify GDM. For details of the test, see the Key Reference.
- Maternal complications with GDM are few, but the fetus is at risk.
- Complications, such as fetal polyhydramnios, macrosomia (6x), prematurity, birth trauma, RDS (2–3x normal rate), neonatal hypoglycemia, or morbidity, are as common with type III diabetes (GDM) as with type I diabetes (insulin-dependent)
- ICD-9-CM Code: 648.0#

**Etiology**
- Occurs in genetically susceptible individuals
- Pregnancy, through secretion of substances from uterus, exerts diabetogenic effects

**Usual Treatment**
- Use of insulin in GDM is now more common as tighter control seems beneficial.
- Diet and exercise have been used in management.
- Insulin can be started if fasting glucose exceeds 105 mg/dL despite diet control.
- Oral hypoglycemic agents have been used, and there has been some success with glyburide.
- Many clinicians obtain a single HbA1c level at 6–12 wk gestation. In pts with mildly elevated plasma glucose levels and normal concentration of HbA1c, dietary modification alone and a modest increase in exercise are often sufficient to normalize plasma glucose levels.

**Postoperative Period**
- Usually GDM cured by delivery
- Women with GDM need a follow up GTT at 6–12 wk after delivery

**Anticipated Problems/Concerns**
- Fetal dysfunction, esp hypoglycemia and acidosis, if maternal hypoglycemia present
- Rapid changes in maternal blood glucose can accompany the pain and/or exertion of vaginal delivery of fetus and accompany the endocrine changes of uterine delivery.
Diabetes Insipidus

Risk
- Hereditary/familial (rare)
- Nephrogenic diabetes insipidus (DI) usually X-linked recessive transmission, but autosomal recessive and autosomal dominant forms exist; males/females
- May also be part of developmental syndromes (Wolfram syndrome, Lawrence-Moon-Beidel syndrome)
- Acquired
  - Trauma and/or surgery; infection; inflammatory, infectious, infiltrative, and/or neoplastic process affecting the hypothalamic-neurohypophyseal region
  - Renal disease (chronic renal failure, polycystic kidney disease, obstructive uropathy, renal transplantation)
  - Systemic conditions (multiple myeloma, sickle cell disease)
  - Electrolyte imbalances (hypokalemia, hypercalcemia)
  - Medications (amphotericin B, colchicine, loop diuretics, demeclocycline, lithium) or toxins (methoxyflurane/fluoride)
  - Gestational due to pregnancy-induced acceleration of vasopressin metabolism

Perioperative Risks
- Dehydration, hyperosmolality, hypernatremia
- Altered mental status/seizures
- Hemodynamic instability
- Bladder distention, hydrourerter

Worry About
- Fluid and electrolyte imbalance
- Contributing drugs and/or toxins (methoxyflurane/fluoride, lithium)
- Postop onset esp following pituitary surgery (1–6 d postop)

Overview
- Polyuria due either to insufficient production of vasopressin or inadequate renal tubular response to vasopressin
- Polyuria, excessive thirst, polydipsia; dehydration rarely present in competent pts with access to water
- Inadequate fluid replacement leads to hypernatremia, hyperosmolality, and dehydration causing fatigue, weakness, altered sensorium, hemodynamic instability, seizures, and possible death

ICD-9-CM Codes: 253.5, S88.1
ICD-10 Codes: E23.2, N25.1

ASSESSMENT POINTS

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<tbody>
<tr>
<td>CV</td>
<td>Hypotension Tachycardia Myocardial ischemia</td>
<td>Fatigue Weakness Reduced exercise tolerance</td>
<td>Orthostatic hypotension</td>
<td>ECG</td>
</tr>
<tr>
<td>ENDO</td>
<td>Anterior pituitary dysfunction</td>
<td>Pituitary surgery, neoplastic or infiltrative disease</td>
<td>Multisystem effects due to hormone deficiencies</td>
<td>Tests of anterior pituitary function, hormone levels</td>
</tr>
<tr>
<td>RENAL</td>
<td>Polyuria</td>
<td>Copious production of dilute urine</td>
<td>Urine volume and specific gravity</td>
<td>24-hr urine collection; simultaneous measurements of plasma and urine osmolality; exclude hyperglycemia, hypercalcemia, hypokalemia</td>
</tr>
<tr>
<td>CNS</td>
<td>Altered sensorium Visual disturbance</td>
<td>Excessive thirst Polydipsia</td>
<td>Neurologic function MRI</td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Perioperative Preparation**
- Vigorous 0.9N saline infusion (1–2.5 L) to restore hemodynamic stability, then use 0.5N saline, esp if serum osmolality is >310 mOsm/L.
- Insulin Rx usually begins with 0.1 U of regular insulin/kg IV bolus (in adults) followed by infusion of 0.1 U/kg/hr. Adjust insulin infusion to decrease glucose by 10% or 50 mg/dL per hr.
- Sodium bicarbonate not generally indicated, administer if pH <6.9, hyperkalemia, or pt hemodynamically unstable with pH <7.1.

**Monitoring**
- Check glucose, electrolytes hourly; check pH frequently: Foley catheter to determine urine output reliability during periop period; CVP catheter for fluid management, possibly PA catheter if pt has pre-existing myocardial dysfunction or CAD; consider TEE in hemodynamically unstable pt.

**Airway**
- Potential stiff joint syndrome with difficult intubation; at risk for aspiration.

**Induction**
- Hemodynamic instability likely if intravascular volume depletion not corrected; pts frequently have pre-existing autonomic neuropathy and CV dysfunction.

**Maintenance**
- Protect end-organs, esp heart, renal, and CNS as they are often compromised by DM.

**Exubtation**
- Awake. May require mechanical ventilation and ICU admission if pH <7.2, compromised mental status and/or high risk of aspiration.

**Adjuvants**
- See under Diabetes in Diseases section.

**Postoperative Period**
- Potential for hypoglycemic injury from rapid increase in insulin sensitivity when surgical cause of DKA corrected.
- Subsequent medical management should be continued by physician with expertise in diabetes.

**Anticipated Problems/Concerns**
- Hemodynamic instability from combined volume deficiency, acidosis, and pre-existing CV disease.
- CNS dysfunction from metabolic and electrolyte abnormalities, both early and late.
Diaphragmatic Hernia (Congenital)

Overview
- Classified by site of herniation
  - Posterior defects (Bochdalek) are left-sided (largest and associated with greatest degree of pulm hypoplasia. Morgagni hernias rare; parasternal, less symptomatic, therefore, diagnosed at later age.
  - Between 4–9 wk of age the pleuropertitoneal membrane forms with the left closing after the right. In Bochdalek and Morgagni normal development of the diaphragm and digestive tract does not occur.
  - Degree of lung hypoplasia determined by time of defect during fetal development and amount of abdominal contents in chest. Though ipsilateral lung most affected, both lungs are abnormal and result in decreased numbers and function of alveoli, hypoplastolic lung with smaller pulm artery and decreased arterial branching causes high vascular resistance.

ICD-9-CM Code: 756.6 (Congenital anomalies of diaphragm)

Risk
- Occurs in ~1/2500–5000 births; 12–25% have associated anomalies in particular cardiac (20%), chromosomal (5–16%), and neurologic
- Parents who have one child with isolated defect have 2% chance with next child
- Usually left sided (90%) due to defect in foramen of Bochdalek and are more common in boys. Morgagni hernias (2–5%) located anterior are more common in girls. Remainder through esophageal hiatus.

Perioperative Implications

Perioperative Management
- ECMO provides temporary support until perinatal circulation matures and less sensitive to vasocostrictive stimuli (1–2 wk)

Preoperative Preparation
- Avoid triggers for pulm vasoconstriction
- Goals incl a PaO₂ >80, Paco₂ 25–30, normal or elevated pH, and normothermia (hypothermia increases O₂ consumption)
- For pulm Htn can also use nitric oxide 20–80 ppm, sildenefil
- Avoid gaseous distension of stomach with early placement of NG tube
- Compromised neonates, ET intubation, sedation, paralysis and ECMO may be required if conventional or high-frequency ventilation fails
- All neonates with resp distress require invasive monitoring using preduectal Rt Radial a-line. Severe consider pre- and postductual a line and pulse oximetry

- IV access best in upper extremities to avoid possible IVC obstruction from increased intra-abdominal pressure
- Watch for pneumothorax (sudden deterioration in BP or oxygenation); consider prophylactic contralateral chest tube; equipment needed should be available.

Anesthetic Technique
- Opioids well tolerated; inhaled halogenated anesthetics may cause significant hypotension; avoid N₂O which distends gas-filled intestines
- Avoid peak pressures more then 25 cm H₂O
- High frequency, low tidal volume preferred
- Continue nitric oxide if given preop
- Lung mechanics change during surgery; may require hand ventilation

Indications/Usual Treatment
- Initial treatment involves determining the severity of associated congenital anomalies and degree of illness.
- Goal is semi-elective surgery when pt is medically stable.
- Posterior defects require surgical repair (does not resolve the pulmonary dysfunction)
- Small defects closed primarily; larger defects use artificial diaphragm, which contributes to postop resp failure.
- Most cases abdomen is closed primarily after correction but a silastic pouch may be used with increased intra-abdominal pressures.
- Fetal surgery has been accomplished in those severely affected with increased degree of pulm hypoplasia. Fetal Endoluminal Tracheal Occlusion (FETO) can be done to trigger lung growth. No maternal complications but preterm rupture of membranes is a drawback (20% <34 wk). Randomized control trials are ongoing to compare survival with controls.

Postoperative Considerations
- Continued muscle relaxation/opioids and ventilatory support
- Once stable, assess need for continued resp support
- If A—aDO₂ gradient >400 mmHg or if cardio-pulmonary deterioration, continue resp assistance
- Persistent hypoxemia while on high FIO₂ suggests persistent pulm Htn with Rt to Lt shunting
- Minimize ET suctioning, correct metabolic acidosis
- Deliver adequate nutrition
- High degree of neurologic problems, whether or not infants placed on ECMO; seizures, developmental delay, and hearing loss in 20–30% but pulm outcomes are usually good

ASSESSMENT POINTS

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<tr>
<td>CV</td>
<td>Mediastinal shift, associated ASD,VSD, coarctation, tetralogy of Fallot (23%)</td>
<td>Displaced cardiac impulse</td>
<td>CV exam</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp distress, pulm Htn</td>
<td>↓ Breath sounds on affected side</td>
<td>Pulmonary exam</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Malrotation, atresia (20%)</td>
<td>Prominent ipsilateral chest</td>
<td></td>
<td>ABG</td>
</tr>
<tr>
<td>GU</td>
<td>Hypospadias</td>
<td>Scaphoid abdomen</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Spina bifida, hydrocephalus, anencephaly (28%)</td>
<td>Inspection and neurologic exam</td>
<td>US, CT scan</td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Acidosis, hypoxemia, hypercarbia</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Diarrhea, Acute and Chronic

**Risk**
- Incidence in the USA: 200–300 million new cases/y of acute, with >900,000 hospital admissions
- Chronic: 1–5% of population; increasing with age
- Acute: male = female
- Chronic: female > male

**Perioperative Risks**
- Hypovolemia with hemodynamic instability
- Electrolyte abnormalities, esp hypokalemia
- Acid-base abnormalities: May be non–anion gap acidosis or alkalosis, depending on underlying cause
- Worry About
  - Chronic: Underlying disease, especially iatrogenic (e.g., infection with antibiotic-induced diarrhea, end-stage liver disease with lactulose-induced diarrhea, or disaccharide [usually lactose] intolerance)
  - Hormone-producing tumors (e.g., carcinoid, VIPomas, gastrinomas)
  - Vitamin K malabsorption with coagulopathy
  - Extra-intestinal manifestations of inflammatory bowel disease (IBD) (e.g., deforming arthritis, cholangitis)
  - Stress steroid therapy in IBD

**Overview**
- Acute: Ablupt onset of loose stools in healthy individual; Viral—self-limited, 1–3 d causing changes in small intestinal cells with a shortened transit time; bacterial—tends to occur in groups of individuals (if within 12 h of a meal, usually due to preformed toxins); protozoan—prolonged watery diarrhea from contaminated water supply in endemic area
- Chronic: Too-frequent passage of stools that are too loose for too long; >200 g/day of stool for >4 wk
- Multifactorial medical problem that requires supportive therapy and attention to the underlying etiology
- Only one in a spectrum of medical problems associated with an underlying disease or with treatment of disease. Supportive therapy includes fluid and electrolyte repletion and attention to acid-base balance.

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hypovolemia</td>
<td>Dysrhythmia 2° to electrolyte abnormalities</td>
<td>Postural symptoms, quantitation of bowel movements</td>
<td>Orthostatic changes, Narrow pulse pressure, Tachycardia, Dry mucous membranes</td>
</tr>
<tr>
<td>RESP</td>
<td>Compensatory hyperventilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Derangement dependent on underlying cause</td>
<td>BUN/Gr</td>
<td>Lab values include Ca²⁺, Mg²⁺, K⁺, HCO₃⁻, Na⁺</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Prerenal azotemia</td>
<td>Anemia—can be acute or chronic from acute GI losses or chronic disease state</td>
<td></td>
<td>Range from drowsiness to obtundation</td>
</tr>
<tr>
<td>CNS</td>
<td>Profound electrolyte abnormality</td>
<td>Stool guaiac</td>
<td>Stool guaiac</td>
<td></td>
</tr>
</tbody>
</table>


**Induction**
- Hemodynamic instability and decrease drug dosage if not repleted
- Sympatholytic drugs and sympathectomy with regional anesthesia can shorten transit time and increase diarrhea

**Maintenance**
- Tailor IV fluids to electrolyte and acid-base status (e.g., avoid normal saline if patient already has hyperchloremic acidosis)
- Continue electrolyte repletion if necessary

**Exsudation**
- Routine, dependent on underlying condition

**ICD-9-CM Code**: 558.9

**Etiology**
- Chronic:
  - Osmotic: Laxatives, indigestible carbohydrates
  - Secretory: Hormone-producing tumors
  - Exudative: Inflammatory bowel disease, pseudomembranous colitis
- Decreased mucosal contact/mixing: short bowel syndrome, irritable bowel syndrome, hypermotility secondary to vagotomy, diabetic neuropathy
- Malabsorption: Pancreatic exocrine insufficiency, celiac disease, Whipple's disease, small-bowel bacterial overgrowth
- Acute
  - Viral or bacterial (with or without toxin) or protozoan (see Overview)

**Usual Treatment**
- Volume and electrolyte replacement, including Na⁺, K⁺, PO₄⁻, Mg²⁺
- Although acid-base correction often follows above, may occasionally need replacement
- Seek and treat underlying cause

**Adjuncts**
- Acid-base status and electrolytes may affect muscle relaxant duration and ability of antagonists to reverse block

**Anticipated Problems/Concerns**
- Most operations do not affect underlying condition, but narcotics can make diarrhea less problematic, but use with caution in severe IBD as they may promote toxic megacolon
- Regional anesthesia that causes sympathectomy leaves parasympathetic system unopposed, which can cause shortened transit time and increase diarrhea

**ASSESSMENT POINTS**

- Toxidrome—extreme manifestation of inflammatory or infectious bowel disease is a surgical emergency. Pts often septic.
Dilated Cardiomyopathy (DCM)

**Risk**
- Incidence: 5 to 8 cases per 100,000 per year
- Racial predominance: African-Americans > Caucasians
- Gender predominance: Male > female
- Marked limitation of exercise capacity is a reliable predictor of mortality

**Perioperative Risks**
- Increased periop morbidity and mortality, particularly in high-risk surgery cases
- CHF exacerbation
- Renal failure
- Systemic or pulm embolization from dislodged intracardiac thrombi

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Arrhythmias CHF</td>
<td>Palpitations DOE Orthopnea</td>
<td>Narrow pulse pressure, pulsus alternans Displaced PMI</td>
<td>ECG, EPS ECHO</td>
</tr>
<tr>
<td></td>
<td>Myocardial ischemia</td>
<td>Angina</td>
<td>Systolic murmur (MR), S3, S4</td>
<td>Coronary angiography</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>Dyspnea</td>
<td>Rales, wheezes</td>
<td>CXR ABGs</td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulopathy</td>
<td>Bruising</td>
<td></td>
<td>PT/PTT</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Oliguria</td>
<td></td>
<td>BUN/Crea</td>
</tr>
<tr>
<td>CNS</td>
<td>Cerebral infarcts</td>
<td>Stroke</td>
<td>Focal neurologic deficits</td>
<td>CT, MRI</td>
</tr>
</tbody>
</table>


**Etiology**
- Cause of idiopathic dilated DCM remains unclear, but three possible basic mechanisms of damage: Familial and genetic factors; viral myocarditis and other cytotoxic insults; and immunological abnormalities

**Usual Treatment**
- Medical treatment primarily based on CHF treatment with diuretics, vasodilators, and β-adrenergic receptor-blocking agents; anticoagulants for thromboembolic prophylaxis; antiarrhythmics or ICD implantation for management of tachyarrhythmias
- Surgical treatment: Mitral valve annuloplasty, cardiomyoplasty, LVAD placement, heart transplant

**Perioperative Implications**

**Preoperative Preparation**
- Optimization of cardiac condition for anesthesia (consider cardiology consultation)

**Monitoring**
- ECG with ST-segment analysis
- Arterial line dependent on invasiveness of surgery
- Consider PA catheter if anticipation of large fluid shifts
- TEE is the monitor of choice for the assessment of biventricular function and AV valve regurgitation in invasive surgical cases

**Airway**
- None

**Worry About**
- Autonomic instability
- Malignant tachydysrhythmias
- Worsening LV systolic and/or diastolic function, RV dysfunction

**Overview**
- Syndrome characterized by dilatation and impaired systolic function of left, right, or both ventricles
- LV systolic and diastolic dysfunction (noncompliant ventricle), RV dysfunction; possibly pulm Htn and AV valvular regurgitation
- High risk of sudden cardiac death

**ICD-9-CM Code:** 425.4 (Cardiomyopathy, idiopathic)

### Diphtheria

**Risk**
- Incidence in USA: ~0.001 cases per 100,000 since 1980 (~5 cases a year)
- Endemic in developing countries
- Still common in countries where mass immunization programs are not enforced
- Risk factors for diphtheria outbreaks: Older age, lack of vaccination, alcoholism, low socioeconomic status, crowded living conditions, and Native American ancestry.

**Perioperative Risks**
- Early (days after exposure): Resp compromise; resp arrest; airway obstruction and hemorrhage; shock, coma, and death
- Late (2–6 wk): Myocarditis and neuritis

**Worry About**
- Resp diphtheria early toxic manifestations: Neck edema, pharyngitis, large pseudomembranes, massive swelling of the tonsils, bull-neck diphtheria (with massive edema of the submandibular and parapharyngeal region and foul breath, thick speech, and stridorous breathing), hoarseness and difficulty breathing are associated with severe advanced disease/poor prognosis, and with a significant early risk of airway obstruction
- Late toxic manifestations of diphtheria: Polynuropathy (resembles Guillain-Barré syndrome) and myocarditis (cardiac arrhythmias or CHF)
- Other complications: Septic arthritis, pneumonitis, renal failure, endocarditis, encephalitis, cerebral infarction, and pulpal embolism
- Fatal pseudomembranous diphtheria typically occurs in pts with nonprotective antibody titers and in immunized pts. Death occurs in 5–10% of resp cases. Risk factors for death incl “bulla-neck” diphtheria, myocarditis with ventricular tachycardia, atrial fibrillation or complete heart block, an age of >60 y or <6 mo, alcoholism, extensive pseudomembrane elongation, and laryngeal, tracheal, or bronchial involvement, and delayed antitoxin administration

#### Overview
- Prompt consideration of diphtheria: Severe pharyngitis, difficulty swallowing, resp compromise, or signs of systemic disease incl myocarditis or generalized weakness, and presence of a pharyngeal pseudomembrane or an extensive exudate
- Resp diphtheria: A sore throat with low-grade fever and an adherent membrane of the tonsils, pharynx, or nose. Occasionally, weakness, dysphagia, headache, and voice change. The diphtheritic pseudomembrane is gray or whitish, sharply demarcated and tightly adherent to the underlying tissue. Attempts to dislodge the membrane may cause bleeding.
- Systemic manifestations of diphtheria: Neuritis and polyneuropathy (cranial nerve involvement, resp and abdominal muscle weakness, generalized sensorimotor polyneuropathy and autonomic manifestations), and myocarditis (dysrhythmia of the conduction tract and dilated cardiomyopathy).
- Cutaneous diphtheria: Infected skin lesions and nonhealing or enlarging skin ulcers which lack a characteristic appearance. Cutaneous diphtheria has a low mortality rate, rarely associated with myocarditis or peripheral neuropathy.

**ICD9-CM: 032.**

### Etiology
- Corynebacterium diphtheria, an aerobic nonencapsulated, nonmotile, nonsporulating gram-positive bacillus
- Two human isolate phenotypes: Nontoxic and toxigenic. Toxigenic strains express diphtheria toxin (mechanism of pathogenesis during human systemic infection). Toxin produced in the pseudomembranous lesion and distributed to all organ systems through the blood. Toxigenic strains cause pharyngeal/resp diphtheria and systemic diseases, nontoxigenic strains cause cutaneous diseases.
- Direct person-to-person transmission via the aerosol route. Cutaneous lesions are also important in transmission. There are no significant reservoirs other than humans. The incubation period for resp diphtheria is usually 2–5 d.

#### Usual Treatment
- Prompt hospitalization in resp isolation with close monitoring of cardiac and resp function. Cardiac workup recommended.
- Start treatments as soon as possible even before confirmatory tests are completed due to the high potential for mortality and morbidity.
- Diphtheria antitoxin (DAT) available in USA only through CDC. DAT reduces the extent of local disease as well as the risk of complications of myocarditis and neuropathy. Rapid institution of DAT is associated with a significant reduction in mortality risk.
- Antimicrobial therapy: Procaine penicillin G 600,000 units (for children, 12,500–25,000 U/kg) IM 12 hr/ PO penicillin V 125–250 mg 6 hr daily to complete a 14-d course; or erythromycin 500 mg IV 6 hr (for children, 40–50 mg/kg/d IV in two or four divided doses) / PO erythromycin 500 mg Q 6 hr daily to complete a 14-d course alternative agents: Rifampin and clindamycin
- Sustained campaigns for vaccination of children and adequate boosting vaccination of adults.
- Resp diphtheria remains a notifiable disease in USA (National surveillance through the National Electronic Telecommunications System for Surveillance [NETSS]), whereas cutaneous diphtheria is not.

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>&quot;Membrane&quot; spread and hemorrhage</td>
<td>Altered speech, resp distress, croupy cough, hoarseness, chills, sore throat</td>
<td>Neck edema, fever, pharyngitis, large pseudomembranes, massive swelling of the tonsils, &quot;bull-neck&quot; diphtheria</td>
<td>Gram stain, culture of &quot;membrane,&quot; indirect laryngoscopy</td>
</tr>
<tr>
<td>CV</td>
<td>Conduction abnormalities, weak, laryngeal involvement, neck edema, fevers, pharyngitis</td>
<td>Dyspnea with minimal exertion, symptoms of CHF, palpitations</td>
<td>Tachycardia, ectopic beats, atrial fibrillation, signs of CHF</td>
<td>ECG, CXR, serum troponin I, echocardiography</td>
</tr>
<tr>
<td>RESP</td>
<td>See HEENT</td>
<td>Tachypnea, dyspnea, presence of membrane</td>
<td>Progressive resp compromise</td>
<td>Indirect laryngoscopy</td>
</tr>
<tr>
<td>HEME/IMMUNO</td>
<td>Systems compromised</td>
<td></td>
<td></td>
<td>CBC, blood cultures, PCR assays</td>
</tr>
<tr>
<td>GU</td>
<td>Proteinuria</td>
<td></td>
<td></td>
<td>UA</td>
</tr>
<tr>
<td>CNS</td>
<td>Interference with phonation, swelling, respirations</td>
<td>Symptoms depend on involved nerves</td>
<td>Cranial nerves (most often III, VI, VII, X), peripheral nerves (motor &gt; sensory)</td>
<td></td>
</tr>
</tbody>
</table>


### Perioperative Implications

#### Preoperative Preparation
- Initiate prompt treatments with diphtheria antitoxin and antimicrobial therapy
- Assessment of resp distress/airway compromise
- Assessment of cardiac involvement (for early detection of rhythm abnormalities. Initiate electrical pacing for clinically significant conduction disturbance and provide pharmacologic intervention for arrhythmias or for heart failure)
- Assessment of neurologic involvement
- Assessment of immunization status of exposed health care workers

#### Monitoring
- Maintain close monitoring of cardiac activity for early detection of rhythm abnormalities.
- Provide 2 large-bore IVs for pts with a toxic appearance; provide invasive monitoring and aggressive resuscitation for pts with septicemia
- Initiate electrical pacing for clinically significant conduction disturbance and provide pharmacologic intervention for arrhythmias or for heart failure.
• Consider pulm artery catheter or trans-esophageal echocardiography to assess degree of myocardial involvement

**Airway**
• Secure definite airway for pts with impending resp compromise or the presence of laryngeal membrane (careful manipulation as membrane will bleed if manipulated)
• Early airway management allows access for mechanical removal of tracheobronchial membranes and prevents the risk of sudden asphyxia through aspiration.

**Induction and Maintenance**
• Compensate for problems of exotoxin shock and possible CHF; cardiac arrhythmia

**Extubation**
• Early: May need prolonged ventilation
• Late: Cardiogenic shock/extensive polyneuritis may necessitate prolonged ventilatory support

**Adjuvants**
• Cardiac pacemaker for arrhythmia control and/or complete heart block
• Minimize use of sedative-hypnotics, as development of resp difficulties may be obscured

**Postoperative Period**
• Careful observation for resp, cardiac, and neurologic compromises

**Anticipated Problems/Concerns**
• Airway obstruction requiring tracheostomy/intubation
• Myocardial conduction problems that may necessitate electrical pacing
• Cardiogenic shock/CHF
• Neuritis that can present as a Guillain-Barré-like syndrome
Disseminated Intravascular Coagulation (DIC)

Risk
- Most common coagulopathy in the ICU.
- Incidence of DIC in severe sepsis is nearly 35%.
- The most important initiators of DIC are sepsis; trauma (hypovolemic shock, extensive tissue damage, fat embolism, head injury); surgery (neurosurgery, CPB); obstetric emergencies (hemorrhage, pre-eclampsia, retained products, amniotic fluid embolism); malignancy (acute promyelocytic leukemia, disseminated metastases) and severe liver disease. Vascular abnormalities, immunological reactions, toxins, and drugs can also cause DIC.
- Gender and race predominance: None.
- Mortality: High and dependant on underlying condition.

Perioperative Risks
- Underlying disease process and nature of surgical intervention
- Existing coagulopathy
- Organ dysfunction

Worry About
- Uncontrollable bleeding from surgical and anesthetic access sites
- Further ischemic damage to end-organs
- Transfusion complications

Overview
- A syndrome characterized by the pathological imbalance of the thrombotic and fibrinolytic systems leading to systemic intravascular coagulation and the deposition of fibrin in the microcirculation.
- Ongoing activation of the coagulation system results in the depletion of clotting factors and severe bleeding.
- Dx: There are no specific laboratory tests for DIC. DIC can be diagnosed clinically on the basis of the presence of a suitable risk factor along with a selection of laboratory findings: a rapidly falling platelet count or a count <100,000/mm^3; prolongation of clotting times (APTT, PT, INR); the presence of fibrin degradation products (FDPs); a reduction in plasma concentration of coagulation inhibitors (ATIII, Protein C).
- Serial testing showing temporal trends are valuable.

ICD9-CM: 286.6 (Defibrination syndrome)

Etiology
- DIC is initiated in one of two ways:
  - A systemic inflammatory response resulting in the activation of the complement pathway and the release of cytokines leading to systemic coagulation.
  - The activation of the extrinsic pathway of coagulation by the presence of increased concentrations of tissue factor.
- An impairment of fibrinolysis, which normally keeps coagulation localized, also plays its part in the progression of the syndrome.

Usual Treatment
- The primary goal is to remove the initiating stimulus and aggressive organ support.
- Mechanical ventilation, invasive monitoring and inotropic support are often required.
- Surgery intended to remove the cause of the DIC should be delayed no longer than is necessary to ensure hemodynamic stability.
- The early involvement of a hematologist.
- Continuous monitoring of the coagulation tests will guide the use of blood products.
- Blood products:
  - PRBCs for significant hemorrhage
  - FFP for clotting factor deficiencies
  - Cryoprecipitate infusions to maintain fibrinogen >100 mg/dL.
  - Platelet infusions to keep level >20,000/mm^3 (without hemorrhage) or >50,000/mm^3 (with hemorrhage)
- Pharmacological agents (controversial):
  - Heparin inhibits the activation of the coagulation process. Clinical trials have not shown improved outcomes but may have a role in the management of chronic (thrombotic) DIC.
  - Antithrombin III concentrates are also used to dampen down the process of thrombosis. Trials have shown improvements in the DIC, which haven’t translated into improved mortality.
  - Activated Protein C helps to restore the anticoagulation pathway and has been shown to improve mortality in the severest of DIC cases.
- ε-Aminocaproic acid is an antifibrinolytic agent and can be used in pts who continue to bleed.

Perioperative Implications

Preoperative Preparation
- Treat underlying cause
- Guided antibiotic therapy
- Correct coagulation where indicated
- Ensure blood product availability

Monitoring
- Routine
- Invasive where indicated by condition
- Serial CBC, coagulation studies and TEG

Airway
- Careful intubation to avoid trauma

Induction
- Consider full stomach
- Consider CV instability in shocked pts

Maintenance
- Resuscitation using invasive monitoring and laboratory tests to guide interventions

Exubtation
- Organ dysfunction and/or failure may necessitate a protracted period of mechanical ventilation in an ICU

Adjuvants
- Hepatic and/or renal failure increases the duration of action of most muscle relaxants

Anticipated Problems/Concerns
- Postop management is best conducted in an ICU environment.
- Hemorrhage may continue into the postop period.
- End-organ damage from ischemia inducing microthrombi may indicate prolonged organ support.
Diverticulosis

**Risk**
- More prevalent in developed countries. Common in the United Kingdom and other parts of northern Europe, North America, Australia, and New Zealand, but uncommon in southern Africa, the Middle East, the Far East, and the Pacific Islands.
- Prevalence in developing countries between 5–45% depending on age of population and method of diagnosis. African and Asian countries with prevalence around 0.2%.
- Prevalence increases with age. In USA, seen in less than 5% of pts younger than 40. 30% by age 60, 65% by age 85.
- Low-fiber diet is the highest risk factor. High fat and/or meat diets are high risk.
- Under age 50 y more common in men. Over 50 y more common in women.
- Colonic motility disorders contribute.

**Perioperative Risks**
- Pts who present with diffuse peritonitis or fail nonoperative management of acute diverticulitis may require emergency surgery.
- Risks may incl full stomach, obstruction, sepsis, and bleeding

**Overview**
- Multiple saclike herniations through weak points in the intestinal wall. Typically does not contain all layers of the wall but is a herniation of the mucosa and submucosa through the muscle layer.
- Vast majority (>90%) found in the sigmoid colon. Limited to the sigmoid in 65%, approx 25% involving sigmoid and other segments.
- Of pts with significant diverticulosis, 70% remain asymptomatic and without related complications.

**ICD-9-CM Code: 562.1 (Colon)**

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hypotension</td>
<td>Fatigue</td>
<td>Auscultation</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>Tachycardia</td>
<td>Weakness</td>
<td></td>
<td>BP</td>
</tr>
<tr>
<td></td>
<td>Hemodynamic instability</td>
<td>Angina</td>
<td></td>
<td>Pulm artery catheter</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxemia</td>
<td>Tachypnea</td>
<td>Auscultation</td>
<td>SpO₂</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dyspnea</td>
<td></td>
<td>ABGs</td>
</tr>
<tr>
<td>GI</td>
<td>Perforation</td>
<td>Abdominal pain</td>
<td>Diffuse abdominal tenderness</td>
<td>Free air under diaphragm if perforation</td>
</tr>
<tr>
<td></td>
<td>Obstruction</td>
<td>N/V</td>
<td>Rebound</td>
<td>CT scan</td>
</tr>
<tr>
<td></td>
<td>Abscess</td>
<td>Fever</td>
<td>Absent bowel sounds</td>
<td>Ultrasonography</td>
</tr>
<tr>
<td></td>
<td>Fistula</td>
<td>Abdominal rigidity</td>
<td>Abdominal rigidity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hemorrhage</td>
<td>Rectal bleeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, leukocytosis, DIC with sepsis</td>
<td>May pass air with urine if perforation into urinary bladder</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Colovesicular fistula</td>
<td></td>
<td>Urinalysis</td>
<td></td>
</tr>
</tbody>
</table>

**CNS**
- Disorientation with sepsis


**Perioperative Implications**

**Monitoring**
- Routine, incl urine output
- With sepsis, monitor arterial pressure; consider PAC monitoring

**Maintenance**
- Optimize intravascular volume and high O₂ content

**Postoperative Period**
- Maintain intravascular volume
- Continued monitoring of CV variables and urine volume

**Adjuvants**
- Antibiotics
- Volume expanders
- Component therapy if DIC develops
- Vasopressor support if required; no interactions

**Anticipated Problems/Concerns**
- Condition is chronic so flare-ups may occur. Diverticulitis may progress to uncomplicated diverticulitis and evolve to the complicated form (abscess, perforation, obstruction, bleeding, fistula)
- Any surgical intervention and bowel resection would therefore have the anticipated side effects and complications expected from that procedure.
Do Not Resuscitate (DNR) Orders

**Risk**
- Violation of pt autonomy and self-determination if DNR orders aren’t reconsidered and honored for the periop period
- Increasing numbers of pts have some form of advance directive
- Approx 15% of surgical pts have DNR orders

**Perioperative Considerations**
- Resuscitation preferences can change based on pt status and prognosis
- DNR orders do not become automatically suspended or continued when a pt goes to surgery
- Intraop arrests tend to have better outcomes because they are witnessed, acted upon quickly, and are often due to reversible causes
- Pts with DNR orders often undergo vascular access procedures, gastrostomy tube placement, tracheostomy, palliative procedures, repair of pathological fractures, and surgery for emergent conditions (bowel obstruction, appendicitis, etc.)

**ASSESSMENT POINTS**
- What are the pts wishes?
- When was the DNR order written/last updated?
- Why was the DNR order initiated?
- Did the pt have a terminal condition?
- Did the pt have correct prognostic information?
- Who discussed/wrote the DNR order with the pt?
- Did the physician influence the decision to have the DNR order?


**Perioperative Implications**
- Review “required reconsideration” of the DNR orders
- All changes to DNR status must be communicated to all members of the periop team and documented in the pt’s medical record.
- Best if discussion of DNR orders can be done preop to develop a better pt-doctor relationship, avoid production pressure influences, and to allow time to contact all appropriate parties (surrogate, surgeon, primary care physician).
- This discussion should incl what procedures are essential for the anesthetic and operation (i.e., intubation paralysis, etc.); iatrogenic arrest; and if the DNR order is modified when and if it should be reinstated.
- The document for Informed Consent for Anesthesia Care in The Patients with An Existing Do-Not-Resuscitate Order created by The American Society of Anesthesiologists Committee on Ethics provides three resuscitation options during the periop time period:
  - Full resuscitation
  - Limited resuscitation: Procedure-directed, documents specific procedures the pt refuses
  - Limited resuscitation: Goal-directed, allows resuscitation if the anesthesiologist and surgeon believe the adverse events are temporary and reversible. Allows resuscitation if the anesthesiologist and surgeon believe the resuscitation efforts support specified and documented goals of the pt
  - Consider consultation with an ethics expert if there is disagreement or concern about DNR orders and the surgery isn’t emergent.

**Anticipated Problems/Concerns**
- Anesthesiologists rarely have an established relationship with the DNR pt, but must discuss and clarify resuscitation wishes.
- Aspects of anesthesia care (intubation, vasoressors, IV fluid therapy, transfusion, etc.) are resuscitative therapies.
- Medications used for anesthesia may cause cardiac depression, resp and cardiac arrest
- Anesthesiologists may be morally conflicted with the pt’s desire for limited intervention. For a nonemergent case, the anesthesiologist can decide not to perform the anesthetic as long as there is another available physician and the change is not detrimental to the pt.

**Worry About**
- Ethical and legal obligation to honor and follow pt’s wishes as well as provide optimal medical care
- Appropriateness of the DNR order
- Delineation of anesthesia care and resuscitation
- Iatrogenic events
- Intraop deaths
- Liability

**Overview**
- The Patient Self-Determination Act (1990) was established to allow pts to avoid undesired medical interventions. It requires federally-funded healthcare institutions to ask pts about advance directives when admitted and provide information about their right to have one (Medicare and Medicaid are federally funded).
- The 1983 Report of the President’s Commission for the Study of Ethical Problems in Medicine justified the “favoring of resuscitation of hospitalized pts with unexpected cardiac arrest”— which conveys implicit pt consent for CPR
- CPR is the only medical intervention that requires an M.D. order to be withheld.
- A DNR order is a limited advance directive which prevents resuscitative intervention in the event of a cardiopulmonary arrest.
- Many pts with DNR orders are terminally ill or have advanced disease.
- Policies should be set in place for re-evaluation of DNR orders for pts requiring surgery. These policies should be institutional, written, unambiguous, and flexible to individual pt needs.
- Anesthesiologists should be familiar with their institution’s policies as well as state and federal laws.
Double Aortic Arch

**Risk**
- Vascular rings account for <1% of CV malformations that require surgical correction; double aortic arch is the most common form of complete ring that encircles both the trachea and the esophagus.
- Race and gender predilection: None

**Perioperative Risks**
- Recurrent resp infections often aggravate chronic airway obstruction.
- Baseline dynamic tracheal compression can progress to complete airway obstruction upon induction and muscle relaxation.
- Persistent postop airway obstruction requiring prolonged mechanical ventilation and CPAP

**Worry About**
- Esophageal obstruction: Dysphagia, choking, emesis, aspiration, FTT
- Tracheal obstruction: Chronic cough, wheezing, barky-brassy cry, inspiratory stridor; acute episodes of severe resp distress, apnea, cyanosis, ALTE
- Associated cardiac anomalies (10–20%): VSD, interrupted aortic arch, transposition of the great arteries, tetralogy of Fallot, truncus arteriosus, complex univentricular sinus, complex univentricular defects incl endocrine abnormalities (hypocalcemia, thyroid/parathyroid dysfunction, short stature), palatal and laryngotracheal abnormalities, developmental delay/neurologic abnormalities, renal tract malformations, thrombocytopenia, T-cell deficiencies, and autoimmune disorders.

**Overview**
- Vascular rings can be classified as complete or incomplete; double aortic arch is the most common form of complete ring that encircles and compresses both the trachea and esophagus.
- Symptoms usually occur at birth or within the first 3 mo of life; the degree of tracheal and esophageal compression will dictate the severity of resp and GI perturbation.
- Initial work-up with CXR and UGI can reveal tracheal deviation and/or narrowing and proximal esophageal distention/indentation. Once diagnosis suspected, ECHO is used to examine arch anatomy and rule-out other intra-cardiac anomalies. Both MRI and CT are very useful in further delineating vascular, airway, and GI anatomy. Catherization is now reserved for assessing complex cardiac defects that require additional hemodynamic information. Bronchoscopy is often performed at the time of repair to evaluate the location, degree, and extent of airway obstruction, which may help identify those pts at risk for postop resp compromise. ICD-9-CM Code: 747.21

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Chromosome 22q11 deletion syndrome (20%): Deletion features:</td>
<td>Previous difficulties w/anesthesia or intubation</td>
<td>Low set ears, short philtrum,</td>
<td>Flexible bronchoscopy</td>
</tr>
<tr>
<td></td>
<td>Facial abnormalities</td>
<td>FTT</td>
<td>hypertelorism, small mouth,</td>
<td>Direct laryngoscopy/bronchoscopy</td>
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<tr>
<td></td>
<td>Palatal abnormalities</td>
<td>Nasal regurgitation of formula; delayed speech/poor articulation (childhood)</td>
<td>Cleft palate</td>
<td></td>
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<tr>
<td></td>
<td>Velopharyngeal incompetence</td>
<td></td>
<td>Hypernasal speech (childhood)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Congenital laryngeal web</td>
<td>Noisy breathing, abnormal cry</td>
<td>Insp/expiratory stridor; aphon</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Depends on presence of associated cardiac anomalies (10–20% cases)</td>
<td>Cyanotic spells, CHF, dyspnea, diaphoresis, FTT</td>
<td>weak high-pitched cry; hoarsness</td>
<td>Pulse oximeter, EKG</td>
</tr>
<tr>
<td></td>
<td>None if only double aortic arch present</td>
<td></td>
<td>(childhood)</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac MRI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac catheterization</td>
</tr>
<tr>
<td>RESP</td>
<td>Airway obstruction</td>
<td>Murmur, cyanosis, 4-limb NIBP discrepancy, grunting, rales/wheezes, hepatosporen</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recurrent resp infection</td>
<td></td>
<td>hemoglobinemia/</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Dyspnea, apnea, intermittent cyanosis, ALTE</td>
<td></td>
<td>CXR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Coughing; wheezing</td>
<td></td>
<td>Bronchoscopy</td>
<td></td>
</tr>
<tr>
<td>GL</td>
<td>Esophageal obstruction</td>
<td></td>
<td>MRI</td>
<td></td>
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<td>CT</td>
<td></td>
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<tr>
<td></td>
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<td>UGI</td>
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</tr>
</tbody>
</table>

**Induction**
- Inhalation induction without neuromuscular blockade until airway maintenance is documented by mask and/or ETT is placed distal to area of obstruction.
- Bronchoscopy during spontaneous ventilation allows for direct assessment of tracheal pathology and degree of dynamic airway collapse, thus identifying pts at risk for postop resp compromise.

**Potential for hemodynamic and resp instability warrant placement of arterial catheter; presence of an aberrant subclavian artery may affect appropriate catheter site**

**Preoperative Preparation**
- O2 therapy if decreased arterial O2 saturation present
- Antibiotics for bronchopneumonia

**Monitoring**
- Bilateral upper extremity pulse oximetry and Doppler probe are useful for assessing subclavian, carotid and temporal pulses during temporary occlusion of the arch that is to be resected.


**DISEASES**
Maintenance
• Balanced technique of narcotics and volatile agent is usually well-tolerated

Extubation
• Extubation at end of case if tracheomalacia and stenosis absent

Postoperative Period
• Good pain control essential for stable hemodynamics and avoidance of resp complications; IV opioids, rectal acetaminophen, intercostal nerve blocks, one-shot caudal, and caudal epidural catheters have all been used with success.

Anticipated Problems/Concerns
• Despite surgical correction, persistent postop airway obstruction requiring prolonged mechanical ventilation and CPAP can occur secondary to edema, mucosal friability and/or reactivity, and long-segment tracheomalacia.
Down Syndrome

Risk
- Incidence in USA: >300,000
- 80% of children with this condition survive beyond 1 y
- Number >50 y will increase by 200% by the year 2010
- Male-female ratio 3:2
- No racial preponderance

Perioperative Risks
- Related to specific abnormalities in individual

Worry About
- Congenital heart disease: 50% born with congenital heart disease (CHD), 8% with cyanotic CHD (usually tetralogy of Fallot)
  - May become profoundly hypoxic with right to left shunting; accidentally injected air bubbles may exit into systemic circulation (coronary and cerebral air emboli)
  - Adults less likely to have CAD
- Upper airway obstruction
  - Soft tissue obstruction of upper airway common immediately on induction of GA due to large tongue, small mandible, short neck

Overview
- Not a disease
- Incidence decreased by prenatal screening and elective termination of pregnancy
- Wide variation in abilities and disabilities; neurologic development enhanced by external stimulation
- Institutionalized individuals have high incidence of seropositivity for hepatitis B
- More people living in group homes in community and becoming more self-sufficient in ADL

ICD-9-CM Code: 758.00

Etiology
- Genetic: trisomy 21
- Risk of parenting a Down syndrome fetus greatest in older (>35 y) parents (well characterized in mothers)

Usual Treatment
- Depends on pathophysiology

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Large tongue, Subglottic stenosis, Hearing deficit in 66%</td>
<td>Hx of snoring, Sleep apnea, Intubation Hx</td>
<td></td>
<td>Audiology</td>
</tr>
<tr>
<td>CV</td>
<td>Tetralogy of Fallot in 4%</td>
<td>Sx of CHF “Tet spells”, Cyanosis, Murmur</td>
<td></td>
<td>ECHO</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypothyroidism, Obesity</td>
<td>Hypothermia, Obesity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Subluxation of C1/C2 Joint laxity</td>
<td></td>
<td></td>
<td>Cervical spine radiographs (controversy over whether these should be routine)</td>
</tr>
</tbody>
</table>


Perioperative Implications

Monitoring
- Temperature (hypothermia); ECG (arrhythmias, ischemia); treat bradycardia from halothane or sevoflurane

Airway
- Have variety of alternative airway management devices available (e.g., oral and nasal airways, laryngeal mask, Bullard laryngoscope)
- Avoid neck extension during laryngoscopy if possible

Anticipated Problems/Concerns
- Hypoxia if right to left shunting develops
- Resistance to separation from caregiver
- Life-threatening upper airway obstruction with difficult vascular access
- Spinal cord ischemia with neurologic damage

System Effect
HEENT
CV
ENDO
MS

Assessment by Hx
PE
Test

Hx of snoring, Sleep apnea, Intubation Hx
Sx of CHF “Tet spells”, Cyanosis, Murmur
Hypothermia, Obesity

Cervical spine radiographs (controversy over whether these should be routine)
Diseases

134

Risk
• True prevalence of LSD use impossible to determine.
• People began using it for recreational and spiritual purpose in 1960s. LSD is still illegally used as a major hallucinogen worldwide.
• LSD-related hospital visits remain low compared with those related to other major illicit drugs.

Perioperative Risks
• Acute intoxication has evidence of sympathetic nervous system stimulation incl mydriasis, increase body temp, systemic Htn, tachycardia, anxiety, agitation, vomiting, aspiration, apnea, and unrecognized injuries.
• May prolong succinylcholine neuromuscular blockade and delay metabolism of ester local anesthetics (speculated inhibition of plasma cholinesterase).
• May potentiate analgesics.

Worry About
• Systemic: Htn, tachycardia, hyperthermia, hyperglycemia, salivation, nausea, vomiting, seizures, and apnea
• Psychiatric: Hallucinations (visual, auditory, and tactile), labile mood, acute panic attacks, agitation, and hypertonla

Overview
• Lysergic acid diethylamide (LSD) is a semi-synthetic product of lysergic acid, a natural substance from the parasitic rye fungus Claviceps purpurea
• LSD is physiologically well tolerated, psychological complications result from overdose or uncontrolled use by layman
• There is high degree of psychological dependence but no evidence of physical dependence or withdrawal symptoms when acutely discontinued.
• Classified under Schedule I of the Controlled Substance Act

Perioperative Implications

Preoperative Preparation
• Rule out associated traumatic injury
• Hemodynamic control
• Aspiration prophylaxis
• Sedation if agitation is severe

Monitoring
• Temp
• Neuromuscular blockade

Airway
• Aspiration risk

Preinduction/Induction
• Salivation, N/V, which may warrant rapid sequence induction

• Ketamine should be avoided, which may have synergic effects with LSD
• Succinylcholine should be avoided
• Exaggerated response to endogenous and exogenous catecholamines

Maintenance
• Maintain normothermia

Exubation
• At risk for aspiration
• Continue supportive reasurance

Adjuvants
• May have exaggerated response to sympathomimetic agents

• Psychologic effects begin in 30–60 min and may last 8–12 hr

ICD-9-CM Code: 305.3

Etiology
• Mechanism of action is still unknown. Stimulation of sympathetic and parasympathetic system, central stimulation of sympathetic system, activation of higher cortical centers causes typical clinical effects.

Usual Treatment
• Supportive reassurance, transfer pt to calm, quiet area with minimum external stimuli
• Benzodiazepines seem to be the most effective agents for treating LSD psychosis and visual disturbances
• Rare cases require hemodynamic control, intubation, ventilatory and supportive care

Drug Overdose, Rat Poison (Warfarin Toxicity)

Risk
• Major risk is hemorrhage, esp CNS or GI.
• Incidence: Risk of hemorrhage in 1.0%-7.4% of pts chronically anticoagulated. Risk is dose-related and proportional to PT prolongation. Risk of hemorrhage doubles as INR increases from 2.0-2.9 to 3.0-4.4. It further quadruples as INR increases from 3.0-4.4 to 4.5-6.9. (2) Age is associated with increased sensitivity to warfarin and increased incidence of bleeding complications.
• Rx for DVT, cerebral vessel atherosclerosis, prosthetic heart valves, mitral stenosis, paroxysmal atrial fibrillation.

Perioperative Risks
• Bleeding
• Drugs that potentiate anticoagulant effects: Antibiotics (esp metronidazole, sulfonamides, cephalosporins), NSAIDs, phenytoin, cimetidine, barbiturates, alcohol

Worry About
• Bleeding complications of invasive procedures
• Drug interactions

Preoperative
• Increased effect: Antibiotics, NSAIDs, oral hypoglycemic, diazepam, cimetidine, diuretics, phenytoin
• Decreased effect: Methylxanthines, rifampin, antihistamines, corticosteroids, barbiturates
• Major surgical procedures warrant discontinuation of drug 1-3 d preop with a target PT within 20% of nl range. Alternatively, pt may be admitted 1-2 d prior to surgery. Warfarin is discontinued and heparin therapy instituted. Heparin is discontinued 6 hr prior to surgery.
• For emergency surgery, pt may be given 10-20 mL/kg of FFP and 5-10 mg of vitamin K, with additional amounts of both given as needed

Adjuvants/Regional Anesthesia/Reversal
• Regional block: Relatively contraindicated without reversal of anticoagulation
• Peripheral block: Relatively contraindicated without reversal of anticoagulation

Anticipated Problems/Concerns
• Relatively minor surgical procedures may be performed without reversal of warfarin anticoagulation.
• The appropriate use of prothrombin concentrate complex (PCC) therapy for warfarin toxicity is controversial. PCC contains a combination of factors II, VII, IX, and X, and are given at a dose of 25-50 U/kg. Benefits incl smaller infusion volumes, no need for blood group testing, the fact that it is a virally inactivated product, and higher levels of factor IX compared to FFP. Evidence for significantly more rapid and more complete correction of INR with the use of PCC versus FFP in the setting of intracranial hemorrhage.
• Recommendation in the European Stroke Initiative guidelines to preferentially use PCC rather than FFP in the setting of warfarin-associated intracranial hemorrhage.


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<tbody>
<tr>
<td>HEME</td>
<td>Abnormal levels of factor II, IV, IX, X, and protein C, protein S</td>
<td>Easy bruising, prolonged bleeding time</td>
<td>Ecchymoses</td>
<td>PT</td>
</tr>
</tbody>
</table>

Possible Drug Interactions
• Transient protein C deficiency preceding effect on procoagulant levels at initiation of warfarin therapy leading to thrombotic complications
• True poisoning with rodenticides (so-called super-warfarins) may result in prolonged clotting abnormality with abnormal PT values weeks to months post event because of the enormously long half-lives of these drugs

Overview/Pharmacology
• Vitamin K antagonist.
• Cleared by hepatic and renal transformation and excretion. T1/2 is ~40 hr. Duration of action is 2-5 d.
• Onset of effect is delayed by 8-12 hr because of time required to clear already synthesized clotting factors. For similar reasons, peak effect of a dose occurs 48 hrs post-administration.

ICD-9-CM Code: 286.9 (Coagulation defect)

Drug Class, Mechanism of Action, Usual Dose
• Blocks vitamin K–mediated carboxylation of factors II, VII, IX, X (procoagulants); protein C, protein S (anticoagulants)
• Carboxylation of coagulation factors oxidizes vitamin K. The vitamin K epoxide must be reduced to become active again. Coumarin anticoagulants block reduction of the epoxide. Thus, large and/or repeat doses of vitamin K are needed for large overdoses or for long-acting forms
• Chronically taken for systemic anticoagulation for DVT, CVA, prosthetic valves, and atrial fibrillation either paroxysmal or associated with mitral stenosis
• Usual doses: Loading regimen varies, but maintenance dose is 2.5-10 mg/d
• Alternatives: None. An oral direct thrombin inhibitor investigated as a possible alternative to warfarin, ximelagatran, was withdrawn from distribution in 2006 because of hepatotoxicity. Heparin is drug of choice for acute anticoagulation, but must be given parenterally, usually as loading dose with an infusion

* Not all PCC formulations are identical. There may be relative factor VII deficiency, requiring concurrent VII administration. PCC also generally considered thrombogenic, although newer formulations may incl the coagulation inhibitors Protein C and Protein S.
• rFVIIa has also been reported to successfully correct INR in the setting of warfarin toxicity. The existing literature contains case reports and case series that describe administration of 20-135 ucg/kg of rFVIIa in various clinical settings, leading to correction of INR.
• Hypothermia will potentiate anticoagulant effect.
Duchenne Muscular Dystrophy  
(Pseudohypertrophic Muscular Dystrophy)

**Overview**
- Most boys die from pneumonia but CHF is also seen in the later stages.
- Gradual onset of muscle wasting, eventually replaced by fat causing pseudohypertrophy.
- Hyperkalemic response to depolarizing NMBs may develop years before the onset of DMD symptoms. The infant may appear entirely normal, with only mild gross motor delay.
- Increased sensitivity to nondepolarizing NMBs.
- Use of Ca²⁺-channel blocker (e.g., verapamil) may prolong or even cause NMB.
- Tendon releases for contractures.
- Exploratory laparotomy for ileus.

**Risk**
- Males, 1/3500; few known cases in females.
- Often undiagnosed until age 3–5 y.
- Deterioration through puberty to death usually before age 25 y.

**Perioperative Risks**
- Resp failure, prolonged mechanical ventilation.
- Muscle weakness.

**Worry About**
- Poor cardiac function, dilated cardiomyopathy, cardiac arrhythmias, MVP.
- Use of Ca²⁺-channel blocker (e.g., verapamil) may prolong or even cause NMB.
- Up to a quarter of pts may have mitral valve prolapse.
- Resting tachycardia common; cardiac involvement in 70% of cases, cardiac debilitation usually late.

**ICD-9-CM Code:** 359.1

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**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Conduction</td>
<td>Tachycardia (Hx CHF Sx: Orthopnea, DOE, PND)</td>
<td>Opening snap (MVP)</td>
<td>ECG, 24-hr ambulatory ECG, ECHO, MUGA</td>
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<tr>
<td>RESP</td>
<td>↓ Volume and flows</td>
<td>SOB</td>
<td>Unreliable</td>
<td>PFTs, SaO₂ on RA, sleep study ECHO</td>
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<tr>
<td>GI</td>
<td>Dysmotility, gastric dilatation, paralytic ileus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Bladder paralysis</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>CNS</td>
<td>↓ IQ</td>
<td>Progressive weakness</td>
<td>Mental status exam</td>
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<tr>
<td>MS</td>
<td>Scoliosis, kyphosis, Contractures, Muscle destruction, Macroglossia, Poor IV access</td>
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</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Avoid or limit preprocedure sedation.

**Monitoring**
- Consider PA catheter and/or TEE based on EF and surgical procedure.
- Nerve stimulator.

**Induction**
- Succinylcholine contraindicated because of hyperkalemia.
- Avoid volatile anesthetics 2° to MH-like response.
- Avoid depressants of cardiac contractility.
- Consider long gastric emptying times, possible full stomach.

**Maintenance**
- Variable response to NM blockers; titrate to effect.
- Consider a total IV anesthesia approach.
- Regional or neuraxial anesthesia when possible over GA.
- Recommended to allow spontaneous recovery, as response to reversal agents varies.
- Avoid hypoxygenia, large fluid shifts, and anemia to prevent uncompensated cardiomyopathy.

**Emergence**
- Potential for prolonged ventilator dependence greatest when vital capacity <30% of predicted.
- For GA cases consider extubating directly to BIPAP and/or CPAP, weaning later.
- Late resp depression reported (cause unclear); may make outpatient surgery inadvisable.

**Anticipated Problems/Concerns**
- Resp failure.
- CHF.
- Supraventricular tachydysrhythmias.
- Rhabdomyolysis, hyperkalemia, and cardiac arrest in response to succinylcholine and volatile agents have been described in boys years before clinical signs of DMD present.
- Late resp depression reported (cause unclear); may make outpatient surgery inadvisable.
### Duodenal Atresia

**Risk**
- Incidence 1/10,000–40,000 live births
- M:F incidence is equal
- 20–30% have trisomy 21
- 45% are premature infants of pregnancy complicated by polyhydramnios
- Mortality 10%, due not to duodenal atresia but to associated CHD or prematurity

**Perioperative Risks**
- Hypoxemia associated with immature lungs
- Hypoxemia due to CHD, persistent fetal circulation

**Worry About**
- Ventilation problems associated with prematurity

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<tbody>
<tr>
<td>CV</td>
<td>CHD—ASD, VSD, AV canal, Persistent fetal circulation</td>
<td>Trisomy 21</td>
<td>Murmur, Cyanosis</td>
<td>ECHO, CXR, Pulse oximetry</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp distress syndrome of prematurity</td>
<td>Polyhydramnios Gestational age &lt;36 wk</td>
<td>Tachypnea Retractions Flaring Grunting</td>
<td>CXR, Pulse oximetry</td>
</tr>
<tr>
<td>GI</td>
<td>Duodenal obstruction Associated esophageal atresia</td>
<td>Bilious vomiting No gas in abdomen</td>
<td>Scaphoid abdomen</td>
<td>Abdominal x-ray, Unable to pass OG tube</td>
</tr>
<tr>
<td>RENAL</td>
<td>Structural anomalies</td>
<td>Palpation of kidneys</td>
<td>Abdominal US</td>
<td></td>
</tr>
</tbody>
</table>


### Perioperative Implications

**Preoperative Preparation**
- OG tube to decompress stomach, reduce gastric contents
- IV catheter placement with hydration (20 mL/kg NS) if diagnosis delayed beyond 24–48 hr; correct electrolyte abnormalities
- Surfactant for premature infants with significant lung disease
- Confirm intramuscular vitamin K given as part of normal newborn care

**Monitoring**
- Arterial monitoring for ABGs, electrolyte, and Hgb determination only in premature infants with significant lung disease, those with CHD, or those with extreme dehydration due to protracted vomiting; otherwise NIBP sufficient as minimal blood loss expected
- Temperature
- Urinary catheter (small feeding tube) may be helpful in assessing adequacy of fluid resuscitation
- Pulse oximetry, ECGs, and end-tidal carbon dioxide and gas monitoring

**Anesthetic Technique**
- Suction OG tube with infant supine and in left and right decubitus positions prior to induction, intubation
- Awake intubation after preoxygenation only for actively vomiting, volume-depleted infants with abnormal airway anatomy
- Rapid-sequence induction after preoxygenation for normovolemic pts with normal airway anatomy
- Avoid N₂O to prevent intestinal distention

- Other associated anomalies in 50% of cases: Esophageal atresia (7%), other intestinal atresias, renal anomalies (5%), malrotation of the gut (25%), volvulus, imperforate anus (3%), annular pancreas (25%)
- CHD associated with trisomy 21 (ASD, VSD, AV canal)
- Aspiration on induction of anesthesia 2° to bowel obstruction
- May be associated with cystic fibrosis
- Late presentation can be associated with dehydration, hypovolemia, and hypochloremic alkalosis

**Overview**
- Frequently premature infant of pregnancy complicated by polyhydramnios
- Vomiting after birth: May be copious and bile stained
- Flat abdomen
- Dx is made by “double bubble” on abdominal x-ray

**ICD-9-CM Code:** 751.1 (Atresia, small intestine)

**Etiology**
- Unknown in sporadic cases
- More common in trisomy 21

**Usual Treatment**
- Surgical repair is curative
- Surgical technique may be open laparotomy or laparoscopic

**Anticipated Problems/Concerns**
- Prematurity/respiratory distress syndrome/apnea
- CHD
- Hemorrhage, air, or gas embolus may occur at start of laparoscopic procedure
- Risk of aspiration may continue postop—leave OG or NG tube in place
- Later risk of GE reflux higher than normal (17%)
- Adequate fluid replacement
- Other associated anomalies
Echinococcosis

Risk (Epidemiology)
• Men = women
• *Echinococcus granulosus* causes cystic echinococcosis (hydatid disease) in people exposed to feces of dogs and other canids in endemic areas of nearly every continent.
• *E. multilocularis* causes alveolar echinococcosis in people exposed to infected dogs living in colder regions of the northern hemisphere.
• *E. vogeli* and *E. oligarthus* cause polyctic echinococcosis in people exposed to infected dogs and wild carnivores in rural Central- and South America.

Perioperative Risks
• Hydatid cyst rupture leads to anaphylaxis and spread of encapsulated organisms, which implant in exposed tissues (e.g., peritoneal cavity), later causing disseminated hydatidosis (bowel obstruction, cachexia, death).
• Failure to resect all echinococcal tissue due to microscopic or extensive disease extension.
• Hemorrhage (if cyst attached to liver or major blood vessel).
• Systemic reactions to toxic agents instilled into cyst cavity; air embolism if cyst attached to a vein or hydrogen peroxide instilled into cyst cavity.
• Postop jaundice, cholangitis, bacterial superinfection, vascular compression, hepatic failure.

Overview and Etiology
• Parasitic disease caused by organism classified as flatworm (adult stage). Parasite cycles through 4 different stages (adult tapeworm, egg, oncosphere, metacestode) each adapted to maximize survival in the 2 host organisms:
  - *Definitive host*: Carnivore; intestines contain adult flatworms releasing eggs into feces.
  - *Intermediate host*: Herbivore/omnivore (sheep, small rodents, man); ingests minute amount of feces of definitive host, eggs hatch in stomach and release *oncospheres* (first larval stage), which penetrate gut blood vessels and distribute to potentially any organ, esp liver and lung. Develop into slowly expanding fluid-filled cysts (*metacestodes*). Inner (germinal) layer of metacestode buds off tiny encapsulated *protopolocyes* [Gr: juvenile heads] which accumulate to form *hydatid sand*.
• A definitive host eats infected organs of intermediate host; *protoceles* are released into intestinal lumen; these evaginate; anterior parts attach to intestinal epithelium and become adult tapeworms.

Adult *E. granulosus* (2–11 mm) inhabits small intestine of canid (dog, wolf, coyote, dingo, jackal); eggs distribute to grass eaten by sheep, goats, camels, yaks, cattle, pigs, horses, marsupials; man becomes infected via hand—oral contact with fecally-contaminated object. Cysts of volume up to 1000 mL form within intermediate host (or man—sometimes called *dead end host*), physically compromising organ function.

Adult *E. multilocularis* (1–5 mm) inhabits small intestine of fox (occasionally dog, bush dog, rarely cat); intermediate host usually a rodent. Cysts become multiple and invade target organs.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GEN</td>
<td>Liver mass (70%), biliary</td>
<td>Pt from endemic area, fever, itching, family Hx</td>
<td>Fever—if high, possible superinfection</td>
<td>US, CT, or MRI imaging of any part of body</td>
</tr>
<tr>
<td>GI</td>
<td>Abdominal pain dyspepsia,</td>
<td>Jaundice; signs of cirrhosis</td>
<td>Abdominal US, CT or MRI; PT/INR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>vomiting fatigue; previous</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>surgery for same disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Lung mass (20%) bronchial</td>
<td>Chest pain, cough, SOB, hemoptysis</td>
<td>Fever (superinfection)</td>
<td>CXR; thoracic US, CT, or MRI; sputum microscopy for protoscolices</td>
</tr>
<tr>
<td></td>
<td>obstruction, pulm Htn</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Urateral obstruction</td>
<td></td>
<td>Abdominal US, CT, MRI</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Eosinophilia, antibodies</td>
<td>Duration of albendazole therapy (marrow toxicity)</td>
<td>CBC, eosinophil count; plt count; antibody-based tests (e.g., ELISA), newer DNA-based tests: problems with cross-reactivity, false negativity</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Obstruction, anaphylaxis</td>
<td></td>
<td>Echocardiography</td>
<td></td>
</tr>
<tr>
<td>GYN</td>
<td>Incidental occurrence</td>
<td>Last menstrual period</td>
<td>Signs of pregnancy</td>
<td>Blood or urine pregnancy test</td>
</tr>
<tr>
<td>CNS</td>
<td>Cyst (1.5%)</td>
<td>Seizure</td>
<td>Localizing neurologic findings, gait abnormality,hydrocephalus</td>
<td>Head CT, MRI</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
• Review all imaging studies.
• Ensure entire surgical team aware of nature of disease.
• If liver disease, OR table capable of intraop cholangiography; if cirrhosis, normalize coagulation status (vitamin K, ffp); ensure intraop availability of prbc (possibly ffp, platelets, cryoprecipitate).
• Know anatomic extent of disease, proposed surgical approach (position, laparoscopy/incision); know backup plan if disease more extensive than thought.
• Pt to take oral benzamidazole anthelminthic (albendazole) 1 week preop, 3 mo postop.

Monitoring
• Based on planned/potential procedure
• Consider urinary catheter; if possibly extensive, consider invasive hemodynamic monitoring (art line, cent line), serial hct/coag/abg, precath Doppler or TEE to diagnose embolism (air, CO₂, cyst contents), serial Na⁺ if hypertonic NaCl used
• *Observe* for SQ emphysema

Airway
• Tracheal intubation for laparoscopic or open procedure. Double-lumen tube to protect non-diseased lung if pulmonary echinococcosis

Induction
• Rx choice based on general health status and concurrent diseases

Maintenance
• Large-bore venous access and fluid warmer(s) if hemorrhage risk
• Consider gastric tube (whether laparoscopic or open)
• Immobile operative field essential, esp during portions of procedure where cyst spillage could occur
• Have on-hand in case of anaphylactic or hemorrhagic shock: Epinephrine, vasopressin, other inotrope/vasopressor, CaCl₂, and NaHCO₃, adequate crystalloid/collod/blood products.
• If gas embolism suspected, aspirate central line; if none, consider subcostal insertion of spinal needle attached to large aspirating syringe directly into right ventricle.

James M. Riopelle
Andrew Hemphill

ICD-9-CM Codes: 122.0—122.9 (depending on parasite species and organ affected)
Extubation
• Base on usual criteria, extent of operative procedure, pt age/physical condition, concurrent disease

Postoperative Period
• Base pain control plan on nature and extent of resection; regional anesthesia an option if coagulation status permits
• Base monitoring on extent of resection, blood loss, preop health status

• Watch for pneumothorax, subphrenic abscess, pneumonia, bronchobiliary fistula, jaundice, hepatic failure, septicemia

Anticipated Problems/Concerns
• If the pt is being treated in a non-endemic area, surgical team may be unfamiliar with disease; anthelmintic medications may require special order well in advance of procedure.

• Consider Echinococcus in any pt from endemic area presenting for surgical excision of cyst; search for others using US imaging; consider ID consult, serologic testing
• Arrange US imaging in family/neighbors/farm animals capable of being intermediate hosts
• Examine stool of companion canids for eggs/segments.
Eclampsia

Risk
• Incidence 0.01–0.1 % in developed countries
• 90% of women with eclampsia have accompanying manifestations of severe preeclampsia
• Risk factors incl age <20 y old, nulliparity, and multiple gestations

Perioperative Risks
• Eclampsia is a factor in up to 10% of all maternal deaths in developed countries.
• Maternal complications incl pulm aspiration, pulm edema, cerebral vascular accident, cerebral hemorrhage, acute renal failure, and cardiopulmonary arrest.
• Fetal complications incl placental abruption, severe prematurity, and intraterine growth restriction.

Worry About
• Risk of pulm aspiration and hypoxemia with seizure.
• Fetal bradycardia may occur during or following seizure activity.

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Htn</td>
<td>Dyspnea, peripheral edema</td>
<td>Htn, peripheral edema</td>
<td>ECHO if suspect LV dysfunction</td>
</tr>
<tr>
<td>RESP</td>
<td>Airway edema</td>
<td>Snoring, stridor</td>
<td>Tachypnea, dyspnea, rales</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>Pulm edema</td>
<td>Dyspnea, orthopnea</td>
<td></td>
<td>ABG</td>
</tr>
<tr>
<td>RENAL</td>
<td>Proteinuria</td>
<td>Rapid wt gain, decreased urine output</td>
<td>Nondependent edema</td>
<td>24 hr urine protein, BUN, Cr, urine acid</td>
</tr>
<tr>
<td></td>
<td>Renal failure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenia</td>
<td>Mucosal bleeding, easy bruising</td>
<td>Petechiae, bleeding from puncture sites</td>
<td>Hgb, Hct, Ptt, fibrinogen, and FSP</td>
</tr>
<tr>
<td></td>
<td>DIC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEURO</td>
<td>Seizure</td>
<td>Headache, visual disturbances</td>
<td>Hypermecitability, hyperreflexia</td>
<td>CT/MRI if focal deficits or prolonged coma</td>
</tr>
<tr>
<td></td>
<td>Coma</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FETUS</td>
<td>Fetal distress</td>
<td></td>
<td></td>
<td>Fetal heart monitor</td>
</tr>
</tbody>
</table>


Perioperative Implications

Monitoring
• Standard maternal monitors incl non-invasive BP, pulse oximetry, and urine output
• Invasive pressures indicated if (1) maternal BP poorly controlled, (2) need for frequent blood sampling, or (3) infusion of rapid-acting vasodilators (nitroprusside or nitroglycerin)
• Pre-eclampsia/eclampsia is not an indication for invasive central monitoring
• Electronic fetal heart monitoring

Regional Anesthesia for Labor and Delivery
• Early epidural decreases likelihood of airway manipulation in the setting of emergency cesarean delivery and increases uteroplacental perfusion
• Coagulation studies, as outlined above, should be checked prior to both placement and removal of epidural catheter

• Eclampsia often occurs with severe preeclampsia and its associated complications.

Overview
• Occurrence of seizure activity or unexplained coma during pregnancy or postpartum in a woman without a pre-existing neurologic disorder
• Eclamptic convulsions can occur antepartum, intrapartum, or postpartum.
• Onset of eclampsia is generally preceded by signs of severe pre-eclampsia but 10–15% present without Htn and/or proteinuria.

ICD-9-CM Code: 642.6

Etiology
• Etiology unknown: Two prevailing theories
• Forced dilation theory: The presence of blood pressures exceeding the upper limit of cerebral autoregulation leads to vasodilation and subsequent hyperperfusion and interstitial edema.
• Vasospasm theory: Severe acute Htn causes cerebral overregulation with resultant ischemia, infarction, and cytotoxic edema.

Usual Treatment
• Establish patent airway and ensure maternal oxygenation
• Prophylaxis against further seizure activity with magnesium sulfate (4–6 g bolus over 20 min followed by 1–2 g/hr infusion)
• BP management with labetalol (10 to 20 mg IV) and hydralazine (5 to 10 mg IV)
• Severe Htn associated with an increased risk of cerebral hemorrhage
• Maintain CPP (MAP – ICP) to avoid further neurologic injury
• Expeditious (preferably vaginal) delivery via induction or augmentation of labor
• Immediately delivery indicated with persistent fetal bradycardia

Postoperative Period
• Continue magnesium infusion for 24 hr after delivery and/or last convulsion
• Increased risk of pulm edema as extracellular fluid is mobilized leading to increased intravascular volume
• Most eclamptic pts have complete resolution of neurologic abnormalities

Anticipated Problems/Concerns
• 10% will have recurrent seizures in the absence of initial prophylaxis with initial seizure
• Eclamptic seizures can occur up to 4 wk postpartum
• Cerebral hemorrhage accounts for 15–20% of deaths from eclampsia
Eisenmenger’s Syndrome

Inna Maranets

Risk
- 8% of all congenital heart disease (CHD) pts
- 11% of pts with intracardiac or aortopulmonary shunt allowing continuous exposure of pulm vasculature to systemic arterial pressure
- VSD is the most common lesion

Perioperative Risks
- High risk of CV complications when undergo non-cardiac surgery, mortality reaching 30%
- Severity of pulm Htn, cyanosis, tricuspid regurgitation and right ventricular dysfunction are important factors
- Additional acquired cardiac and systemic diseases, such as CAD and renal dysfunction
- Underlying pathology, urgency, duration of surgery and anesthetic choice contribute to the risk
- Bleeding due to platelet dysfunction
- Mortality rate of pts with ES carrying pregnancy to viability is 27–30%, most often at delivery or postpartum
- Fetal risks: ↑ Risk of preterm labor, intrauterine growth retardation; fetal demise of 75%
- C-section carries higher mortality: 70% versus 30% for vaginal delivery

Worry About
- R—L shunt, pulm Htn, right and left ventricular failure, hypoxemia, polycythemia
- Minor decrease in SBP can cause increase in R—L shunt, decrease pulm blood flow, hypoxia, and cardiovascular collapse
- Increased blood viscosity can lead to thromboembolic phenomena, paradoxal emboli, hemoptysis
- Arrhythmias, ventricular and supraventricular
- May not tolerate positive pressure ventilation
- Decreasing systemic vascular resistance of pregnancy worsens R—L shunt

Overview
- Eisenmenger’s syndrome is defined as pulm Htn at systemic level due to high pulm vascular resistance with reversed or bidirectional shunt through communication between the two circulations
- Communication may be at aortic level (PDA, aortopulmonary window), intracardiac (ASD, VSD, AV canal, TAPVR) or single ventricle
- Uncorrected L—R shunt leads to irreversible fixed pulm vascular obstructive disease
- Characterized by pulm Htn, R—L shunt, and right ventricular dysfuntion
- Overall poor prognosis; mean age at death: 25 y
- Syncope, increasing right-sided filling pressures, and systemic arterial desaturation below 85% indicate poor prognosis
- 3%–70% of pregnant pts die in association with pregnancy
- Some pulm vascular reactivity may exist in the pulm vasculature of pregnant women; may be due to systemic hormonal changes of pregnancy

ICD-9-CM Code: 416.8 Pulmonary hypertension, secondary

Ecology
- Individuals with large unrestricted intracardiac or aortopulmonary communication have large L (systemic)—R (pulmonary) shunts
- Uncorrected L—R shunt overloads pulm vasculature and RV
- Continuous exposure to systemic pressure leads to pulm arteriolar medial hypertrophy, intimal proliferation and fibrosis
- Progressive pulm capillary and arteriolar occlusion leads to fixed increased PVR
- As pulm pressure exceeds systemic, shunt reverses to R—L

Usual Treatment
- Repair of intracardiac lesion is contraindicated
- Supplemental O2, to decrease PVR
- Avoidance of medications that can cause hypotension, worsening cyanosis or hemorrhage (Ca channel blockers, antipatelet agents, anticoagulants)
- Phlebotomy to treat hyperviscosity, extreme erythrocytosis (Hct >65%) and bleeding diathesis
- Single or bilateral lung transplantation with repair of the primary cardiac defect
- Combined heart-lung transplant in select pts
- With expected high maternal mortality, pregnant pts with ES should initially be counseled to terminate pregnancy
- For the pt who wishes to continue with pregnancy:
  - Hospital admission early in 3rd trimester
  - Anticoagulation with heparin: SQ heparin 5000–10,000 U bid
  - Pts with O2 sat <80% on room air should be fully anticoagulated
  - O2 Rx
  - Monitor for preterm labor
  - Medical Rx: Diuretics, antiarrhythmics, inotropes

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<tbody>
<tr>
<td>CV</td>
<td>R—L shunt</td>
<td>DOE, fatigue, syncope edema, orthopnea, anjinal chest pain, arrhythmias</td>
<td>Elevated jugular venous pressure, increased intensity of S2, split S2 and S3, decrescendo murmurb of pulmonic regurgitation, holosystolic murmurb of tricuspid regurgitation, rales. Right parasternal heave</td>
<td>ECG, CXR, ECHO, MRI, Cardiac catheterization</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm Htn</td>
<td>Dyspnea, hemoptysis</td>
<td>Palpable pulm artery Cynosis, clubbing</td>
<td>Pulse oximetry, ABGs, Hct (polycythemia)</td>
</tr>
<tr>
<td>NEURO</td>
<td>Neurologic abnormalities</td>
<td>Headache, dizziness, visual disturbances, CVAs</td>
<td>Neurologic exam</td>
<td>CT scan, MRI</td>
</tr>
<tr>
<td>HEME</td>
<td>Polycythemia, Hyperviscosity</td>
<td>Headache, weakness, blurred vision, pruritus</td>
<td>Splenomegaly, facial erythema, bleeding gums</td>
<td>CBC</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Continue antiarrhythmic medications, withhold diuretics
- Discontinuation of heparin; consider reversal with protamine
- Endocarditis prophylaxis depends on the type of operation (AHA Guidelines)
- In pregnant pts avoid aortocaval compression at all times
- IV lines must be carefully de-aired, consider placing air filters

Monitoring
- Pulse oximetry
- With uncorrected patent ductus arteriosus, use simultaneous right hand (preductal) and foot (postductal) pulse oximetry to estimate changes in shunt fraction
- Arterial line for early recognition of sudden alteration of BP and repeated blood gas sampling
- CVP catheter
- PA catheter use must be balanced against potential complications:
  - Difficult to position in PA
  - High risk of arrhythmias, thrombi, paradoxical emboli, PA hemorrhage
  - Misleading data: Unreliable PCWP and measurement of CO with shunt

Airway
- Preop administration of Bictria, metoclopramide, and ranitidine if needed
- NPO for 8 hr (if possible)
- Coaxial technique: Initial intrathcal dose of narcotic

Preinduction/Induction
- No one best technique reported
- Goal of any technique is to maintain both cardiac output and SVR
- Combining short-acting IV narcotic (fentanyl), low-dose induction agent (sodium thiopental or ketamine) and inhalational agent (sevo- or isoflurane) with muscle relaxant devoid of CV effects (vecuronium or rocuronium)
- For labor:
  - Provision of effective analgesia prevents increased release of catecholamines, which increases PVR
  - Coaxial technique: Initial intrathcal dose of narcotic
Diseases

For C-section:
- Regional: Slow induction of epidural anesthesia; counteract sympathectomy with vasopressor and maintenance of preload
- General anesthesia: Avoid rapid-sequence with risk of precipitating increase in PVR or inducing myocardial depression; maintain cricoid pressure through induction; avoid increase in PVR, decrease in SVR, hypoxia, hypercarbia, and myocardial depressants

Maintenance
- GA: Narcotic, low-dose inhalational agent, muscle relaxant
- Avoid hypotension (↓ SVR, acidosis, hypercarbia and hypoxia (↑ PVR)
- For labor:
  - Epidural infusion with low-dose local anesthetic/narcotic solution
  - Avoid Valsalva maneuver, pushing; delivery with vacuum or forceps
- For cesarean:
  - High-dose narcotic technique
  - Amnesia with benzodiazepine
  - Avoid halogenated agents: Myocardial depression, decreasing SVR
  - Avoid nitrous oxide: Increasing PVR, higher FIO₂

Extubation
- High-dose narcotic technique may necessitate postop ventilation

Adjutants
- Avoid N₂O
- Maintain SVR with dilute solution of phenylephrine
- Inotrope, vasodilator for treatment of failure
- Cautious use of oxytocin (systemic vasodilation)
- Avoid prostaglandin F (increasing in PVR)
- Resume anticoagulation in postpartum period

Postoperative Period
- Pain management is critical
- In pregnant pts death most often occurs at delivery or postpartum
- Possible hemodynamic changes:
  - Excessive blood loss; replace volume
  - Autotransfusion; treat with vasodilator, inotrope, judicious use of diuretic
  - Arrhythmias: Sinus bradycardia, AV block, EMD
  - Pulm emboli
  - Postpartum increase in PVR; reason unknown

Anticipated Problems/Concerns
- Unresponsive, increase in PVR or decrease in SVR with loss of oxygenation
- CHF
Emphysema

Risk
• Incidence in USA: 3.1 million
• Prevalence, incidence, mortality increase with age
• Higher in males than females
• Higher in whites than nonwhites

Perioperative Risks
• Intraop bronchospasm
• N₂O expansion of bullae
• Postop resp failure
• Postop pulmonary infection

Worry About
• Worsening of baseline pulm function, caused by
  • Bronchospasm
  • Acute bronchitis or pneumonia
  • Pulm embolism
• Worsening of baseline cardiac function caused by right heart failure

Overview
• Anatomic: Destruction of interalveolar septa and loss of pulm elastic recoil, leading to formation of bullae and development of irreversible expiratory airflow obstruction

• The “pink puffer” has dyspnea, hyperinflation, distant breath sounds, low diffusing capacity (decreasing D₂CO to <60% predicted)
• “Blue bloater” pts have chronic bronchitis, leading to hypoxemia, polycythemia, and CO₂ retention.
• Hypoxia, hypercarbia, cor pulmonale are late developments.
• Mucociliary clearance is often worsened after inhalational anesthetics.
• Diaphragmatic mechanics are impaired by anesthetics, sedatives, NMBs, interscalene blocks, supine positioning.

ICD-9-CM Code: 492.8 (Other Emphysema)

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<tbody>
<tr>
<td>HEENT</td>
<td>Tumors 2° to smoking</td>
<td>Voice change</td>
<td>Hoarseness, stridor, inspiratory obstruction</td>
<td>Flow-volume loops</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cor pulmonale (late)</td>
<td>Edema, severe dyspnea</td>
<td>Signs of pulm Htn, Hepatosplenomegaly, Pedal edema, cyanosis, pleural effusions, usually without pulmonary edema</td>
<td>CXR, ABGs</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchospasm</td>
<td>Recent ↑ in dyspnea or ↓ in exercise tolerance</td>
<td>↑ Resp rate, ↑ Expiratory time, ↑ Accessory muscle use</td>
<td>Spirometry pre- and post-bronchodilators</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>Fever, dyspnea, ↑ sputum</td>
<td>Signs of pulm consolidation</td>
<td>CXR, WBC</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
• Optimize bronchodilatation
• Eradicate any underlying bacterial infection
• Encourage smoking cessation if this can occur >6 wk before surgery
• Consider regional anesthesia where appropriate

Monitoring
• Be cognizant of potential for increased gradient between PETCO₂ and PaCO₂

Airway
• None, unless tumor present in airway

Preinduction/Induction
• If patient has airway reactivity, consider issues related to asthma/chronic bronchitis
• Avoid N₂O when expansion of bullae is a risk
• May avoid high concentrations of desflurane if airway reactivity is of concern

Maintenance
• Recumbent positions impair chest wall muscle function, and abdominal muscle function usually needed for spontaneous ventilation
• Ventilator settings: Long expiratory times may be required; try to avoid high positive pressures (consider pressure control ventilation), esp. if bullae are present.

Extubation
• Residual anesthetics may compromise the ventilatory response to CO₂, increasing the risk of postop resp failure
• Pre-extubation bronchodilators
• Unreolved incisional pain, esp. after abdomino or thoracic surgery, will impair breathing—consider postop epidural analgesia.
• Consider regional block and/or NSAIDs to lessen risk of resp depression.
• Pts may be semiconscious and combative owing to hypoxia and hypercarbia on emergence.

• Genetic: Absent or abn API, also known as a₁-antitrypsin deficiency, which accounts for a small fraction of cases

Usual Treatment
• Smoking cessation (>6–8 wk may lessen anesthetic risk)
• Relief of symptoms by treatment of bronchospasm and infection
• In advanced cases, if hypoxia and cor pulmonale have developed: O₂ therapy may improve quality of life and survival.
• Lung volume reduction surgery may be considered for those with predominantly upper lobe disease and/or low exercise tolerance.

Evaluate whether postop ventilation may be the safest approach until the residual anesthetic effects have dissipated. Exubation to non-invasive positive pressure ventilation (NIPPV) may be useful in such cases.

Adjuncts
• β₂-Adrenergic agonists, anticholinergic agents for airway reactivity (may consider theophylline)
• Oral or inhaled steroids in selected pts

Anticipated Problems/Concerns
• Postop resp failure (consider NIPPV rather than reintubation in selected pts)
• Tension pneumothorax from ventilator-induced barotrauma
• Airway plugging from secretions
Encephalitis

Risk
- Increased by exposure in endemic areas via human or zoonotic contacts. Transmission can be person-to-person, fecal-oral, via infected mosquito or animal bite, or infected saliva or secretions.
- Increased during seasonal variation and epidemic outbreaks
- Assoc with 1° viral infection or reactivation, post-infectious/immune response, paraneoplastic syndromes or treatment-related immunosuppression

Perioperative Risks
- Assoc with mental status alteration, seizures, increased ICP, SIADH
- Assoc with increased sensitivity to sedative and amnestic effects of anesthetics and adjunct drugs
- Unrecognized, unexpected deterioration in mental status

Worry About
- Delayed awakening, postop delirium, clinical and subclinical seizures
- Hyperkalemic response to succinylcholine
- Transient myocardial dysfunction
- Electrolyte abn 2° to SIADH and CPM with rapid correction of Na abn

Overview
- Inflammation of brain parenchyma
- May be 1° manifestation of disease process or a component of another CNS or systemic illness
- Organisms enter CNS via bloodstream or peripheral nerves
- Symptoms include altered mental status, altered consciousness, with or without focal neurologic abn, behavioral changes, in the presence of fever, headache, photophobia, nuchal rigidity, vomiting, disorientation, lethargy, confusion, hallucinations, memory loss, clinical or sub-clinical seizures, myoclonus, coma
- Dx is established by symptoms, epidemiologic Hx (exposure, season, geographic location), CSF culture, CSF bacterial and viral antigens, CSF viral PCR, virus specific DNA sequencing, MRI, EEG, CT scan. Brain biopsy is rarely performed.

ICD-9-CM Code: 064 (Viral encephalitis transmitted by other and unspecified arthropods), 054.3 (Herpetic meningoencephalitis), 052.0 (Postvaricella encephalitis)

Etiology
- Infectious
  - Viral: Herpes, varicella, CMV, EBV, influenza, RSV, enteroviruses, arboviruses, HIV, JC virus, rabies
- Non-viral: Bacteria, protozoa, nematodes, fungi
- Noninfectious
- Post-infectious/immune mediated
- Autoimmune
- Paraneoplastic

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<tbody>
<tr>
<td>HEENT</td>
<td>Virus access to CNS from nasal mucosa to olfactory bulb and olfactory tracts</td>
<td>Preceding URI</td>
<td>Labile BP, HR</td>
<td>Nasopharyngeal swab</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Autonomic dysfunction Neurogenic stunned Myocardium</td>
<td>Transient myocardial dysfunction</td>
<td>EKG, troponin, CK, echocardiography, LV angiography</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>↑ or normal WBC</td>
<td>Serum Na⁻ and osmolality</td>
<td>CBC, WBC differential, serum antibody titers</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>SIADH</td>
<td>Water intoxication</td>
<td>No evidence of volume depletion</td>
<td>Urine Na⁻ and osmolality</td>
</tr>
<tr>
<td>CNS</td>
<td>Focal, global neurologic disturbances</td>
<td>Fever, Headache, Seizure, Personality change, Memory loss, Confusion, Weakness, Sleep/awake abn</td>
<td>Focal neurologic deficits, altered mentation, papilledema, anisocoria; if spinal cord involvement: flaccid paraplegia, increased DTRs</td>
<td>CSF: Cell count (WBC, lymphocyte predominance), protein ↑, Gram stain, viral and bacterial culture, antibodies, antigens, viral polymerase chain reaction (PCR), viral DNA sequencing, MRI (temporal lobe involvement, hemorrhagic lesions, mass effect), EEG, CT</td>
</tr>
</tbody>
</table>


Preoperative Implications

Preoperative Preparation
- Document neurologic exam
- Elicit Hx of increased ICP or seizure
- If SIADH present, correct electrolyte and free water abn
  - Sodium administration or fluid restriction depending on severity of hyponatremia
- Beware of central pontine myelinolysis with rapid correction of hyponatremia

Monitoring
- Electrolytes
- Fluid I/O
- Consider ICP monitoring, EEG monitoring

Preoperative preparation

Examination
- None

Induction
- Potential for hyperkalemic response to depolarizing NMbs if myopathy or paralysis, prefer use of nondepolarizing NMbs
- Autonomic instability and labile hemodynamics

Maintenance
- If pt is receiving seizure prophylaxis (e.g., phenytoin), be aware of potentiation of sedative effects and alteration of hepatic metabolism of anesthetics and muscle relaxants.

Exubation
- Delayed awakening
- Seizures on emergence

Postoperative Period
- Delirium
- Possible progressive deterioration

Anticipated Problems/Concerns
- Delayed awakening
- SIADH, careful selection of replacement fluid
- Hyperkalemic response to succinylcholine
- Universal precautions for contact with infected materials, sterilization of reusable instruments
Encephalopathy, Hypertensive

**Risks**
- Most common clinical presentations of hypertensive emergencies are cerebral infarction (24.5%), pulm edema (22.5%), hypertensive encephalopathy (16.3%), and CHF (12%)
- Rapidly developing, fluctuating, or intermittent Htn carries a particular risk for hypertensive encephalopathy; pts with untreated or undertreated chronic Htn, renal failure, or preeclampsia-eclampsia are at increased risk
- Longstanding Htn disorders, coexisting renal disease, and immunosuppressive therapies are also risk factors

**Perioperative Risks**
- Increased risk of myocardial ischemia, ventricular dysrhythmias, heart failure, cerebral hemorrhage, coma, long-term neurologic disability, aortic dissection, renal failure, or sudden death

**Worry About**
- Myocardial ischemia or infarction
- Cerebral infarction (ischemic or hemorrhagic)
- Aortic dissection
- CHF
- Acute renal failure
- Pulm edema
- Subarachnoid hemorrhage
- HELLP syndrome in eclamptic parturients
- Microangiopathic hemolytic anemia

**Overview**
- A relatively rapidly evolving syndrome of severe Htn in association with headache, N/V, visual disturbances, confusion, and—in advanced cases—stupor and coma that may be rapidly fatal
- More common with chronic hypertensive pts, renal failure, and eclampsia
- Occurs when the BP is elevated beyond autoregulatory thresholds of mean arterial pressures greater than 150–160 mmHg (‘autoregulation breakthrough’)
- Dx of exclusion; stroke and SAH also produce encephalopathy with acutely elevated BP; stroke is by far the most likely Dx; elevated BP, headache, papilledema, and altered consciousness are also seen with intracranial hemorrhage
- Pts with chronic Htn can tolerate higher mean arterial pressures before they have disruption of their autoregulation system; such pts, however, also have increased cerebrovascular resistance and are more prone to cerebral ischemia when flow decreases, esp if BP is decreased into normotensive ranges.
- BP >250/150 mmHg is usually required to precipitate the syndrome in pts with chronic Htn, while previously normotensive pts are affected at pressures as low as 160/100 mmHg (i.e., as with eclampsia or acute renal failure)
- Has been termed “hypertensive posterior reversible encephalopathy syndrome” or PRES

**ICD-9-CM Code: 437.2**

**Etiology**
- A sudden increase in BP, with or without pre-existing chronic Htn, resulting in failure of cerebral autoregulation, leading to cerebrovascular endothelial dysfunction and vasogenic edema that renders the pt encephalopathic
- Most common in pts with untreated or undertreated chronic essential arterial Htn
- Renovascular disease or renal parenchymal lesions
- Endocrine causes: Pheochromocytoma, renin-secreting tumor, Cushing’s disease, Conn’s syndrome
- Preeclampsia and eclampsia
- Status post carotid endarterectomy (CEA hyperperfusion syndrome)
- Thrombocytopenic purpura
- Acute intermittent porphyria
- AIDS
- Autonomic hyperreactivity, with spinal cord lesions
- Drug induced: D/C of antihypertensive drugs, erythropoietin, immunosuppressive therapy (cyclosporine, tacrolimus, cisplatin, interferon-alfa), MAOs
- Ingestion of cocaine, amphetamine, or LSD
- Often multifactorial from the previous list

**Preoperative Implications**

**Preinduction**
- Determine medications, compliance with antihypertensive regimens, and adequacy of BP control
- Evaluate for end-organ damage

**Monitoring**
- Arterial catheter
- Central venous catheter or pulm artery catheter may be used if extensive surgery is planned or there is evidence of other end-organ damage (LV dysfunction, renal failure)

**General Anesthesia**
- Volatile anesthetics are useful in attenuating sympathetic nervous system pressor responses; there is no evidence to suggest one volatile agent over another for control of intraop Htn
- Nitrous oxide–opioid technique can be used be used for maintenance of anesthesia in pts with labile pressure while under GA; a volatile agent may be needed during periods of abrupt changes in surgical stimulation
- Antihypertensive agent by bolus or continuous infusion is an alternative to volatile agent BP control

**Induction**
- Induction of anesthesia may produce an exaggerated decrease in BP due, esp in the face of diastolic Htn (intravascular volume depletion)


**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Htn</td>
<td>Hx of Htn</td>
<td>S&lt;sub&gt;i&lt;/sub&gt;</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>CHF</td>
<td>Hx of preeclampsia</td>
<td>S&lt;sub&gt;i&lt;/sub&gt; gallop</td>
<td>EKG</td>
</tr>
<tr>
<td></td>
<td>Myocardial ischemia</td>
<td>Angina</td>
<td>Rales</td>
<td>Arteriogram</td>
</tr>
<tr>
<td></td>
<td>Aortic dissection</td>
<td>Hx of CHF</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pain radiating to the back</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>SOB</td>
<td>Rales</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>FEV, FVC</td>
<td>Frothy sputum</td>
<td></td>
<td>O&lt;sub&gt;2&lt;/sub&gt;, sat</td>
</tr>
<tr>
<td>GI</td>
<td>Aortic dissection</td>
<td>Orthopnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>N/V</td>
<td>Abd pain</td>
<td>US or arteriogram (if indicated)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Back pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal failure</td>
<td>Anuria</td>
<td></td>
<td>Measurement of UO, BUN/Cr</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>May occur without presence of proteinuria</td>
</tr>
<tr>
<td>CNS</td>
<td>Cerebral hemorrhage</td>
<td>Mental status exam</td>
<td>Retinal arteriolar spasm on ophthalmoscopy</td>
<td>CT</td>
</tr>
<tr>
<td></td>
<td>Severe headache</td>
<td></td>
<td>Papilledema</td>
<td>MRI</td>
</tr>
<tr>
<td></td>
<td>Visual disturbances</td>
<td></td>
<td>May have a normal fundoscopic exam</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Generalized or focal seizures</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Paralysis</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Stupor, coma</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

**Key: S<sub>i</sub>**
• Direct laryngoscopy and tracheal intubation can produce significant Htn; limit duration of DL and consider use of opioid, lidocaine, blocker, and vasodilator to blunt autonomic response

**Maintenance**
• Control BP and minimize wide fluctuations in BP, as aggressive treatment of Htn may worsen other end-organ function
• Monitor for myocardial ischemia

**Regional Anesthesia**
• Epinephrine-containing solutions may place the hypertensive pt at risk or worsen an existent hypertensive crisis (e.g., epidural in a pre-eclamptic parturient)

**Postoperative Period**
• Extubate with careful BP control
• Continue IV antihypertensive therapy and monitoring of BP and mental status

• Maintain monitoring for other end-organ morbidity such as myocardial ischemia, cardiac dysrhythmias, CHF, stroke, and bleeding

**Anticipated Problems/Concerns**
• Particular caution is necessary with elderly pts and pts with preexisting Htn; over-aggressive reduction in BP may be accompanied by worsening neurologic status and stroke.
**Encephalopathy, Metabolic**

**Risk**
- 3.4–11% of medical ICU admissions
- 12–33% of multiple-organ dysfunction pts

**Perioperative Risks**
- With predisposing conditions, e.g., hepatic insufficiency, risk of developing or exacerbating metabolic encephalopathy
- Increasing severity of pre-existing encephalopathy

**Worry About**
- Worsening hepatic insufficiency causing hepatic encephalopathy
- Diabetics becoming hypoglycemic or with DKA/hyperosmolar coma
- Postop hyponatremia
- Deteriorating renal insufficiency leading to uremic encephalopathy
- Pre-existing encephalopathy may be exacerbated by anesthetics, e.g., benzodiazepines, in hepatic encephalopathy
- Undiagnosed sepsis, hypothermia, high fever, CNS-acting drugs, incl overdose
- CNS cause: Brainstem CVA, meningitis, occult head trauma, encephalitis, brain tumor

**Overview**
- Altered sensorium, stupor, or coma without any other explanation in the setting of a metabolic disturbance
- Process affects global cortical function by altering brain biochemistry
- Distinguished from structural lesions by a non-focal neurologic exam
- EEG shows diffuse background slowing, triphasic waves in hepatic encephalopathy
- Increased spontaneous motor activity: restlessness, asterixis, myoclonus, tremors, rigidity

**ICD-9-CM Code:** 348.3 (Unspecified encephalopathy)

**Etiology**
- Hypoglycemic encephalopathy: Most commonly caused by accidental or deliberate overdosing with insulin or oral hypoglycemic agents or prolonged ethanol intoxication
- Hepatic encephalopathy: Acute or chronic hepatic insufficiency, Reye syndrome
- Uremic encephalopathy: Renal failure. After dialysis, disesequilibrium syndrome caused by acute fluid and electrolyte shifts.

**Perioperative Implications**

**Preoperative Preparation**
- Assess and document preop mental status and neurologic function
- Uremic encephalopathy: Preop dialysis, if possible
- Hyperthyroidism, hypothyroidism: Initial treatment, if possible

**Monitoring**
- Routine
- In hyperosmolar states, uremia and liver failure with ascites may need central monitoring

**Preinduction/Induction**
- Benzodiazepines should be avoided in hepatic encephalopathy
- Increased potential for aspiration; consider rapid-sequence

**Maintenance**
- Carefully titrate anesthetics to avoid overdosing
- Careful attention should be paid to intravascular volume status, blood glucose, and lights
- During and after TURP and hysteroscopy, sodium concentrations and volume status should be monitored
- Correction of hypernatremia and hyponatremia should be gradual
- In renal and hepatic failure, appropriate drugs and doses should be used. Long-acting drugs should be avoided. May be increased bleeding

**Diabetes:** Monitor intraoperative blood glucose to avoid hypoglycemia. Too rapid correction of hyperglycemia can lead to cerebral edema

**Exubation**
- Exubate only if pt is able to protect airway and maintain adequate ventilation

**Anticipated Problems/Concerns**
- Poor mental status at the conclusion of surgery may require continued intubation
- Hyponatremia is a cause of postop metabolic encephalopathy
Encephalopathy, Postanoxic

Risk
- After successful prehospital cardiac resuscitation: 59–65% of pts remain comatose
- 0–5% of successful resuscitations result in chronic vegetative state

Perioperative Risks
- Worsening of neurologic status; blindness most common residuum
- Postpone surgery in all but emergency situations
- Do what is necessary to treat precipitating cause and to decrease sequlæ (e.g., treat elevated ICP)

Worry About
- Repeat of events that initially caused encephalopathy (e.g., arrhythmias leading to cardiac arrest)
- Hypotension, hypercapnia, hypoxia, and sepsis that can exacerbate encephalopathy.

Overview
- Definition: Brain injury resulting from prolonged period of insufficient cerebral oxygenation
- Clinical picture ranges from mild confusion to brain death
- Chances for acceptable neurologic recovery—1% with continued coma after 24 hr and lack of two of the following reflexes: pupillary, corneal, and ocuulovestibular
- Absence of brainstem function 72 hr after event associated with irreversible coma.
- Good prognosis seen in 50% of pts awakening within 24 hr of insult.
- Seizures occur in 25% of pts.
- Anoxic damage may have been sustained by other organs (e.g., MI, shock liver, acute renal failure, stress ulcers, ARDS)
- Diabetes insipidus poor prognostic sign

ICD-9-CM Code: 348.1 (Anoxic brain damage)

Etiology
- Caused by inadequate O₂ delivery to CNS due to inadequate cardiac output, resp dysfunction, severe anemia, and/or increased ICP
- Most often 2° to 1° cardiac (MI or arrhythmia) or pulm (asthma, pulm embolism) event
- May also be result of CO poisoning, suffocation, and cyanide poisoning

Perioperative Implications

Preoperative Preparation
- Assess and document neurologic function and mental status
- Review cause of anoxic event
- Assess damage to other organs
- If pt hypothermic beware of possible increased blood loss

Monitoring
- If arrest was due to cardiac arrhythmias or MI/ischemia or if pt is hemodynamically unstable, may need specialized monitoring

Airway
- Assess potential for aspiration: Gag reflex, ability to cough and clear secretions

Induction
- Avoid succinylcholine

Maintenance
- Must consider that pts may have pain perception and will require analgesia
- Do what is appropriate to decrease sequlæ (e.g., treat increased ICP); therapeutic hypothermia

Extubation
- If unable to maintain patent airway or sustain adequate minute ventilation, pt should remain intubated.

Adjuvants
- Avoid long-acting anesthetics so that neurologic status can be assessed soon after surgery.
- Avoid drugs that decrease seizure threshold.

Anticipated Problems/Concerns
- Repeat of events (e.g., arrhythmias) that initially led to anoxic encephalopathy
- Worsening of neurologic condition during periop period
- Postpone all but emergency surgery if fluctuating neurologic deficits or acute encephalopathic condition exists.


ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>MI</td>
<td>Assess if cardiac disease was cause of arrest</td>
<td></td>
<td>ECG, other cardiac assessment</td>
</tr>
<tr>
<td>RESP</td>
<td>ARDS</td>
<td>Assess if resp disease was cause of arrest</td>
<td>Wheezing, stigmata of COPD</td>
<td>Pre-arrest PFTs</td>
</tr>
<tr>
<td>GI</td>
<td>Shock liver, Stress ulceration</td>
<td>Hx of GI bleeding</td>
<td>Jaundice</td>
<td>AST, ALT, bilirubin, alkaline phosphatase</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal failure</td>
<td>Assess if electrolyte abn or acidosis caused initial event</td>
<td>UO</td>
<td>BUN/Gr</td>
</tr>
<tr>
<td>CNS</td>
<td>Altered mental status, diffuse and focal neurologic abn</td>
<td>Changes in neurologic signs since hypoxic event</td>
<td>Neurologic and mental status exams, apnea test, brainstem reflexes</td>
<td>CT scan/CT angiography, MRI/MRA, EKG, SSEP, BAER</td>
</tr>
<tr>
<td>MS</td>
<td>Myoclonus, posturing</td>
<td>Hx of abnormal movements, posturing</td>
<td>Decerebrate or decorticate postures</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Contractures</td>
<td>Prolonged immobility</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Usual Treatment
- Prevent recurrence of inciting event
- Ventilatory and hemodynamic support, as needed
- Stress ulcer prophylaxis
- Treatment of seizures (with anticonvulsants, e.g., phenytoin) and myoclonus
- Therapeutic hypothermia (esp. after cardiac arrest with initial VF or VT) to 32–34°C for 12–24 hr.
- BP should be maintained at normotensive or mildly elevated levels in normotensives and higher in hypertensives.
- Treat fever promptly with antipyretics.
Endocardial Cushion Defect

Risk

- 2% of CHD is endocardial cushion defect (ECD). No gender predilection.
- 30% of ECD assoc with Down syndrome

Perioperative Risks

- Shunt reversal caused by anesthetic drugs, airway stimulation during light anesthesia, or airway obstruction
- Paradoxical embolism, particularly with shunt reversal
- Subacute bacterial endocarditis; recommend antibiotic prophylaxis
- Pulmonary hypertensive crisis in pts with reactive pulm vasculature
- AV valve regurgitation, arrhythmias following surgical repair of ECD

Worry About

- Development of pulm vascular obstructive disease, reversal of shunt and RV failure
- Development of atrial arrhythmias due to significant atrial enlargement
- Extreme sensitivity to myocardial depressant effects of inhaled agents

Overview

- ECD causes various combinations of Ostium Primum atrial septal defects, ventricular septal defects in inlet septum, and clefts in anterior mitral and/or septal tricuspid valve leaflets
- Causes shunting at atrial or ventricular (or both) sites with or without assoc AV valvular regurgitation. Leads to heart failure (R > L), failure to thrive and repeated resp infections
- ECD can lead to shunt reversal, pulm vascular obstructive disease, and Eisenmenger's syndrome which can limit surgical therapy
- Dx confirmed by TEE and cardiac catheterization

ICD-9-CM Code: 745.6

ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Feeding difficulties</td>
<td>Failure to thrive</td>
<td>&lt; Normal wt/ht for age</td>
<td>Comparison of wt/ht to published values</td>
</tr>
<tr>
<td>CARDIO</td>
<td>CHF</td>
<td>Diaphoresis, coughing</td>
<td>Wheezing, rales, hepatosplenomegaly</td>
<td>Cardiac catherization, ECG with RVH, TEE</td>
</tr>
<tr>
<td></td>
<td>Pulm Htn</td>
<td>Tachycardia, murmur</td>
<td>Worsening CHF</td>
<td>TEE, cardiac catheterization</td>
</tr>
<tr>
<td>RESP</td>
<td>CHF</td>
<td>Dyspnea, tachypnea</td>
<td>Wheezing, rales</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal dysfunction due to heart failure</td>
<td></td>
<td></td>
<td>BUN, Cr</td>
</tr>
<tr>
<td>MS</td>
<td>Decreased exercise compared with peers</td>
<td></td>
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</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation

- Prophylactic antibiotics for subacute bacterial endocarditis
- Premedication to minimize anxiety and possible shunt reversal
- Diuretic if prone to CHF

Monitoring

- Intra-arterial catheter and central venous catheter if required by surgical procedure
- TEE if available and appropriate to anesthetic and surgical procedure

Airway

- May be difficult if associated congenital anomalies such as Down syndrome are present

Preinduction/Induction

- Meticulous air removal to avoid paradoxical embolism
- IV or inhalation induction depending on pt preference/cooperation. Choice of IV induction agent depends on severity of CHF and pulm Htn.

Maintenance

- Volatile agents that decrease systemic vascular resistance may worsen R to L shunting. Combinations of narcotic with low concentrations of volatile agents may be appropriate in pts with moderate disease.

Exubation

- Possible in pts without CHF or pulm Htn. Proceed cautiously esp if reactive pulm vasculature, may develop pulm hypertensive crisis requiring hyperventilation, ↑ FIO₂, sedation, or even NO or ECMO, which is more easily accomplished during mechanical ventilation.

Adjutants

- NO, nitroglycerin, prostaglandin to control pulm vascular tone. Inotropes for heart failure.

Postoperative Period

- Observe left and right atrial pressures, as LAP more than 6 mm > RAP suggests mitral valve incompetence and/or stenosis.

- Residual shunting at atrial or ventricular level should be excluded by TEE.
- Heart block or other conduction defects may result from surgical repair.
- Effective analgesia to minimize pulm hypertensive crisis, which may incl epidural or spinal analgesia in some centers

Anticipated Problems/Concerns

- Pts with partial or complete AV canal defects 2° to endocardial cushion defects who have CHF are likely to have moderate to severe AV valvular incompetence or pulm Htn following surgical repairs.
- Significantly increased pulm blood flow 2° to L to R cardiac shunting increases the risk of developing pulm vascular obstructive disease and postop pulm hypertensive crisis.

Etiology

- AV canal defects result from failure of the endocardial cushions to grow and fuse with portions of the interatrial and interventricular septa in the 5–6 mm embryo
Epidermolysis Bullosa

Risk
- 1/17,000, 50% dystrophic form
- Racial distribution equal

Perioperative Risks
- Difficult IV access, airway, intraop positioning, reflex, steroid dependence, intraop hemorrhage, sepis, iatrogenic corneal abrasion, blister formation, airway obstruction

Worry About
- Problems similar to pts with severe skin burns. Pts are severely compromised.
- Difficult intubation (23%) 2° to microstomia
- Establishing monitoring, IV access
- Dehydration, malnutrition
- Anemia, hypoalbuminemia, electrolyte imbalance, thoracomyocytosis
- Septicemia
- Renal and adrenal dysfunction

Overview
- Characterized by epithelial blistering as a result of minor trauma by lateral shearing forces, not pressure
- 3 types: Simplex (SEB), junctional (JEB), dystrophic (DEB)

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Enamel hypoplasia: Blisters, microstomia, ankyloglossia, supraglottic ulceration or narrowing</td>
<td>Delayed eruption, caries of teeth; painful peri- and intraoral lesions; hoarseness, resp obstruction; painful swallowing, spasm, food impaction</td>
<td>Poor oral hygiene, malocclusion; tongue atrophy; obliteration of vestibular sulci, stricture, webs, vocal cord lesions</td>
<td>Airway assessment, endoscopy</td>
</tr>
<tr>
<td>GI</td>
<td>Bullae</td>
<td>Anal pain, tenesmus, constipation</td>
<td>Anal fissure or stricture</td>
<td>Endoscopy</td>
</tr>
<tr>
<td>GU</td>
<td>Blisters</td>
<td>Urinary diversion</td>
<td>Obstruction, sepsis</td>
<td>Renal function</td>
</tr>
<tr>
<td>MS</td>
<td>Contractures, growth retardation</td>
<td>Movement limitations, stature</td>
<td>Flexion contracture, pseudodactyly</td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Blisters</td>
<td>Age at onset, Hx of remissions, infections</td>
<td>Scars, milia, nail dystrophy, cancer</td>
<td>Skin biopsy</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Careful planning of monitoring, IV placement, positioning in the OR, prevention of reflex, and airway management

Monitoring
- No contraindication to pulse oximeter use
- Protect blisters on the face with foam adhesive inverted to pad mask
- Pad automated BP cuff heavily, limit intervals
- Cut off adhesive from ECG leads; hold in place with debubillarimeter jelly pads
- Suture invasive monitoring and IVs or wrap in place with petrolatum gauze
- Esophageal stethoscope may damage mucosa
- Avoid excessive heat or sweating, which increases risk of blisters

Induction
- Regional anesthesia encouraged; use spray antiseptics or pour prep solutions; no intradermal local anesthetics
- No GA or muscle relaxant specifically contra-indicated

Airway
- All airway management techniques reported successful
- Mask (or nasal mask) lubricated and padded with petrolatum gauze; pad chin under fingers; bullae occurred in 1 in 50
- LMA one size too small, heavily lubricated, cuff soft with audible leak, extubated deep to prevent trauma; lingual bulla occurred in 1 in 57
- Intubation less frequent; blind nasal, fiberoptic, and oral techniques; heavily lubricated small tube; lubricate laryngoscope heavily; cricoid pressure without lateral movement permissible; 66% class I or II view of larynx, 7–23% difficult airway incidence; soft lubricated gauze to prevent tube movement in mouth, no lateral forces on mouth corners by tube, no tape; trachea lined with columnar epithelium, so less likely to blister

Emergence
- Aim for a quiet emergence
- No suction on intraoral mucosa

Anticipated Problems/Concerns
- Positioning: Pt performs if possible; lateral shear forces from lifting cause blisters
- Corneal abrasion: Poor eyelid retraction; use ointment generously, protect eyes in prone position
- Treat hemorrhage with epinephrine or thrombin-soaked sponge
- Avoid sweating; warming devices, if unavoidable, no warmer than skin temperature
- Extremity tourniquets, IM or rectal medications, EMLA can be used
- Common procedures: Release of syndactyly, dressing change, squamous cell carcinoma, esophageal dilation, dental surgery

Etiology
- SEB: Inherited autosomal, usually dominant, mutation producing abnormal keratin intermediate filament proteins 5 or 14 which weaken the epidermal architecture. In MD form abn plectin (cytolinker protein) is the cause.
- JEB: Inherited autosomal recessive mutation producing abn laminin 5, abn type XVII collagen, and abn α,β integrin
- DEB: Inherited autosomal dominant or recessive mutation producing abn type VII collagen
Epiglottitis

Overview
- An acute, potentially life-threatening cause of upper airway obstruction (etiologic agents may incl bacteria other than Haemophilus influenzae type B)
- Produces inflammatory edema of epiglottis and other supraglottic structures
- Onset usually rapid; progression to severe obstruction can occur in several hours
- High fever, sore throat, and dysphagia frequently so severe that swallowing is inhibited and drooling results
- Differential diagnosis also incl retropharyngeal abscess (a bacterial infection), which can have same presentation. It can be differentiated from epiglottitis by presence of torticollis and trismus and with radiographic studies (contrast CT). Treatment is with antibiotics and surgical drainage.

ICD-9-CM Codes: 464.30; 464.31 (With obstruction); 478.24 (Retropharyngeal abscess)

Etiology
- H. influenzae type B is most often traditional assoc pathogen, though can be caused by β-hemolytic streptococci, group A Streptococcus pneumoniae, Staphylococcus aureus, Klebsiella pneumoniae

Usual Treatment
- Antibiotic therapy against bacteria (usually H. influenzae) and airway support, which generally requires tracheal intubation
- Because of high incidence of ampicillin-resistant strains, ampicillin plus a β-lactamase inhibitor (such as sulbactam) and/or chloramphenicol, cefuroxime, cefazidime, or another penicillinase-resistant antibiotic as indicated by blood and epiglottitis culture results
- Tracheal intubation classically performed in OR in a controlled fashion with surgical support for possible tracheotomy or cricothyrotomy present and gown

Risk
- Children 1–7 y, although epiglottitis (sometimes called supraglottitis) does occur in adults. (Decreasing incidence in children >3 y, related to vaccine against Haemophilus influenzae type B, but still found, particularly if not immunized.)
- Adult incidence remains constant with organisms group A Streptococcus pneumoniae, Staphylococcus aureus, Klebsiella pneumoniae

Perioperative Risks
- Acute deterioration of airway patency resulting in complete obstruction worse in children
- Difficulty in tracheal intubation due to severe edema of epiglottis and arytenoids

Worry About
- Airway compromise in children who appear toxic, with increasing distress, drooling, hypoxemia. The acute risks of airway compromise (of concern in small children) appear to be less critical in adults, most likely because of larger airway.
- Loss of airway control and aspiration

Assessment Points

| Differentiation between epiglottitis and croup (laryngotracheobronchitis): |
|-----------------------------|-----------------------------|
| Croup                      | Epiglottitis                |
| AGE                        | 3 mo–3 y                   | 1–7 y                      |
| ONSET                      | Gradual                    | More rapid (usually <24 hr) |
| FEVER                      | Low-grade                  | High-grade                 |
| COUGH                      | Characteristic barking     | None                       |
| SORE THROAT                | Occasional                 | Frequently severe          |
| POSTURE                    | Any                        | Frequently sitting forward, mouth open, drooling |
| AIRWAY SOUND               | Inspir stridor             | Inspiratory stridor        |
| VOICE                      | Normal                     | Muffled                    |
| APPEARANCE                 | Nontoxic                   | Toxic                      |
| SEASONALITY                | Peak winter, epidemic      | Year-round                 |

Radiographic studies may be helpful, because AP view of trachea appears normal but lateral neck view usually shows a markedly swollen, edematous epiglottis (“thumbprinting”).


Perioperative Implications

Preoperative Preparation
- With suspected epiglottitis, other personnel on pt care team can set up care (e.g., OR or ICU). Radiographs can be obtained, but a team member capable of monitoring and securing the airway should be present.
- Allow to remain in a position of comfort (often sitting with parent). Direct exam of oropharynx generally avoided, as are attempts to secure vascular access, because these may cause agitation leading to acute tracheal obstruction.
- Humidified O₂ should be delivered as tolerated.
- Aerosol therapy with racemic epinephrine may provide slight improvement of Sx, but not definitive. If Dx is confirmed, pt is taken to the location for intubation (most commonly OR).

Post Airway Management Plans
- Once airway secured, cultures of blood and epiglottis are obtained, antibiotic therapy is initiated, and sedation plans are instituted.

Anticipated Problems/Concerns
- Resp support often for 24–72 hr until swollen epiglottis returns to normal
- Usually require sedative management to facilitate tolerating mechanical ventilation
- Many pts (~25%) have assoc pneumonia that requires treatment
Familial Dysautonomia (Riley-Day Syndrome)

### Risk
- Rare due to improved genetic screening measures. Incidence likely 1:10,000–20,000 in Jews originating from Eastern Europe (Ashkenazi)
- Carrier frequency in Ashkenazi Jews 1:27–32

### Perioperative Risks
- Hemodynamic instability 2° to an erratic ANS
- Pulm insufficiency 2° to a relative insensitivity to hypoxemia and hypercarbia
- Altered response to hypoxia resulting in bradycardia and hypotension
- Impaired renal function in older pts due to renal hypoperfusion

### Worry About
- Dysautonomic crisis: Triggered by physiologic and psychologic stressors and characterized by intractable vomiting, Htn, tachycardia, diaphoresis, and erythematous skin blotching

### Perioperative Implications

#### Preoperative Preparation
- Consider regional anesthesia or adjunctive use of regional techniques combined with general anesthesia for improved intraoperative and postop pain control.
- Difficulty swallowing: Abundant secretions plus diminished laryngeal reflexes. Treat with antialgalogues
- Avoid medications that interact with the ANS
- Dysautonomic crisis: Prevent with anxiolytics.
  - If symptoms develop, first-line treatment with midazolam, second-line treatment with clonidine for residual Htn or agitation
  - H2 blockers can decrease gastric volume and acidity.
  - Phenothiazines are assoc with erratic hemodynamics at induction.
  - Treat chronic dehydration 2° to dysphagia and emesis. Increased insensible losses 2° to increased sweating and drooling may require greater maintenance fluid volumes to maintain normovolemia.
  - Insensitivity to hypoxia, hypercarbia: Minimize narcotics as premedication
  - Insensitivity to superficial pain: Lines placed without discomfort. Poor thermal discrimination may affect assessment of regional blocks.

#### Monitoring
- Routine
- Consider arterial line

#### Induction
- Rapid sequence induction preferred. Titration of induction agents should be attempted to minimize risk of hypotension.
  - Use of nondepolarizing agents must be balanced against the risk of postop hypotonia and unpredictable effect of reversal agents on ANS.
  - Lubricate eyes to avoid corneal abrasions 2° to alarina.

#### Maintenance
- Dysfunctional regulation can require fluids and vasoactive drug therapy.
  - Titrate volatile anesthetic with processed EEG monitor to avoid overdose, supplement with short acting opioid.
  - Aggressively treat blood loss, as hemodynamic instability exacerbated by decreasing intravascular volume
  - Very sensitive to effects of exogenous catecholamines. Fluid boluses preferred over vasopressors. However, if vasopressors are required, use direct-acting agents.
  - Consider controlled ventilation.

#### Emergence
- Titrations of analgesics for pain control
- Spontaneous breathing may be delayed. PaCO2 levels are a poor trigger for breathing 2° to chemoreceptor dysfunction.
- Aggressive pulm toilet: pts are at greater risk for atelectasis and bronchiectasis.

#### Postoperative Care
- Although peripheral pain sensation is diminished, visceral pain sensation is usually intact and presents as anxiety, Htn, or tachycardia and can precipitate dysautonomic crisis. Treat pain with careful titration of narcotics and/or consider NSAIDs.
- Maintain CV stability with fluids, plasma expanders, blood products, and mineralocorticoids as needed.
- Consider resp optimization with assisted ventilation, deep suctioning, inhalational therapies, and/or chest physiotherapy.

### Anticipated Problems/Concerns
- Resp function often compromised by aspiration, hypotonic musculature, and scoliosis, and abn response to hypoxemia and hypercarbia; some authors advocate endotracheal intubation until pain Rx no longer needed
  - Autonomic instability characterized by hypotension and/or Htn, arrhythmias, central sleep apnea, and temp dysregulation

#### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Orthostatic hypotension</td>
<td>Dizziness, syncope</td>
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<td>Autonomic function, EKG</td>
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<td></td>
<td>QTC prolongation</td>
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<td>Arrhythmias</td>
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<tr>
<td>RESP</td>
<td>Pneumonia</td>
<td>Pleuritic chest pain</td>
<td>Wheezing, clubbing of digits</td>
<td>CXR</td>
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<td></td>
<td>Bronchiectasis</td>
<td>Secretions</td>
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<td>GI</td>
<td>Poor swallowing</td>
<td>Dromesis, vomiting, Hx of “attacks”</td>
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<td>Swallow study</td>
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<td></td>
<td>Aspiration pneumonia</td>
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<tr>
<td>GU</td>
<td>Dehydration</td>
<td>Emesis, nocturia, diaphoresis</td>
<td>Dry mucosa, skin turgor</td>
<td>Electrolytes, serum BUN, Cr</td>
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<td>Glomerulosclerosis</td>
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<tr>
<td>CNS</td>
<td>Seizure</td>
<td>Seizure</td>
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<td>EEG</td>
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</table>

Familial Periodic Paralysis (Hyperkalemic)

Risk
- Rare, probably about 1/200,000
- Race appears to be exclusively Caucasian
- Presents usually in childhood

Perioperative Implications
- No reported increase in mortality with any procedure, but severe myotonia could create resp difficulty
- Succinylcholine may not give relaxation, and therefore an unexpected difficult intubation may result
- Succinylcholine may also cause hyperkalemia and cardiac arrhythmia

Preoperative Preparation
- 24 hr furosemide for K+ depletion
- Temp (esophageal) (keep warm)
- ECG (detection of hyperkalemia)
- Neuromuscular (minimize relaxant dose)

Monitoring
- Temp (esophageal) (keep warm)
- ECG (detection of hyperkalemia)
- Neuromuscular (minimize relaxant dose)

Airway
- No special difficulty, but may need support

Preinduction/Induction
- Regional techniques are appropriate
- Avoid ketamine and succinylcholine
- Relaxation with nondepolarizing agents as indicated

Maintenance
- Keep warm
- Warm all IV fluid, use glucose 5% as maintenance

Extubation
- Normal reversal as indicated clinically
- Evidence of muscle weakness should be treated with IV calcium gluconate or chloride 10% 10 mL slowly over 5 min.

Adjuvants
- Some experimental evidence suggests that condition, e.g., postop weakness, may be helped by phenytoin or by salbutamol.
- Anticipate normal analgesic requirements for age and surgery.
- Regional techniques are appropriate.

Anticipated Problems/Concerns
- Severe myotonia may create resp difficulty.
- Succinylcholine may not give relaxation, and therefore intubation may be difficult.
- Cold or hypoglycemia can trigger hyperkalemic attack.
- Hyperkalemia can cause cardiac arrhythmia.

Overview
- Intrinsic defect in muscle membrane allows depolarization of the muscle, but Na+ channel does not close. Membrane thus remains inexcitable and a variable K+ efflux continues.
- Pt may experience profound global stiffness and weakness after succinylcholine, exposure to cold, or spontaneously.
- Dx by family Hx

ICD-9-CM Code: 359.3

Etiology
- Na+ channel in skeletal muscle membrane has a defective α subunit.
- Defect associated with chromosome 17 is substitution of a single base pair, usually methionine replacing threonine in fifth transmembrane segment of second domain.

Antus Treatment
- Avoid succinylcholine.
- Avoid cooling during anesthesia.
- Avoid hypoglycemia.
- Do not give K+-containing solutions.
- Preop treatment with furosemide has been used.
- Severe postop weakness may be alleviated with Ca2+.

Assessment Points

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>Weakness</td>
<td>Exercise, fatigue</td>
<td>Limb tone</td>
<td>Electromyography (discharges) K+ load</td>
</tr>
</tbody>
</table>

Familial Periodic Paralysis (Hypokalemic)

### Risk
- Rare, about 1 in 100,000
- Appears to occur in most races
- Presents usually in childhood or adolescence

### Perioperative Risks
- Assoc with supraventricular or conduction defect–type cardiac arrhythmias
- Treatment with lidocaine is contraindicated.
- Weakness may be enhanced or precipitated by β-adrenergic blocking drugs
- Resp muscle weakness may occur postop.

### Worry About
- Attacks after glucose intake or insulin administration
- Cold triggers attacks
- Serum K+ levels should be maintained above 4.0 mEq/L.
- Cardiac dysrhythmias, esp bradycardias, during an attack

### Overview
- Any severe hypokalemia may induce paralysis in susceptible persons even if they do not have familial disease. Limb weakness and paralysis have been reported after thyrotoxicosis, starvation, autoimmune and renal disease.
- Familial hypokalemic periodic paralysis is an autosomal dominant condition with reduced penetrance in females.
- Usually the pt will be aware of the onset of weakness
- Prompt treatment with K+ will usually abort an attack, although as much as 40 mEq of K+ may be required hourly
- Attacks are most likely with anything that increases muscle activity; can be precipitated by exercise and also cold, presumably because of the increased muscle activity in shivering. Usually there will be a CK increase during an attack.
- The symptoms in many pts can be controlled by regular K+ supplements and acetazolamide.

#### ICD-9-CM Code: 359.3

### Etiology
- The intrinsic defect in muscle membrane appears to be assoc with gene localized to 1q11–1q12 region near the dihydropyridine receptor gene.
- Unrelated to familial hyperkalemic disease
- Gene defect substitutes an arginine and impairs voltage-sensitive Ca2+ channel in about 70% of pts. Less commonly, in about 10% the Na+ channel is affected. The change causes a compensatory increase in the Na+/K+/Cl− co-transport and a reduced overall efflux in K+

### Usual Treatment
- Avoid cooling during anesthesia, hyperglycemia
- Give solutions containing K+. Up to 30 mEq IV over 1 hr, aim for K+ 4–5 mEq/L.
- Acetazolamide should be considered, if not already being given.
- Severe postop weakness may be aggravated by Ca2+.
- Ventilation during anesthesia should be normocarbic to avoid K+ shifts.
- Maintenance by IPPV if evidence of weakness in postop phase

#### ASSESSMENT POINTS

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<tbody>
<tr>
<td>RESP</td>
<td>Inadequate</td>
<td>Noticeable SOB</td>
<td>Resp rate high</td>
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<tr>
<td>MS</td>
<td>Weakness</td>
<td>Exercise, fatigue</td>
<td>Limb tone</td>
<td>Serum K+</td>
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<td></td>
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<td>elevation</td>
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</tbody>
</table>


### Perioperative Implications

#### Preoperative Preparation
- 24-hr acetazolamide if not already given. Only glucose-free solutions IV. If Hx of frequent instability, prepare infusion with K+.

#### Monitoring
- Temp (esophageal). Keep warm.
- ECG (detection of hypokalemia may not be seen until late)
- NM (minimize relaxant dose)

#### Airway
- No special difficulty, but may need support

#### Preinduction/Induction
- Regional techniques are appropriate.

#### Induction
- Successful relaxation with succinylcholine and with atracurium has been reported.

#### Maintenance
- Use warming blanket.
- Warm all IV fluid, use glucose-free solutions as maintenance.

#### Extubation
- Normal reversal as indicated clinically
- Evidence of muscle weakness should be treated with IV potassium chloride up to 40 mEq/hr

#### Adjuvants
- Ca2+–channel blockers do not appear to be contraindicated in pts with concomitant CV disease.
- Anticipate usual analgesic requirements for age and surgery.

#### Anticipated Problems/Concerns
- May have assoc supraventricular or conduction defect arrhythmias
- Resp muscle weakness may occur postop.
- Cold triggers attack.
- Must maintain serum K+ above 4.0 mEq/L
**Fat Embolism**

**Risk**
- Long bone fractures, pelvic fractures
  - 80–100% fat embolism
- 0.2–19% fat embolism syndrome (FES)
- Male > female
- Adult >> pediatric
- Multiple fractures > single fractures
- Pathologic fractures > traumatic fractures
- Total hip, total knee replacement, intramedullary nailing:
  - 27–100% fat embolism
- Unknown incidence FES
- Unusual causes: Liposuction, fat injection, bone marrow harvest and/or transplantation, vertebroplasty, cardiopulmonary bypass, CPR, burns, pancreatitis, sickle cell disease, osteomyelitis, fatty liver, soft tissue injury

**Perioperative Risks**
- FES: 7–20% mortality
- Pre-existing FES: Respiratory failure/ARDS, RV dysfunction, shock, coagulopathy, neurologic dysfunction
- Intraop fat embolism: Shock, hypoxemia

**Worry About**
- Pre-existing FES: Hypoxemia, reduced pulmonary compliance, hypotension, cardiac arrest, Pulm Htn, RV failure, abnormal CNS response to anesthetic, coagulopathy
- Intraop embolism: Hypotension, right ventricular failure, hypoxemia, paradoxical embolization, neurologic dysfunction

**Overview**
- Fat particles (globules of marrow fat) traveling into blood and lung
- Must distinguish fat embolism, which is common, from FES, a much less common consequence of fat embolism
- FES can produce mild pulm dysfunction to severe ARDS.
- Pulm Htn and acute right ventricular failure may occur in severe cases of FES.
- Typically, the onset of signs and symptoms of FES is delayed up to 72 hr following injury.
- Fat embolism occurs commonly during femoral reaming and cementing in hip arthroplasty.
- FES is confounded with cement reaction during arthroplasty.

**ICD9-CM: 673.8 (Other pulmonary embolism)**

**Etiology**
- Most frequently follows orthopedic trauma with release of marrow fat into venous circulation
- Pathology produced by mechanical obstruction by intravascular fat passing into the pulm and systemic arterial circulation and by production of endogenous inflammatory mediators

**Usual Treatment**
- Early fracture fixation to decrease embolization
- Use of noncemented prosthesis or venting of femoral shaft may reduce embolization during hip arthroplasty
- Unreamed nailing for fracture fixation to reduce embolization
- O2 therapy to maintain SaO2 >90%
- Low tidal volume ventilation strategy with PEEP for ARDS
- Aggressive hemodynamic support with fluid and/or inotropes for shock and/or RV failure
- Factor replacement for coagulopathy with bleeding
- Corticosteroids, heparin, ethanol, dextran, aspirin, prophylactic venous calf filter: Unproven benefit

**ASSESSMENT POINTS**

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<td>Fever</td>
<td>?Fat staining of blood</td>
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<td>Hypoperfusion</td>
<td>Syncope</td>
<td>TEE, CVP, ?PA catheter</td>
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<td>Pulm Htn</td>
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<td>Lactic acidosis</td>
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<td>RV failure</td>
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<td>Anemia</td>
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<td>Bleeding (rare)</td>
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<td>Coma (rare)</td>
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**Perioperative Implications**

**Preoperative Preparation**
- Avoid sedatives and/or narcotics if hypoxicemic and not mechanically ventilated or with obtundation

**Monitoring**
- Arterial catheter
- TEE, CVP, ?PA catheter to diagnose and manage right ventricular failure and/or pulm Htn

**Airway**
- May already be intubated and ventilated in severe cases
- Decreased FRC, O2 reserve and tolerance of apnea with ARDS

**Induction**
- Minimize myocardial depression

**Maintenance**
- CV: Anticipate decrease in BP with femoral reaming/cementing: anesthetic reduction, fluid, vasopressors; pts with RV dysfunction may require longer-term inotropic support
- Resp: pts with ARDS may require increased FIO2 and PEEP; utilize lung protective strategy with low tidal volume ventilation
- Heme: Factor replacement for coagulopathy with bleeding

**Exubation**
- Maintain intubation and mechanical ventilation in hemodynamically unstable pts and those requiring increased FIO2, PEEP or with reduced compliance
- Pts with CNS involvement may have a prolonged or exaggerated response to anesthetics and narcotics and may require intubation postop for airway protection/patency

**Anticipated Problems/Concerns**
- Embolism during femoral reaming, prosthesis cementing
- FES may be delayed by up to 72 hr following fat embolism
- Pts with ARDS may be difficult to ventilate and oxygenate
- Hypotension is due to RV dysfunction and pulm Htn
Foreign Body Aspiration

Overview
• Acute presentation, with parent or caretaker witnessing the child swallowing or aspirating a foreign body and immediately developing cough, dyspnea, and stridor
• Foreign body aspiration into the airway or esophagus is one of the most frequent and threatening pediatric surgical emergencies
• Morbidity and mortality ranges from 0–4.8%

Risk
• Transfer to a specialized facility should be considered if a stable resp status.
• Urgent cases should go directly to the OR

Perioperative Management
• Divided into three time periods: preop, intraop, and postop
• NPO period: The stomach will not empty, so proceed expeditiously.
• An IV should be started if possible and an anti-cholinergic agent administered. If the child has pneumonia, addition of antibiotics is indicated.
• Rapid sequence intubation for full stomach situations
• Light anesthesia should be avoided.
• Small amounts of PEEP (3–5 cm H2O) useful for any degree of obstruction
• Topical anesthesia of larynx and cords with 4% solution of lidocaine, 5 mg/kg (4% Lidocaine contains 40 mg lidocaine/mL) prior to laryngoscopy, so no response to introduction of ventilating bronchoscope
• If an IV is present, small doses of propofol (1 mg/kg) to make inhalation induction smoother

Perioperative Risks
• Risk of aspiration is present but is very small. The danger period for vomiting or regurgitation with aspiration is primarily during the induction and recovery from anesthesia.
• Light anesthesia should be avoided.
• Small amounts of PEEP (3–5 cm H2O) useful for any degree of obstruction
• Topical anesthesia of larynx and cords with 4% solution of lidocaine, 5 mg/kg (4% Lidocaine contains 40 mg lidocaine/mL) prior to laryngoscopy, so no response to introduction of ventilating bronchoscope
• If an IV is present, small doses of propofol (1 mg/kg) to make inhalation induction smoother

Monitoring
• ETCO2 (also the wave form) may be elevated into the 80s or 90s. As long as O2 saturation remains in 85–95 range, the CO2 is usually not a problem.
• Using a ventilating bronchoscope with a sidearm attachment for anesthesia circuit, bronchoscope advanced through larynx into trachea and often into main stem bronchus. Desaturation may result from inadequate ventilation of contralateral lung. If this occurs consider administering PEEP until saturation can be returned to reasonable range.
• With pneumonia, saturations may not be able to be raised higher than the low 90s. Saturation of 85–90 is acceptable as long as it is stable. If rapidly falling O2 saturation, bronchoscope must be withdrawn from trachea and ventilation assisted with PEEP.
• The critical part of the surgery occurs when the surgeon extracts the foreign body from the airway. If the child starts to move or cough, management is either releasing foreign body and deepening the anesthetic; or administering either muscle relaxant such as succinylcholine, or 1 mg/kg of propofol, lidocaine, or ketamine (1 mg/kg) to deepen anesthesia.

Anticipated Problems/Concerns
• Hypercarbia
• Hypoxia
• Atelectasis
• Bronchitis

Usual Treatment
• Rigid bronchoscopy
• Foreign bodies found 70–90% of the time

Key References:

Perioperative Management
• Done in OR without presence of parents
• A technique of spontaneous ventilation usually with sevoflurane
• If child is struggling to breathe or cyanotic, induction is with sevoflurane and O2.
• If only mild airway distress, nitrous oxide used for initial inhalation induction to facilitate administration of sevoflurane. Sevoflurane is the ideal induction agent. In addition, small doses of ketamine or a propofol infusion may be added to stabilize the maintenance anesthetic.

Perioperative Concerns
• A technique of spontaneous ventilation usually with sevoflurane
• If child is struggling to breathe or cyanotic, induction is with sevoflurane and O2.
• If only mild airway distress, nitrous oxide used for initial inhalation induction to facilitate administration of sevoflurane. Sevoflurane is the ideal induction agent. In addition, small doses of ketamine or a propofol infusion may be added to stabilize the maintenance anesthetic.

Preoperative Concerns
• A technique of spontaneous ventilation usually with sevoflurane
• If child is struggling to breathe or cyanotic, induction is with sevoflurane and O2.
• If only mild airway distress, nitrous oxide used for initial inhalation induction to facilitate administration of sevoflurane. Sevoflurane is the ideal induction agent. In addition, small doses of ketamine or a propofol infusion may be added to stabilize the maintenance anesthetic.
Friedreich’s Ataxia

**Risk**
- Prevalence: 2/100,000; 80–90% have cardiac involvement

**Worry About**
- Cardiac involvement does not correlate with neurologic involvement
- Electrophysiologic disturbances
- Cardiac dysfunction and failure

**Overview**
- Progressive degeneration of posterior columns and corticospinal and posterior spinocerebellar tracts
- Muscle weakness
- Abnormal glucose homeostasis

**Risk**
- Usual onset in childhood
- Proprioceptive sensory loss, areflexia, ataxia of limbs, Babinski’s sign
- Pes cavus and scoliosis
- Cardiomyopathy

**ICD-9-CM Code: 334.0**

**Etiology**
- Inherited: Usually autosomal recessive, but occasionally dominant
- Nucleotide has been mapped
- Fratoxin (mitochondrial iron content protein) deficiency

**Overview**
- Progression: Degeneration of posterior columns and corticospinal and posterior spinocerebellar tracts
- Muscle weakness
- Abnormal glucose homeostasis
- Usual onset in childhood
- Proprioceptive sensory loss, areflexia, ataxia of limbs, Babinski’s sign
- Pes cavus and scoliosis
- Cardiomyopathy

**Perioperative Implications**

**Preoperative Preparation**
- Usual premedication

**Monitoring**
- Train of four to monitor effects of neuromuscular blocking agent with unpredictable response due to neuromuscular disease

**Airway**
- None

**Preinduction/Induction**
- Case report of sensitivity to curare (0.06 mg/kg caused 90 min apnea)
- Possibility of hyperkalemia and cardiac arrhythmias after succinylcholine

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>LV hypokinesia</td>
<td>Severe</td>
<td>Cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>Concentric and asymmetric hypertrophy</td>
<td>Hypokinesia and asymmetry</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Severe scoliosis</td>
<td>Noncardiac dyspnea</td>
<td>Lung functions</td>
</tr>
<tr>
<td>MS</td>
<td>Pes cavus</td>
<td>Ability to walk without assistance</td>
<td>Cardiomyopathy</td>
</tr>
</tbody>
</table>


**Usual Treatment**
- Usually untreatable and progressive
- Medical management of cardiac abnormalities
- Scoliosis repair
- Can be mistaken for metabolic disorders (hexosaminidase A deficiency, adrenomyeloneuropathy, vitamin E deficiency)
- Clinical trials of coenzyme Q10 (CoQ10)/vitamin E
Gastrinoma

Risk
- Mean age of onset in the fourth decade
- Slight male predominance
- Less than 0.1% of all PUD caused by gastrinomas
- 60% of gastrinomas are malignant

Perioperative Risks
- Risks associated with PUD
- Assoc tumors (MEN type I)
- Risks assoc with metastatic disease (regional lymph nodes, liver, bone)

Worry About
- Large gastric fluid volume
- Esophageal reflux (common)
- Intravascular volume depletion

Overview
- Gastrinoma is a gastrin-secreting neuroendocrine tumor (non-beta islet cell tumor) occurring most commonly in duodenum or pancreas
- Gastrin release stimulates gastric acid hypersecretion which causes symptoms of abdominal pain (due to refractory peptic ulcer disease), diarrhea and GERD known as Zollinger-Ellison syndrome
- Diagnosis often delayed several years from onset of symptoms because of difficulties distinguishing it from other cases of PUD

Perioperative Implications

Preoperative Preparation
- Assure adequate treatment of gastric hypersecretion
- Evaluate for other endocrinopathies of MEN I syndrome
- Assess volume status
- Check electrolytes and coagulation tests
- Consider preop NG tube placement

Monitoring
- May need central venous pressure monitoring and arterial line due to intravascular volume depletion causing hypotension and potential for fluid shifts. UO should be measured with a bladder catheter.

Assessment Points

<table>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Abdominal pain, dizziness</td>
<td>Vital signs</td>
<td>Orthostatics</td>
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<tr>
<td>GI</td>
<td>Gastric acid hypersecretion</td>
<td>Abdominal pain, esophageal reflux, diarrhea</td>
<td>Abd exam</td>
<td>Fasting gastrin levels, Secretin stimulation test</td>
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<tr>
<td>FEN</td>
<td>Hypokalemia</td>
<td>Weakness, muscle cramps</td>
<td>Electrolytes, EKG</td>
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<tr>
<td>ENDO*</td>
<td>Hyperparathyroidism</td>
<td>Multiple systems involved</td>
<td>Serum parathyroid hormone</td>
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<tr>
<td>RENAL*</td>
<td>Nephrolithiasis</td>
<td>Flank pain, hematuria</td>
<td>Costovertebral angle tenderness</td>
<td>Urinalysis</td>
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<tr>
<td>CNS*</td>
<td>Pituitary adenoma</td>
<td>Headaches, visual changes</td>
<td>Visual fields</td>
<td>MRI; prolactin level</td>
</tr>
<tr>
<td>MS*</td>
<td>Weakness, arthralgias</td>
<td>Proximal muscle weakness</td>
<td>Motor strength, Hyperreflexia</td>
<td>Ca²⁺ levels</td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulation disorder</td>
<td>Bleeding abn</td>
<td>PT, PTT</td>
<td></td>
</tr>
</tbody>
</table>

* If gastrinoma presents as component of MEN I.


Induction
- Treat as full stomach due to increased gastric acid volumes and increased risk for aspiration
- Rapid-sequence induction with cricoid pressure

Exubation
- Assess adequate ventilatory function and recovery from neuromuscular blockade prior to extubation

Adjuvants
- May consider epidural catheter for postop pain control

Postoperative Period
- Decreased vital capacity and FRC due to pain and ileus
- Concern for continued symptoms if surgical resection not curative

Anticipated Problems/Concerns
- Lower cure rates after resection for pts with gastrinomas assoc with MEN I or in the presence of metastatic liver disease

ICD-9-CM Code: 235.5
See also Multiple Endocrine Neoplasia (MEN) Types I and II in Diseases section

Etiology
- May be inherited as autosomal dominant trait when associated with MEN I

Usual Treatment
- Control gastric acid hypersecretion with proton pump inhibitors and H₂ blockers
- Surgical exploration and resection
**Gastroesophageal Reflux in Children**

**Risk**
- Symptoms of gastroesophageal reflux (GER) persist past 6 wk of age in 1/300 infants
- 60% resolve by age 18 mo; 30% persist beyond age 4 y
- 5% develop esophageal stricture
- 5% die of complications of GER
- 10% of pyloric stenosis pts
- After diaphragmatic hernia, tracheoesophageal fistula, and esophageal atresia repairs
- Neurologically impaired or developmentally delayed children with spastic quadriplegia, hypoxic brain damage, or trisomy syndromes

**Overview**
- GER is defined as regurgitation without pathological consequences. GER disease (GERD) is defined as regurgitation resulting in esophagitis, nutritional compromise, and/or resp complications.
- Presence of a hiatal hernia does not necessarily mean pt will have GER
- Older children may complain of heartburn and chest pain
- Degree of reflux, duration of acid exposure in the esophagus, and ability of the esophagus to clear the reflux material determine extent of mucosal damage and degree of esophagitis
- Esophagitis may lead to bleeding, which may result in hematemesis, iron-deficiency anemia, and esophageal stricture. Also, predisposes to Barrett esophagus
- GER may be a cause of neonatal apnea.
- Diagnostic procedures incl upper GI series, esophagoscopy, and esophageal pH probe.

**ICD-9-CM Code**: 530.81

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Chronic aspiration</td>
<td>Cough, cyanotic episodes, apnea</td>
<td>Rales, rhonchi</td>
<td>CXR, ABGs (if indicated)</td>
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<tr>
<td></td>
<td>RAD</td>
<td>Dyspnea, wheezing, cough</td>
<td>Wheezing ↓ BS, prolonged expiration</td>
<td>CXR, peak flow ABGs (if indicated)</td>
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<tr>
<td>HEME</td>
<td>Iron deficiency</td>
<td></td>
<td>Pallor</td>
<td>CBC</td>
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<tr>
<td>GENERAL</td>
<td>Malnutrition</td>
<td>Wt loss ↓ SQ tissue</td>
<td></td>
<td>Serum albumin</td>
</tr>
</tbody>
</table>

**Perioperative Risks**
- Aspiration during induction of anesthesia
- Severe bronchospasm in pts with reactive airway disease (RAD)
- Decreased pulm reserve 2° to chronic aspiration and pneumonia

**Worry About**
- Pulm complications from aspiration pneumonia and RAD
- Anemia and malnutrition

**Perioperative Implications**

**Preoperative Preparation**
- Assess the severity of pulm compromise
- Optimize resp status: Treat pneumonia and control bronchospasm
- Correct anemia
- Improve nutritional status
- Confirm availability of blood
- Continue acid-suppressing therapy

**Monitoring**
- Consider arterial line

**Induction**
- At risk for aspiration. Rapid-sequence induction with cricoid traction.
- For pts with RAD, ensure adequate depth of anesthesia prior to instrumenting the airway

**Maintenance**
- No one anesthetic preferred
- Avoid N₂O in laparoscopic procedure
- Esophageal bougie may be required
- Watch for possible pneumothorax, trauma to viscera, hemorrhage, and vena cava compression or laceration. Air embolism may occur during laparoscopic procedures.

- During laparoscopic procedures, intra-abdominal pressures of ≤12 mmHg should be maintained.

**Extubation/Postoperative Period**
- May be extubated after uncomplicated surgery
- Pts with severe resp compromise preop or with neurologic impairment may require a period of postop ventilation.
- Analgesic requirements will be less after laparoscopic procedures.

**Surgical Procedure**
- Fundus of the stomach is wrapped around the lower part of the esophagus. May be accomplished either open or laparoscopically.
- Pyloroplasty may be performed for associated delayed gastric emptying.
- Pneumoperitoneum created during laparoscopic surgery will result in increased SVR, increased CVR, increased CO₂, and increased BP. Intra-abdominal pressures >20 mmHg will decrease venous return and decrease CO, but the BP will remain unchanged due to increased SVR.
- Pneumoperitoneum will also elevate the diaphragms, which will decrease lung volumes, decrease FRC, decrease compliance, increased airway resistance, and increased V/Q mismatch.
- Pneumoperitoneum should not exceed 12 mmHg. Pts are placed in the reverse Trendelenburg position. This will help ameliorate both diaphragmatic elevation and the CVP elevation.
- Pneumoperitoneum is accomplished by the insufflation of CO₂, which may necessitate increased minute ventilation.
- Laparoscopic procedures are associated with reduced rates of postop resp and wound complications and analgesic requirements, and shorter hospital stays.

**Anticipated Problems/Concerns**
- Resp compromise
- Unable to vomit postop and up to 3 mo after surgery. Therefore, intestinal obstruction in the postop period should be treated as a dire emergency.
### Glaucoma, Closed-Angle

**Risk**
- Worldwide prevalence: 15.7 million pts with angle closure glaucoma (ACG) in 2009
- Second most common cause of irreversible blindness
- Risk factors: Eskimo, Fin, or Indian race, age (>60), female gender (69%), family Hx

**Perioperative Risks**
- Further damage to the optic nerve

**Worry About**
- Glaucoma pts may be at increased risk of sight-threatening complications from orbital injections because the optic nerve is already compromised and vulnerable to pressure/ischemic damage. Rather than a retrobulbar or peribulbar block, less invasive techniques of local anesthesia may be employed (anterior sub-Tenon, subconjunctival, topical, or intracameral placement.)

**Overview**
- ACG is a chronic (or sometimes acute) condition characterized by progressive pressure/ischemic damage to the optic nerve head, due to an obstruction to the outflow of aqueous humor and consequent rise in intraocular pressure. Acute ACG is an ophthalmologic emergency.

#### ASSESSMENT POINTS

<table>
<thead>
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<th>Hx</th>
<th>PE</th>
<th>Test</th>
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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Acute ACG</td>
<td>Sudden unilateral pain</td>
<td>Ocular injection</td>
<td>Penlight</td>
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<td></td>
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<td>Blurred vision</td>
<td>Hazy cornea</td>
<td>Gonioscope</td>
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<tr>
<td></td>
<td></td>
<td>Photophobia</td>
<td>Mid-dilated pupil</td>
<td>Slit-lamp</td>
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<tr>
<td></td>
<td></td>
<td>Colored halos around lights</td>
<td>Headache</td>
<td>US biomicroscopy</td>
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<tr>
<td>Subacute ACG</td>
<td>Headaches (often mistaken for migraine) or asymptomatic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic ACG</td>
<td>Generally asymptomatic</td>
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</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Avoid mydriasis, either due to stress, dim lighting, or drugs.
- Check electrolytes if pt is on a diuretic.
- Preop antisympathetic agents or scopolamine are generally ok.
- Many pts undergoing ophthalmic surgery take anticoagulants; the relative risks of a thrombotic complication must be weighed against possible bleeding. Glaucoma surgery is of intermediate risk for serious hemorrhagic complications. A consensus is developing that cataract surgery may be safely performed while maintaining pts on warfarin. For intermediate risk procedures, such as some glaucoma surgeries, stopping warfarin for 4 d preop is indicated. A pt may continue their clopidogrel.
- Echothiopate should be D/C 4 to 6 wk prior to surgery. Systemic absorption can inhibit plasma cholinesterase (causing prolonged muscle paralysis after succinylcholine) as well as inhibit metabolism of ester local anesthetics (LA), predisposing a pt to LA toxicity.

**Induction**
- Frequently, these cases may be done under very light sedation and topical anesthesia and possibly a block performed by the surgeon. Anesthetic goals center on minimizing interventions that may increase IOP or cause further damage to the optic nerve.
- General anesthesia may be required by the surgeon or due to individual pt factors. Succinylcholine increases IOP by 6–12 mmHg for 5 to 10 min, and may be used. Endotracheal intubation causes a similar increase in IOP. Bucking, coughing, and vomiting increase IOP by 30–40 and should be avoided. ET intubation causes a similar increase in IOP. An LMA may be preferred.

**Maintenance**
- Avoid hyperventilation, which will cause choroidal vascular congestion.
- Be prepared for bradycardia and/or arrhythmia due to elevated IOP and surgical pressure

**Anticipated Problems/Concerns**
- 50–75% of pts will have ACG in the contralateral eye within 1 y; prophylactic iridotomy greatly reduces this risk.
- There is a phenomenon of severe visual loss after surgery, with no obvious cause, known as wipe out, or sniff syndrome. Local anesthetic injections are a putative cause, perhaps due to unnoticed direct trauma to the optic nerve from the needle, or to a hematoma in the nerve sheath, or simply due to the volume of the LA itself. High pressure around the nerve could potentially occur even with a low volume of LA if it were to be trapped between fascial layers and cause a compartment syndrome. Epinephrine in the LA mixture may contribute to ischemia due to a marked reduction in blood flow to the anterior optic nerve. This effect is not seen with anterior (e.g., subconjunctival) placement of LA.
- Topical β-blockers (esp. Timolol) may have systemic effects, and have been shown to exacerbate asthma and CHF, and produce bradycardia.

**ICD – 9CM Codes:**
- 365.20 (Primary ACG, unspecified), 365.21 (Intermittent ACG), 365.22 (Acute ACG), 365.23 (Chronic ACG), 365.24 (Residual Stage of ACG)

**Etiology**
- Multifactorial: Once thought to be primarily due to occulted drainage angles in the anterior chamber of the eye (leading to elevated IOP), optic nerve ischemia has been found to occur in pts with either elevated or normal IOP (which is 10–20 mmHg). Causative etiologies for the damage may also include excitotoxicity, neurotophin insufficiency, inflammatory cytokines, or immune abn.
- Precipitating factors: Dim/indoor light, anti-cholinergic agents (atropine, cyclopentolate, tropicamide, antihistamines, antipsychotics, anti-depressants, antiparkinsonian, and GI spasmyotics), adrenergic agents (topical, e.g., epinephrine and phenylephrine), or systemic, (e.g., vasoconstrictors, central nervous system stimulants, bronchodilators, appetite depressants, and hallucinogenic agents), emotional stress

**Usual Treatment for Acute ACG**
- Topical β-blocker, α2-agonist, pilocarpine 2% or 4% (pilocarpine is effective in inducing miosis only when iris ischemia is relieved, i.e., when intraocular pressure falls to <50 mmHg)

**Usual Treatment for Chronic ACG**
- IV/oral acetazolamide 5–10 mg/kg (alternatives: hyperosmotic agents, e.g., IV 20% mannitol 1–2 g/kg, oral 50% glycerol 1–1.5 g/kg (contraindicated in diabetics), oral isosorbide 1.5–2.0 g/kg)
- Topical steroids
- Lie pt supine (to allow lens-iris diaphragm to move posteriorly)
- Analgesia and antiemetics
- After 1–2 hrs, if the attack is broken and corneal edema resolves (or if it is not broken but the cornea is clear), perform laser iridotomy.
- If attack is not broken and cornea is still hazy, pt should have laser iridoplasty first, followed by laser iridotomy later when cornea edema resolves.

Glaucoma, Open-Angle

Risk
- Open-angle glaucoma is the leading cause of blindness among African Americans and the second leading cause overall in the USA.
- African American race, advanced age, elevated intraocular pressure (IOP), myopia, low diastolic perfusion pressures, and family Hx of open-angle glaucoma increase the risk for primary-open-angle glaucoma.
- Incidence in USA: Estimates suggest over 2.25 million Americans over age 40 have open-angle glaucoma.

Perioperative Risks
- Vision loss secondary to optic nerve damage from pressure or ischemia.

Worry About
- Interactions between ophthalmologic drugs and anesthetics

Overview
- Glaucoma is a degenerative optic neuropathy characterized by optic-nerve cupping that results in progressive vision loss and possibly blindness if not treated. Treatment does not reverse the blindness.
- Elevated IOP is often found in glaucoma but is not required for the diagnosis. Nonetheless, treatment for all forms is aimed at maintaining a low-normal IOP.
- Onset is gradual, bilateral, and often unnoticed. While juvenile forms exist, it is much more common in those above 40 y of age.

ICD-9-CM Code: 365.10

ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Optic nerve damage, increased IOP</td>
<td>Visual changes, family Hx of glaucoma, myopia</td>
<td>Decreased visual acuity, increased optic cup-to-disk ratio, visual field losses</td>
<td>Slit lamp exam</td>
</tr>
<tr>
<td>CV</td>
<td>Excessive beta blockade</td>
<td>Fatigue, syncope or near-syncope, SOB, chest pain</td>
<td>Hypotension, bradycardia</td>
<td>Tonometry Visual fields Visual acuity</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preinduction
- Maintain miosis by continuing topical and systemic treatment medications with the exception of echothiophate, which should be stopped four weeks prior to elective surgery.
- Pts taking acetazolamide, a carbonic anhydrase inhibitor, should have electrolytes checked preop with specific attention to Na⁺, K⁺, and bicarbonate levels.
- Antisialogogue premedication with glycopyrrolate or atropine is not contraindicated; however, several texts suggest scopolamine should be avoided due to its greater mydriatic effect.

Induction
- Blunt increases in IOP during laryngoscopy and intubation, which tend to decrease IOP. Controversy surrounds ketamine's effect.
- Succinylcholine is safe for induction and intubation, provided echothiophate has been discontinued.
- Hypotension should be avoided due to optic nerve perfusion concerns.

- Increases in IOP
- Periop derangements in electrolytes secondary to ophthalmologic drugs

Etiology
- Likely caused by sclerosis of the trabecular meshwork at the canal of Schlemm which decreases aqueous humor outflow and elevates IOP
- Normal pressure, open-angle glaucoma is thought to be caused by insufficient blood flow leading to optic nerve damage, but treatment is the same as with primary open-angle glaucoma.

Usual Treatment
- Treatment goal is to maintain a low-normal IOP. Treatment is most successful if disease is detected early.
- Medical treatment incl topical timolol, beta-blockers, echothiophate, or dipivefrin and oral acetazolamide.
- Surgical treatment incl laser trabeculoplasty, trabeculectomy, Baerveldt and Ahmed device implantation, and cycloablative.

Regional Anesthesia
- Ester local anesthetics should be avoided in pts taking echothiophate due to reduced plasma cholinesterase activity and altered metabolism.

Exubation
- Avoid coughing and bucking, which can cause acute increases in IOP.
- Neuromuscular blockade reversal agents and antimuscarinics in usual dosages are considered safe.

Postoperative Period
- If emergency surgery is required in pts currently taking echothiophate, expect the need for prolonged postop ventilation.

Anticipated Problems/Concerns
- Avoid increases in IOP.
- Echothiophate therapy produces decreased plasma cholinesterase activity and should be stopped 4 wk prior to surgery to avoid a prolonged paralysis with the use of succinylcholine.
- Be aware that topical beta-blockers are systematically absorbed and can have systemic effects.
Glomus Jugulare Tumors

Risk
- 0.6% of head and neck tumors
- M:F ratio: 1:2.5
- Slow-growing
- Can co-exist with other paragangliomas
- Histologically benign but can be malignant with metastases

Overview
- Tumors of neural crest at base of skull in jugular bulb area
- Highly vascular
- May extend into the posterior fossa
- May cause hydrocephalus
- May damage the lower cranial nerves (IX–XII)
- May involve internal carotid artery
- May grow into lumen of the jugular vein, as far as the right atrium
- May secrete catecholamines: 5%
- May secrete serotonin, histamine

ICD-9-CM Codes: 194.6 (Malignant); 237.3 (Paraganglia)

Etiology
- Congenital (usually benign) hypertrophied arteriovenous anastomosis
- Epithelial cells with abundant capillary network

Usual Treatment
- Resection
- Embolization, alone or pre-resection
- Radiation
- Gamma knife

Worry About
- Multiple locations, persistence of symptoms after resection of the tumor

Perioperative Risks
- Hypothermia
- Massive blood loss
- Venous air embolism
- Htn
- Bradycardia
- Hypotension
- Bronchospasm
- Tumor-parts embolization

Perioperative Implications

Preoperative Preparation
- Control Htn (in catecholamine-secreting tumors). Preparation is similar to pheochromocytoma (see under Pheochromocytoma in Diseases section)
- Treat pneumonia
- Metoclopramide for delayed gastric emptying
- Adequate venous access for rapid fluid infusion

Monitoring
- Consider A-line, CVP
- Monitor for venous air embolism (frequent ABG, ETCO₂, N₂; precordial Doppler)
- Cerebral oximetry
- Facial nerve

Maintenance
- Watch out for massive blood loss, Htn, hypotension, bradycardia, bronchospasm, venous air embolism, tumor-parts embolization
- Provide controlled hypotension if needed
- Measure to decrease the ICP for intracranial extension:
  - Mannitol
  - Hyperventilation
  - Optimize venous return from brain
  - CSF drainage

Exubation
- Evaluate for cranial nerves (IX–XII) injury

Adjuvants
- Controlled ventilation
- Muscle relaxants to prevent spontaneous ventilation intraop
- Controlled hypotension

Anticipated Problems/Concerns
- Loss of upper airway reflexes
- Airway obstruction
- Aspiration
- Delayed gastric emptying
- Ileus
- CNS insult
- CSF leak


ASSESSMENT POINTS

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<tbody>
<tr>
<td>HEENT</td>
<td>Cranial nerve injury</td>
<td>Hoarseness Dysphagia Tinnitus Vertigo</td>
<td>Tongue movement Soft palate motion Gag reflex Hearing test</td>
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<tr>
<td>CV</td>
<td>Htn Intravascular growth</td>
<td>Headache Palpitations</td>
<td>BP</td>
<td>Catecholamines level MRI/CT scans Angio (if indicated)</td>
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<tr>
<td>RESP</td>
<td>Aspiration</td>
<td>Cough Fever SOB</td>
<td>Rhonchi, wheezing</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Delayed gastric emptying</td>
<td>Heartburn Regurgitation</td>
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<td></td>
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<tr>
<td>GU</td>
<td>Intracranial extension</td>
<td>Hearing loss Headache Dizziness</td>
<td>CT scan MRI (if indicated)</td>
<td>Paragangliomas in other locations</td>
</tr>
</tbody>
</table>

Glossopharyngeal Neuralgia

R. David Todd

**Overview**
- Rare: Constitutes <1% of cases of facial pain
- Sudden and unilateral pain involving the pharynx, tonsils, base of tongue, with radiation to the throat and/or ear structures (cranial nerve IX and X distribution).
- Attacks may be triggered by chewing, swallowing (cold fluids), talking, coughing, or sneezing.
- Sudden paroxysms usually are <1 min and average 5 per hr but can be longer lasting and more frequent.
- Clusters of attacks can last weeks to months.
- Dx made when application of topical anesthetic solution to oropharynx relieves pain.

**ICD-9-CM Code:** 352.1

**Etiology**
- Usually idiopathic
- Secondary causes incl:
  - Vascular compression of the glossopharyngeal nerve
  - Neoplasms: Cerebellopontine tumors, laryngeal and tongue carcinomas
  - Infection and/or inflammation: Tonsillitis, pharyngeal abscess, arachnoiditis
  - Trauma: Tonsillectomy, dental extraction, impacted wisdom tooth

**Usual Treatment**
- Conservative treatment: Anticonvulsants-carbamazepine, gabapentin, phenytoin
- Microvascular decompression is the preferred surgical method with high success in pts with typical symptoms
- Surgical alternative incl sectioning of the glossopharyngeal (IX) nerve

**ASSESSMENT POINTS**

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<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Bradycardia, tachycardia, syncope, hypotension</td>
<td>Syncope, palpitations</td>
<td>BP, HR</td>
<td>EKG or biotelemetry to capture pain attacks</td>
</tr>
<tr>
<td>CNS</td>
<td>Pain in IX/X distribution</td>
<td>Paroxysmal pain attacks in IX/X distribution with various triggers</td>
<td>Attempt to trigger pain and find distribution</td>
<td>MRI/MRA to ID etiology and vascular compression</td>
</tr>
</tbody>
</table>


**Preoperative Implications**

**Preoperative Evaluation**
- Assessment of triggers and subsequent pain with emphasis on Hx of bradycardia, palpitations, syncope, seizures

**Monitoring**
- Preinduction arterial line in pts with significant CV symptoms and central venous catheter when temporary pacemaker might be indicated

**Airway**
- Direct laryngoscopy can trigger an attack.
- Topical anesthesia to oropharynx prior to laryngoscopy can blunt CV symptoms.
- Glossopharyngeal nerve block is an alternative to topical anesthesia for prophylaxis.

**Maintenance**
- Vigilance and promptness to treat cardiac symptoms and labile blood pressure
- Watch for sudden arterial hypotension, bradycardia, cardiac arrhythmias

**Extubation**
- Look for possible IX/X nerve palsy and subsequent vocal cord paralysis following microvascular decompression surgery.

**Anticipated Problems/Concerns**
- Direct laryngoscopy triggering a pain attack with hypotension, bradycardia, and cardiac arrhythmias
- Periop pain attack with severe uncontrolled pain
- Some pts have long Hx of narcotic use
Gonorrhea

Risk
- Decreasing; 120 per 100,000 as of 2006
- Most common in people ages 20–24 y, large urban areas, people with low socioeconomic status and/or low levels of education
- Incidence higher in men, prevalence higher in women

Overview
- Sexually transmitted disease
- High incidence of co-existing chlamydial infection

Clinical Features
- Local infection: Purulent, profuse urethral discharge; possible epididymitis, prostatitis, or proctitis in men. Often asymptomatic in women, may have cervical discharge, vaginitis, salpingitis, or proctitis. Ascending infection may lead to pelvic inflammatory disease (PID).
- Disseminated infection: Fever/rash, tenosynovitis/arthrosis (common), conjunctivitis (usually from autoinoculation), possible myopericarditis, toxic hepatitis or peripancreatitis (Fitz-Hugh–Curtis syndrome), rarely endocarditis or meningitis

ICD-9-CM Code: 098

Etiology
- Neisseria gonorrhoeae: Gram-negative intracellular diplococcus, usually found inside polymorphonuclear leucocytes
- Humans only natural hosts for N. gonorrhoeae

Usual Treatment
- Diagnosis gold standard: Isolation of organism by culture, testing for antimicrobial resistance
- Test for other STDs: Syphilis, HIV; test partners as well
- Penicillins and tetracyclines not recommended as first line agents due to resistance
- Fluoroquinolones no longer recommended as first-line therapy due to increasing resistance, esp. in men who have sex with men

Uncomplicated cervicitis/urethritis: Ceftriaxone is drug of choice; other 3rd generation cephalosporins (cefotaxime, cefpodoxime) also commonly used. Spectinomycin can be used in penicillin allergic pts.
- Add doxycycline or azithromycin for co-existing chlamydial infections
- Symptoms may subside without treatment, leaving chronic asymptomatic carrier state.
- Pharyngeal infection frequently asymptomatic; may clear spontaneously over several weeks, even without therapy. Ceftriaxone and trimethoprim-sulfamethoxazole can be used for treatment.
- Complicated infections: Penicillin G IV x 5 d or ceftriaxone x 5 d. Oral fluoroquinolones may be used provided susceptibility.
- PID requires second generation cephalosporin such as cefotetan or cefoxitin, or combination of clindamycin and gentamicin. Treat for chlamydial co-infection.
- Resolution of symptoms after treatment suggests cure; follow-up cultures are recommended.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Conjunctivitis, ophthalmia neonatorum, adult gonococcal conjunctivitis Pharyngeal infection</td>
<td>Exudative tonsillitis</td>
<td>Cultures</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Anorectal infections Proctitis</td>
<td>Pain, pruritus</td>
<td>Purulent discharge, bloody diarrhea</td>
<td>Cultures</td>
</tr>
<tr>
<td>GU</td>
<td>Women</td>
<td>Abn vaginal discharge, dysuria, urinary frequency, lower abd pain, labial pain, abn menstruation</td>
<td>Mucopurulent cervicitis</td>
<td>Cultures from urethra and vagina</td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>Acute epididymitis Prostatitis</td>
<td>Pain</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Gonococcal endocarditis</td>
<td>Possible murmur</td>
<td>Echocardiography</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Perihepatitis (Fitz-Hugh–Curtis syndrome)</td>
<td>RUQ tenderness</td>
<td>Liver enzyme elevation</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Women PID</td>
<td>Lower abdominal pain, vaginal discharge, fever, palpable adnexal mass</td>
<td>Severe pain to palpation</td>
<td>Endocervix cultures</td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>Dysuria</td>
<td>Purulent urethral discharge</td>
<td>Cultures from urethra</td>
</tr>
<tr>
<td>CNS</td>
<td>Gonococcal meningitis</td>
<td>Meningeal signs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Septic arthritis</td>
<td>Most common cause of septic arthritis in young adults, tends to involve single joints</td>
<td>Warmth, tenderness of affected joint(s)</td>
<td></td>
</tr>
<tr>
<td>SKIN</td>
<td>Disseminated lesions</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications
- Universal blood and body fluid precautions and/or barrier precautions

Monitoring
- Awareness: Foley catheter/temp probe placement

Airway
- Awareness if pharyngitis exists

Positioning
- Awareness of joint involvement

Maintenance
- Awareness of extent of disease

Adjuvants
- Vary with hepatic involvement

Anticipated Problems/Concerns
- No vaccine available
- Follow-up cultures
- Effective antibiotics
- Testing isolates for antibiotic susceptibility
- Routine culturing of high-risk populations
- Diligent contact tracing and prompt referral; treatment of sexual partners
- Education targeted at high-risk groups
- Use of condoms or other barrier methods
Guillain-Barré Syndrome

Gordon N. Finlayson
Jay B. Brodsky

Risk
- Prevalence: Both sexes, all races, all ages but mostly affects young and middle-aged adults.
- Worldwide incidence, occurs all times of year.
- Mortality rate 5–20%. Most pts eventually fully recover, 20% have significant residual weakness.

Perioperative Risks
- Resp failure 2° to polyneuropathy
- Autonomic dysfunction with profound CV instability

Worry About
- Rapidity of symptoms—resp paralysis may occur within 24 hr of onset
- Pulm complications

Overview
- Polyneuropathy often encountered in critical care practice
  - Pts present initially with lower limb weakness that spreads

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Inability to close eyes</td>
<td>Dry eyes</td>
<td>Dry eyes</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Fluctuating hypo- and Htn, postural hypotension, sinus tachycardia, arrhythmias DVT risk</td>
<td>Orthostatic Sx Palpitations</td>
<td>BP/pulse</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp failure 2° to weakness</td>
<td>Stamina—for breathing</td>
<td>Strength on repeated ventilation Inability to sustain head lift</td>
<td>VC Maximum inspiratory pressure Maximum expiratory pressure Doppler US</td>
</tr>
<tr>
<td>GI</td>
<td>Bowel obstruction</td>
<td>Constipation</td>
<td>Abdominal exam</td>
<td>Abdominal x-ray</td>
</tr>
<tr>
<td>CNS</td>
<td>Autonomic dysfunction</td>
<td>Early satiety</td>
<td>BP lying and standing</td>
<td>ECG with R-R interval on deep breathing</td>
</tr>
<tr>
<td></td>
<td>Pain: Acute nociceptive and chronic neuropath</td>
<td>Orthostatic hypotension Lack of sweating Pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Weakness, joint fixation</td>
<td>Lack of stamina</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Avoid rapid turning of pt: Autonomic instability and postural hypotension may result.
- Avoid head-up (reverse Trendelenburg) position: Inability of pt to maintain CV stability with tilt.
- Increased gastric acidity: Treat with antacid and metoclopramide, 10 mg/70 kg.
- Maintain appropriate environmental temp.
- Coagulopathy and hypocalcemia may complicate plasma exchange therapy.

Monitoring
- Arterial line for continuous pressure monitoring started prior to anesthetic induction
- CVP or PA line to monitor for potential fluid shifts that result from positional changes and cardiac dysrythmias
- Temperature: Pts may become poikilothermic
- Neuromuscular monitoring: Sensitive to relaxants

Airway
- Most pts have early tracheostomy; airway access should not be a problem; previous pts may have tracheal stenosis.
- Endotracheal suction may provoke bradysrythmias and asystole.

Induction
- Avoid barbiturates and phenothiazines, which may produce profound CV depression.

Maintenance
- Local anesthesia preferred
- GA: Non-sympatholitic technique
- Sensitive to positive pressure ventilation: May result in autonomic instability

Exudation
- Continue to ventilate postop if pt required ventilatory support preop
- Residual weakness from anesthetic agents and muscle relaxants may necessitate postop ventilation in pts not ventilated preop

Adjutants
- Muscle relaxants
  - Avoid succinylcholine; can cause hyperkalemia with cardiac arrest

Usual Treatment
- Basis of treatment is symptomatic care and plasma exchange or IVIG
- Daily bedside evaluation of vital capacity and resp muscle strength; pts with ↓ resp reserve should be moved to ICU
- Elective tracheal intubation and mechanical ventilatory support when signs of resp distress are present even before Paco, rises or vital capacity falls
- Anticipating requirement for ventilatory support
  - Vital capacity <20mL/Kg or reduction of 30% from baseline
  - Maximum inspiratory pressure <30cmH2O
  - Maximum expiratory pressure <40cmH2O
  - Facial and/or bulbar weakness, autonomic dysfunction, rapid disease progression
- Plasmapheresis or IVIG reduces hospital stay and time spent on ventilator if given to pts who do not improve or who worsen within first 2 wk of symptom onset

Worldwide illness, occurs all times of year.
- Neurological involvement.
  - Case reported of newborn with GBs features following delivery by affected mother.

ICD-9-CM Code: 357.0

Etiology
- Evidence points to infection induced aberrant immune response
- Typically antecedent illness within 4 wk of onset (resp or GI infection in 60–70% of cases)
- Other predisposing factors incl surgery, pregnancy, malignancy, acute seroconversion to HIV
- Epidural or spinal anesthesia may be antecedent event or associated with recurrence

Anticipated Problems/Concerns
- Autonomic instability
- Resp failure
- Parturient: Third trimester and postpartum, risk of exacerbation; for labor a regional anesthetic indicated to avoid exaggerated hemodynamic response to pain from autonomic dysfunction. Aspiration pneumonitis and resp failure may result in premature labor and maternal mortality. For C-section a regional anesthetic relatively contraindicated even for pt with mild resp involvement. Case reported of newborn with GBS features following delivery by affected mother.
- Fecal impaction
- Stress ulcers

• Pts have increased sensitivity to nondepolarizing muscle relaxants
  - May have residual muscle weakness after apparent full recovery from GA
  - Volume
  - Maintain blood volume
  - CVP < 5 cm H2O

DISEASES

Hashimoto’s Thyroiditis

Effect
- Dehydration,
- Tachy-
- Other
- autoin-
- Immune
dysfunc-
tion
- Anemia
- Tilt	table
test
- ECG
- PE
- Hgb,
- Hct
- Rough,
pale
skin
Assessment by Hx
- Arthralgias	and	myalgias
- Weakness
- Decreased	resp	muscle	strength

Diseases
- Table	as
treatment	for	hyroid	storm)
- Monitoring
- •	Assess	for
to-morbidities	autoimmune/
adrenal/
- •	Assess	fluid	status.
- •	Ensure	that
to-euthyroid
to-avoid
to-depressants)

Overview
- Hashimoto’s	thyroiditis	or	chronic	auto-
- Immune	thyroiditis	is	an	autoimmune
disease
- involving	progressive	thyroid
dysfunction
due
to
- autoimmune-mediated
destruction
do.
- thyroid
gland
- through
- apoptosis
- thyroid
epithelial
cells.
- Typical
- manifestations
- of
- the
disease
can
- h-pass
- high
- serum
- antibodies
- against
- one
- or
- more
- thyroid
- antigens,
diffuse
- lymphocytic
- infiltration
- of
- the
- thyroid,
destruction
- of
- thyroid
gland
- resulting
- in
- thyroid
- failure.
- Chronic
- inflammation
- of
- thyroid
- (painful
- or
- painless)
- with
- lymphocytic
- infiltration
- due
to
- autoimmune
- factors
- Acute
- inflammation
- results
- in
- increased
- release
- of
- preformed
- hormone
- with
- hyperthyroidism
- Chronic
- inflammation
- results
- in
- decreased
- thyroid
- gland
- function
- by
- resistant
- hyperthyroidism.

ICD-9-CM Code: 423.9

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Swollen, tender neck</td>
<td>Neck pain, hoarseness</td>
<td>Examine airway and neck</td>
<td>Lateral neck x-rays or CT of neck</td>
</tr>
<tr>
<td></td>
<td>Enlarged tongue</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tracheal compression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Dehydration, tachy- or bradydysrhythmias</td>
<td>Orthostatic symptoms</td>
<td></td>
<td>Tilt table test ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Decreased resp muscle strength</td>
<td>SOB</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dyssnea on exertion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Ileus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Constipation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Acutely hyperthyroid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chronically hypothyroid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Cold intolerance</td>
<td></td>
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<tr>
<td></td>
<td>Slow or fast movement, depending on stage</td>
<td></td>
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<tr>
<td>SKIN</td>
<td>Rough, pale skin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Coarse, dry hair</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Careful inspection of hair and skin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Arthralgias and myalgias</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GEN</td>
<td>Other autoimmune dysfunction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Weakness</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Inability to arise from chair without using hands</td>
<td></td>
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</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Assess NPO status (may have poor gastric emptying)
- Cautious use of preop drugs (increased sensitivity of central nervous and resp system to depressants)
- Ensure that pt is euthyroid to avoid thyroid storm.
- Assess fluid status.
- Assess for co-morbidities (autoimmune/adrenal/pancreatic dysfunction).

Monitoring
- Temp (consider placing cooling blanket on OR table as treatment for thyroid storm)
- Consider invasive monitoring if CV or resp compromise

Airway
- If normal preop, consider routine management
- If displaced or distorted, consider awake fiberoptic and armored tube intubation

Induction/Maintenance
- No data indicate one technique preferred over another

Extravasation
- Consider extubation in optimal situation for reintubation

Postoperative Concerns
- Routine monitor and treatment of co-morbidities if co-existing autoimmune disease

Adjuncts
- Esmolol for acute hyperthyroidism
- Steroids sometimes necessary for adrenal dysfunction
- Oral hypoglycemics (chronic use) can cause hypoglycemia for longer duration and of greater severity in periop pt.

Postoperative Monitoring
- NSAI

Risk
- Hashimoto's thyroiditis is the most common cause of hypothyroidism in iodine-sufficient countries and primary hypothyroidism in adults
- Incidence in USA: Approx 100,000–400,000 new diagnosis each year
- Causes thyroid failure in 10% of pts
- Prevalence increases with age, but also the most common cause of hypothyroidism in children as early as 1 to 2 years of age
- No documented ethnic predominance
- Gender predominance: F:M ratio: 7:1; age 30–50 years

Perioperative Risks
- Increased risk of thyroid storm even if euthyroid preop as the inflammatory process in the disease progression may involve enough apoptosis of thyroid follicles and cause thyroid hormone release. Clinical diagnosis of life-threatening illness if hyperthyroidism severely exacerbated by the stress of operation, typically manifested by hyperpyrexia, tachycardia, and alterations in consciousness
- Risk of resp failure or insufficiency and increased bleeding periop

Etiology
- Autoantibodies against thyroid peroxidase, thyroglobulin, or TSH receptors causing immune-mediated destruction of thyroid epithelial cells, though a small percentage of pts do not have the presence of such antibodies.
- Assoc with other autoimmune diseases: Sjögren's syndrome, SLE, RA, pernicious anemia, autoimmune endocrinopathies, Addison's disease, hypoparathyroidism, diabetes mellitus, gonadal failure
- Increased incidence in pts with a family Hx and with chromosomal disorders such as Turner's, Down, or Klinefelter's syndromes
- Also has been linked to several polymorphisms in genes for HLA and T-cell antigen receptors
- Precipitating causes: Thyroid injury (infection, radiation, and drugs), stress, steroids, pregnancy, and excessive iodine intake

Standard Treatment
- Thyroid hormone replacement chronically in hypothyroidism
- NSAIDs in acute thyrotoxicosis for pain and propranolol to control symptoms of hyperthyroidism

Overview
- Hashimoto’s thyroiditis or chronic autoimmune thyroiditis is an autoimmune disease involving progressive thyroid dysfunction due to autoimmune-mediated destruction of the thyroid gland through apoptosis of thyroid epithelial cells. Typical manifestations of the disease may encompass high serum concentrations of antibodies against one or more thyroid antigens, diffuse lymphocytic infiltration of the thyroid, and destruction of thyroid gland resulting in thyroid failure.
- Chronic inflammation of thyroid (painful or painless) with lymphocytic infiltration due to autoimmune factors
- Acute inflammation results in increased release of preformed hormone with hyperthyroidism
- Chronic inflammation results in decreased thyroid gland function with resistant hypothyroidism.

ICD-9-CM Code: 423.9


Preoperative Preparation
- Assess NPO status (may have poor gastric emptying)
- Cautious use of preop drugs (increased sensitivity of central nervous and resp system to depressants)
- Ensure that pt is euthyroid to avoid thyroid storm.
- Assess fluid status.
- Assess for co-morbidities (autoimmune/adrenal/pancreatic dysfunction).

Monitoring
- Temp (consider placing cooling blanket on OR table as treatment for thyroid storm)
Headache, Migraine

**Risk**
- Incidence in USA: >28 million; maximum prevalence 25–55 y of age
- Can start as early as 1 y of age, 10–20% of children by 20 y of age, male = female
- In adults: More frequent in women after age 11 y; approx 3:1; female: male; prevalence declines after age 40 y
- Familial aggregation; CACNA1A (P/Q voltage-gated calcium channel; ATP1A2 (Na+-K+ ATPase), and SCN1A (Na, 1.1 voltage-gated sodium channel) genes implicated in genetic predisposition for variations of familial hemiplegic migraine
- Can be assoc with sinusitis; AVM; stroke; patient foramen ovale; epilepsy; ischemic myocardial infarction; depression; anxiety disorder; sensitivity to foods rich in tyramine, phenylethylamine, or octopamine (chocolate, wine, dairy products); electroencephalographic ab
- Socioeconomic status: Inversely related to household income and education

**Perioperative Implications**

**Perioperative Risks**
- ↑ Incidence of hypertension, stroke, CAD
- Gastric stasis
- Drug toxicity and side effects

**Worry About**
- Toxie and side effects of antimigrainous preparations, adverse interaction with anesthetic drugs
- Assoc intracranial disorders

**Overview**
- Freq occurrence, frequently unilateral, throbbing head pain with strong family Hx
- Often associated with increased sensitivity to touch, N/V, phonophobia and/or photophobia
- May be preceded by a visual, sensory or motor aura; headache and aura may present independently
- Dx is Hx dependent in the absence of 2+ causes
- Migrainous infarction with permanent neurologic damage is rare.

**ICD-9-CM Codes: 346.0 (Classic migraine); 346.1 (Common migraine). 5th digit subclassification: 0 without mention of intractable migraine, 1 with intractable migraine, so stated.**

**Etiology**
- Central or peripheral mechanisms incited by internal or external stimuli
- Lowering Mg2+ levels ↑ the affinity and release of serotonin at cerebrovascular and neuronal sites as well as NO production and activation of NMDA receptors
- Precipitated by trigger factors
- Cerebral and extracerebral arteries are most likely sources of pain.
- Pain results from exaggerated pulsations in association with trigeminal release of substance P (sP), calcitonin gene-related peptide (CGRP), and vasointestinal peptide (VIP) and sensitization of nociceptors around blood vessels.

**Usual Treatment**
- No permanent cure
- Elimination of trigger factors when possible; chronobiologic regulation
- Abortive therapy: NSAIDs, barbiturates, ergotamines, triptans, phenothiazines, dihydroergotamine, sphenopalatine ganglion block, nonopioid and opioid analgesics
- Prophylactic therapy: Effective ➤ β-blocking agents (metoprolol, propranolol, timolol), TCA (amitriptyline), anti-epileptic drugs (AED; topiramate, divalproex sodium), serotonin agonists (frovatriptan; short-term prevention in menstrual migraine), pethes (butterbur); probably effective—ACE inhibitors (lisinopril, candesartan), AED (gabapentin), β-blocking agents (atenolol, nadolol), antidepressants (fluoxetine, venlafaxine), serotonin agonists (naratriptan, zolmitriptan; short-term prevention in menstrual migraine), histamine, cyproheptadine, MIG-99 (feverfew), vitamins (riboflavin, CO-Q10, Mg2+); possibly effective—α-agonists (clonidine, guanafacine), Ca2+-channel blockers (verapamil, nicardipine, nifedipine, nimodipine), AED (carbamazepine); conflicting evidence for efficacy of MAOIs; botulinum toxin probably not effective
- Behavioral treatment with biofeedback, self-hypnosis, relief by dark surroundings, sleep

**ASSESSMENT POINTS (Mainly side effects and toxicity of antimigrainous therapy.)**

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<th>Test</th>
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<tbody>
<tr>
<td><strong>CARDIO</strong></td>
<td>Ergotamine, sumatriptan</td>
<td>Symptoms of angina and peripheral vascular insufficiency</td>
<td>ECG</td>
<td>Stress ECG</td>
</tr>
<tr>
<td></td>
<td>• Worsening of Htn, ischemic heart disease, PVD, serotonin syndrome β-adrenergic receptor blocking agents and Ca2+-channel blockers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Excessive depression of myocardial function</td>
<td>Methysergide (no longer available)</td>
<td>S, Rales</td>
<td>CXR, ECHO</td>
</tr>
<tr>
<td></td>
<td>• Pericardial fibrosis, cardiac valvular fibrosis</td>
<td>TCA's and Ca2+-channel blockers</td>
<td>↓ Heart sounds</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>• Cardiac conduction abnormalities</td>
<td>Syncope</td>
<td>Q-T prolongation</td>
<td>ECG</td>
</tr>
<tr>
<td><strong>RESP</strong></td>
<td>β-blockers</td>
<td>Dyspnea</td>
<td>Expir wheezing</td>
<td>CXR, ABGs</td>
</tr>
<tr>
<td></td>
<td>• Worsening of COPD</td>
<td>Dyspnea</td>
<td>Rapid shallow breathing</td>
<td>PFTs</td>
</tr>
<tr>
<td></td>
<td>Methysergide (no longer available)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>• Pleurapulmonary fibrosis</td>
<td></td>
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</tr>
<tr>
<td><strong>GI</strong></td>
<td>Gastroparesis</td>
<td>Early satiety</td>
<td>Focal deficit</td>
<td>Neuroimaging</td>
</tr>
<tr>
<td><strong>CNS</strong></td>
<td>Intracranial disorders</td>
<td>Tachycardia, dry mouth</td>
<td></td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>TCA's</td>
<td>Blurred vision, urinary</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anticonvulsants</td>
<td>Somnolence, diplopia, ataxia, cognitive impairment</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>MAOIs</td>
<td>Retention, delayed gastric emptying</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Key Reference:** Silberstein SD, Lipton RB, Dodick DW, eds. Wolff’s headache and other head pain. 8th ed. New York: Oxford University Press; 2008.

**Exubation**
- Increased risk of CNS stimulation with sumatriptan, ergotamine, TCAs, and MAOIs

**Postoperative Period**
- Pain management may be critical
- Avoid withdrawal syndromes

**Anticipated Problems/Concerns**
- Possible adverse interactions of anesthetic drugs and antimigrainous preparations
- No unique hazards of anesthesia administered to pts with migraine
HELLP Syndrome

David J. Birnbach

Risk
- If severe preeclampsia, 20% may exhibit HELLP syndrome
- Preeclampsia occurs in 2–10% of pregnancies

Perioperative Risks
- High maternal and fetal morbidity and mortality
- Increased C-section rate (up to 94%)
- Urgent delivery after diagnosis to prevent maternal and fetal death

Worry About
- Confused with hepatitis, thrombotic thrombocytopenic purpura, gallbladder disease, and acute fatty liver of pregnancy. Malaria may also be mistaken for HELLP syndrome.
- Thrombocytopenia and coagulopathy increase risk of hematoma after regional anesthetic. Report of spinal hematoma associated with a HELLP pt who was not coagulopathic but had a platelet count of 91 X 10^9/L.
- High risk of hemorrhagic complications
- Upper airway and laryngeal edema leading to airway obstruction and difficult or failed intubation. Fluid management difficult; pulm edema may ensue.

Overview
- HELLP is an acronym for the findings that suggest hepatic involvement in preeclampsia pt: Hemolysis, Elevated Liver enzymes, Low Platelets.
- Diagnostic criteria incl hemolysis, defined by abnormal peripheral smear and increased bilirubin levels, elevated liver enzymes (SGOT >70 U/L, LDH >600 U/L), and thrombocytopenia (<100,000/mm^3).
- Failure to treat may lead to eclampsia or death due to hepatic hematoma or rupture.
- Not always associated with Htn

ICD-9-CM Code: 642.5 (Severe preeclampsia)

Etiology
- Poorly understood
- May be severe form of preeclampsia resulting from abnormal prostaglandin control, intravascular plt activation, and microvascular endothelial damage. Macroangiopathic hemolytic anemia usual.

Usual Treatment
- Definitive treatment is delivery as quickly as possible.
- After delivery, many experience uneventful recovery, with plt counts returning to normal within 1 wk.
- Glucocorticoids may accelerate fetal lung maturity and may also improve mother’s plt count and reduce liver enzyme abnormalities
- Plts, FFP, and cryoprecipitate administered as needed
- Magnesium sulfate for CNS irritability and antihypertensives for Htn

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Upper airway edema</td>
<td>Dyspnea, voice change</td>
<td>Poor visualization on airway exam</td>
<td>Mallampati assessment</td>
</tr>
<tr>
<td>CV</td>
<td>LV failure</td>
<td>Dypsnea, desaturation</td>
<td>Adventitious sounds</td>
<td>CVP and/or LVEDP</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp depression</td>
<td>Magnesium administration</td>
<td>↓ Reflexes</td>
<td>MgSO4 level</td>
</tr>
<tr>
<td>GI</td>
<td>Liver swelling Subcapsular hematoma</td>
<td>Epigastric pain N/V</td>
<td>Elevation of SGOT, SGPT</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenia</td>
<td>Bruising</td>
<td>Bleeding (IV site oozing)</td>
<td>Plt count</td>
</tr>
<tr>
<td></td>
<td>Hemolytic anemia</td>
<td>Pallor, jaundice</td>
<td></td>
<td>LDH, bilirubin</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Peripheral smear</td>
</tr>
<tr>
<td>RENAL</td>
<td>Acute renal failure</td>
<td>Oliguria</td>
<td></td>
<td>Elevated uric acid, BUN, serum Cr</td>
</tr>
<tr>
<td>CNS</td>
<td>Eclampsia, cerebral edema</td>
<td>Seizures</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Testing
- Obtain CBC, PT, PTT, fibrinogen, SGPT, SGOT, LDH, BUN, Cr

Monitoring
- Consider arterial line
- Consider CVP or PA catheter if oliguria despite fluid administration or CHF

Airway
- Assess airway early and repeat airway exam periodically.
- Laryngeal edema may preclude normal tracheal intubation in the event of emergency C-section.
- Difficult intubation equipment should be readily available.
- Consider pre-emptive epidural or continuous spinal catheter.

Induction
- Controlled neuraxial anesthesia with incremental dosing of catheter, if not contraindicated due to coagulation abnormalities. Recent reports suggest that spinal techniques can be safely used in severe preeclampsia.
- If general anesthesia is required, the hypertensive surge associated with ET intubation can often be avoided by pretreatment with magnesium, antihypertensives, or opioids.

Adjuncts
- If significant Htn, antihypertensive therapy prior to laryngeal intubation
- If receiving magnesium sulfate and needs GA, small doses of neuromuscular blocking agents with close monitoring
**Hemophilia**

**Risk**
- Incidence: Hemophilia A, factor VIII (FVIII) deficiency: 1/10,000 male births. Hemophilia B, factor IX (FIX, Christmas disease) deficiency: 1/100,000 male births
- Prevalence: Hemophilia A is 1/10,000 male births; Hemophilia B, 1/30,000 male births
- Hemophilia A, FVIII deficiency, affects 80–85% of hemophiliacs; remainder has hemophilia B due to factor IX deficiency.
- Hemophilia A and B are X-linked recessive hereditary disorders.
- Females may be asymptomatic carriers of the hemophilia gene and may have partial deficiency of FVIII or FIX.
- Hemophilia is without ethnic or geographic predilection.

**Perioperative Risks**
- Prolonged and potentially fatal hemorrhage both during and after surgery
- Closed-space bleeding can lead to nerve injury, vascular or airway obstruction.
- Surgery should not proceed without adequate supply of factor concentrate to support the procedure and postop course.

**Worry About**
- Spontaneous bleeding
- Intra- and postop hemorrhage despite optimal replacement therapy of deficient coagulation factor
- Development of FVIII and FIX inhibitor antibodies (approx 20% for FVIII, 3% for FIX)
- Viral transmission from plasma derived factor replacement therapy

**Overview**
- Hemophiliacs can have severe deficiency (<1% nml levels), moderate deficiency (1–5% of nml levels), or mild deficiency (5–30% of nml levels)
- Congenital disorder, inherited as an X-linked recessive trait, affecting males almost exclusively
- Acute and chronic complications often due to recurrent spontaneous bleeding, the hallmark of which is bleeding into the joints (e.g., cycle of joint hemorrhage, inflammation, synovial proliferation, and erosion of cartilage, causing pain and disability)
- Pts with hemophilia generally have nml prothrombin time (PT), normal bleeding times, and a prolonged partial thromboplastin time, (aPTT). Specific laboratory factor assays make the distinction and plasma concentrations of FVIII or FIX determine the severity.
- Treatment generally follows bleeding episodes. New approaches to treatment involve the prophylactic use of clotting factors.

**ICD-9-CM Code:** 286.0.

**Etiology**
- Hereditary disorder, X-linked recessive
- Acquired hemophilia is the development of FVIII inhibitors (autoantibodies) in persons without a Hx of FVIII deficiency

**Usual Treatment**
- Desmopressin (DDAVP injection of Stimate nasal spray) whenever possible for mild hemophilia A
- Recombinant FVIII and FIX products
- Plasma concentrations of deficient factors maintained at minimum of 40–70% throughout the periop period (7–10 d postop) for adequate hemostasis
- Cryoprecipitate is no longer recommended as a treatment alternative except in life-threatening emergencies
- Recombinant factor VIIa (NovoSeven) for use in pts with inhibitors to FVIII of FIX
- Gene insertion therapy shows promising future

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Pharyngeal bleeding</td>
<td>Often seen in children</td>
<td>Tongue and mouth lacerations</td>
<td>Exam</td>
</tr>
<tr>
<td>GI</td>
<td>GI bleeding not common</td>
<td>When it occurs, bleeding can be excessive</td>
<td>Stool exam, endoscopy</td>
<td>Hemoccult, angio</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, hematoma formation, bruising</td>
<td>Lethargy, SOB, skin discoloration</td>
<td>Hematomas</td>
<td>PT/PTT, plt count, bleeding FVIII and FIX assay, gene analysis</td>
</tr>
<tr>
<td>GU</td>
<td>Hematuria</td>
<td>Blood in urine</td>
<td>Any sign or symptom of head injury or trauma</td>
<td>Urinalysis, cysto, IVP</td>
</tr>
<tr>
<td>CNS</td>
<td>Intracranial hemorrhage</td>
<td>Head trauma, headache, change in mental status</td>
<td>Any sign or symptom of head injury or trauma</td>
<td>Head CT</td>
</tr>
<tr>
<td>MS</td>
<td>Joint hemorrhage</td>
<td>Painful distention of the joint</td>
<td>Hemarthroses, Limited ROM Tenderness</td>
<td>Physical exam X-ray</td>
</tr>
</tbody>
</table>

Hepatic Encephalopathy (HE)

**Risk**
- Incidence in pts with hepatic cirrhosis (about 0.1% of the population) is 50–70% of pts. Frequently subclinical, but can be exacerbated in the postop period by the surgical stress response, dehydration and postop infection.
- HE is acutely worsened, in about 20% of pts, following surgical portocaval shunts, minimally invasive transjugular intrahepatic portosystemic shunt (TIPS) and hepatic resections.

**Perioperative Risks**
- Precipitation of encephalopathy from benzodiazepines, surgical procedure (portocaval shunt), postop infection, GI hemorrhage, or erosive gastritis.
- In pts with severe underlying liver disease, Childs Class B and C, or high MELD score.

**Worry About**
- Preop resp depression from benzodiazepine premedication
- Hemorrhage from underlying hepatic dysfunction
- Underlying precipitating factor (infection, bleed) may create hemodynamic instability. HE in absence of precipitating factor, or when accompanied by seizure or focal nerologic deficit, should prompt brain imaging to rule out intracerebral bleed.
- Undiagnosed cerebral edema with risk of cerebral ischemia in fulminant hepatic failure presenting for liver transplantation

**Overview**
- A syndrome of alteration in mental status, from impaired concentration to coma, caused by portosystemic shunting, usually in the presence of liver failure. Hyperammonemia from protein breakdown is almost always present and the degree of hyperammonemia generally correlates with the degree of encephalopathy.
- Multifactorial in origin but altered neurotransmission and elevated levels of endogenous benzodiazepines and opioids appear important contributors. Although not effective in improving outcome, administration of flumazenil and naloxone temporarily improves mental status in about 50% subjects with HE.
- Underlying hepatocellular injury may arise from multiple etiologies but the most common are chronic alcohol abuse, chronic viral hepatitis, non-alcoholic steatohepatitis (NASH).

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Impaired concentration, drowsiness, coma</td>
<td>Amnesia</td>
<td>Transition of reflexes from hyperactive to hypoactive, and disappearance of asterixis, signify onset of severe HE</td>
<td>Plasma ammonia, CT scan</td>
</tr>
<tr>
<td>CV</td>
<td>Hypotension</td>
<td>Liver failure</td>
<td>Systolic BP 90 may be acceptable in liver failure</td>
<td>BP</td>
</tr>
<tr>
<td>RESP</td>
<td>Hyperventilation, hypoxemia</td>
<td>Dyspnea</td>
<td>Ascltes, pleural effusions</td>
<td>CXR, ABG</td>
</tr>
<tr>
<td>METAB</td>
<td>Hyponatremia, hypokalemia</td>
<td>Correction of hyponatremia or worsening of hypokalemia can further impair mental status</td>
<td>Free water excess exacerbates ascites and anasarca</td>
<td>BMP</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, coagulopathy</td>
<td>GI bleeding</td>
<td>Pallor, splenomegaly</td>
<td>Hemoglobin, plt count, prothrombin time</td>
</tr>
</tbody>
</table>


**Perioperative Implications for Liver Transplantation**
- Recurrent or persistent HE predicts poor survival in cirrhosis and indicates decompensated liver disease which is best treated by liver transplantation.
- When severe, particularly in association with fulminant hepatic failure, HE is frequently associated with cerebral edema. The resulting intracranial Htn may be underestimated by CT scan and intracranial pressure monitoring is indicated to ensure adequate cerebral perfusion pressure periop.

**Perioperative Implications for Other Surgeries**
- Mental capacity may be impaired to degree that consent is problematic.
- Pt may be hypovolemic from impaired ability to maintain PO intake, lactulose therapy causing diarrhea, diuretic therapy for associated ascites, or recent GI bleed. Maintenance of hydration is important to prevent acute tubular necrosis the incidence of which is increased in liver failure.

- HE usually reflects advanced hepatic dysfunction and is frequently seen in pts awaiting liver transplantation.

**ICD9-CM: 572.2 (Hepatic coma)**

**Etiology**
- Underlying liver disease with identifiable hyperammonemic precipitating cause in over 90% of cases: GI hemorrhage, infection, azotemia, diuresis, constipation, sedatives esp benzodiazepines
- Elevated levels of endogenous benzodiazepines, γ-aminobutyric acid agonists and opioids
- Direct ammonia neurotoxicity

**Usual treatment**
- Identify and treat precipitating cause.
- Reduce plasma ammonia with lactulose: 20g q6-12 hr orally or by NG tube until softening of stool; reduce dose if diarrhea. Alternately, 300 mL lactulose mixed with 700 mL tap water given as retention enema in pts with severe HE that cannot protect their airway.
- Consider combining lactulose with oral antibiotic such as metronidazole or neomycin.
Hepatitis, Alcoholic

Alan Kaye
Amir Baluch

Risk
- Incidence in the USA: 8.5% of adults met DSM-IV criteria for current alcohol use disorder. 30.5% of adults met DSM-IV criteria for lifetime alcohol use disorder. Roughly 10–15% of alcoholics will develop alcoholic hepatitis and cirrhosis.

Perioperative Risks
- Mortality rate of 60–100% of pts undergoing surgery during active alcoholic hepatitis
- Poorer prognosis when accompanied by increased bilirubin, increased Cr, PT >1.5x control, asites, or encephalopathy
- >10% develop delirium tremens (DTs) without prophylaxis

Worry About
- Anemia and coagulopathy
- Pulm shunting leading to arterial hypoxemia
- Altered mental status and/or hepatic encephalopathy

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>High CO</td>
<td>Exercise tolerance</td>
<td>Hyperdynamic cardiac exam</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td></td>
<td>Low SVR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Low CO (in advanced disease)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm shunts</td>
<td>Orthodeoxia</td>
<td>Effusions on CXR, ascites on abdominal exams</td>
<td>Resp alkalosis on ABG</td>
</tr>
<tr>
<td></td>
<td>Restrictive disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pulm effusions</td>
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<tr>
<td></td>
<td>Central hyperventilation</td>
<td></td>
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</tr>
<tr>
<td>GI/HEPATIC</td>
<td>Disrupted synthetic and metabolic function</td>
<td>Anorexia, N/V, malaise, wt loss, fever</td>
<td>Jaundice, ascites, tender hepatomegaly, splenomegaly</td>
<td>Elevated transaminases (AST/ALT &gt;2), PT, alk phos, bilirubin Decreased albumin</td>
</tr>
<tr>
<td>RENAL</td>
<td>Mg&lt;sup&gt;2+&lt;/sup&gt; and PO&lt;sub&gt;4&lt;/sub&gt;&lt;sup&gt;2-&lt;/sup&gt; wasting</td>
<td>Ascites</td>
<td>Serum Mg&lt;sup&gt;2+&lt;/sup&gt; and PO&lt;sub&gt;4&lt;/sub&gt;&lt;sup&gt;2-&lt;/sup&gt; Hyponatremia</td>
<td>Glucose</td>
</tr>
<tr>
<td>ENDO</td>
<td>Insulin resistance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia and thrombocytopenia</td>
<td>Bruising/bleeding</td>
<td>Splenomegaly</td>
<td>Hgb/Hct, platelets</td>
</tr>
<tr>
<td>CNS</td>
<td>Decreased clearance of amines</td>
<td>Altered mental status</td>
<td>Neurologic exam</td>
<td>NH&lt;sub&gt;4&lt;/sub&gt; levels</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Pt should be assessed via Child-Pugh or MELD score. Elective procedures should be postponed for Child-Pugh score >7 or MELD >8
- Increased sensitivity to sedative medications (increased cerebral uptake of benzodiazepines)
- Asites may be treated by diuretics (spironolactone) or percutaneous drainage
- Hypokalemia and hyponatremia should be corrected slowly (over 24–36 hr)
- Correct coagulopathy with vitamin K, FFP, and platelets (if needed)

Monitoring
- Consider CVP or PA catheter: Following the removal of large amounts of ascitic fluid, IV colloid fluid replacement is often necessary to prevent profound hypotension and renal shutdown.
- Monitor blood glucose closely due to deranged insulin production secondary to liver pathology
- Arterial catheter for hemodynamic lability, frequent blood gas sampling, large fluid shifts.

Airway
- Rapid sequence intubation: Some pts at risk for aspiration due to ascites (increased abdominal pressure).
- Hemodynamic instability secondary to DTs
- Insulin resistance

Overview
- Most common form of liver disease in USA
- Usually preceded by period of heavy alcohol consumption
- An intermediate stage between fatty liver and alcoholic cirrhosis
- Can vary from mild (with only elevated liver function tests) to severe liver inflammation (prolonged prothrombin time, and liver failure).
- Characteristic clinical features: Fever, hepatomegaly, jaundice, anorexia, abdominal bruit over liver (heard in >50% pts)
- Mortality is 50% within 30 d of onset with pts having hepatic encephalopathy, derangement in renal function, hyperbilirubinemia, and prolonged PT

ICD9-CM: 571.1 (Acute alcoholic hepatitis)

Etiology
- A daily intake of more than 40 g of alcohol in men and 20 g in women significantly increases the risk of alcoholic hepatitis.
- Inflammatory process via leukocytic infiltration which leads to hepatocellular necrosis with intracellular deposition of alcoholic hyaline in the cytoplasm of liver cells (Mallory Bodies).
- Repeated episodes precipitous to cirrhosis after healing and scar tissue formation

Treatment
- Abstinence with counseling
- Nutritional support: Diet, multivitamin and mineral supplementation
- Medications: Pentoxifyline, steroids (may reduce mortality in pts with severe alcoholic hepatitis or encephalopathy)
- Supportive care incl diet adjustment, multivitamin supplementation, lactulose, and neomycin if needed

Exubitation
- Exubate when pt fully awake to ensure highest degree of airway protection.

Adjuvants
- Multivitamins, minerals, and vitamin K 10 mg SQ or IM.

Postoperative Period
- Regional pain control ideal so as to avoid pharmacokinetic disturbances of systemic agents
- Maintain low threshold for transfer of pt to ICU environment.
- Vigilant observation for signs of acute hepatic decompensation (jaundice, encephalopathy, and ascites), delerium tremens, and sepsis (with secondary DIC).

Anticipated Problems/Concerns
- Increased risk of postop complications: Acute hepatic failure, sepsis, bleeding, renal dysfunction
- Need for prolonged airway protection because of altered mental status and pulm dysfunction
- Acute withdrawal from alcohol
- Multiple coagulation abnormalities due to synthetic dysfunction and hypersplenism
**Hepatitis, Halothane**

**Risk**
- Multiple exposures to halothane is the most important risk factor
- Prior Hx of jaundice or fever after anesthesia
- Female sex
- Obesity
- Age
  - Rare in pts <6 years old (3% of all cases)
  - Pts <30 years of age make up about 10% of all cases
  - Most cases occur in pts >40
  - In older pts the disease is more devastating
- Genetics: There is a strong family linkage associated with halothane hepatitis

**Perioperative Risks**
- Type or duration of surgery not a risk factor
- Hx of non-halothane related liver disease also not a risk factor

**Worry About**
- Induction of cytochrome P450 2E1 enzyme by alcohol, barbiturates, or isoniazid

**Overview**
- Estimated incidence
  - First exposure: 0.3 to 1.5 per 10,000
  - With multiple exposures: 10 to 15 per 10,000
  - F:M ratio: 2:1
  - Latency period before clinical symptoms
  - After first exposure –6 d, with overt jaundice in –11 d
  - After multiple exposures –3 d, with overt jaundice in –6 d

**Presenting symptoms**
- Fever 75%
- Leukocytosis, eosinophilia 20–60%
- Myalgias 20%
- Rash 10%
- Jaundice 25%
- Ascites, coagulopathy, GI hemorrhage 20% to 30%
- Liver enzyme markers
  - Alanine aminotransferase 25–250× upper limit of normal
  - Aspartate aminotransferase 25–250× upper limit of normal
  - Alkaline phosphatase 1–3× upper limit of normal
- Histological liver findings
  - Zone 3 necrosis (massive in 30% of cases, submassive in 70% of cases)
  - Inflammation, granulomas, eosinophilic infiltrates
  - Clinical course
  - Mortality rate in preliver transplant era as high as 80% if encephalopathy present
  - Recovery becomes evident as symptoms resolve over 5–14 d, with full recovery taking weeks to months.

**ICD-9-CM Code: 997.4 (Postoperative acute)**

**Etiology/Pathophysiology**
- Two distinct types of hepatitis are associated with halothane exposure
  - Type I
    - Subclinical disease with mild elevation of liver enzymes, no jaundice
    - Caused by the anaerobic, reductive metabolism of halothane
    - May occur in up to 30% of pts receiving halothane
  - Type II
    - Fulminant liver failure with massive zone 3 liver necrosis
    - Caused by oxidative metabolism of halothane
    - Trifluoroacetyl intermediates conjugate liver proteins
    - In susceptible individuals, antibodies to the metabolite-liver protein complex are formed causing an immune response
    - Incidence is 10 times higher in second exposure cases and severity of illness greater if second exposure follows soon after first exposure

**Drug Class/Metabolism**
- Halothane: A nonvolatile anesthetic—a halogenated hydrocarbon
- Enflurane: 2%
- Isoflurane: 0.2%
- Desflurane: 0.02%
- There are a few case reports of Type II hepatitis associated with isoflurane and desflurane in the world literature.

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</tr>
</thead>
<tbody>
<tr>
<td>N/V, malaise</td>
<td>Jaundice (about 6 days after second exposure, longer if 1st exposure)</td>
<td>Eosinophilia, leukocytosis E elevated liver enzymes: 1) Aspartate aminotransferase 2) Alanine aminotransferase (25 to 250× upper limit of normal) 3) Alkaline phosphatase (1–3× upper limit of normal)</td>
<td>Liver biopsy: Zone 3 Centrilobular necrosis</td>
</tr>
</tbody>
</table>

**Preoperative Implications**
- Prior records should be reviewed and prior exposure documented
- Avoid volatile anesthetics in a pt with a confirmed Hx of postop liver dysfunction from halogenated agents
- Total IV anesthesia is one approach if general anesthesia is planned
- Regional anesthesia not contraindicated

**Anticipated Problems/Concerns**
- How to evaluate postop liver dysfunction
  - Incidence: 25–75% of surgical pts may have some form of hepatic dysfunction, from mild elevation in liver enzymes to global liver failure
  - Up to 50% of pts with cirrhosis may have postop jaundice
- Categories of postop liver dysfunction
  - Hepatocellular injury (elevated alanine aminotransferase, +/- hyperbilirubinemia)
  - Etiologies: Inhalational anesthetics and other drugs, hypotensive shock, transfusion reactions, unrecognized preop liver dysfunction
    - Cholestatic jaundice (elevated alkaline phosphatase, +/- elevated ALT; direct hyperbilirubinemia)
    - Etiologies: Benign postop cholestasis, prolonged cardiac bypass, sepsis, prolonged administration of total parenteral nutrition, cholecystitis, cholangitis, microlithiasis, drugs (esp antibiotics)
    - Indirect hyperbilirubinemia (other liver enzyme markers often normal)
    - Etiologies: Multiple transfusions, hemolysis, glucose-6-phosphate dehydrogenase deficiency, Gilbert’s syndrome
  - Steatohepatitis: Alcohol or nonalcoholic (NASH)
  - Autoimmune hepatitis
  - Wilson’s disease
  - Periop disorders
  - Drug reactions
  - Hypotensive shock, other causes of liver ischemia
  - Second rule: In pts with drug-induced liver injury, jaundice may herald impending global liver failure and should be considered life threatening.
  - Third rule: Treatment is supportive in nature and orthotopic liver transplant may be life saving
  - Fourth rule: In a pt with documented or suspected AIH, avoiding all volatile anesthetics is the safest course for future anesthetics, due to immune cross reactivity, the possibility of trace amounts of volatile anesthetics in the anesthesia circuit, as well as the many unanswered issues regarding this disorder.

**Differential Diagnosis for Inhalational Anesthetic Induced Hepatitis [AIH]**
- First rule: AIH is a diagnosis of exclusion
  - Preexisting liver disease
  - Viral hepatitis

**Mark G. Mandabach**

**A.J. Wright**

Hepatitis A

Risk
- Most common form of acute viral hepatitis in many parts of the world and about one third of the USA population has antibody to HAV.
- With more widespread use of the HAV vaccine, the rate has decreased from 150,000 in 1999 to 25,000 in 2007.
- Very common infection in economically developing countries of Africa, Asia, and Latin America; children are frequently sources for outbreaks in crowded households, day care centers, and institutions; increased risk of disease is associated with travel to developing countries, men who have sex with men, users of injecting and non-injecting drugs, persons with clotting-factor disorders.
- While health care workers do not appear to be at increased risk for occupationally acquired infection.
- Although pts with chronic liver disease are not at increased risk for HAV infection, they are at risk for fulminant hepatitis A.

Perioperative Risks
- Elective surgery should not be performed on pts with acute HAV infection.
- Worsening liver function

Worry About
- With fulminant hepatitis A and acute liver failure; there may be coagulopathy, encephalopathy, cerebral edema, and multiple organ failure; mortality rate of greater than 40%.
- Maintenance of liver blood flow and O2 delivery; metabolism of drugs with hepatic clearance; increased bioavailability of IV drugs if serum albumin concentration is decreased
- Pts with acute liver failure require intensive support and may require liver transplantation.

Overview
- HAV replicates in the liver and is shed in the stool; the concentration in the stool is highest during the 2-wk period before to 1 wk after the onset of clinical symptoms; the risk of transmission of infection via the fecal-oral route is greatest during this time.
- Symptoms do not occur until the viral load in the stool begins to decrease and most pts with hepatitis A do not require hospitalization for treatment.
- In children less than 6 y of age, most HAV infections are asymptomatic while among older children and adults, most infections are symptomatic with jaundice occurring in over 80%.
- The two most common physical findings are jaundice and hepatomegaly. In symptomatic pts, the most common laboratory findings are elevated levels of serum alanine aminotransferase and bilirubin.
- Chronic HAV infection does not occur; most acute infections resolve within 2 mo; 10–15% of symptomatic pts may have a relapse of illness for up to 6 mo.
- Fulminant hepatitis with acute liver failure occurs in about 0.5% of all pts with HAV infection; the rate is 1.8% among adults greater than 50 y of age; pts with chronic liver disease are at increased risk for fulminant hepatitis when infected with HAV.

Assessment by Hx

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hypovolemia</td>
<td>N/V, GI bleed</td>
<td>Tachycardia, hypotension</td>
<td>Orthostatic BP changes; measure CO, SVR</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxemia</td>
<td>Hx of bleeding</td>
<td>Tachypnea</td>
<td>O2 saturation, ABG</td>
</tr>
<tr>
<td>GI</td>
<td>Bleeding</td>
<td>Dark urine</td>
<td>Hemocult + material</td>
<td>Hct, endoscopy</td>
</tr>
<tr>
<td>N/V</td>
<td>Jaundice</td>
<td>N/V</td>
<td>Icteric sclera</td>
<td>Bilirubin</td>
</tr>
<tr>
<td>Hx</td>
<td>Hypoalbuminemia</td>
<td>Abdominal pain</td>
<td>Edema, ascites</td>
<td>Serum albumin</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypoglycemia</td>
<td>Altered consciousness</td>
<td>Blood glucose</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td>Tachycardia</td>
<td>Bruises</td>
<td>Hct</td>
</tr>
<tr>
<td></td>
<td>Thrombocytopenia</td>
<td>Easy bruising</td>
<td>Bleeding in wounds</td>
<td>Plt count</td>
</tr>
<tr>
<td></td>
<td>Immunosuppression</td>
<td>Infections</td>
<td></td>
<td>PT (low factor V, VII, IX, X, fibrinogen)</td>
</tr>
<tr>
<td></td>
<td>Coagulopathy</td>
<td>Abnormal bleeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Hepatorenal syndrome</td>
<td>Oliguria</td>
<td>Urinary Na low</td>
<td>Serum Na+</td>
</tr>
<tr>
<td></td>
<td>Hyponatremia</td>
<td>Altered consciousness, seizures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalopathy</td>
<td>Mental status exam</td>
<td>Level of consciousness</td>
<td>Serum ammonia level</td>
</tr>
<tr>
<td></td>
<td>Cerebral edema</td>
<td></td>
<td>Level of consciousness</td>
<td>Measure intracranial pressure</td>
</tr>
</tbody>
</table>


Perioperative Implications for Patients with Acute Hepatitis A or Fulminant Hepatitis A

Preoperative Preparation
- Elective surgery should be postponed in pts with acute hepatitis A
- Correction of clotting abnormalities with FFP, Ptt, cryoprecipitate as needed
- Administration of vitamin K to facilitate production of coagulation factors (prolonged PT), if time permits
- Premedication depressive or sedative drugs should be avoided.

Monitoring
- Arterial line for ABG, electrolytes, glucose, and BP
- Consider central venous or pulm artery catheter

Airway
- Consider rapid sequence induction if N/V, or upper GI bleeding exist

Preinduction/ Induction
- Consider ketamine or etomidate in hypovolemic pts
- Acute liver failure is not likely to reduce plasma cholinesterase levels so succinylcholine may be used if indicated
- Increased bioavailability of IV drugs if serum albumin concentration is decreased
- Limit sedative drugs
**Maintenance**
- Inhalation agent with high inspired O₂ concentration useful for maintaining hepatic blood flow and O₂ supply; should probably avoid halothane
- The effect of muscle relaxants with hepatic clearance may be prolonged
- Increased blood loss with coagulopathy

**Extubation**
- May need postop mechanical ventilation to ensure time for adequate metabolism of depressant drugs

**Adjuvants**
- Hypocalcemia may occur with citrate administration

**Anticipated Problems/Concerns**
- Worsening of hepatic or renal function
- Delayed awakening from prolonged drug metabolism or encephalopathy
- Need to protect airway with reduced consciousness
- Hypoglycemia
Hepatitis B

**Risk**
- Incidence in USA: 3–5% have had the disease
- 0.3–1.0% are carriers of hepatitis B virus (HBV)
- High-risk groups incl immigrants from endemic areas, IV drug users, homosexual men, household contacts of HBV carriers, pts on hemodialysis, clnts in mental institutions
- Before introduction of hepatitis B vaccine, about 20% of susceptible anesthesiologists had serologic evidence of prior hepatitis B infection

**Perioperative Risks**
- Depends on activity and stage of infection
- Worsening liver function, hepatic encephalo-
athy, coagulopathy

**Worry About**
- With acute hepatic failure or end-stage liver disease: Coagulation abn, decreased hepatic metabolism of drugs, decreased levels of plasma cholinesterase, hypoxemia from pulm shunting and edema, ascites and Na+ overload, hypokalemia, hepatic encephalopathy and cerebral edema, impaired glucose metabolism and hypoglycemia, portal Htn and GI bleeding, acute renal failure and hepatorenal syndrome, infection and sepsis, malnutrition
- Maintenance of liver and cerebral BF and O₂ delivery

**Overview**
- Hepatotropic viral infection: 90% have self-limiting acute hepatitis; 10% become chronic HBV carriers with about half of those progressing to chronic active hepatitis, cirrhosis, or hepatocellular carcinoma; 0.5% of pts with acute infection develop fulminating hepatic failure.
- 70% with acute infection have subclinical hepatitis; symptomatic infection may produce jaundice, malaise, nausea, abd pain.
- HBV carriers are diagnosed by persistent positive serology for hepatitis B surface antigen (HBsAg).
- Hepatitis B surface antibody (anti-HBs) confers immunity (after resolution of infection or with immunization)

**ICD-9-CM Code: 070.3 (Viral hepatitis B without mention of hepatic coma)**

**Assessment Points**

<table>
<thead>
<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hyperdynamic circulation</td>
<td>Tachycardia, Skin spiders</td>
<td>Tachypnea, SpO₂</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxemia</td>
<td>Hx of bleeding, Increasing abd girth</td>
<td>Acutes, pedal edema, Abd pain</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Bleeding, Ascites, Jaundice, Hypoalbuminemia, Hepatitis</td>
<td>Increased urinary Na+</td>
<td>Hct, endoscopy, Bilirubin, Serum albumin, SGPT, SGOT</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypoglycemia</td>
<td>Altered consciousness</td>
<td>Blood glucose</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, Thrombocytopenia, Immunosuppression, Coagulopathy</td>
<td>Easy bruiseability, Infections, Abn bleeding</td>
<td>Bruises, Pt count (low factors V, VII, X, K, fibrinogen)</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Hepatorenal syndrome, Hyponatremia, Hypokalemia</td>
<td>Altered consciousness, seizures, Taking diuretics</td>
<td>Oliguria, Urinary Na⁺, Serum Na⁺, Serum K⁺</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalopathy</td>
<td>Mental status exam</td>
<td>Level of consciousness, Asterixis</td>
<td></td>
</tr>
</tbody>
</table>

Hepatitis C

Risk
- HCV accounts for about 20% of cases of acute viral hepatitis in the USA.
- HCV is the most common cause of chronic liver disease in the USA and is the most frequent indication for liver transplantation.
- HCV is transmitted through percutaneous exposure to infected blood (IV drug use); occupational and sexual transmission of HCV can occur; pts on hemodialysis are at increased risk for infection.
- About 2–3% of cases occur in health care workers.

Perioperative Risks
- Worsening liver function, hepatic encephalopathy, coagulopathy
- Risk of transmission of HCV from carrier to anesthesia personnel is ~2% after percutaneous exposure.

Worry About
- With end-stage liver disease: Coagulation abn, decreased hepatic metabolism of drugs, decreased levels of plasma cholinesterase, hypoxemia from pulm shunting, ascites and Na+ overload, hepatic encephalopathy, glucose metabolism, portal Htn and GI bleeding, hepatorenal syndrome
- Maintenance of liver blood flow and O₂ delivery
- In addition to use of universal precautions by anesthesia personnel, use of sharp devices for invasive procedures should be minimized, and/or safety devices should replace standard sharp devices.

Overview
- Hepatotropic insidious viral infection; fulminant acute hepatitis C rare
- 60–70% of individuals with acute HCV infection are asymptomatic or have only a mild clinical illness.
- Over 80% of pts remain HCV-RNA positive with the majority having persistent elevation of liver enzymes.
- 70–85% of HCV infected pts develop chronic infection; cirrhosis develops in up to 50% of individuals with chronic hepatitis C and hepatocellular carcinoma in 1–5%.
- Pts over age 50 may have a more rapid progression of liver injury; alcohol use increases the risk of liver injury.
- Serologic testing for HCV infection incl testing for HCV RNA or immunoassay for anti-HCV; HCV RNA can be detected in serum within days after infection and HCV RNA quantification is useful in the management of treatment; ELISA tests for anti-HCV become positive about 8 wk after exposure, but anti-HCV is not associated with resolution of infection and does not confer immunity

ICD-9-CM: 070 (Viral hepatitis)

Etiology
- HCV (30–60-nm RNA virus with at least 6 genotypes) carried in and transmitted by exposure to blood and body fluids

Usual Treatment
- For pts with acute HCV infection in which HCV RNA has not cleared after 3 mo, treatment with standard interferon or pegylated interferon α-2b, with or without ribavirin, should be considered.
- Some pts with chronic HCV infection may benefit from treatment with standard interferon or pegylated interferon, with or without ribavirin, but current guidelines should be consulted for specific details.
- Protocols should be in place for health care workers to report on and have follow-up of percutaneous or permucosal exposures to blood or bloody body fluids; immune globulin and antiviral agents are not recommended for postexposure prophylaxis after occupational HCV exposure.
- Currently there is no vaccine for prevention of HCV infection.
- Orthotopic liver transplantation for end-stage liver disease

### ASSESSMENT POINTS

The following are for patients with end-stage liver disease from cirrhosis or chronic hepatitis.

<table>
<thead>
<tr>
<th>System</th>
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</tr>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Hyperdynamic circulation Pulm Htn</td>
<td>Tachycardia Telangiectases</td>
<td>Measure CO, SVR Pulm artery pressure</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxemia Hepato-pulm syndrome</td>
<td>Tachypnea</td>
<td>ABGs, SpO₂</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Bleeding Ascites Jaundice Hypoalbuminemia Hepatitis</td>
<td>Hx of bleeding Increasing abdominal girth Dark urine Icteric sclera Ascites, pedal edema Abdominal pain</td>
<td>Hemocult + material Fluid wave on abdominal exam Icteric sclera Ascites, pedal edema Abdominal pain</td>
<td>Hct, endoscopy Bilirubin Serum albumin ALT, AST</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypoglycemia</td>
<td>Altered consciousness</td>
<td>Blood sugar</td>
<td></td>
</tr>
<tr>
<td>HHEME</td>
<td>Anemia Thrombocytopenia Immunosuppression Coagulopathy</td>
<td>Easily bruised Infections Abn bleeding</td>
<td>Bruises</td>
<td>Hct Ptl count PT (low factors V, VII, IX, X, fibrinogen)</td>
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<td>Hepatorenal syndrome Hyponatremia Hypokalemia</td>
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<td>Oliguria</td>
<td>Urinary Na⁺ low Serum Na⁺ Serum K⁺</td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalopathy Cerebral edema</td>
<td>Mental status exam Level of consciousness Asterixis</td>
<td>Intracranial pressure; cerebral perfusion pressure</td>
<td>Serum ammonia</td>
</tr>
</tbody>
</table>


**Perioperative Implications for Patients with End-Stage Liver Disease from Hepatitis C**

**Preoperative Preparation**
- Correction of clotting abn with FFP, plt, cryoprecipitate, rFVIIa as needed
- Administration of vitamin K to facilitate production of coagulation factors (prolonged PT), if time permits
- Paracentesis if resp compromise from massive ascites

**Monitoring**
- Arterial line for ABGs and BP
- Consider central venous or pulm artery catheter (useful for diagnosis of pulm Htn)

**Airway**
- Consider rapid-sequence induction with ascites or upper GI bleeding

**Preinduction/Induction**
- Ketamine or etomidate in hypovolemic pts
- Duration of action of succinylcholine may be prolonged
- Increased bioavailability of IV drugs with low serum albumin
- Limit sedative drugs

**Maintenance**
- Inhalation agent with high FIO₂ useful for maintaining hepatic blood flow; should probably avoid halothane
- Choose muscle relaxants not dependent on liver metabolism
- Increased blood loss with coagulopathy
Extubation
• May need postop ventilation to ensure time for adequate metabolism of depressant drugs

Adjuvants
• Hypocalcemia may occur with citrate administration

Anticipated Problems/Concerns
• Worsening of hepatic or renal function
• Fluid overload
• Delayed awakening from prolonged drug metabolism or encephalopathy

• Need to protect airway with reduced consciousness, esp. with upper GI bleeding
• Hypoglycemia
Hereditary Hemorrhagic Telangiectasia (Osler-Weber-Rendu Disease)

Etiology
- Autosomal dominant trait with varying penetrance and expressivity

Usual Treatment
- Epistaxis is medically treated with Fe supplementation, estrogen therapy, and humidification. With intractable epistaxis ablative therapy with ND: YAG laser is effective, although multiple treatments required
- Multiple transfusions
- Pulm AVMs with feeding artery diameter greater than or equal to 3 mm require treatment with transcatheter embotherapy with coils.

Risk
- Affects varied in racial and ethnic groups, wide geographic distribution
- Men and women affected equally
- In Vermont, frequency 1:16,500
- Europe and Japan: 1:5,000–8,000

Perioperative Risk
- Excessive bleeding
- Paradoxical air, bland, or septic embolism to brain

Worry About
- Chronic anemia due to hemorrhage, esp. recurrent epistaxis
- Due to danger of intrapartum or postpartum pulm hemorrhage, a pregnant woman with HHT who has not had a recent pulm evaluation should be evaluated as soon as pregnancy is recognized.

Overview
- Mucocut and visceral vascular dysplasia
- Result from the combination of defective perivascular connective tissue, insufficient smooth muscle contractile element, endothelial cell junction defects, and increased endothelial tissue plasminogen activator impairing thrombus formation in case of vascular damage
- International consensus diagnostic criteria (Curacao criteria): HHT diagnosis classified as definite if 3 criteria present, possible or suspected if 2 criteria present and unlikely if, one criterion present. The criteria are:
  - Epistaxis: Spontaneous recurrent nosebleeds
  - Mucocut telangiectasia
  - Visceral involvement (i.e.; GI telangiectasia, pulm AVM, hepatic AVM, cerebral AVM, spinal AVM
  - Affected 1° relative
  - Manifestations of HHT are not present generally at birth, but develop with increasing age, with epistaxis usually being the earliest sign that may lead to chronic anemia. 90% of pts have signs and symptoms by age 40.

ICD9-CM: 448.0

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Telangiectasia of nasal mucosa, conjunctival telangiectasia, retinal vascular malformations</td>
<td>Recurrent frequent epistaxis</td>
<td>Rales, neurologic deficits</td>
<td>CXR</td>
</tr>
<tr>
<td>CV</td>
<td>High-output heart failure, thromboembolism</td>
<td>Fatigue, SOB</td>
<td>Cyanosis, clubbing, neurologic deficits</td>
<td>CXR, CT, detection of R to L shunt via radionucleide perfusion scans or contrast ECHO</td>
</tr>
<tr>
<td>PULM</td>
<td>AVMs with R to L shunt leading to hypoxemia, absence of filtering capillary bed allowing particulate matter to reach systemic circulation, fragile vessels may hemorrhage into bronchus or pleural cavity</td>
<td>Fatigue, dyspnea on exertion, hemoptysis, embolic cerebral events</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, coagulopathy, associated with von Willebrand disease</td>
<td>Recurrent epistaxis</td>
<td>Pallor</td>
<td>CBC, PT/INR, PTT</td>
</tr>
<tr>
<td>CNS</td>
<td>Cerebral AVM, aneurysms, cavernous angiomas paradoxical embolism, spinal AVM, migraines</td>
<td>CVA, brain abscess</td>
<td>Headache, seizure, hemorrhage, ischemia of the surrounding tissues due to a steal effect</td>
<td>MRI</td>
</tr>
<tr>
<td>HEPATIC</td>
<td>Hepatomegaly, high output heart failure, portal Htn, encephalopathy, biliary disease</td>
<td>Hemorrhage, sepsis</td>
<td>Jaundice</td>
<td>LFTs, PT/INR, PTT</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Preop cardiac and pulm evaluation to exclude high-output cardiac failure and pulm AV malformations
- CBC for anemia from bleeding or polycythemia from pulm shunt
- Check liver and renal function
- Perform neurologic assessment to exclude previous paradoxical emboli and severe brain AVM
- Debubble IV lines to prevent paradoxical air emboli
- Use meticulous aseptic technique
- For regional technique, assess any possibility of AVMs in the neuraxial region prior to performing the technique

Monitoring
- Avoid or use with great caution TEE, gastric succioning or esophageal stethoscope if esophageal varices or AVMs are present

Airway
- If oropharengeal AVMs are present, there is a high risk of airway bleeding
- Nasal intubation contraindicated if nasal telangiectasias are present

- Well-lubricated smaller size ETT to prevent any tissue trauma

Maintenance
- Risk of high-output heart failure, liver failure, modify anesthetic management
- Pulm AVMs could be large enough to lead to heart failure and polycythemia.
- Key aspects of anesthetic management are interventions to maintain nml hemodynamic parameters and to prevent bleeding and the formation of emboli.

Postoperative Period
- Avoid immobilization for prolonged periods of time to avoid thromboembolism to CNS

Adjuvants
- Watch for incompatible drugs in IVs or peripheral veins to avoid particulate matter precipitation and embolization to the brain
- Broad spectrum antibiotic prophylaxis to decrease risk of CNS infections
- NSAIDs may precipitate GI or mucosal bleeding and impair renal function

Anticipated Problems/Concerns
- Anemia due to recurrent bleeding
- Transfusion is complicated: Low Hct may increase the risk of high-output CHF by increasing extent of arteriovenous shunting (decreasing viscosity effect), but a high Hct may increase risk of thromboembolism
- Coagulopathy: Multiple hemostatic defects, incl low-grade DIC, reduced plt aggregation, and factor XI deficiency, may aggravate bleeding caused by local vessel wall pathology
- Paradoxical embolism: Owing to pulm AVMs, peripheral microemboli (air, bland, or septic) bypass nml pulm capillary filtering and embolize, causing transient or permanent neurologic defects or brain abscess
- Special attention should be paid to pregnant women with the diagnosis of HHT. In the rare instances, deterioration of pre-conception AVMs and the development of new AVMs will present with clinically silent but potentially life threatening complications of the disorder. These are most commonly located in the pulm vasculature, followed in frequency by the cerebral, GI, and spinal circulation. With CV and hormonally induced enlargement of certain AVMs there is concurrent risk of rupture, as well as shunt-induced high cardiac output failure and systemic embolism.
Herniated Nucleus Pulposus

Risk
- Incidence of symptomatic disc herniation is 1–2% in the general population
- Most common age is during third and fourth decades of life
- Smoking (leads to a decrease in O₂ tension 2° to vasoconstriction with decreased nutrient supply to the nucleus pulposus)
- Chronic increases in disc strain (i.e., chronic coughing, sitting without lumbar support, heavy lifting)
- Poor posture combined with poor body mechanics stresses the lumbar spine and affects the distribution of the body’s weight
- Obesity and sedentary behavior

Overview
- Located between vertebral bodies
- The intervertebral disc is the largest avascular structure in the body.
- The nucleus pulposus is composed of H₂O, collagen, and proteoglycans (PGs). PG molecules are important because they attract and retain H₂O, making a hydrated gel–like matter that resists compression. The amount of H₂O in the nucleus varies throughout the day depending on activity. It decreases with age, leading to degenerative disc disease.
- The annulus fibrosus is an annular structure composed of concentric sheets of collagen fibers connected to the vertebral endplates. The sheets are oriented at various angles and enclose the nucleus pulposus.
- Disc herniation occurs when the annulus fibrosus breaks open or cracks, allowing the nucleus pulposus to escape. This is called a herniated nucleus pulposus (HNP) or herniated disc. The herniated disc material initiates an inflammatory reaction.
- Disc herniation typically gives rise to radicular pain, which is pain in the distribution of the nerve root affected by the herniation with a strong inflammatory and neuropathic component, with or without neurologic change. If radicular changes take place, the presentation is that of a radiculopathy.
- Lumbar region: L4–5 most common site (59%), followed by L5–S1 (30%), L3–4 (9%)

Etiology
- The H₂O-retaining ability of the nucleus pulposus progressively declines with age.
- Nuclear material that is displaced into the spinal canal is associated with a significant inflammatory response (proinflammatory molecules interleukin-1 (IL-1), IL-8, and TNF α)
- Macrophages respond, which results in scar production, and an increase in substance P.
- Symptoms do not always correlate with herniation size (asymptomatic herniations frequent)

Disease Presentation
- Frequently present with a combination of back pain along with radicular symptoms; neurologic signs such as weakness or sensory deficits are possible (isolated low back pain may also be the sole presentation)
- Pts often describe popping sensation prior to radicular symptom.
- Neural impingement is responsible for dysfunction (compression of a motor nerve results in weakness, and compression of a sensory nerve results in numbness).
- Radicular pain is caused by inflammation of the nerve (which can explain the lack of correlation between herniation size and pain symptoms).
- Ideal imaging modality is MRI, although CT may also be helpful, EMG/NCS aid in identifying nerve root (there is not always correlation between findings on imaging studies and clinical presentation).
- Maneuvers which increase intrathecal pressure (coughing, sneezing, prolonged sitting) aggravate pain.

Treatment
- Conservative therapies
  - NSAIDs are literature supported
  - Systemic corticosteroids, opioids, muscle relaxants, neuropathic agents (empirical data, limited EBM data)
  - Contrary to prior beliefs, activity is now preferred over bed rest
- Surgery
  - Most common procedure for a herniated or ruptured intervertebral disc is a microdiscectomy
  - 200,000 lumbar discectomies performed annually
  - Cauda equina syndrome or a high degree of motor dysfunction are surgical emergencies.
  - Most recently a large study showed short-term benefit from surgery; however at long-term follow-up showed no benefit over conservative therapy.

ASSESSMENT POINTS

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</tr>
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<tbody>
<tr>
<td>MS</td>
<td>↓ ROM, pain</td>
<td>Lumbar sprain: Stiffness, ↓ ROM</td>
<td>Muscle tenderness</td>
<td>MRI/CT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Annular tear: Axial pain, difficulties sitting</td>
<td>↓ ROM referred</td>
<td>MRI/CT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HNP: numbness, weakness or simply pain</td>
<td>dermatomal pain</td>
<td>EMG/NCS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cauda Equina</td>
<td>↓ Reflexes, sensory loss</td>
<td>Surgical emergency</td>
</tr>
<tr>
<td>NEURO</td>
<td>↓ Reflexes or ↑ reflexes with severe spinal stenosis</td>
<td>“Saddle anesthesia”</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSYCHOSOCIAL</td>
<td>Anxiety, chronic opioid intake, litigation issues</td>
<td>Medications preop</td>
<td>If opioid abruptly stopped, may present with withdrawal</td>
<td>Need for multimodal analgesia</td>
</tr>
</tbody>
</table>


Perioperative Considerations
- Pts on high doses of opioids may present a challenge intraop and postop.
- Multimodal analgesia is the best option incl the continuation of the preop dose of opioids as a baseline, gabapentin, NSAIDs (as allowed per surgical team), acetaminophen, clonidine (as allowed by CV status), and ketamine IV for its NMDA antagonist properties.
Herpes, Type I

Risk
- 500,000 new cases each year in USA; 58% of people worldwide are seropositive
- Symptoms are typically minor or absent except in immunocompromised pts

Perioperative Risks
- Theoretical risk that spinal anesthesia can spread HSV-1 infection to new dermatomes

Worry About
- Transmission of infection to health care workers or other pts
- Reactivation after organ transplantation and initiation of immunosuppression
- 2nd infection of herpetic lesions with bacteria or fungi

Overview
- Transmission occurs after contact with lesions or mucus.
- Primary infection associated with fever/malaise; mean duration 19 d. Recurrences are milder, with a mean duration of 10 d.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Pneumonitis</td>
<td>Aspiration of oral secretions; previous HSV esophagitis</td>
<td>Bilateral crackles</td>
<td>CXR—bilateral interstitial infiltrates</td>
</tr>
<tr>
<td>GI</td>
<td>Esophagitis</td>
<td>Odynophagia, dysphagia, substernal pain</td>
<td>Multiple shallow mucosal ulcers</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Cystitis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalitis, meningitis</td>
<td>Headache, confusion, lethargy</td>
<td>Anosmia, memory loss, expressive aphasia, focal seizures</td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Cutaneous ulcers</td>
<td>Recurrent painful skin or mucosal ulcers</td>
<td>Multiple vesicular lesions on an erythematous base with subsequent ulceration</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stevens-Johnson syndrome</td>
<td>Extensive painful skin lesions</td>
<td>Deep bullous erosive lesions</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Cover exposed herpetic lesions
- Strict adherence to universal precautions

Monitoring
- Avoid disturbing active lesions

Regional Anesthesia
- Needle should not be inserted through lesion. Theoretical risk of spreading herpes from one infected ganglion to another, but regional anesthesia is not contraindicated

Postoperative Period
- Thorough disinfection of any surface area that might have been in contact with oral secretions or herpetic lesions. Most disinfectants are effective, incl chlorine and alcohol.

Anticipated Problems/Concerns
- No effective pre- or postexposure prophylaxis
- Acyclovir may reduce effectiveness of phenytoin.
- C-section should be offered for pregnant women with active HSV.
- Vaginal delivery is acceptable for women in remission; acyclovir is often used.

Epstein-Barr virus (HHV-4), cytomegalovirus (HHV-5), and human herpes virus 6 (HHV-6)
- No animal/insect reservoir or vectors exist for HSV-1.
- Infection is usually mild in pts with an intact immune system.
- Dx is by viral culture, PCR, fluorescent antibody testing, or serology.

Usual Treatment
- Acyclovir, valacyclovir, and famcyclovir are effective as episodic therapy when initiated within 72 hr of appearance of symptoms. Reduce viral shedding, lesion healing time, and symptoms.
-Suppressive therapy (lower dose initiated while asymptomatic) is effective in reducing frequency and severity of recurrences, as well as transmission to an uninfected partner.
- Foscarnet or vidarabine may be used in acyclovir-resistant herpes infections.
Herpes, Type II

Risk
- Incidence within USA: Estimated 40–60 million (20% of sexually active adults)
- Highest prevalence in women, African-Americans, and lower socioeconomic groups
- Frequency and severity of infection increased in immunocompromised pts, incl HSV encephalitis
- Incidence of neonatal HSV infection estimated at 1/2000–1/5000 deliveries

Perioperative Risks
- Vertical transmission from infected mother to fetus during vaginal birth
- Intrauterine fetal infection after rupture of membranes

Worry About
- Transmission of infection to health care personnel resulting in herpetic whitlow via inoculation of virus into fingers
- Neonatal herpes infection during vaginal births
- Viremia 2° to needle placement within infected area during regional anesthesia
- Extension of genital infection to adjacent areas during exam and instrumentation
- 2° bacterial or fungal infection of herpetic lesions

Overview
- Causative primarily of infections below waist transmitted by sexual contact
- Maternal primary HSV-2 infection associated with spontaneous abortion
- Newborns infected with HSV-2 during vaginal delivery from the mother's genital infection (high neonatal mortality)
- Primary genital HSV-2 infection with highest incidence of systemic symptoms (malaise, fever, headache, myalgias)
- Latent infection remains dormant in sensory ganglia innervating infected area until reactivation
- Recurrent infection involves vesicular, ulcerative lesions in genital tract, labia, vulva, perineum, cervix, urethra
- No increased risk of reactivation of HSV-2 associated with neuraxial anesthesia
- Chronic recurrent HSV-2 infection associated with development of cervical and vulvar cancer
- Dx by viral culture (gold standard) most sensitive and specific (rapid Dx by Tzanck smear)

ICD-9-CM Codes: 054.9 (Infection); 771.2 (Congenital)

ASSESSMENT POINTS (Primary and Recurrent)

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Pharyngitis (primary)</td>
<td>Cervical adenopathy</td>
<td>Mucosal ulceration</td>
<td></td>
</tr>
<tr>
<td>GU (mucous membranes)</td>
<td>Cystitis (primary)</td>
<td>Dysuria</td>
<td>Vaginal or urethral discharge</td>
<td>Viral culture (gold standard)</td>
</tr>
<tr>
<td></td>
<td>Genital ulcers (recurrent)</td>
<td></td>
<td>Ulcerated lesions of penis or labia or cervix</td>
<td>Tzanck smear; direct immunofluorescent assay</td>
</tr>
<tr>
<td>Lymphatics</td>
<td>Lymphadenopathy</td>
<td></td>
<td>Tender inguinal nodes</td>
<td>Biopsy; intranuclear inclusion bodies</td>
</tr>
<tr>
<td>SKIN</td>
<td>Herpetic whitlow (recurrant)</td>
<td>Painful vesicular or popular lesion</td>
<td>Pain</td>
<td>Tzanck smear</td>
</tr>
<tr>
<td>CNS</td>
<td>Aseptic meningitis (primary)</td>
<td>Headache</td>
<td>Cauda equina syndrome</td>
<td>Proctsigmoidoscopy</td>
</tr>
<tr>
<td>Rectal</td>
<td>Herpes proctitis (primary)</td>
<td>Constipation</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Perioperative Implications

Preoperative Preparation
- Universal precautions

Monitoring
- Routine

Regional Anesthesia
- Needle placement in infected area contraindicated 2° to risk of viremia and local extension into deep tissues
- Preferred in pregnant women with recurrent infection, no systemic symptoms, and no infection in area of block placement

Postoperative Period
- Universal precautions

Anticipated Problems/Concerns
- Difficulty identifying asymptomatic carriers of HSV-2 with viral shedding
- No effective prophylaxis for newborns


Etiology
- Double-stranded DNA virus in family of Herpesviridae
- Acquired genital infection primarily by sexual transmission of HSV-2
- Immunosuppression and increased number of sexual partners are risk factors for acquisition
- Diagnosed by multinucleated giant epithelial cells (polynuclears) with intranuclear (Cowdry type A) inclusion bodies on Giemsa stain smears (Tzanck preparation) taken from vesicle or tissue biopsy

Usual Treatment
- IV acyclovir for neonatal HSV-2 infection
- Oral acyclovir and topical cream shorten duration of lesions for recurrent infections
- Most recommend that full-term parturients with visible genital lesions (esp. primary infection) undergo abdominal delivery to decrease incidence of neonatal HSV infection. Neonates exposed to asymptomatic shedding of HSV during parturition (4-fold increase in HIV seropositive women) may also rarely acquire neonatal HSV.
Hirschsprung’s Disease

Overview
- 90% of pts have delayed (>48 hrs) passage of meconium
- 80% of pts present in the first few mo of life with constipation, poor feeding, and progressive abdominal distention
- Presentations vary from mild symptoms to severe disease with toxic megacolon, enterocolitis, peritonitis, or perforation.
- Dx may be made by radiographic imaging (transition zone seen with contrast enema), anorectal manometry, or histologic findings (definitive diagnosis) on rectal suction or full thickness biopsies.

ICD-9-CM Code: 751.3

Etiology
- Disease is caused by the failure of neural crest cells to migrate cephalocaudally causing an absence of ganglion cells in all or part of the colon.
- Several genes incl RET and EDNRB have been linked to the disorder.
- Varying lengths of the distal colon are unable to relax, causing functional colonic obstruction over time.
- Aganglionic segment extends proximally from the anus and is limited to the rectosigmoid region in 75% of cases; however 10% of pts may have total colonic aganglionsis.

Usual Treatment
- Nasogastric decompression, antibiotics, volume resuscitation and correction of electrolyte abn
- Rectal irrigation to decompress bowel and prevent enterocolitis
- Healthy newborns with non-distended colons and short-segment disease can undergo a primary ileoanal pull-through procedure.
- If child has enterocolitis or a significantly dilated colon, a colostomy can be placed; with pull-through procedure being performed 4–6 mo later.
- Swenson’s was the original pull-through procedure; newer techniques (Duhamel, Soave, and Boley) help preserve the nerve supply to the rectum and bladder.
- Recent advances incl laparoscopic-assisted endorectal pull-through and primary single-stage transanal pull-through without colostomy or intra-abdominal dissection. The colon and rectum are mobilized laparoscopically while the endorectal dissection is done through the anus. The laparoscopic approach minimizes postop analgesia requirements and hospital stay. Intraop pathologic evaluation of the bowel is essential for single-stage repair to ensure complete resection of aganglionic bowel.

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Congenital hypoventilation “Ondine’s curse”</td>
<td>Apnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIAC</td>
<td>Hypovolemia, septic shock, tetralogy of Fallot</td>
<td>IV replacement, Extent of vomiting</td>
<td>Mucous membranes, Vital signs/UO, Murmure, cyanosis</td>
<td>BUN and Cr, BUN/Cr ratio, ECHO</td>
</tr>
<tr>
<td>GI</td>
<td>Intestinal obstruction</td>
<td>No meconium, Constipation, Diarrhea, Vomiting</td>
<td>No feces in rectum, abdominal distention, malnutrition</td>
<td>Electrolyte panel, Abdominal films, Barium enema</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Implications
- Assessment of volume status due to bowel preparation, vomiting, and diarrhea
- Other congenital anomalies: Cardiac malformations (2–5%) and trisomy 21 (5–15%) are associated with congenital aganglionsis, consider cardiac evaluation and genetic testing

Monitoring
- Routine

Airway
- No significant issues other than those related to newborns or associated syndromes

Induction
- Rapid-sequence induction required if bowel obstruction present
- Monitor BP
- Some IV and inhalational anesthetics may be poorly tolerated if hypovolemic

Maintenance
- Prevent heat loss with forced warm air device and heating lamps. Pts may be uncovered from mid chest down and procedures may be lengthly (4–6 hr)
- Need for muscle relaxation
- Use isotonic IVFs. Consider checking an intraop blood glucose
- Avoid N2O
- Consider combined general and regional anesthesia for profound relaxation of anal sphincter muscles and reduction of need for IV narcotics or muscle relaxation

Extubation
- Can routinely be performed at the end of surgery. Pts should be awake.

Postoperative Period
- Apnea may occur in newborns receiving narcotics postop
- Association with congenital hypoventilation syndrome

- Consider postop pain management with continuous epidural/caudal anesthesia. The back may be prepped into the surgical field. Placement of regional technique may need to occur at the end of surgery.

Anticipated Problems/Concerns
- Aspiration pneumonitis
- Early postop surgical complications incl anastomotic leak (1–10%), pelvic abscess (5%) and wound dehiscence (3%).
- Late complications incl enterocolitis, constipation, incontinence, and stricture formation.
- Postop enterocolitis is the most serious and potentially life-threatening complication. Overall postop mortality is low at 2%.
- Definitive treatment of complications may require operative management involving a redo pull-through procedure, fecal diversion, or anorectal myectomy.
Histiocytosis

Risk
- Langerhans cell histiocytosis (LCH)
- Incidence: 1 in 250,000
- M:F ratio: 1.5:1
- Seen in all ages, but peak incidence at 1–3 y of age
- Pulm LCH common in young adults

Perioperative Risks
- Dependent on organ systems involved and extent of dysfunction

Worry About
- Specific organ dysfunction caused by infiltration with histiocytes incl: Liver, lungs, hematopoietic system, pituitary, spleen, and bone
- Can involve single or multiple sites and organs
- Treated with steroids and chemotherapy; at risk for adrenal insufficiency and may require stress steroids
- Diabetes insipidus due to posterior pituitary involvement
- Cervical instability if lesions present in cervical vertebrae

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Soft tissue distortion of airway, loose teeth, mucosal ulceration</td>
<td>Airway and dental evaluation</td>
<td>CXR, ABGs, PFTs, CT with cysts or nodular infiltrate</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Spontaneous pneumothorax, reactive airways, infiltrates, fibrosis, pulm Htn</td>
<td>Tachypnea, dyspnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Ulceration, obstruction, hepatic dysfunction</td>
<td>Jaundice Hepatomegaly</td>
<td>Bilirubin, albumin AST, ALT INR</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Diabetes insipidus, neuropathy, exophthalmos</td>
<td>Polyuria, polydipsia</td>
<td>Neuro exam</td>
<td>Urine and serum Osm, electrolytes</td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenia, anemia, leukopenia</td>
<td>Bruising or bleeding</td>
<td>Splenomegaly</td>
<td>CBC</td>
</tr>
</tbody>
</table>


Perioperative Implications

Monitoring
- Routine

Preinduction/Induction
- Airway soft tissue or mandibular involvement may distort anatomy.
- Cervical vertebrae lesions may cause cervical instability.
- Ensure adequate preoxygenation, esp. if there is significant pulm involvement.
- Usual precautions depending on severity of organ involvement

Maintenance
- For pts with DI consider aqueous ADH infusion, frequent sodium monitoring, and isotonic crystalloid fluids.
- Stress dose steroids if patient has had steroid therapy.
- Usual precautions depending on severity of organ involvement

Exsuffation
- Consider awake extubation if anatomy distorted and airway difficult
- Severe pulm involvement may delay extubation

Regional Anesthesia
- Follow ASRA precautions if thrombocytopenic or elevated INR

Postoperative Period
- May need continued stress dose steroid coverage for several days postop.
- Closely monitor oxygenation and ventilation when pulm disease present

Anticipated Problems/Concerns
- Organ dysfunction (hepatic, pulm, hematologic, hypothalamic, or bone)
- DI
- Adrenal suppression due to chronic steroid therapy. May have intraop hypotension without stress steroids.
- Severe pulm involvement may increase risk of pneumothorax and complicate extubation.
Hydrocephalus

Overview
- Excess accumulation of CSF due to obstruction in normal CSF flow pattern from ventricular system to cortical surface (obstructive hydrocephalus); or from impaired realabsorption of CSF at arachnoid villi (communicating hydrocephalus)
- Slow progressive hydrocephalus well tolerated for weeks with slowly worsening symptoms (headache, nausea, papilledema)
- Acute hydrocephalus results in acute symptoms and may be life-threatening owing to herniation of brain with catastrophic ischemic injury; bradycardia, Htn, depressed level of consciousness, depressed airway reflexes and resp drive, and gastric atony

ICD-9-CM Codes: 331.4 (Obstructive); 331.3 (Communicating)

Etiology
- Congenital: Anatomic abnormalities: Aqueductal stenosis, Arnold-Chiari malformation, Dandy-Walker syndrome

Anticipated Problems/Concerns
- Posthemorrhagic/Post-traumatic: Intraventricular hemorrhage (newborns or adults) with block clot in ventricular system
- Neoplastic: Brain tumor obstructing nml CSF flow
- Postinflammatory: Meningitis, abscess, meningoen cephalitis, intracranial hemorrhage

Perioperative Implications

Preoperative Preparation
- Assessment of urgency of presentation. Catastrophic increased ICP requires emergent intubation and hyperventilation. In young infants, direct neurosurgical needle puncture of a proximal lateral ventricle or previously inserted shunt may diminish ICP sufficiently to avoid a catastrophe.
- Secure IV access if possible

Monitoring
- Level of consciousness
- Routine

Airway
- Head up 10–20° and midline may diminish ICP
- Aspiration risk due to gastric atony

Preinduction/Induction
- Sedatives usually not indicated so that resp compromise or sedation does not increase ICP. Minimal sedation or use of local anesthetic to secure IV access without causing increased ICP due to pain, crying, or struggling.
- Rapid-sequence IV induction preferred (because of aspiration risk) unless in doubt of airway anatomy

Postoperative Period
- Usually unremarkable; depressed level of consciousness is concern for periop ischemic insult or hemorrhage.
- EBL: Minimal

Adjuvants
- Lidocaine, mannitol, furosemide, spontaneous hyperventilation by pt


Risk
- Newborns and children with anatomic CNS abnormalities (incl myelomeningocele)
- Head trauma and intracranial hemorrhage (prematurity, SAH, other causes)
- CNS tumors
- Meningitis
- Recurrent VP shunt malfunction

Perioperative Risks
- Cerebral ischemia and neurologic sequelae
- Impaired airway reflexes, level of conscious ness, gastric emptying
- Cardiorespiratory arrest

Worry About
- Intracranial Htn
- Persistent N/V
- Bradycardia
- Decreased level of consciousness

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Bradycardia, Htn</td>
<td>Late signs</td>
<td>Pulse, BP</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Impaired resp drive and airway reflexes</td>
<td>Cranial nerve exam, stridor, swallowing abn</td>
<td>Pulse oximetry</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>N/V, aspiration, abnormal feeding</td>
<td>Hx of progression of N/V</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Depressed level of consciousness, increased ICP, headache</td>
<td>Timing of onset</td>
<td>Arousalability and neurologic exam</td>
<td>CT scan</td>
</tr>
</tbody>
</table>

Timothy E. Smith
Joseph R. Tobin

Postoperative monitoring.
- Failure of achieving above criteria may require CT scan and/or ICU monitoring.

Extubation
- Ensure return of airway reflexes, level of consciousness, and resp drive.

Posthemorrhagic/Post-traumatic
- Intraventricular hemorrhage (newborns or adults) with block clot in ventricular system
- Blood loss.
- Use of local anesthetic to secure IV access
- Hyperventilation to minimize increase in ICP due to laryngoscopy and endotracheal intubation.

Maintenance
- Stable patient with blood clot in ventricular system
- Use of local anesthetic to secure IV access
- Hyperventilation to minimize increase in ICP due to laryngoscopy and endotracheal intubation.

Preoperative Monitoring
- Secure IV access if possible
- Preinduction/Induction
- Sedatives usually not indicated so that resp compromise or sedation does not increase ICP. Minimal sedation or use of local anesthetic to secure IV access without causing increased ICP due to pain, crying, or struggling.
- Rapid-sequence IV induction preferred (because of aspiration risk) unless in doubt of airway anatomy

Postoperative Monitoring
- Failure of achieving above criteria may require CT scan and/or ICU monitoring.
Hyperaldosteronism (Secondary)

James Duke

Risk
- Cause of Htn in as many as 20% of hypertensive pts, however, the numerical risk of 2° Htn cannot be estimated.
- These are high renin states and the greater risks may be associated with the primary problem leading to hyperreninemia.
- May have severe Htn refractory to therapy.

Perioperative Risks
- Htn, possibly refractory to treatment
- The volume status of these pts is uncertain. Some have edematous states with decreased plasma volume and some may be frankly hypovolemic.

Worry About
- The primary problem that leads to increased renin (and hence, aldosterone) secretion
- Hypokalemia and associated muscle weakness
- Metabolic alkalosis
- Renal insufficiency
- Pts with CHF can have 2° aldosteronism, so impaired myocardial function may be a concern.

Overview
- As opposed to primary hyperaldosteronism, where increased aldosterone is 2° to (usually) adrenal adenoma or adrenal cortical hyperplasia, increased aldosterone is a result of increased renin secretion.

Assessment Points

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<tbody>
<tr>
<td>CARDIO</td>
<td>Htn, often resistant to therapy; increased sympathetic activity, cardiac output often increased except where heart failure is an etiology. May have unexplained congestive failure.</td>
<td>Exercise tolerance, dyspnea, hypertension- headache</td>
<td>BP (compare R to L or arms with legs with coarctation)</td>
<td>BP, ABG, CXR, ECG</td>
</tr>
<tr>
<td>HEME</td>
<td>Hypovolemia (or decreased plasma volume in edematous states) may result in hypotension, hypokalemia, metabolic acidosis</td>
<td>Postural hypotension, tachycardia, weakness</td>
<td>Orthostatic BP</td>
<td>Serum electrolytes, bicarbonate, ABGs</td>
</tr>
<tr>
<td>RENAL</td>
<td>Increased renal tubular sodium absorption Azotemia may be caused by decreased renal perfusion or ineffective plasma volume. In setting of renal artery stenosis, ACE inhibitors may also result in renal failure; Hypokalemia</td>
<td>Htn, chronic renal disease, weakness</td>
<td>Abdominal bruit suggests renal artery stenosis Edema</td>
<td>BUN, creatinine, electrolytes, ABG</td>
</tr>
<tr>
<td>LIVER</td>
<td>Cirrhosis is an edematous state and pt may have decreased effective blood volume, altered metabolism of medications, hypoalbuminemia, coagulation abn</td>
<td>Alcoholism, hepatitis, other liver disease</td>
<td>Ascites, liver may be small, spider angiomas</td>
<td>Liver functions, coagulation tests, serum albumin</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preinduction/Induction/Maintenance
- Correct severe hypokalemia; this may require larger than usual supplementation. Assess and treat other electrolytes disturbances.
- Correction of Htn with mineralocorticoid antagonists, ACE inhibitors, angiotensin receptor blockers
- Assess volume status and cardiac function and determine choice of induction agents on this assessment.
- Largely dependent on primary medical problems that led to increased renin secretion

Monitoring
- Consider intra-arterial monitoring
- Central venous or pulm arterial monitoring should be predicated on assessments of intravascular volume and cardiac function.
- Urine output
- Neuromuscular blockade

General Anesthesia
- Consider co-morbidities and their impact on the pt
- Hypokalemia may potentiate muscle relaxants.

Regional Anesthesia
- Consider volume status.
- Direct acting sympathomimetics may exacerbate pre-existing Htn; titrate carefully to effect.

Postoperative Period
- Appropriate care predicated on surgical procedure, co-morbidities, hemodynamic stability
- Evaluate volume status, electrolytes, BP, myocardial function.
- If decreased liver function or renal function is the cause of 2° hyperaldosteronism, pt may have altered metabolism and excretion of medications and prolonged effects.
- Consider appropriate postop monitoring if sleep apnea is a concern.

Anticipated Problems/Concerns
- Labile blood pressure
- Pts with severe, longstanding Htn are at increased risk for left ventricular hypertrophy, myocardial infarction and stroke.
- Where CHF is an etiology, decreasing cardiac output under general anesthesia may exacerbate failure.
- Increased sympathetic activity leads to activation of the renin-angiotensin system.
- Decreased renal perfusion is a concern, esp. with renovascular Htn
Hypercalcemia

**Risk**
- Pts with hyperparathyroidism
- Pts with cancer: Breast cancer accounts for 25–50% of malignancy-related hypercalcemia. Other cancers associated with hypercalcemia incl lung cancer, squamous cell carcinomas of the head, neck, and esophagus, gynecological tumors, renal cell carcinoma, and multiple myeloma.

**Perioperative Risks**
- Pts with nml renal and CV function who have moderate hypercalcemia (11.5–13 mg/dL) have no special preop problems but may exhibit lethargy, anorexia, nausea, and polyuria.
- Severe hypercalcemia (>13 mg/dL) carries risk for:
  - Hypovolemia and acid-base abn; therefore, normal intravascular volume and electrolyte status should be restored prior to surgery
  - Neurologic symptoms with muscle weakness
  - Neurologic disturbances ranging from poor concentration to coma
  - CV effects incl Htn, dysrhythmias, heart block, cardiac arrest, and digitalis sensitivity
- Total serum Ca++ >14 mg/dL is a medical emergency and requires immediate treatment and delay of elective surgical procedures.

**Worry About**
- Volume status (hypovolemia 2° to polyuria, fluid overload 2° to treatment)
- Electrolyte disturbances
- Dysrhythmias and/or ECG changes
- Organ system manifestations of hypercalcemia (see table) and underlying disease

- Longstanding hypercalcemia can lead to calcification in the myocardium, blood vessels, brain, and kidneys. Beware of seizures from cerebral calcifications. Polyuria that is unresponsive to vasopressin may result from renal calcifications.

**Overview**
- Total body Ca++ is stored in bone (99%) and in the serum (1%).
- Total serum Ca++ exists in 3 fractions: 50% protein-bound (mainly to albumin), 40–50% free or ionized (the physiologically active fraction), and 5–10% anion-bound (to phosphate or citrate).
- The normal range for total serum calcium is 8.6 to 10.4 mg/dL; the normal range for ionized calcium is 4.7 to 5.3 mg/dL. Hypercalcemia is defined as total serum Ca++ >10.4 mg/dL.
- The total serum Ca++ level should be corrected for serum albumin level: for every 1 mg/dL decrease in serum albumin, there is a 0.8 mg/dL increase in Ca++.
- Normal serum Ca++ is regulated by several hormones:
  - Parathyroid hormone, which increases bone resorption and renal tubular resorption of calcium
  - Calcitonin, which inhibits bone resorption
  - Vitamin D, which augments intestinal absorption of Ca++

**ICD-9-CM Code**: 275.4

**Etiology**
- Disorders with increased bone resorption of Ca++: 1° or 2° hyperparathyroidism (common cause), malignancy (common cause – solid tumors elicit a PTH-like hormone that stimulates osteoclastic activity), hyperthyroidism, and immobilization
- Disorders with increased Ca++ absorption from the GI tract: Milk-alkali syndrome, vitamin D and A toxicity, and granulomatous diseases such as sarcoidosis
- Disorders with decreased renal Ca++ excretion: Thiazide diuretics, lithium therapy, familial hypocalciuric hypercalcemia, and renal failure

**Usual Treatment**
- Initiated in pts with total serum Ca++ >14 mg/dL or symptomatic pt with total serum Ca++ <14 mg/dL
- Volume expansion with saline to correct fluid deficit (from polyuria) and to increase urinary excretion of Ca++
- Loop diuretics to increase urinary excretion of sodium and Ca++
- Discontinuation of offending drugs, dietary Ca++ restriction, and increased physical activity
- Calcitonin, bisphosphonates, or mithramycin may be required in disorders associated with osteoclastic bone resorption
- Hydrocortisone may be used to reduce GI absorption of Ca++ in granulomatous disease, vitamin D intoxication, lymphoma, and myeloma. Hydrocortisone is not helpful in pts with hypercalcemia due to hyperparathyroidism or other malignancies.
- Dialysis may be required for life-threatening hypercalcemia.
- Surgical removal of the parathyroid glands to treat 1° or 2° hyperparathyroidism
- Treat underlying cause

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Postural symptoms, palpitations, fatigue, poor exercise tolerance, dizziness, syncope, headache</td>
<td>Orthostatic vital signs, narrowed pulse pressure, tachycardia</td>
<td>ECG</td>
</tr>
<tr>
<td>RENAL</td>
<td>Polyuria; polydipsia; renal tubular acidosis; nephrogenic diabetes insipidus</td>
<td>Confusion, obtundation and/or coma</td>
<td>Mini-mental exam</td>
<td>EEG</td>
</tr>
<tr>
<td>MS</td>
<td>Muscle weakness</td>
<td>Muscle weakness</td>
<td>Decreased muscle strength and tone, depressed deep tendon reflexes</td>
<td>X-ray (lytic lesions or pathologic Fx), densitometry (DXA)</td>
</tr>
<tr>
<td>ENDO</td>
<td>Excess PTH or production of PTH-related hormone</td>
<td>Poor appetite, nausea and/or vomiting</td>
<td>Abdominal pain</td>
<td>Radioimmunoassay of PTH or PTH-related peptides</td>
</tr>
<tr>
<td>GI</td>
<td>Anorexia, nausea and/or vomiting, bowel hypomotility and constipation, pancreatitis</td>
<td>Abdominal pain</td>
<td>Abdominal X-ray or CT scan, colonoscopy, LFTs (amylase and lipase)</td>
<td>EGD</td>
</tr>
</tbody>
</table>

Perioperative Implications

Preinduction
• Acquire knowledge of and treat the underlying cause.
• Determine if the hypercalcemia is acute or chronic.
• Assess volume status: Hydrate to attain normal intravascular volume and to promote renal excretion of Ca++.
• Administer diuretics to increase urinary Ca++ excretion if serum Ca++ >14 mg/dL.
• Correct other electrolyte imbalances: Hypophosphatemia, hypokalemia, and hypomagnesemia

Induction
• No specific anesthetic drug or technique has advantages in a pt with hypercalcemia however hemodynamic instability may occur if standard dosing is used in a hypovolemic pt.

Monitoring
• Standard ASA monitors
• Volume status (urine output and fluid administration); depending on the severity of hypercalcemia, underlying cause, the pt’s CV status, and type of surgery, additional monitors of volume status (CVP or TEE) should be considered.
• Electrolytes (whether sampled via venous or arterial access)
• ECG to monitor for shortened Q-T interval, S-T changes, decreased T wave amplitude or T wave inversion
• BP to monitor for Htn (approx one third of hypercalcemic pts have Htn that usually resolves with treatment of the 1st disease)

General Anesthesia/Maintenance
• Routine maintenance tailored to the comorbidities of the pt and the surgical needs
• Continued hydration and electrolyte replenishment to attain normal intravascular and acid-base status
• Hypercalcemia may be associated with decreased sensitivity to muscle relaxants and thus a shortened time course of neuromuscular blockade; however, associated electrolyte disturbances or renal insufficiency may prolong neuromuscular blockade.
• Careful positioning of the anesthetized pt is important because osteopenia/lytic bone lesions predispose these pts to pathologic bone fractures
• If the pt is mechanically ventilated, avoid resp alkalosis because alkalosis lowers plasma K+, which would leave hypercalcemia unopposed.

Regional Anesthesia
• General anesthesia is most commonly used for parathyroid surgery; however, a cervical plexus block or local anesthesia with hypnosis has also been used.

Postoperative Period
• Continue to monitor the same intraop parameters
• After parathyroid surgery, monitor for bleeding, recurrent laryngeal nerve injury or hypocalcemia (2nd to profound decrease in PTH).

Anticipated Problems/Concerns
• Fluid and electrolyte disturbances: Beware that Mg++, phosphate, and K+ levels may be altered with treatment of hypercalcemia.
• ECG changes
• Neurologic impairment: Altered mental status may impair ability to protect airway
• When hypercalcemia is severe, do not hesitate to consult a specialist (endocrinologist, nephrologist, or cardiologist) and postpone surgery if possible.
**Hypercholesterolemia**

**Risk**
- Incidence in USA: 102.2 million Americans age 20 and older have total cholesterol levels of 200 mg/dL or higher.
- Risk factors: Male age >45 y, women age >55 y, family Hx of premature CAD, current cigarette smoking, DM, obesity, Htn, CAD, HDL level <40 mg/dL.
- The LDL-C level of 130 to 159 mg/dL is considered borderline high, and level of ≥190 mg/dL is considered very high risk.
- An HDL (good) cholesterol level below 40 mg/dL in adults is considered low and is a risk factor for CHD and stroke.
- A triglyceride level >150 mg/dL in adults is considered elevated and is a risk factor for CHD and stroke.
- Familial hypercholesterolemia, an autosomal dominant trait (LDL >260 mg/dL), risk for premature CHD.
- Familial combined hyperlipoproteinemia elevated LDL and/or VLDL, increase risk of coronary disease.

**Perioperative Risks**
- Risk of acute coronary syndrome, myocardial ischemia and infarction.
- Cardiac events, worsened CHF.
- Stroke or death.

**Overview**
- The mean level of LDL (bad) cholesterol for American adults ≥20 y is 115.0 mg/dL.
- Normal total cholesterol < 200 mg/dL, borderline high 200–239 mg/dL, high ≥ 40 mg/dL.
- Suggested treatment goals in high-risk pts with CHD or CHD risk equivalents is an LDL level <100 mg/dL, 2 or more risk factor LDL level <130 mg/dL, 0–1 risk factor LDL level <160 mg/dL along with triglyceride <120 mg/dL and HDL >45 mg/dL.
- Intensive reduction of LDL-C to <70 mg/dL and ApoB <80 mg/dL is reasonable in highest risk pts.
- HDL ≥60 mg/dL is high and is considered protective.
- Periop statins use is not assoc with rhabdomyolysis or myopathy.
- Preop treatment with statins is assoc with improvement in early clinical outcome in pts undergoing cardiac surgery.

**ICD-9-CM Code: 272.0**

**Perioperative Implications**

**Preoperative Preparation**
- Assess for CAD, DM, and PVD.
- Pts currently on statins and undergoing noncardiac surgery, statins should be continued.
- Pts undergoing vascular surgery with or without clinical risk factors, initiation of statins should be considered.
- Assessment for statin related myopathy and liver damage.
- Assess and screen for obesity, related OSA and metabolic syndrome.

**Monitoring**
- Consider appropriate invasive monitoring in presence of large fluid shifts, ischemic Hx, and high-risk surgery.
- ST-T measurement or mapping in pts with CHD or risk factor for CHD.
- May be overweight and difficult to intubate or ventilate.
- Hypovolemia may lead to hypotension.
- Aggressive treatment for tachycardia, Htn, or hypotension during induction.
- Maintain hemodynamic stability without hypothermia or anemia (ideal Hct may be >27%).
- No anesthetic agent or technique proven superior.
- Monitor for ischemia and CHF.

**Etiology**
- Can be 1° or 2° to systemic illness such as diabetes, nephrotic syndrome, chronic renal failure, hypothyroidism, or drugs that increase LDL such as anabolic steroids.

**Usual Treatment**
- Life style modification: Dietary, physical exercise, and wt control.
- Cholesterol and lipid statistics checked every year in high-risk pts.
- HMG CoA reductase inhibitors (rosuvastatin, lovastatin, pravastatin, simvastatin, atorvastatin) are the drug of choice in most of the pts with hypercholesterolemia as they reduce LDL level effectively.
- High-risk pts with high triglyceride or low HDL level—consideration can be given to combine a fibrate or nicotinic acid with an LDL lowering drug.
- Gemfibrozil or nicotinic acid may be better choice in pts with significant hypertriglyceridemia.
- The combination treatment with reductase inhibitor and cholesterol absorption inhibitor (ezetimibe) highly synergistic in treating 1° hypercholesterolemia.

**ASSESSMENT POINTS**

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<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Myocardial ischemia and infarction</td>
<td>Angina or its equivalents</td>
<td>Displaced PMI S&lt;1</td>
<td>ECG, CXR, stress testing, ECHO, coronary angio</td>
</tr>
<tr>
<td></td>
<td>LV dysfunction</td>
<td>Dyspnea, edema, exercise intolerance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>CHF</td>
<td>Dyspnea, orthopnea, cough</td>
<td>Rales and rhonchi</td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Lipid deposits</td>
<td></td>
<td>Xanthelasma, xanthoma, arcus juvenilis</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Impaired renal perfusion</td>
<td>Nighttime urinary frequency</td>
<td>Cr</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Cerebrovascular atherosclerosis</td>
<td>TIAEs</td>
<td>Carotid bruit</td>
<td>Carotid US and angio</td>
</tr>
</tbody>
</table>


**Anticipated Problems/Concerns**
- ST-T measurement or mapping in pts with CHD or risk factor for CHD.
- May be overweight and difficult to intubate or ventilate.
- Hypovolemia may lead to hypotension.
- Aggressive treatment for tachycardia, Htn, or hypotension during induction.
- Maintain hemodynamic stability without hypothermia or anemia (ideal Hct may be >27%).
- No anesthetic agent or technique proven superior.
- Monitor for ischemia and CHF.

**Exubation**
- For noncardiac surgery, this is the period of greatest risk for ischemia.

**Postoperative Period**
- High incidence of tachycardia, ischemia, and MI for several days after noncardiac surgery.
- Treat pain, unstable hemodynamic and biochemical abn aggressively.

**Adjuvants**
- Depends on end-organ disease.
Hyperglycemia

Risk
- Incidence in USA: Can occur in virtually any anesthetized or critically ill pt
- Race with the highest prevalence: None

Perioperative Risks
- Increased likelihood of neurologic injury following brain ischemia, and perhaps traumatic brain injury and spinal cord injury
- Dehydration resulting from osmotic diuresis
- Increased infection rate
- Diminished wound healing

Worry About
- Electrolyte abn, particularly hypo-kalemia, while treating hyperglycemia
- Hypoglycemia following insulin, resulting in insult to the CV system and CNS

Overview
- Is not a disease
- Typically produces adverse effects by three mechanisms: Increases in plasma osmolality, increases in postischemic tissue lactic acidosis, and inhibition of white blood cell function
- Dx made by measuring blood glucose concentrations
- In acute setting, blood glucose concentrations can be estimated using indicator-impregnated strips or other point-of-care methods; confirmation can be made by mechanized techniques in a reference laboratory.

ICD-9-CM Code: 790.6

Etiology
- Results from DM (both insulin-requiring and non-insulin–requiring), other endocrinopathies (Cushing’s syndrome, acromegaly, obesity, pheochromocytoma), physiologic stress, drug administration (particularly corticosteroids), and glucose-containing fluid infusions

Usual Treatment
- Insulin
- Isotonic IV crystalloid solutions to treat hypovolemia and dilute existing blood glucose
- If possible, treat underlying cause (e.g., D/C infusion of glucose-containing solutions, D/C corticosteroids, reduce physiologic stress to pt)

ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Dehydration in extreme cases</td>
<td>Dry mucosa in extreme cases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Mild positive inotropic effect with mild hyperglycemia</td>
<td>Tachycardia, orthostatic hypotension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Polydipsia in extreme cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Osmotically induced diuresis</td>
<td>Polyuria, urinary frequency</td>
<td>Elevated urine glucose</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>See under Etiology</td>
<td>Elevated blood glucose</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Diminished WBC activity; changes in serum sodium concentrations</td>
<td>Serum sodium concentration decreases 1.6 mEq/L for each 100 mg/dL increase in glucose concentration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Altered consciousness, neurologic deficits</td>
<td>Plasma osmolality</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Glucose reduction with insulin
- Hydration
- Normalization of lytes

Monitoring
- Blood glucose concentrations in all cases
- In severe cases, blood lytes, blood osmolality, UO

Airway
- Abn typically related to DM (reduced range of motion and abn atlanto-occipital contractions), acromegaly (distorted anatomy), or chronic corticosteroid use or Cushing’s syndrome (cushingoid Sx, friable tissues)

Maintenance
- Maintain hydration
- Insulin therapy
- K+ replacement

Extubation
- No special considerations, other than those related to underlying disease

Adjutants
- Limit attempted reduction of blood glucose concentration to ~75 mg/dL/hr to avoid problems with osmotic injury to brain and lyte disturbances.
- Monitor ECG during correction of profound hyperglycemia.

Postoperative Period
- Variations in physiologic stress, fluid administration, and drug usage make postop blood glucose concentrations difficult to predict and control.

Anticipated Problems/Concerns
- Increases in blood glucose concentrations by a mere 40 mg/dL may worsen outcome following cerebral ischemic insult. Hyperglycemia may also harm wound healing, increase infection rates, and worsen outcomes after myocardial infarction. In contrast, hypoglycemia resulting from excessive use of insulin may result in pt morbidity and mortality from neurologic and other causes, independent of ischemic events.
Hyperkalemia

Overview
- Condition that can be due to total body K+ content or alterations in distribution between intracellular and extracellular sites

ICD-9-CM Code: 276.7

Etiology
- Diminished renal excretion
- Acute oliguric renal failure
- Chronic renal failure
- Addison disease
- Hyporeninemic hypoaldosteronism
- Drugs: Potassium-sparing diuretics, nonsteroidal anti-inflammatory drugs, heparin, ACE inhibitors, angiotensin-receptor antagonists, K+-containing antibiotics
- Ingestion of K+-rich foods, salt substitutes in pts with renal insufficiency
- Transcellular shifts
- Acidosis—resp or metabolic
- Cell destruction—trauma, burns, rhabdomyolysis, hemolysis, tumor lysis, reperfusion of ischemic limb or organ
- Hyperkalemic periodic paralysis
- Diabetic hyperglycemia

Risk
- Any pt with plasma K+ concentration >5.5 mEq/L

Perioperative Implications

Preoperative Preparation
- Normal K+ levels before elective surgery
- Avoid sedatives (↓ ventilation) prior to K+ normalization

Monitoring
- ECG
- Plasma K+ levels
- ABG concentration
- Peripheral nerve stimulator

Maintenance
- Adequate ventilation to avoid respiratory acidosis
- Avoid metabolic acidosis: Arterial hypoxemia or excessive depths of anesthesia
- IV fluids: Avoid lactated Ringer’s or others containing K+

Adjuvants
- Muscle relaxants: Avoid depolarizing agents; increase K+ 0.3–0.5 mEq/L with succinylcholine
- Dose of nondepolarizing relaxants required is unclear—may need diminished dose

Common Problems/Concerns
- Acute increases in K+ leading to acute ECG changes or adverse cardiac effects. Rx: see Usual Treatment.
- Avoid use of depolarizing muscle relaxants in pts with burns, spinal cord transection, catatonia with immobility, muscle trauma, or denervating muscle

Key Reference:
Cooper RC, Baumann PL, McDonald WM. An unexpected hyperkalemic response to succinylcholine during electroconvulsive therapy for catatonic schizophrenia. Anesthesiology. 1999;91:574–575.

Usual Treatment
- Promote transfer of K+ from ECF to ICF
- Glucose and insulin: 25–50 gm glucose with 10–20 units regular insulin/70 kg
- Sodium bicarbonate: 50–100 mEq/70 kg
- Hyperventilation: With each pH change of 0.1, there is an inverse change in K+ of 0.5 mEq/L.
- Dialysis: With each pH change of 0.1, there is an inverse change in K+ of 0.5 mEq/L
- Antagonism of cardiac effects: Ca++ gluconate—10–30 mL of a 10% solution over 10–20 min/70 kg counteracts cardiac effects
Hypermagnesemia

Risk
- Pts with renal insufficiency, esp. those receiving Mg²⁺-containing cathartics or antacids
- Parturients on MgSO₄ therapy
- “Runaway” infusion of Mg²⁺ during transporta-
tion to the OR can cause acute, life-threatening
event/magnesemia. Risk of developing very high serum Mg²⁺ levels in such cases can be reduced by
always using a small volume Burette device in pts
receiving IV Mg²⁺ therapy.

Therapeutic Uses
- Treatment of pre eclampsia, eclampsia, and
preterm labor
- Recent evidence indicates that Mg²⁺ therapy reduces the risk of cerebral palsy in women at risk of preterm delivery
- Treatment of ventricular dysrhythmias
- Treatment of severe asthma in pts who have not responded to initial therapy
- Treatment of migraine
- Lower risk of metabolic syndrome

Perioperative Implications

Preoperative Preparation
- D/C MgSO₄ unless being used to treat seizures or ventricular dysrhythmias
- Check serum level
- ECG, creatinine, electrolytes

Monitoring
- Routine

Airway
- Use full dose of succinylcholine for intubation
- Reduce dose of non-depolarizing NMBs by one
third to one half

Preinduction/Induction
- Avoid sedative premedications
- Ensure full denitrogenation of lungs
- Avoid pre-curarization or priming dose of NMB

Maintenance
- May decrease requirement for anesthetics owing to decreased neurotransmitter release

Exubation
- Ensure full return of train-of-four, ability to
sustain head lift and vital capacity >10 mL/kg
- Ensure pt responsiveness

Adjuvants
- Hypermagnesemia may exacerbate hypotension associated with hypovolemia, Ca²⁺-channel block-
ers, volatile inhalation anesthetics, butyrophenones, lumbar epidural, or subarachnoid anesthesis
- Treat with IV calcium gluconate 1 gm and fluid
load and diuretics

Postoperative Period
- Beware of excessive sedation, weakness, hypventilation, cardiac arrest

- May cause or aggravate neonatal hypotonia and
hypotension
- May reduce postop analgesic requirements by
antagonism of N-methyl-D-aspartate

Anticipated Problems/Concerns
- Hypermagnesemia potentiates action of non-
depolarizing NMBs by inhibiting release of ace-
tyline from motor nerve terminal, decreasing sensitivity of postjunctional membrane, and reduc-
ing excitability of muscle fibers
- Many common anesthetic drugs exacerbate
weakness and sedation associated with hyper-
magnesemia
- Potentiates local anesthetic toxicity
- Excessively high plasma Mg²⁺ concentrations can cause cardiorespiratory arrest

ASSESSMENT POINTS

The side effects of hypermagnesemia are more serious as the serum level of magnesium increases.

<table>
<thead>
<tr>
<th>System</th>
<th>Signs and Symptoms</th>
<th>Serum Mg²⁺ Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENERAL</td>
<td>Normal</td>
<td>0.7–1.1 mmol/L (normal range)</td>
</tr>
</tbody>
</table>
| CARDIO   | Warmth, flushing, headache, nausea, dizzi-
ness                                      | 2–3 mmol/L (range during parenteral treatment) |
|          | Decreased AV and intraventricular conduc-
tion                                    | >2.5 mmol/L                     |
|          | ECG—prolonged PQ and widening of QRS    |                                |
|          | Possible hypotension                     |                                |
|          | Cardiac arrest in diastole*              | >12.5 mmol/L                   |
| CNS      | Sedation                                 | 2–3 mmol/L                     |
| MS       | Absent deep tendon reflexes              | 4–5 mmol/L                     |
|          | Progressive muscle weakness and resp arrest | 6–7.5 mmol/L                   |

*The ability of this degree of hypermagnesemia to cause cardiac arrest is uncertain if ventilatory support and normal acid-base balance are maintained.


Risk
- The side effects of hypermagnesemia are more serious as the serum level of magnesium increases.

Diseases

Cardiovascular
- May exacerbate local anesthetic toxicity

General
- May cause or aggravate neonatal hypotonia and
hypotension
- May reduce postop analgesic requirements by
antagonism of N-methyl-D-aspartate
Hypernatremia

Risk
- Older age, infants, prior brain injury, DM, surgery, diuretic therapy, altered mental status; insufficient water intake, DI, hypertonic sodium solution (incl sodium bicarbonate), hyperalimentation, hyperaldosteronism, Cushning syndrome, hypothalamic injury

Perioperative Risks
- Increased incidence of morbidity and mortality, seizures, coma, cerebral bleeding, subarachnoid hemorrhage

Worry About
- Increased risk of hospital death, residual and/or permanent neurologic disability
- If Na⁺ corrected too rapidly, cerebral edema, seizures, and death

Overview
- Hypernatremia is a relative deficit of body H₂O in relation to body sodium content.
- Serum Na⁺ is preserved within a fine physiologic range (138–142 mEq/L).
- Sodium metabolism is regulated by the kidney through the interaction of the renin-angiotensin-aldosterone system, sympathetic nervous system, atrial natriuretic peptide, brain natriuretic peptide, effective circulating volume, and serum H₂O content. H₂O metabolism is tightly regulated by arginine vasopressin.
- Most commonly found in pts with impaired sense of thirst (brain injury, altered mental status), lack of access to H₂O, diuretic therapy, and severe GI losses of H₂O.

Etiology
- Lack of access to H₂O
- Impaired thirst mechanism
- DI (central and nephrogenic)
- Osmotic diuresis (mannitol, glucose); diuretics (furosemide, thiazides)
- Insensible losses from the dermal or resp systems
- GI losses from diarrhea or osmotic cathartics (lactulose, sorbitol), vomiting, or nasogastric suctioning
- Seizures or severe exercise (transient intracellular shift of H₂O)
- Excess sodium administration; hyperalimentation
- Hyperaldosteronism and Cushning syndrome

Usual Treatment
- H₂O replacement (see below); central DI can be treated with desmopressin (5–20 mcg intranasal once or twice per day), nephrogenic DI can be treated with thiazide diuretics.
- Free H₂O deficit = total body water X [(serum Na⁺/140)−1]
- Total body water is approx 0.6 and 0.5 x the lean body weight for men and women, respectively. Replace ½ of the free H₂O deficit over the first 24 hr as an initial starting point. Note that the free H₂O deficit does not take into account on-going losses, so ultimately the rate of H₂O replacement must be guided by serial measurements of serum Na⁺.
- Rate of correction of Na⁺ to level of 145 mmol/L:
  - If hypernatremia developed acutely, Na⁺ can be corrected rapidly (1 mmol/L per hr, with a limit of 12 mmol/L per 24 hrs)
  - If hypernatremia developed slowly, Na⁺ can be corrected at a maximum rate of 0.5 mmol/L per hr (or in the case of life-threatening complications, at 1 mmol/L/hr with a limit of 12 mmol/L per 24 hrs)
- Measurement of Na⁺ at least every 4–6 hr and adjustment of the rate of H₂O replacement is important to ensure safe and expeditious correction of Na⁺.

ASSESSMENT POINTS

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<tbody>
<tr>
<td>HEENT</td>
<td>Dry mouth/mucous membranes</td>
<td></td>
<td>Mouth exam</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Tachycardia/hypotension</td>
<td>Orthostatic changes</td>
<td>HR/BP</td>
<td>ECG</td>
</tr>
<tr>
<td>CNS</td>
<td>Restlessness, irritability, lethargy, seizures, coma</td>
<td>CNS exam</td>
<td>EE G</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>NVD</td>
<td>Urinary frequency/color</td>
<td>Serum and urine Na⁺, K⁺, osmolality</td>
<td></td>
</tr>
</tbody>
</table>

Renal

Perioperative Implications

Preoperative Preparation
- Correct electrolytes, replace H₂O deficit, assess neurologic status.
- Consider delaying elective surgery until serum Na⁺ is normal. If surgery cannot be delayed, care must be taken to avoid rapid correction of Na⁺.

Monitoring
- Electrolytes

Airway
- None

Maintenance
- Restore circulatory volume
- Maintain uo
- Correct electrolytes

Exsuffation
- Assess neurologic status to determine whether the pt is a candidate for exsuffation.
- Possible muscular weakness

Adjuvants
- In central DI, vasopressin 5 units IVP will dramatically reduce UOP for 1–2 hr, making it possible to catch up on IV fluids.
- Caution must be used to avoid too-rapid correction of Na⁺.

Postoperative period
- Assess for lethargy, irritability, muscular weakness, confusion.
- Monitor serum Na⁺.

Anticipated Problems/Concerns
- Too rapid correction and resultant neurologic effects

**Hyperglycemic Hyperosmolar State (HHS)**

**Risk**
- Elderly pts with DM, usually type II
- Debilitated pts who cannot care for themselves
- Chronically ill diabetic pts who experience exacerbation of an underlying co-morbidity
- Incidence increased in African Americans, Hispanics, Native Americans

**Perioperative Risks**
- Severe hypovolemia and hemodynamic instability
- Presence of diffuse organ system damage from poor glycemic control
- Altered mental status and increased risk of pulmonary aspiration
- Poirop stress causing further elevations in serum glucose

**Worry About**
- Cause of hyperglycemic hyperosmolar state
- Volume status and potential hemodynamic instability
- Electrolyte and acid-base abn increase the risk of cardiac arrhythmias

**Overview**
- Serious metabolic condition characterized by hyperglycemia, hyperosmolarity, and dehydration
- Is one of several potentially fatal states associated with poorly controlled DM
- Requires aggressive treatment and close electrolyte and hemodynamic monitoring

**ICD9-CM: 250.2 (Hyperosmolar [nonketotic] coma)**

**Etiology**
- Inadequate insulin production and increased counter-regulatory hormone production (cate-cholamines, glucagon, cortisol) in the setting of an acute insult leads to severe hyperglycemia, dehydration and electrolyte abn
- Triggering event may be infection, dehydration, CVA, inadequate dosing of insulin, silent myocardial infarction, pancreatitis, or drug ingestion (drugs that affect carbohydrate metabolism)

**Usual Treatment**
- Aggressive volume resuscitation with isotonic fluids to re-establish end-organ perfusion
- Insulin replacement and correction of electrolyte abn (start dextrose-containing fluids when serum glucose approaches 250 mg/dL to help prevent hypoglycemia and cerebral edema)
- Identify and treat underlying cause of hyperglycemic state
- Frequent evaluation of volume resuscitation and metabolic status in an ICU setting

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEURO</td>
<td>Altered mental status, obtundation, coma, seizures</td>
</tr>
<tr>
<td>CARDIAC</td>
<td>Hypovolemia and shock</td>
</tr>
<tr>
<td>PULM</td>
<td>Hyperventilation if severe metabolic acidosis, hypoventilation if brainstem malperfusion</td>
</tr>
<tr>
<td>ENDO</td>
<td>Insulinopenia, hyperglycemia, hyperosmolarity</td>
</tr>
<tr>
<td>RENAL</td>
<td>Polyuria progressing to anuria, metabolic acidosis, electrolyte abnormalities</td>
</tr>
</tbody>
</table>

**Assessment by Hx**
- Progression of mental status changes over days
- Polyuria progressing to anuria, sense of thirst, headaches, dry mouth
- DM, recent infection or stress
- Polyuria progressing to anuria, metabolic acidosis, electrolyte abnormalities

**PE**
- Mental status exam, airway reflexes
- Orthostatic hypotension, tachycardia, dry mucous membranes
- Resp rate and pattern of ventilation
- Serum glucose and osmolarity

**Test**
- Head CT, CSF culture
- CVP, PAP, ECHO
- ABG


**Perioperative Implications**

**Monitoring and Intravenous Access**
- Large bore IV access for volume resuscitation, particularly before induction
- Invasive monitoring incl arterial line and CVP may be useful to guide volume replacement and allow for frequent glucose and electrolyte sampling.

**Induction**
- Aggressive volume resuscitation before induction
- Rapid sequence induction if altered mental status and concern for aspiration
- Limited use of succinylcholine if metabolic acidosis and hyperkalemia are present

**Anticipated Problems/Concerns**
- Be prepared for exaggerated hemodynamic changes with induction despite adequate volume resuscitation.
- Smaller doses than usual of induction agent if pt is obtunded
  - Close follow serum glucose, electrolytes, acid-base status.
  - Continue volume resuscitation until UO is adequate and hemodynamics have stabilized.
  - Assessment of airway reflexes and ability to protect airway before tracheal extubation
- Ensure metabolic and electrolyte status is corrected, and pt meets the usual criteria for extubation.

**Postoperative Period**
- Continued insulin therapy and observation for worsening hyperglycemia due to surgical stress response

**Usual Treatment**
- Aggressive volume resuscitation with isotonic fluids to re-establish end-organ perfusion
- Insulin replacement and correction of electrolyte abn (start dextrose-containing fluids when serum glucose approaches 250 mg/dL to help prevent hypoglycemia and cerebral edema)
- Identify and treat underlying cause of hyperglycemic state
- Frequent evaluation of volume resuscitation and metabolic status in an ICU setting
Hyperparathyroidism

Overview
- Endocrinopathy associating elevation in PTH levels
- Primary problem is hypercalcemia
- Dx supported by increased PTH level associating with hypercalcemia
- Most pts with primary hyperparathyroidism are hypercalcemic but asymptomatic
- Hyperparathyroidism in pregnancy is associating with high (50%) maternal and fetal morbidity and can lead to neonatal hypocalcemia and tetany

ICD-9-CM Code: 252.0

Etiology
- Primary hyperparathyroidism usually associating with benign parathyroid adenoma (80–90%), hyperplasia (15%), or parathyroid carcinoma (uncommon)
- May be manifestation of multiple endocrine neoplasia Type 1 or 2a
- 2° hyperparathyroidism may be seen in pts with chronic renal disease

Usual Treatment
- Surgically with parathyroidectomy
- Recent advances such as nuclear imaging for correct localization of parathyroid tumors, quick hormone assays, and radiologically-guided or video-assisted surgical techniques are allowing minimally invasive parathyroidectomy under local/regional anesthesia
- Medically with saline hydration, furosemide, and phosphate repletion in emergency situations to restore serum Ca++ to a safe level (<14 mg/dL)
- Other Ca++ lowering modalities such as calcitonin, cinacalcet, bisphosphonates (inhibit bone resorption), mitramycin (for more resistant hypercalcemia; toxic effects limit use), glucocorticoids, or hemodialysis
- Pregnant women with primary hyperparathyroidism should be treated with parathyroidectomy ideally in the second trimester

Risk
- Incidence in USA: 100,000 pts per year
- Race with highest prevalence: None
- M:F ratio: 1:2
- Prevalence: 50–100/100,000 (increases with age)
- 0.8% in pregnancy

Perioperative Risks
- Hypovolemia and lyte abn
- Increased risk of cardiac dysrythmias 2° to hypercalcemia
- Aspiration from full stomach
- Postop hypocalcemia

Worry About
- Signs of hypercalcemia and other lyte irregularities
- Intravascular volume changes
- Fluid overload and Na+ retention in CV fragile pts
- Renal, cardiac, skeletal, and CNS abnormalities
- Pancreatitis 2° to hypercalcemia

Preoperative Preparation


ASSESSMENT POINTS

System Effect Assessment by Hx PE Test
CARDIO Htn, dysrhythmias Palpitation, headache Abn pulse rate and/or rhythm, ↑ BP ECG, lytes, total and ionized Ca++, QTc interval
RESP ↓ Bronchial clearance of secretions Cough Adventitious sounds
GI Peptic ulcers, pancreatitis Constipation, anorexia, N/V, epigastric pain
RENAL Nephrocalcinosis, nephrolithiasis → renal dysfunction Polyuria, polydipsia, hematuria BUN, Cr
CNS EEG abn, seizures Depression, personality change, psychomotor retardation, memory impairment Psychosis, disorientation, obtundation, coma
MS Hyporeflexia, osteopenia, osteitis fibrosa cystica Weakness, bone pain Muscular atrophy, arthritis, pathologic fractures

Airway
Possibility of pathologic fractures requires careful positioning for laryngoscopy

Preinduction/Induction
- No preferred agents or techniques
- Avoid ketamine in pts with psychosis due to hypercalcemia.
- Hypervolemia can lead to hemodynamic instability if usual dose of induction agents is given.
- Minimally invasive procedures can be performed using local/regional anesthesia.

Maintenance
- No preferred agents or techniques. Possibility of pathologic fractures requires careful positioning and padding of pressure points.

Extubation
- Airway edema, surgical site hematoma, or recurrent laryngeal nerve injury may cause airway compromise.

Adjuvants
- Response to NM blockers may be unpredictable if Ca++ level elevated

Anticipated Problems/Concerns
- Cardiac arrhythmias due to hypercalcemia
- Postop airway compromise 2° to bleeding or recurrent laryngeal nerve injury
- Pneumothorax 2° to surgical procedure
- Fluid overload and lyte abn from too aggressive hydration

* QTc = QT / R-R

Hypertension

Risk
- Incidence in USA: Affects approx 50 million people
- The incidence of Htn increases with advancing age. Half of people aged 60–69 y and ¾ of people aged over 70 y are affected.
- There is a continuous relationship between BP and the risk of CV diseases incl MI, heart failure, stroke, and kidney disease. For people aged 40–70 y a 20-mm Hg increase in systolic pressure or a 10 mm Hg increase in diastolic pressure doubles the risk of CV disease across the entire range of BPs.

Perioperative Risks
- BPs of up to 180/100 mmHg are not independently associated with an increased risk of periop complications. There are limited data that suggest BPs greater than this may be associated with an increased risk of such complications.
- Intraop CV lability, esp. hypotension, which may precipitate myocardial ischemia or predispose to stroke.

Worry About
- Markedly elevated BP (>180/110 mmHg).
- Possible 2˚ Htn.

Overview
- Approx 95% of people with raised BP have essential Htn whilst in 5% of people an underlying cause for Htn can be identified.
- The aim of the long-term medical management of Htn is to reduce the burden of CV morbidity and mortality associated with chronically raised BP.
- • The 1˚ concern of the anesthetist when managing a hypertensive pt through the periop period is to prevent or curtail the periop myocardial ischemia and BP lability that has been demonstrated to occur in Htn pts undergoing anesthesia and surgery.
- • Target organ damage associated with Htn may of itself increase periop risk
  - Ischemic heart disease
  - Heart failure
  - Cerebrovascular disease
  - Renal impairment
  - Peripheral vascular and aortic disease

ICD-9-CM Code: 401

Etiology
- Essential Htn appears to be a complex multifactorial condition for a single cause has not been identified. Factors that have a role in the development of essential Htn incl genetic factors, race (increased prevalence and severity in African Americans), age, sedentary lifestyle, obesity (in particular visceral obesity), sodium intake, alcohol intake, childhood influences (birth wt, BP tracking). Htn is part of the constellation of disorders that constitute the metabolic syndrome.
- 2˚ Htn is found in approx 5% of people with raised BP. Identifiable causes of Htn incl sleep apnea, drug-induced Htn, chronic renal disease, renovascular disease, primary aldosteronism, Cushing syndrome, chronic steroid treatment, pheochromocytoma, thyroid and parathyroid disease.
- Many pts who are found to have raised BP at presentation for surgery will be found to not to be hypertensive when the BP is rechecked in a less stressful setting.

Usual Treatment
- • Lifestyle modification should be encouraged in all pts with elevated BP.
- • Goals for long-term BP treatment are <140/90 for general CV disease prevention, <130/80 for pts with high CV risk, a Hx of ischemic heart disease, diabetes or chronic renal disease and <120/80 for pts with left ventricular dysfunction.
- • BP reduction is more important than the choice of drug in the 1˚ prevention of CV complications. There is evidence to support ACEIs, angiotensin receptor blocking drugs (ARB), calcium channel blockers, or thiazide diuretics as first-line therapy. Combination therapy is frequently required to achieve and sustain long-term BP control.
- • Specific classes of antihypertensive drugs may provide better 2˚ prevention in pts with compelling indications for BP control. In pts with a Hx of previous MI a beta-blocker (if the pt is hemodynamically stable) and an ACEI or ARB is indicated.

ASSESSMENT POINTS

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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CAD</td>
<td>MI, angina, previous CABG or PCI Dyspnea, orthopnea</td>
<td>Displaced apex beat S1, basal crepitations Rales Pulses Ankle brachial pressure index</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>LVH/LVF</td>
<td>Peripheral vascular/aortic disease Claudication/rest pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Metabolic syndrome</td>
<td></td>
<td>Central obesity</td>
<td>Fasting blood glucose Triglycerides HDL-cholesterol</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal impairment</td>
<td></td>
<td></td>
<td>Creatinine Estimated creatinine clearance Microalbumin urine test</td>
</tr>
<tr>
<td>CNS</td>
<td>TIA/CVA</td>
<td>Hx of TIA/CVA Neurologic signs Carotid bruit</td>
<td></td>
<td>Doppler CT/MRI Angiography/CT angiography/MR angiography</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preinduction/Induction/Maintenance
- • There is no clear evidence to support deferring surgery or for acute management of BP in pts presenting with moderate Htn.
- • Severe Htn (>180/110 mmHg) confirmed on multiple readings should be controlled prior to surgery if the delay necessary to achieve this will not compromise the pt (esp. if the pt has evidence of target organ damage).
- • Consider withholding ACEI and ARBs for 12 hr before surgery as they may be associated with an increased incidence of intraop Htn.
- • Maintain treatment with other antihypertensive medications (in particular beta-blockers) unless the pt is hypotensive or has evidence of postural hypotension.
- • If beta-blockers are started de novo in the periop period begin with a low dose and titrate this up gradually ensuring that the pt does not become hypotensive relative to their usual resting BP.
- • Maintain euvolemia, esp. in pts taking vasodilating drugs such as ACEI or ARB.

Monitoring
- • Standard monitoring
- • Frequent BP readings should be taken at times of potential CV instability such as induction in order to detect sudden changes in BP.
- • Consider direct arterial pressure monitoring if surgery is proceeding in the face of severe Htn.
- • Consider dynamic (e.g., pulse pressure variation) or static (CVP) volume monitoring if significant hypovolemia is suspected.

General Anesthesia
- • Pts may develop profound hypotension at induction and Htn at intubation.
- • Consider a fluid preload prior to induction if relative hypovolemia is suspected.
- • Prepare a short acting vasopressor prior to induction.
- • Consider the use of opiates or short-acting vasoactive drugs to control the response to intubation in pts with significant CV disease.
• Aim to keep intraop BP within 20% of best estimate of preop BP with appropriate use of fluids and vasoactive drugs.
• No anesthetic maintenance technique has been demonstrated to be superior in this setting.

Regional Anesthesia
• Risk of hypotension with neuroaxial blockade
• Consider a fluid preload prior to neuroaxial blockade.
• Take BP readings every 1–2 min immediately after neuroaxial blockade if using non-invasive monitoring.
• As with general anesthesia, aim to keep intraop BP within 20% of best estimate of preop BP.

Postoperative Period
• Resume normal antihypertensive treatment as soon as possible.
• If the pt is not on appropriate CV prevention make appropriate medical referrals to rectify this if possible.

Anticipated Problems/Concerns
• In some cases parenteral treatment of BP may be required if the pt cannot take oral medications.
• Consider parenteral beta-blockade if a pt who is chronically treated with a beta-blocker is unable to resume this treatment.
• In pts with pre-existing CV disease poorly controlled BP in the postop period may precipitate myocardial ischemia and cardiac complications.
Hypertension, Uncontrolled with Cardiomyopathy

**Risk**
- 1.5 billion worldwide in 2005
- USA highest prevalence: African American
- Male=female

**Perioperative Risks**
- Increased risk of MI and/or ischemia
- Increased risk of stroke
- Increased risk of CHF
- Increased risk of renal failure
- Increased blood loss
- Increased risk of cerebral hypoperfusion due to the shift to the right of the curve for the autoregulation of cerebral blood flow
- Prolonged hospitalization

**Overview**
- Possibility of masked hypovolemia

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>LV function LVH</td>
<td>Exercise by Hx</td>
<td>2-flight walk</td>
<td>ECG, CXR ECHO, MUGA Stress thallium</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>Orthopnea Dyspnea</td>
<td>Rales</td>
<td>CXR</td>
</tr>
<tr>
<td>CNS</td>
<td>Stroke</td>
<td>Blackouts Carotid bruit</td>
<td>Carotid study</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Nephropathy Edema</td>
<td>BUN/Cr</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative**
- Continue and/or increase antihypertensive medicine
- Short-acting vasodilators prepared, incl nitroglycerin
- Assess myoccardial and volume status
- Anxiolytics on the day before surgery
- Correct electrolyte imbalance if present

**Monitoring**
- Arterial monitoring
- Foley catheter to monitor urine output for traumatic or long procedures or for procedures with expected significant blood loss
- Volume status monitoring depending on LV function (e.g., CVP, possibly PA catheter, or TEE)
- Consider brainwave monitoring to ensure adequate depth of anesthesia and optimal anesthetic dosing

**Induction**
- Pre-intubation opiates to blunt hypertensive response to laryngoscopy and ETT placement
- Consider administration of the high end of the dose range of the IV induction agent with uncontrolled Htn, with significant cardiomyopathy, consider etomidate to maintain cardiac hemodynamics
- Use of defasciculating dose of non-depolarizing NMB to prevent mesenteric blood mobilization during abdominal muscle contractions during acetylcholine-induced muscular fasciculations

**Exubation**
- Adequate pain relief prior to termination of anesthesia
- Short-acting vasodilator and/or β-blockers to prevent Htn and tachycardia

**Adjutants**
- Regional: May prevent severe increases in BP, since intubation not needed. Severe dehydration may be present, resulting in profound hypotension.
- Continuous infusions of nitroglycerin, nitroprusside, or esmolol
- Severe hypotension may not respond to usual doses of vasoconstrictor due to prior drug treatment.
- Consider use of α-, β-adrenomedimetics
- Inhalational agents, in particular, above 1 MAC can cause dose-dependent increase in heart rate and have different hemodynamic effects

**Postoperative**
- Restart antihypertensive medication as soon as possible in postop period
- Patch therapy for some drugs must be started 12 h prior to anticipated need due to slow absorption from skin, e.g., clonidine and fentanyl
- Effective pain control using opioids and/or NSAIDs or continuous blockade

**Anticipated Problems/Concerns**
- Watch for symptoms of CNS, renal, or myocardial dysfunction
- Preop period affords opportunity to educate pt about importance of complying with antihypertensive therapy
- Hypotension if therapy is continued, esp. angiotensin-receptor blocking drugs and receptor inhibitors; or if pt is untreated and volume depleted
- Rebound Htn if certain medications are discontinued (e.g., clonidine)
# Hyperthyroidism

**Risk**
- Incidence in USA: 440,000/y develop hyperthyroidism plus 5–15% of pregnant females (highest prevalence in 2nd trimester); 1/1000 females; 1/3000 males
- Race with highest prevalence: Unknown

**Perioperative Risks**
- Risk related to occurrence of thyroid storm; increased risk of storm, even if made euthyroid prior to surgery
- Some increased risk of resp insufficiency
- Progressive increased risk of hypothyroidism after surgery on thyroid, radioactive Rx of hyperthyroidism, and thyroiditis

**Worry About**
- Assessing that pt is euthyroid
- Securing airway in pt with large goiter or displaced trachea
- Postop risks of nerve injury (immediate stridor requires immediate reintubation), surreptitious bleeding (examine wound—can drain externally—prior to PACU discharge), and thyroid storm (uncommon without another acute illness or after 3 d postop)

### Overview
- Endocrinopathy with CV disease—tachycardia (commonly idiopathic if no prior Dx of hyperthyroidism has been made), CHF, dysrythmias (AFIB) as major manifestation
- Other target: Resp and CNS (decreases drive to breathe; worsens anxiety, psychoses) and metabolic (hypermetabolism and increased protein turnover resulting in weakened muscles and malnourishment); can present as unintentional wt loss
- If euthyroid prior to operation, risk of storm and of periop CV problems diminished by >90%
- If not euthyroid, try to delay operation until euthyroid
- If emergency (life-threatening trauma, ruptured vissus), use β-blocking agents and iodides to decrease periop effects and decrease further synthesis and release of thyroid hormones; keep in ICU until risk of storm has passed
- ICD-9-CM Codes: 242.9 (Hyperthyroidism [thyrotoxicosis]); 242.0 (Graves’ disease); 245 (Thyroiditis); 193 (Malignant thyroid disease); 198.89 (Metastatic malignant thyroid disease)

### Anticipated Problems/Concerns
- Thyroid storm is life-threatening illness if hyperthyroidism has been severely exacerbated by illness or operation. Manifested by hyperpyrexia, tachycardia, striking alterations in consciousness. Early signs include delirium, confusion, mania, excitement. Differential Dx: malignant hyperthermia, pheochromocytoma crisis, NMS.
- Rx incl supportive care, methimazole or propylthiouracil followed in 1 hr by iodides and propranolol, decrease conversion of the less active T3 to the more active T4
- Surreptitious bleeding behind neck bandages, or into chest if minimally invasive technique is used from axilla, can suddenly compromise airway function or result in CV collapse
- Recurrent laryngeal nerve injuries post thyroidectomy usually result in damage to abductor fibers, which results in hoarseness
- Bulbous glottic edema can require immediate reintubation
- Occasionally late tetany (usually 2–3 d post thyroidectomy) can occur from accidental removal of or damage to parathyroid glands

### ASSESSMENT POINTS

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<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Weakened tracheal rings, distorted/displaced trachea</td>
<td>Snoring, hoarseness, neck pain</td>
<td>Ask to vocalize “e”; examine airway and neck; look at eyes; test for diplopia; change over time in measure of eye protrusion</td>
<td>Check CXR (PA and lateral) lateral neck films; CT scan or US of neck</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Dysrhythmias, AFIB, sinus tachycardia, mitral valve prolapse, CHF, cardiomyopathies</td>
<td>Palpitations; ↑ HR during sleep DOE, orthostatic SOB</td>
<td>Standard exam</td>
<td>Rhythm strip or full ECG; CV system is involved in either Hx or PE</td>
</tr>
<tr>
<td>GI</td>
<td>Wt loss, diarrhea, dehydration</td>
<td>Dizziness on arising; Hx of diarrhea, constipation</td>
<td>Skin turgor; other measures of volume status such as orthostatic vital signs</td>
<td>Increased serum alkaline phosphatase</td>
</tr>
<tr>
<td>HEMΕ</td>
<td>Mild anemia, thrombocytopenia; agranulocytosis 2° to propylthiouracil or methimazole</td>
<td>Skin/mucous membranes for infection/petechiae</td>
<td>CBC with plt count and differential</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Need to assess if euthyroid; malnourished</td>
<td>Reflex speed, tremor, nervousness, mental status</td>
<td>Reflex speed; HR</td>
<td>Free T4</td>
</tr>
</tbody>
</table>

### Key Reference

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**Perioperative Implications**
- See also under thyroidectomy, subtotal

**Preoperative Preparation**
- Assess if euthyroid
- Assess for associated autoimmune diseases

**Preinduction/Induction**
- Prehydrate liberally if CV status will tolerate
- Check and protect eyes

**Anesthetic Technique**
- No one technique has proved superior
- Hyperthyroidism is associated risk factor for halothane hepatitis

**Monitoring**
- Temp (also place cooling blanket on OR table to treat thyroid storm if it occurs)
- Consider invasive monitoring if pt has dilated cardiomyopathy/thyroid storm/severe dysrythmias
Hypertriglyceridemia

Risk
- Elevated triglyceride levels are an independent risk factor for CAD after adjustment for other risk factors. The risk increases proportionally with increasing triglyceride levels.
- 30% of the population have elevated levels at age 20 and 43% have elevated levels by age 50.
- Component of the metabolic syndrome (increased waist circumference, low HDL, HTn, impaired glucose metabolism) each of which are risk factors for CAD.

Perioperative Risks
- Atherosclerosis with coronary and peripheral vascular sequelae.
- Pancreatitis sequelae incl hemorrhage, dysregulation of glucose, electrolyte imbalance, dehydration, pseudocysts, and pancreatic exocrine insufficiency. Levels needed to trigger acute pancreatitis varies but is usually above 1000 mg/dL.

Worry About
- Myocardial ischemia and infarction.
- Alteration of medication 2° to fatty acid sequestration of albumin.
- Pseudohyponatremia.

Overview
- Triglycerides are glycerol molecules with three free fatty acid side chains of variable length and saturation. They are the major form of stored and circulating energy.
- The two main sources of plasma triglycerides are exogenous (dietary) and endogenous (liver). The exogenous form is carried in chylomicrons while the endogenous form is carried in very-low-density lipoprotein (VLDL) particles.
- Normal level is below 150 mg/dL, Borderline high is 150–199 mg/dL, high is 200–499 mg/dL and above 500 is very high.
- The autonomic nervous system regulates lipolysis of adipose cells. Increases in sympathetic stimulation can increase levels.

ICD-9-CM Code: 272.1-3

Etiology
- Primary hypertriglyceridemia incl: familial chylomicronemia, primary mixed hyperlipidemia (both having pathologically increased chylomicron levels), familial hypertriglyceridemia (elevated VLDL), familial combined hyperlipoproteinemia (elevated VLDL and LDL), familial dysbeta-lipoproteinemia (elevated triglyceride remnnants, Beta-VLDL).
- 2° hypertriglyceridemia is caused by obesity (esp. visceral), DM type 2, hyperinsulinemia, metabolic syndrome, alcohol consumption, renal disease, pregnancy, hypothyroidism, autoimmune disorders and medications incl: thiazide diuretics, beta-blockers, oral estrogen compounds, retinoids, protease inhibitors, antipsychotics, corticosteroids.

Usual Treatment
- Lifestyle modification such as increasing exercise, wt reduction and dietary changes such as decreasing intake of fat and refined carbohydrates as well as elimination of alcohol.
- Fibrates: Gemfibrozil, bezafibrate and fenofibrate reduce triglyceride levels by 50%.
- Niacin: 45% reduction but can cause flushing, pruritis, and light-headedness.
- Fish oil (Omega-3 fatty acids): Can reduce triglyceride levels by 20%

Monitor
- Standard ASA monitors unless co-morbid conditions dictate otherwise.

General Anesthesia
- Increasing serum triglyceride levels will increase blood-gas partition coefficient and increase volatile solubility.
- Renal function may be diminished, therefore, uo should be closely observed.

Regional Anesthesia
- Neuraxial anesthetic poses theoretical benefit as sympatholysis will result in decreased lipolysis of adipocytes.
- Associated co-morbidities such as obesity and cardiac sequelae may affect choice.

Anticipated Problems/Concerns
- Known complications of CAD.
- Complications regarding comorbidities.
Hypokalemia

Risk
- Defined as plasma K+ <3.5 mEq/L
- Common conditions and/or treatments place pts at increased risk, incl:
  - Those on diuretics (esp. loop and thiazide diuretics) to treat Htn, CHF, etc
  - Those experiencing significant GI fluid loss (e.g., vomiting, diarrhea, or gastric suction)
  - Those with increased serum pH (metabolic or resp alkalosis)

Perioperative Risks
- Increased risk of cardiac dysrhythmias (with greater concern in those with pre-existing heart disease and in setting of acute onset hypokalemia)
- Increased risk of muscle weakness (which incl possible resp muscle weakness and prolonged neuromuscular blockade)
- Increased risk of GI hypomotility

Worry About
- Cardiac dysrhythmias are the most worrisome complication of hypokalemia.
- Many medications regularly used in periop treatment can cause or worsen hypokalemia (e.g., diuretics, antibiotics, β agonists, epinephrine).
- Pts requiring significant/urgent K+ replacement may require central line placement.
- Over-replacement: Any pt requiring K+ replacement may be at risk for hyperkalemia and thus the malignant dysrhythmias associated with hyperkalemia.

Overview
- K+ ions have essential role in maintaining cellular resting membrane potentials and in generating functional activity in muscle cells, neurons, and cardiac tissue.
- Overall, intracellular K+ concentration is ~30 times greater than extracellular K+ concentration; this ratio is maintained by cell membrane Na+/K+-ATPase.
- Decreases in extracellular K+ impair nml gradients required for membrane potential/action potential transmission.
- Acute/rapid decreases in serum K+ concentration create more concerning derangements in cellular membrane potential physiology than chronic or slowly developing decreases in K+.

ICD-9-CM Code: 276.8

Etiology
- Inadequate K+ intake: Seen in eating disorders, inability to eat, “tea & toast” diet, alcoholism, and those receiving K+-poor TPN.
- Increased K+ excretion
  - Renal losses: Mineralocorticoid excess (1° disorder), Cushing disease, congenital adrenal hyperplasia, hyperreninism, congenital renal disorders (Bartter/Gitelman/Liddle syndromes), medication-induced (loop and thiazide diuretics, carbonic anhydrase inhibitors, amphotericin B, some penicillins, gentamicin)

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<tbody>
<tr>
<td>NERVOUS</td>
<td>Muscle weakness</td>
<td>↓ Mobility, falls, ↓ ADL</td>
<td>↓ Muscle strength</td>
<td>TOF intraop</td>
</tr>
<tr>
<td></td>
<td>Cramping/myalgia</td>
<td>c/o muscle pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESPIR</td>
<td>Respir muscle failure</td>
<td>SOB, hypoventilation, ventilator dependence</td>
<td>Poor insp effort, low TV</td>
<td>ABG, NIF</td>
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<tr>
<td>CV</td>
<td>Dysrhythmias</td>
<td>c/o palpitations, syncope, cardiac arrest</td>
<td>Refractory shock, hypotension</td>
<td>EKG</td>
</tr>
<tr>
<td></td>
<td>Vaso motor instability</td>
<td>Syncope, falls, disorientation</td>
<td></td>
<td></td>
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<tr>
<td>GI</td>
<td>↓ GI motility</td>
<td>Constipation, abd pain</td>
<td>Loss of bowel sounds, abd tenderness and distention</td>
<td>KUB</td>
</tr>
<tr>
<td>RENAL</td>
<td>Poluria</td>
<td>Frequent urination</td>
<td></td>
<td>Urine ammonia</td>
</tr>
<tr>
<td></td>
<td>Polydipsia</td>
<td>Frequent drinking</td>
<td></td>
<td>Urine sodium</td>
</tr>
<tr>
<td></td>
<td>↑ Renal ammonia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Edema and sodium retention</td>
<td></td>
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</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Obtain serum K+ concentration preop if pt presents with risk factors for hypokalemia
- Attempt to identify and/or address the etiology of hypokalemia
- For elective cases, replete serum K+ concentration to ≥2.6 before going to OR. Discuss concerns/implications with pt/family/surgical team.
- Have ACLS meds on hand, and transport with cardiac monitoring.

Monitoring
- EKG/continuous cardiac monitoring (watch for T wave flattening, U waves, PVC, VT/VF)
- BP cuff or arterial line (watch for hypotension related to vasomotor insufficiency)
- Periodic ABG and electrolyte panels as needed (watch for pH and K+ trend)
- Twitch monitor (watch for prolonged neuromuscular blockade)

Maintenance
- Judicious use of medications associated with causing or exacerbating hypokalemia
- Control glucose and fluid volume
- Control ventilation to avoid hyperventilation and resp alkalosis

Usual Treatment
- Identify and attempt to correct the underlying precipitating factors causing the hypokalemia (e.g., adjust diet intake, review/reconsider medications, lower pH of pts with alkalosis by treating 1° disorder)
- K+ repletion: It is reported that each 10 mEq of K+ given will raise serum K+ by 0.1 mEq/L.
- Oral K+: Can use K+ paired with gluconate, phosphate, chlorite, or citrate, with delivery via tablet or solution
- IV K+: Most commonly as K+ chloride. Careful repletion required via programmable infusion pump to avoid hyperkalemic complications — pts receiving >10–20 mEq/hr should have cardiac monitoring in place. Peripheral IV administration can cause burning sensation and vascular epithelial damage; consider placement of central line.

Anticipated Problems/Concerns
- Pts with symptomatic hypokalemia (esp. with cardiac symptoms) that are not well controlled after initial treatments may need elective surgical procedures delayed
- Cardiac dysrhythmias are greatest concern in hypokalemia, as these can be lethal. Risk is greatest when hypokalemia is acute and serum K+ <3.0.
- Preop problems: EKG changes and volume status (related to diuretics or polydipsia)
- Intraop problems: Persistent hypotension after induction (related to refractory vasomotor response to catecholamines), prolonged neuromuscular blockade, resp muscle weakness
Hypomagnesemia

James M. Feld

**Risk**
- General population (up to 25%) may be deficient because of poor eating habits.
- 12% of all hospitalized pts as well as 44–60% of all pts admitted to medical/surgical and pediatric ICUs were hypomagnesemic.

**Associated With**
- Poor nutrition
- Gastrointestinal losses
- Diarrhea
- Malabsorption (steatorrhea, bowel resection, intestinal fistulas)
- Acute pancreatitis
- Renal losses
- Diuretics (esp. loop diuretics)
- Antimicrobials (e.g., aminoglycosides, amphotericin B)
- Chemotherapeutics (e.g., cisplatin, foscarinet, cyclosporine)
- Phosphorus depletion
- Metabolic acidosis
- Alcohol abuse
- Miscellaneous
- Diabetes mellitus
- Prolonged IV therapy
- Massive blood transfusions
- Digitalis

**Perioperative Implications**

**Perioperative Risks**
- Arrhythmias (atrial and ventricular, esp. torsades)
- Worsening cardiac ischemia and CHF
- Increased susceptibility to seizures, bronchoconstriction, and vasospasm
- Inability to correct low K⁺ and Ca²⁺ levels
- Resistance to vasodilators
- Insulin resistance in the diabetic pt

**Worry About**
- Weakness, lethargy, paresthesias, muscle spasms
- Seizures (esp. in preeclampsia)
- Arrhythmias (esp. torsades)
- Hypokalemia and hypocalcemia (may be difficult to treat if hypomagnesemia is the underlying cause)
- Coronary artery spasm and CHF
- During treatment of hypomagnesemia: Burning at IV site, overall sense of warmth and flushing, transient and mild hypotension may occur if MgSO₄ is given too fast

**Overview**
- Hypomagnesemia is defined as plasma Mg²⁺ <1.7 mg/dL. Most symptomatic pts have levels <1 mg/dL.
- Mg²⁺ levels are not routinely checked in screening tests. Hypomagnesemia should be suspected esp. in chronic diarrhea, alcoholism, malnutrition, long-term hospitalization, and hypoalbuminemia.
- Mg²⁺ is primarily an intracellular ion. Plasma levels may not reflect the true magnitude of deficiency. Intracellular shift may occur with the administration of insulin and thyroid hormone.
- Normomagnesemic Mg²⁺ depletion has been described; if clinical suspicion of hypomagnesemia present, Mg²⁺ should be administered, even with normal plasma levels.
- Differentiation of renal from non-renal causes is helpful to investigate etiology. In a 24-hr urine sample, Mg²⁺ loss >3-4 mEq/d supports renal etiology.
- Alternatively, a fractional excretion of Mg²⁺ can be calculated in a spot-urine sample:
  \[ \text{FE}_{\text{Mg}} = \frac{\text{U}_{\text{Mg}} \times \text{P}_{\text{Cr}}}{\text{U}_{\text{Cr}} \times \text{P}_{\text{Mg}}} \times 100 \]
  where \( \text{U}_{\text{Mg}}/\text{U}_{\text{Cr}} \) and \( \text{P}_{\text{Mg}}/\text{P}_{\text{Cr}} \) denote urinary and plasma concentrations of Mg²⁺ and creatinine.
- Usually, \( \text{FE}_{\text{Mg}} > 2\% \) indicates renal etiology. In a study of 74 hypomagnesemic pts, ranges of \( \text{FE}_{\text{Mg}} \) in non-renal and renal causes were 0.5–2.7% and 4–48%, respectively.

ICD9-CM: 275.2 (Disorders of magnesium metabolism)

**System** | **Effect** | **Assessment by Hx** | **PE** | **Test** |
---|---|---|---|---|
CNS | Seizures, cerebral vasospasm (after SAH) | Lethargy, SAH (vasospasm) | Altered mental status | Plasma Mg²⁺, TCD, cerebral angiogram |
CARDIO | Arrhythmias (torsades), wide QRS, tall T waves (flattens in severe depletion), CHF (impaired diastolic relaxation) | Tachyarrhythmia, Htm, dyspnea | | EKG, plasma Mg²⁺, BNP, ECHO |
MS | Hypocalcemia (decreased secretion and resistance to PTH) | Weakness, tetany | Chvostek and Trousseau signs | Plasma Ca²⁺, Mg²⁺ |
ENDO | Insulin resistance, may affect lipid profile | Diabetes (type 1 and 2) | Hyperlipidemia | Glucose, plasma Mg²⁺, HDL, triglycerides |
RESP | Bronchospasm | Asthma | Wheezing | Plasma Mg²⁺ |
RENAL | Hypokalemia (K⁺ loss from loop of Henle) | Alcohol abuse, nephrotoxins (antibiotics, chemo), diuretics | | Creatinine, BUN, Plasma K⁺, Mg²⁺ |


**Perioperative Preparation**
- Check serum Mg²⁺ level (<1.7 mg/dL is hypomagnesemia)
- Obtain 12-lead EKG
- Start replacing Mg²⁺ (e.g., 2 gm over 20 min; faster replacement safe but may cause burning at the IV site)

**Monitoring**
- EKG
- Plasma Mg²⁺ levels (normal range 1.7–2.5 mg/dL)
- TOF monitoring (replacing Mg²⁺ potentiates NMB agents)
- Consider BIS (or other depth of anesthesia) monitoring, since replacing Mg²⁺ may alter anesthesia requirement

**Induction**
- Mg²⁺ IV bolus during induction is safe (e.g., 2–4 gm IV bolus)
- May cause mild and transient drop in BP
- Replacing Mg²⁺ minimizes changes in heart rate and BP during intubation
- Hypomagnesemia may cause or exacerbate bronchospasm

**ASSESSMENT POINTS**

**System** | **Effect** | **Assessment by Hx** | **PE** | **Test** |
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CNS | Seizures, cerebral vasospasm (after SAH) | Lethargy, SAH (vasospasm) | Altered mental status | Plasma Mg²⁺, TCD, cerebral angiogram |
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RESP | Bronchospasm | Asthma | Wheezing | Plasma Mg²⁺ |
RENAL | Hypokalemia (K⁺ loss from loop of Henle) | Alcohol abuse, nephrotoxins (antibiotics, chemo), diuretics | | Creatinine, BUN, Plasma K⁺, Mg²⁺ |

**Perioperative Uses**
- Besides correction of hypomagnesemia, Mg²⁺ replacement can be used.
- CV: Myocardial protection, decreases CHF, improves contractility, diastolic relaxation, attenuates or prevents tachycardic arrhythmias, minimizes changes in heart rate and BP during intubation
- Neurological: May improve memory, decrease cerebral vasospasm, limit any neuro insult to brain or spinal cord
- Endocrine: Attenuates insulin resistance, helps in hemodynamic control in pheochromocytomas, may increase HDL levels
- OB: Widespread use in treatment of preeclampsia, decreases risk of cerebral palsy in preterm infants
- Pulm: Bronchodilation esp. in severe asthmatic
- Anesthesia: Decrease need for inhalation agent to maintain same BIS level
- Pain: Decreases need for postop opioids through its blockade of NMDA receptors
- MS: Relaxes muscle rigidity and decreases autonomic dysfunction in tetanus
- Intoxication/recreational drugs: Helpful to treat CV problems associated with cocaine and methamphetamines
Maintenance
- Replacing Mg\(^{2+}\) decreases anesthetic requirement (i.e., maintain same BIS level with less anesthetic)
- Hypomagnesemia may decrease cardiac contractility and impair diastolic relaxation in pts with CHF; improved by replacing Mg\(^{2+}\).
- Replacing Mg\(^{2+}\) attenuates or prevents tachyarrhythmias.
- Insulin resistance may occur in the hypomagnesemic pt.

Emergence
- Replacing Mg\(^{2+}\) attenuates shivering.
- Replacing Mg\(^{2+}\) maintains hemodynamic stability.
- Titrate NMB agents and reverse residual NMB, esp. if Mg\(^{2+}\) was replaced intraop.

Postoperative Period
- Hypomagnesemia may increase analgesic (i.e., opioid) requirement; Mg\(^{2+}\) replacement leads to decreased opioid consumption.
- Hypomagnesemia may worsen bronchospasm in asthmatic pts.
- Increased catecholamine levels may exacerbate tachyarrhythmias in the hypomagnesemic pt.

Anticipated Problems/Concerns
- Although Mg\(^{2+}\) replacement is usually well tolerated, potential problems with overdose include the following:
  * Levels above 8–10 mg/dL may cause diaphragmatic weakness and above 10–12 mg/dL may cause widening of QRS and conduction blocks. These levels are rarely reached with the above-recommended doses and in the absence of decreased GFR.
  * Potentiation of neuromuscular blockade
Hyponatremia

Risk
- Premenopausal women, esp. those undergoing procedures associated with rapid irrigant absorption, at particularly high risk.
- Conditions associated with syndrome of inappropriate antidiuretic hormone (SIADH), or adrenocortical insufficiency (Addison disease).
- Pts with liver, heart, or renal failure
- Hyponatremia esp. common in elderly pts and associated with increased morbidity and mortality
- Up to 25% of men undergoing TURP
- Infants and/or children receiving multiple tap H2O enemas

Perioperative Implications
- Risk of CV collapse with adrenocortical insufficiency and inability to cope with stress of surgery
- Iatrogenic dilution in TURP and endoscopic gyn procedures associated with CNS, cardiopulmonary, and skeletal muscle abn
- Increased ADH secretion extremely common periop and may cause further decrease serum Na+
- Isotonic saline (0.9%) will result in free H2O gain and decrease in serum Na+ in presence of SIADH.

Worry About
- Acute iatrogenic hyponatremia associated with TURP syndrome
- Development of cerebral and/or pulm edema
- Cardiac dysrhythmia
- Visual or motor disturbances attributed to ammonia intoxication with glycine irrigation

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Dysrhythmias</td>
<td>Palpitations</td>
<td>S3, rales</td>
<td>Oscillation, ECG (wide QRS, ↑ ST, VT/VF)</td>
</tr>
<tr>
<td></td>
<td>CHF, hypervolemia</td>
<td>DOE, orthopnea</td>
<td>Orthostatic hypotension, ↓ CVP, tachycardia</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>Hypovolemia</td>
<td>Lightheadedness, weakness</td>
<td>BP, CVP</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>DOE</td>
<td>S3, rales</td>
<td>CXR</td>
</tr>
<tr>
<td>CNS</td>
<td>Confusion, restlessness, gait disturbance, lethargy, seizures, visual disturbances, obtundation, coma</td>
<td></td>
<td>Urine Na+, serum and urine osmolality</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Weakness, cramps</td>
<td>Weakness, cramps</td>
<td>Reflexes</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Free H2O retention</td>
<td>Salt wasting</td>
<td></td>
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</tr>
</tbody>
</table>


Preinduction
- Ensure medical optimization of co-morbid diseases (hyponatremia greater risk with increasing severity of disease: ASA III and IV)
- Caution with sedatives
- Preop lyres in high risk procedures
- Consider regional in TURP to facilitate monitoring of mental status
- Increased ADH and volume changes associated with surgical trauma likely to decrease Na+ further
- Identify irrigating solution and prepare for irrigant-specific side effects

Monitoring
- TURP
  - Metal status with regional technique
  - Consider EGG with GA
  - Consider invasive monitoring (CVP/PA cath/TEE) with development of TURP syndrome and CHF in elderly pts
  - Visual acuity with glycine irrigation
  - Hyponatremic pts undergoing therapy to correct serum Na+
    - Serum Na+
    - Mental status
    - ECG

General Anesthesia
- Isotonic fluids for volume resuscitation
- Ensure adequate depth of anesthesia as pain and/or stress associated with ADH release

Regional Anesthesia
- Neuroaxial blockade technique of choice for TURP

Postoperative Period
- Ensure adequate pain control
- Monitor serum Na+
- Initiate appropriate therapy in symptomatic or severely hyponatremic pts
- Avoid too rapid correction and associated demyelination syndrome
- Restore blood and/or volume loss if necessary

Anticipated Problems/Concerns
- TURP procedures of long duration and with significant bleeding or increased hydrostatic pressure of irrigant predictive for large amounts of irrigation fluid absorption. (Increased vigilance required)
- Prepare for emergency airway protection if resp distress, seizures, obtundation

Usual Treatment
- Free H2O restriction
- IV or oral sodium chloride (incl hypertonic saline with caution in symptomatic pts)
- Loop diuretics (limits extracellular volume expansion with 3% saline)
- Vasopressin antagonists
- Risk of osmotic demyelination syndrome with overly aggressive correction of plasma Na+ concentration
- Goal: Raise plasma sodium ≤115 mEq/L in first 24 hr; ≤18 mEq/L in first 48 hr
- If needed, dose of sodium required to correct a deficit may be calculated using the following formula: Dose (mEq) = (Weight [kg] × (140 − [Na]) [mEq/L]) × 0.6
- The optimal rate of correction appears to be 0.6 to 1 mmol/L/hr until the Na+ concentration is 125 mEq/L, and then correction proceeds at a slower rate. One half the deficit can be adminstered over the first 8 hr and the next half over 1 to 3 d if symptoms remit.
- Targeted therapy for end-organ dysfunction (diuresis, vasodilation in CHF; airway protection with mental status changes)
- Hypovolemic pts require careful isotonic fluid resuscitation to maintain hemodynamic stability
• Attempt to optimize pts with cardiopulmonary disease preop
• Isotonic saline in SIADH will result in increased free H$_2$O and worsening hyponatremia (close monitoring of serum Na$^+$ required with 0.9% saline administration necessary if unclear diagnosis)
• Premenstrual women at greater risk of both symptomatic hyponatremia and osmotic demyelination with Na$^+$ correction
• Chronic hyponatremia tolerated better than acute hyponatremia
Hypophosphatemia

Risk
- Incidence: 1% of population, 5–20% of hospitalized pts

Perioperative Risks
- Acute resp or cardiac failure, generalized weakness, confusion, or seizures

Worry About
- Periop resp or cardiac failure
- Too rapid correction can cause hypocalcemia or Ca++ deposition in tissues

Overview
- Total body phosphorus is distributed 90% in bone, 10% intracellular and < 1% in the extracellular fluid.
- Normal ionized phosphorus (Pi) is 2.7–4.5 mg/dL. May fall by 30% after administration of carbohydrates/insulin. Higher in childhood and postmenopausal women. Lower in AM than PM.
- Serum concentration does not correlate closely to body stores.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Depressed ATP, impaired response to Norepinephrine/angiotensin</td>
<td>Heart failure</td>
</tr>
<tr>
<td>Blood</td>
<td>Impaired phagocytic, migration and bacteriocidal activity</td>
<td>Sepsis</td>
</tr>
<tr>
<td>WBC</td>
<td>Reduced RBC 2,3 DPG</td>
<td>Thrombocytopenia, impaired clot retraction</td>
</tr>
<tr>
<td>Platelets</td>
<td></td>
<td>Increased Hgb O₂ affinity</td>
</tr>
<tr>
<td>RBC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Neurologic dysfunction</td>
<td>Seizures, coma, hypereflexia, paresthesia, dysarthria</td>
</tr>
<tr>
<td>MS</td>
<td>Resp failure, motor weakness</td>
<td>Proximal &gt; distal, rhabdomyolysis, myoglobinuria</td>
</tr>
</tbody>
</table>


Perioperative Implications
- Correction of severe hypophosphatemia should be done slowly over several hours to days to prevent severe hypocalcemia and vascular and interstitial Ca++ precipitation.
- Consider hypophosphatemia in the pt who is difficult to wean off the ventilator as this might be the cause.

Usual Treatment
- Prefer oral over parenteral because of risk of resultant hypocalcemia or calcification of tissues. Suggested dose of KPHOS 2–5 mg/kg/d.
- If parenteral therapy is required, administration of 10–45 mmol of IV Na⁺ or K⁺ phosphate over 6–12 hr. Important to monitor Ca++, K⁺, and Mg²⁺ levels.

- Decreased absorption and/or intake. Malnutrition, malabsorption syndromes. Crohn’s disease, celiac disease, inadequate replacement in TPN, hemodialysis, Mg²⁺ and aluminum antacids, sulfaftate, Vitamin D deficiency
- Increased losses: Rapid volume resuscitation, steroids, pancreatitis, burns, alcoholism, dialysis, hyperparathyroidism, diuretics
- Redistribution: Shift from serum into cells-hyperglycemia, glucose infusion, hormonal effects-catecholamines, insulin, glucagon, calcitonin
- Resp alkalosis, leukemic blast cell crisis

ICD-9-CM Code: 275.3

Etiology
- Decreased intake, increased loss, redistribution, occasionally genetic

- Normal requirements 1 mmol/kg/d
- 1st absorption of Pi is in the duodenum and the jejunum, stimulated by vitamin D
- Kidney: Primary filtration in the kidney with 1st reabsorption in the proximal tubules with only 10% reabsorption in the distal tubules. Regulated by PTH, cortisol, high dietary intake, and calcitonin. Increased Pi excretion with volume expansion.
- Functions: Phosphates provide the 1st energy bond in ATP, creatine phosphate. Severe Pi depletion can cause cellular energy depletion, lack of cAMP, also important for cellular structures as phospholipids, nucleic acids, and cellular membranes. As part of 2,3 diphosphoglycerate, phosphates promote O₂ release from hemoglobin.

iCD-9-Cm Code: 275.3
**Hypopituitarism**

**Risk**
- Most frequent cause: Pituitary tumor (61%).
- The incidence is 45.5 people out of 100,000.
- 30% of pituitary macroadenomas (>10 mm) cause one or more hormone deficiencies
- 50% of pts after pituitary radiation therapy by 4.2 y have hypopituitarism.

**Perioperative Risks**
- If adequate hormone replacement, surgery presents no increased risk
- If due to secreting tumor, then there is an increased risk of Cushing disease, acromegaly, SIADH, or hyperthyroidism

**Worry About**
- Concerns with manifestations of disease process (Cushing disease, hypercortisolism 2° to an adrenocorticotropic hormone-secreting adenoma), acromegaly (2° to a growth hormone-secreting adenoma), hyperthyroidism in the setting of thyrotrpic adenomas.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Mandibular and oral soft tissue hyperplasia in acromegalis</td>
<td>Airway exam</td>
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<tr>
<td></td>
<td>Catecholamine resistance</td>
<td>Check ring size</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Orthostatic hypotension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Hypoaldosteronism</td>
<td>Anorexia, N/V, wt loss, abdominal pain</td>
<td>Hyperkalemia, hyponatremia, hypocalcemia, hypovolemia</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Decreased ACTH</td>
<td>Fatigue, fever, stress-induced hypotension, and hyponatremia</td>
<td>Fever, hypotension, wt loss, mental status</td>
<td>am cortisol level, rapid ACTH stimulation test, insulin tolerance test</td>
</tr>
<tr>
<td></td>
<td>Decreased LH, FSH</td>
<td>Decreased libido and sexual function, amenorrhea</td>
<td>Regression of 2° sexual characteristics</td>
<td>FSH, LH serum levels, serum estradiol and testosterone</td>
</tr>
<tr>
<td></td>
<td>Decreased GH</td>
<td>Fatigue</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Increased prolactin</td>
<td>Wt gain, cold intolerance, depression, constipation, hair loss</td>
<td>Myxedema, hyporeflexia</td>
<td>TSH, T4;</td>
</tr>
<tr>
<td></td>
<td>Increased GH in acromegalis</td>
<td>Lactation, amenorrhea</td>
<td>Galactorrhea</td>
<td>Serum prolactin</td>
</tr>
<tr>
<td>MS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>↑ Vasopressin</td>
<td>Excessive thirst</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Vasopressin</td>
<td>Increased UO and thirst</td>
<td>Hypovolemia</td>
<td>Dilute urine</td>
</tr>
</tbody>
</table>

**Monitoring**
- Consider arterial line if severe CV compromise, central venous pressures if indicated by inadequate preop correction of fluid status
- Frequent monitoring of electrolytes if hypo or hypernatremia is not corrected preop
- Consider glucose monitoring

**Airway**
- Acromegals with normal airway exam may be difficult to intubate. Have LMA, fiberoptic, or glidescope available.

**Induction**
- Little risk of increased ICP with pituitary adenomas
- No special technique if hormone replacement and volume status are adequate

**ICD-9-CM Codes:** 253.2 or 253.7 (if due to radiotherapy, post ablative, post hypophysectomy, or secondary to hormone therapy)

**Etiology**
- 61% 2° to tumors of the pituitary gland
- 9% due to other types of lesions
- 19% due to other causes (radiation, hemorrhage, infarct, head trauma, infiltrative diseases)
- 11% no cause could be identified

**Usual Treatment**
- Surgical resection of adenoma with appropriate hormonal replacement therapy for ACTH: Prednisone or cortisone PO; for TSH: Thyroxine PO; for LH and FSH: Women: estrogen and progesterone PO; Men: testosterone esters IM; for ADH: Desmopressin intranasal

Postoperative Period

- Polyuria and polydipsia with dilute urine may indicate development of DI
- Postop hypopituitarism may require steroid replacement therapy

Anticipated Problems/Concerns

- Acromegalic should be treated as a difficult airway
- Pt with GH deficiency may manifest hypoglycemia
- Electrolyte abn (K⁺, Na⁺, Ca++) and possible hypovolemia resulting predisposing to arrhythmias and CV instability
## Hypothermia, Mild

### Overview
- Core temp normally protected by responses and sweating, vasoconstriction, shivering.
- *Typical doses of general anesthetics have little effect on the sweating threshold, but decrease vasoconstriction and shivering thresholds 2–4°C, thus increasing the range of temp not triggering protective responses 10-fold from −0.4°C to −4°C.
- Regional anesthesia inhibits thermoregulatory control by preventing peripheral responses (such as vasoconstriction) and centrally by reducing afferent input.

### ICD-9-CM Code: 991.6 (Accidental)

### Etiology
- Initial 0.5–1.3°C decrease in core temp from core-to-peripheral redistribution of body heat.
- Subsequently, slow, linear decrease in core temp from heat loss exceeding heat production.

### Usual Treatment
- Forced-air is the most effective generally available warming method, typically increasing mean body temp 1°C/hr.
- 1 L of crystalloid at 20°C or 1 U of blood at 4°C decreases mean body temp −0.25°C in adults. Fluid warming should be restricted to pts given large volumes of fluid (i.e., ≥2 L/hr).
- Passive insulation (e.g., surgical drapes, cotton blankets) decreases heat loss only 30% which is usually insufficient to maintain periop normothermia.
- Circulating-H₂O mattresses less effective and may cause burns. Airway heating and humidification is ineffective.

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Dx</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Ischemia protection</td>
<td>None</td>
<td>Induce and maintain hypothermia</td>
</tr>
<tr>
<td></td>
<td>Thermal discomfort</td>
<td>Visual analogue scale</td>
<td>Active cutaneous warming</td>
</tr>
<tr>
<td>CARDIAC</td>
<td>Myocardial ischemia (usually postop)</td>
<td>ST-segment depression</td>
<td>Active cutaneous warming</td>
</tr>
<tr>
<td></td>
<td>Arteriovenous shunt constriction; ↑ BP, ↓ HR</td>
<td>Associated with sweating</td>
<td>Active or passive cooling</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fingers feel cold, 10 mmHg increase in mean arterial pressure</td>
<td>Active cutaneous warming</td>
</tr>
<tr>
<td>VASC</td>
<td>Pre-capillary dilation; reduced SVR</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS (shivering)</td>
<td>2–3-fold ↑ metabolic rate</td>
<td>Visual inspection</td>
<td>Prevent hypothermia</td>
</tr>
<tr>
<td></td>
<td>Pt discomfort</td>
<td>O₂ consumption</td>
<td>Meperidine 10–25 mg IV</td>
</tr>
<tr>
<td></td>
<td>Interference with monitoring</td>
<td></td>
<td>Clonidine 75 µg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Active cutaneous warming</td>
</tr>
<tr>
<td>IMMUNO</td>
<td>Incidence of infections increases 2–3-fold</td>
<td>Clinical infections</td>
<td>Prevent hypothermia</td>
</tr>
<tr>
<td>HEME</td>
<td>10% ↑ /°C in blood loss</td>
<td>Bleeding time</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PT/PTT falsely normal</td>
<td>Prevent hypothermia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Defect probably not reversed by FFP and platelet transfusions</td>
</tr>
<tr>
<td>METAB</td>
<td>MAC decreases −5%/°C</td>
<td>Monitor drug action (rather than dose)</td>
<td>Titrmate drug administration to desired endpoint</td>
</tr>
<tr>
<td>(increased drug action)</td>
<td></td>
<td></td>
<td>Monitor twitch depression</td>
</tr>
</tbody>
</table>

### Key Reference

### Perioperative Implications

#### Preoperative Preparation
- Active prewarming for 30–60 min helps prevent redistribution hypothermia.

#### Monitoring
- Four core temp sites are accurate: Pulm, artery, distal esophagus, tympanic membrane, nasopharynx.
- Four additional sites suitable except during cardiopulmonary bypass: Mouth, axilla, rectum, bladder.
- The best site for postop temp monitoring is the mouth.

#### Intraoperative
- Maintain normothermia (core temp ≥36°C) unless otherwise indicated.
- Sufficient passive or active reduction of heat loss will prevent hypothermia, however, active warming is usually required.
- Once triggered, thermoregulatory vasoconstriction effective in preventing further core hypothermia.
- Current standards (Surgical Care Improvement Project and Physicians Quality Reporting Initiative) require that nearly all surgical pts have regional or general anesthesia lasting ≥1 hr be normothermic near the end of anesthesia and/or that active over-body warming be used.

#### Postoperative
- Hypothermic pts should be rewarmed with forced-air.
- Shivering and thermal discomfort can be specifically treated.
- Postop warming not a routine substitute for maintaining intraop normothermia.

#### Anticipated Problems/Concerns
- Thermal discomfort has been proven to cause numerous life-threatening complications and should be actively prevented unless therapeutic hypothermia is specifically indicated.

---

**Risk**
- Greater in infants and children
- Greater in longer, larger operations
- Similar in regional and GA

**Perioperative Risks**
- Myocardial ischemia
- Surgical wound infections
- Coagulopathy
- Reduced drug metabolism
- Prolonged recovery and hospitalization
- Shivering and thermal discomfort

**Benefits**
- Improved neurologic recovery after cardiac arrest
- Improved neurologic recovery in asphyxiated neonates
- Decreases triggering and severity of malignant hyperthermia

**Assessment Points**

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Hypothyroidism

**Risk**
- Subclinical hypothyroidism may be present in as many as 8–10% of adult women and 1–2% of adult men; about 3% of adults receive chronic thyroid replacement.

**Perioperative Risks**
- Potential increased risk for hypothermia, hypotension, cardiac failure, and GI dysfunction
- Periop mortality rate not increased unless overtly hypothyroid
- During pregnancy maternal hypothyroidism associates with adverse obstetric outcomes and developmental delays in the offspring

**Worry About**
- Predisposition to hypothermia
- Neuromuscular weakness may impair weaning from mechanical ventilation

**Overview**
- A common condition, particularly in adult women
- Elevated thyrotropin (TSH) concentration in blood is hallmark laboratory finding and may be present months to years before decreased T4 in blood is hallmark laboratory finding and may be present months to years before decreased TSH concentration.
- Adequacy of T4 replacement defined by TSH concentrations in the low-normal range
- Total and free thyroxine (T4) (and usually triiodothyronine [T3]) concentrations usually reduced
- Symptomatic pts with TSH >10 mU/L should receive maintenance thyroid replacement. (levothyroxine 1.6 μg/kg daily)
- Pts presenting with severe, untreated hypothyroidism or myxedema coma may also demonstrate hypothyria, hypovolemia, hyponatremia, hypotension, heart failure, bowel obstruction, and hypoglycemia

**Assessment by Hx**

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<th>Test</th>
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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Puffiness below eyes, enlarged tongue</td>
<td>Snoring</td>
<td>Enlarged tongue</td>
<td>TSH, T4 (or T3) concentrations</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Bradycardia, ↓ BP, heart failure</td>
<td>Palpitations, myocardial ischemia, arrhythmias, peripheral edema</td>
<td>Bradycardia, tachycardia</td>
<td>TSH, T4 (or T3) concentrations, ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoventilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Ileus, wt gain</td>
<td>Constipation, ascites, ↓ Bowel sounds</td>
<td></td>
<td>TSH, T4 (or T3) concentrations</td>
</tr>
<tr>
<td>RENAL</td>
<td>Decreased free water clearance</td>
<td>Fluid retention, edema</td>
<td>Edema</td>
<td>TSH, T4 (or T3) concentrations, serum Na+ concentration</td>
</tr>
<tr>
<td>CNS</td>
<td>Ophthalmoplegia, depression, muscular weakness, cold intolerance</td>
<td>Lethargy, weakness, mental slowing, Decreased deep tendon reflexes, impaired mental status examination</td>
<td></td>
<td>TSH, T4 (or T3) concentrations</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Chronic thyroid replacement to maintain euthyroid state

**Monitoring**
- Temp
- Other monitors as indicated by surgery

**Airway**
- Rarely incidence of macroglossia

**Maintenance**
- No effect of hypothyroidism on MAC for inhaled anesthetics

**Anticipated Problems/Concerns**
- Only those pts who have been inadequately treated with T4 carry risks; those who chronically receive an appropriate dose of T4 probably have (at most) minimally increased risks compared with other pts.
- Inadequately treated hypothyroidism can lead to lethargy and fatigue, wt gain, dementia, heart failure, resp insufficiency, fluid retention and edema, hyponatremia, clotting abn, and generalized weakness.

**ICD-9-CM Code**: 244.9

**Etiology**
- Hypothyroidism (decreased thyroid hormone secretion) most often results from primary disease of thyroid gland (most commonly autoimmune thyroiditis). Less frequently it results from disorders of the pituitary gland or hypothalamus.
- Previous treatment for hyperthyroidism and previous total thyroidectomy are also relatively common causes of hypothyroidism.
- Pts with critical illness may have reduced total T4 and reduced total and free T4 despite normal TSH concentrations (euthyroid sick syndrome) but usually do not require thyroid hormone replacement.
- Primary TSH deficiency may result from pituitary tumors and cysts or their treatment (either surgery or radiation), pituitary infiltration, necrosis, or infarction; 2° TSH deficiency may result from congenital deficiency of thyrotropin-releasing hormone (TRH), radiation therapy, infections, or tumors or cysts impinging on the hypothalamic-pituitary portal circulation.

**Usual Treatment**
- Maintenance outpatient therapy for adults consists of oral thyroxine 0.1–0.2 mg (1–3 μg/kg) daily.
- There may be a delay of up to 4 wk for TSH to stabilize after T4 dosage adjustment.
- Long T4 half-life of T3 (about a week) permits oral T4 to be withheld safely for several NPO days.
- Chronic rifampin, carbamazepine, phenobarbital, and phenytoin, and increase T4 dosage requirements by increasing metabolism or clearance of T4.
- Pts with known or occult CAD may have increased symptoms unless T4 replacement is initiated at a reduced dose and only cautiously increased.
- Myxedema coma may require use of IV T3 (liothyronine) 0.15–0.3 μg/kg every 6 hr and IV hydrocortisone 0.5–1 mg/kg every 8 hr to cover for possible hypothyroid-impaired adrenal response to stress.
- IV liothyronine may also be indicated in other circumstances when peripheral conversion of T4 to T3 is impaired (e.g., hypothermic cardiopulmonary bypass).
Hypoxemia

Risk
- All pts undergoing anesthesia and surgery (7–35% in large series have PaO₂ <60 mmHg in OR or PACU)
- Pts with pre-existing pulm disease

Perioperative Risks
- Hypoxemia may lead to hypoxia and eventual severe neurologic/cardiac sequelae or death

Worry About
- Inadequate delivery of O₂ to blood—greatest concern to the anesthesiologist is inadequate delivery of O₂ to pt.
- Concern that inadequate delivery of O₂ to blood will lead to inadequate delivery of O₂ to tissues
- Misinterpretation of clinical manifestations of hypoxemia

Overview
- Hypoxemia:—Denotes low PaO₂ in blood (versus hypoxia, which denotes inadequate delivery of O₂ to tissues)
- Hypoxemia defined as resting Po₂ >2 SD below normal for age and FIO₂, SaO₂ <90%, PaO₂ <60 mmHg on room air, and a fall in SaO₂ >5%
- Multiple clues in vital signs and pt symptoms that should be assumed to be due to hypoxemia

ICD-9-CM Code: 799.0 (Hypoxia)

Etiology
- Decreased FIO₂ failure to provide adequate inspired O₂ (e.g., O₂ supply failure, gas machine disconnect, airway disconnect, pts at higher altitude)
- Inadequate alveolar ventilation or alveolar hypoventilation: Venous admixture accounts for majority of causes

Usual Treatment
- Determine cause of decreased O₂ delivery and treat
- Increase FIO₂: This will help in all situations of hypoxemia except those due to R to L shunts

Perioperative Implications

Monitoring
- Routine: Pulse oximetry is mandatory
- ABGs

Airway
- Must ensure patency and intact circuit at all times

Maintenance
- Adequate FIO₂ and alveolar ventilation

Anticipate Problems/Concerns
- Must have a high index of suspicion whenever SaO₂ decreases or any of the clinical subjective or objective signs and symptoms are present. Always assume the decreased SaO₂ does not reflect a problem with the pulse oximeter but signifies a real problem. Stable vital signs may not fully eliminate significant arterial hypoxemia.

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Sympathetic stimulation</td>
<td>Htn</td>
<td>Tachycardia</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Cyanosis</td>
<td>Atelectasis lung collapse</td>
<td>Tachypnea</td>
<td>SaO₂, ABG Decreased PaO₂ CXR</td>
</tr>
<tr>
<td>CNS</td>
<td>Altered mental status</td>
<td>Anxiety</td>
<td>Confusion seizures</td>
<td></td>
</tr>
</tbody>
</table>

IgA Deficiency

**Risk**
- The most common immunodeficiency disorder
- Incidence has been estimated to be 1/100 to 1/1000
- More prevalent among European descendants
- Most pts are clinically normal
- Increased risk of allergies and anaphylaxis
- Increased risk of malignancies

**Perioperative Risks**
- Increased incidence of pulm complications, atopic disorders, and postop infections
- Recurrent sinopulmonary infections leading to decreased pulm reserve
- Associated autoimmune disorders (e.g., lupus, DiGeorge syndrome)

**Worry About**
- Associated autoimmune disorders (e.g., lupus, DiGeorge syndrome)
- Anaphylactic reactions from transfusion of blood products containing IgA

**Overview**
- An immunodeficiency syndrome with increased susceptibility to nosocomial infection
- Cell-mediated immunity is usually nml
- Co-existing diseases may incl atopy, recurrent sinopulmonary infection, GI disease, and autoimmune disease
- Decreased synthesis or secretion of IgA

**ICD-9-CM Code**: 279.01

**Etiology**
- Absence of IgA on mucosal surface
- Decreased IgA blocking antibodies against environmental antigens
- Increased risk of malignancies
- Co-existing diseases may incl atopy, recurrent sinopulmonary infection, GI disease, and autoimmune disease

**Perioperative Implications**

**Preoperative Preparation**
- Consider antibiotic therapy
- Work up any indication of infection
- Optimize any underlying organ dysfunction and volume status

**Monitoring**
- Consider invasive hemodynamic monitoring in dehilitated pts

**Airway**
- Strict aseptic technique
- Universal precautions
- May encounter difficult intubation in pts with associated rheumatoid arthritis

**Induction**
- Hypotension 2° to hypovolemia and/or decreased cardiac reserve
- Wheezing allergies relatively resistant to conventional therapy

**Maintenance**
- May require high inspired O₂
- Regional anesthesia and careful titration of anesthetic agents due to potential underlying CV and pulm diseases
- Use only thoroughly washed RBC transfusions

**Extubation**
- Careful assessment of neuromuscular function due to potential drug-drug interaction

**Adjuvants**
- Depend on organ dysfunction

**Postoperative Period**
- May require intensive pulm therapy
- Maintain strict antiseptic precaution
- Increased suspicion of bacterial infection

**Anticipated Problems/Concerns**
- Anaphylactic reaction from transfusions of blood or blood products containing IgA to individual with IgA antibodies
- Asthmatic pt with IgA deficiency is relatively resistant to treatment
- Increased risk of nosocomial infection

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<tbody>
<tr>
<td>CARDIO</td>
<td>Decreased reserve, hypovolemia</td>
<td>Dypnea or exertion</td>
<td>Tachycardia, orthostatic hypotension</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Recurrent sinopulmonary infection, hemosiderosis, asthma</td>
<td>↓ Exercise tolerance</td>
<td>Wheezing, rales</td>
<td>CXR, PFTs Sinus x-rays</td>
</tr>
<tr>
<td>GI</td>
<td>Chronic gastroenteritis, malnutrition, malabsorption</td>
<td>Chronic diarrhea</td>
<td>Cachexia</td>
<td>Electrolytes, BUN, serum albumin</td>
</tr>
<tr>
<td>HEME</td>
<td>Nonspecific</td>
<td>Depends on the extent of co-existing diseases</td>
<td>Serum IgA, anti-IgA antibody, Coombs' test</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Nonspecific</td>
<td>Varies in severity depending on the extent of co-existing diseases</td>
<td>BUN, Cr</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Degenerative, demyelinating</td>
<td>Mental retardation associated with ataxia-telangiectasia</td>
<td>MRI</td>
<td></td>
</tr>
</tbody>
</table>

**Immune Suppression**

**Risk**
- Incidence in USA: 0.25 to 1.5% of population has HIV or other cause of immune suppression
- 20–25% of HIV infected pts will require surgery
- Major risk factors: Neutropenia, yeast overgrowth, and/or nosocomial colonization of skin and mucosa

**Perioperative Risks**
- 22.2% 30-d mortality in one study of AIDS pts undergoing intra-abdominal surgery
- Mortality greatest at the extremes of age
- Greatest source of morbidity and mortality is 2nd to infection
- Pneumonia accounts for ~40% of all deaths
- Increased incidence of postop pneumonia, wound infection, postop sepsis, resp insufficiency, SIRS, and hypotension due to CV instability
- Increased healing time

**Overview**
- Immune suppression can arise from multiple causes both primary and acquired
- Intraop, surgical trauma, anesthetic agents, blood transfusion with/out severe hemorrhage decreases the immune response

**ICD-9-CM Code:** 279.3 (Immune deficiency)

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<td>HEME</td>
<td>Anemia, neutropenia, lymphocytopenia, hypoglobulinemia, recurrent bacteremia, coagulation abn, thrombocytopenia</td>
<td>Easy fatigue, recurrent fever, sweats, and chills</td>
<td>Pale, petechiae</td>
<td>Hct/Hgb, WBC, platelets, plasma proteins, coagulation studies, special lymphocyte counts (e.g., CD4+ cells)</td>
</tr>
<tr>
<td>CARDIO</td>
<td>SBE, decreased CV reserve, hypovolemia, drug-induced injury (e.g., arabinomycin), mycotic aneurysms, pericardial effusion, vasculitis, pulm Htn</td>
<td>Decreased exercise tolerance</td>
<td>Murmurs, orthostatic hypotension, abn HR</td>
<td>ECG, ECHO</td>
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<tr>
<td>RESP</td>
<td>Recurrent acute pulm infections, pulm fibrosis, pulm obstruction, chronic TB and/or fungal infections</td>
<td>Decreased exercise tolerance</td>
<td>Airway lesions</td>
<td>CXR, spirometry</td>
</tr>
<tr>
<td>GI</td>
<td>Chronic gastroenteritis, chronic malnutrition, severe mucositis</td>
<td>Severe “cramping”, dysphagia, odynophagia diarrhea</td>
<td>Cachexia, leukoplasia</td>
<td>Electrolytes, albumin Blood cultures</td>
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<td>RENAL</td>
<td>Chronic pyelonephritis, bladder infections, chronic cystitis, drug-induced nephropathy (e.g., cyclosporine), end-stage renal pathology</td>
<td>Recurrent UTIs, frequency</td>
<td>Focal deficits, decreased mental function</td>
<td>BUN, Cr, pyelogram</td>
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<tr>
<td>CNS</td>
<td>Mycotic infections, AIDS, dementia and encephalopathy</td>
<td>Minor strokes</td>
<td>Brain scan</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Osteomyelitis</td>
<td>Deep pain located over involved area</td>
<td>Point tenderness</td>
<td>X-ray</td>
</tr>
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**Perioperative Implications**

**Preoperative Preparation**
- Continue or initiate antibiotic therapy and immune therapy
- Assess and optimize underlying organ system dysfunction (HIV-associated cardiomyopathy).
- Assess volume status and electrolytes due to chronic diarrhea
- Involved assessment may be required (pulm function tests, ECHO cardiography).
- Identify timing of administration of immune suppressive drug(s)

**Monitoring**
- Consider arterial line, PA line, or other invasive hemodynamic monitors in severely debilitated pts

**Airway**
- Strict aseptic technique and universal precautions when handling the airway
- Examination of upper airway for potentially obstructive lesions (i.e., Kaposi’s sarcoma)

**Induction**
- Chronic resp injury due to repeated lung infections may cause rapid desaturation.
- Hypotension due to decreased myocardial reserve and/or relative hypovolemia
- Decreased drug requirements 2nd to decreased plasma proteins

**Maintenance**
- Increased inspired O2 may be required due to chronic lung infections.
- Decreased myocardial reserve may require careful selection and titration of anesthetic agents or local or regional anesthesia for peripheral procedures.
- Pre-emptive pain management may protect against additional immune suppression

**Exubation**
- Due to weakness and drug-drug interactions, return of strength should be carefully evaluated.

**Adjuvants**
- Transplantation and anticancer drug interactions need to be considered (e.g., cyclosporine and barbiturates, narcotics, and muscle relaxants); bleomycin and O2 administration

**Postoperative Period**
- Resp adequacy should be carefully followed and may require intensive care monitoring.
- Maintain careful antisepsis procedures for extended periods.

**Anticipated Problems/Concerns**
- The greatest intraop risk to these pts is infection; therefore, strict hygienic practices are required.
- The general state of nutrition, recurrent infections, and the underlying cause of the immune suppression all tend to generally decrease resp reserve and CV stability.
- Risk of transmission of drug-resistant pathogenic microbial agents to medical personnel (needle stick or resp, e.g., drug-resistant TB). Follow CDC recommendations if exposed.
Implantable Cardioverter-Defibrillators (ICDs)

**Epidemiology**
- 450,000 pts/y in USA suffer sudden cardiac arrest (SCA); 550,000 new cases/y of CHF
- Incidence in USA: Approx 300,000 have implantable cardioverter-defibrillator (ICD)
- Incidence worldwide: Approx 500,000 have an ICD
  - ICD therapy for SCA, VT, VF and primary prevention remains substantially better than drug therapy.
  - Associated diseases incl cardiomyopathy, CAD, long QT syndrome, arrhythmogenic right ventricular dysplasia, Brugada syndrome, and LV noncompaction. Some pts will also have sinus node and/or AV nodal disease.
  - New implants (based on registry data) currently exceed 10,000 per mo in USA
  - ICD implant is indicated for any cause cardiomyopathy with EF ≤35% and without evidence of dystrophy; thus some pts undergo ICD implantation for primary prevention.
  - At current implant and survival rates, nearly 700,000 pts in USA with ICD in 2020
  - ALL ICDs can provide pacing for bradycardia; some pts might be pacing dependent.
  - Some ICDs also have atrial, RV, and LV pacing capability for cardiac resynchronization therapy (CRT).
  - Premature ICD failure rate might approach 2%. For the ICD pt without evidence of pacing, determining battery function is difficult.9

**Perioperative Risks**
- No proven increase in risk due to ICD itself, although an inappropriate high voltage therapy (HVT) might induce tachydysrhythmia, injure the myocardium releasing troponin, or both.
- These pts might be at increased risk owing to associated disease(s).
- Risk related to incorrect interpretation of events (pseudomalfuction), which is similar to the issues with pacing
- Risk related to incorrect interpretation of device type, i.e., confusing an ICD for a pacemaker, because ICD pts tend to have more medical issues and ICDs are more complicated

**Worry About**
- EMI entering the ICD on the ventricular channel, which might result in an ICD discharge. For the pacing dependent pt, EMI-induced ventricular oversensing with pacing inhibition can also result in asystole.
- Intraop increase in ventricular pacing owing to EMI entering a dual chamber DDD ICD on the atrial channel with resulant tracking
- Intraop increases in pacing rates with misinterpretation as inadequate anesthesia owing to activation of the exercise sensor, whether due to direct mechanical stimulation (such as preparation of the chest) or pressure on the device (personal leaning)
- Failure to capture (i.e., pacing output without depolarization) due to inadequate preop output (i.e., inadequate safety margin) or increase in pacing threshold, which can result from myocardial ischemia/infarction, drug administration, or electrolyte shifts. Note that any or all chambers can undergo failure to capture, with possible hemodynamic derangement but not apparent outright pacing failure.
- Magnet placement will change pacing rates only in ICDs from ELA (Sorin, Milan, Italy).
- Only Boston Scientific (BOS) ICDs emit tones confirming appropriate magnet placement. NO confirmation of magnet placement is available in Medtronic, St Jude Medical (SJM), or Boston Scientific.
- ICDs from BOS and SJM can have the magnet switch disabled by program.
- Premature ventricular pacing owing to EMI entering a dual chamber DDD ICD on the atrial chamber(s) (A, V, O), which might result in an ICD discharge.

**ASSESSMENT POINTS**

<table>
<thead>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Myocardial ischemia</td>
<td>Angina symptoms</td>
<td>ECG, pulse</td>
<td>Nuclear imaging</td>
</tr>
<tr>
<td></td>
<td>LV dysfunction</td>
<td>Exercise tolerance, DOE</td>
<td>S1, rales</td>
<td>Echocardiography</td>
</tr>
<tr>
<td></td>
<td>Heart rate (guidelines suggest &lt;80 bpm)</td>
<td>Frequency of ICD therapy</td>
<td></td>
<td>B-type natriuretic peptide</td>
</tr>
<tr>
<td></td>
<td>Need for pacing</td>
<td></td>
<td></td>
<td>ICD interrogation</td>
</tr>
<tr>
<td>RESP</td>
<td>Amiodarone toxicity</td>
<td>Exercise tolerance, DOE</td>
<td>SpO2, CXR, PFTs, ABGs</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Amiodarone toxicity</td>
<td></td>
<td>TSH, T3</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Edema</td>
<td>BUN, Cr</td>
<td></td>
</tr>
<tr>
<td>NEURO</td>
<td>CV disease</td>
<td>Stroke, T1As</td>
<td>Bruits</td>
<td>Carotid duplex</td>
</tr>
<tr>
<td>LYTES</td>
<td>Reversible VT/VF</td>
<td>Diuretic</td>
<td>Serum K+ and Mg2+</td>
<td></td>
</tr>
</tbody>
</table>

**Indications and Usual Treatment**
- Primary prevention in a pt with LVEF ≤35% (and more than 40 d from an ischemic event or 3 mo from vascular intervention) who is receiving optimal medical therapy and has a reasonable expectation of survival with good functional capacity for >1 y
- Survivors of cardiac arrest presumably due to VT/VF, not associated with reversible factors, such as acute coronary syndrome
- Pts with inducible VT/VF by EP study and no reversible cause
- Treatment for LV cardiomyopathy should incl (unless a contraindication is noted on the chart): beta blocker and ACE inhibitor and/or angiotensin receptor blocker therapy (see ACC/AHA Heart Failure Guidelines). Many pts will also have antiarrhythmic, diuretic, nitrate, or digoxin therapy.

Perioperative Implications

Preoperative Preparation
- Comprehensive ICD evaluation and/or interrogation should be performed in a timely manner (<3 mo) prior to surgery. An EP pacing and/or ICD service consult might be needed. The remaining battery life, tachy zones and therapies, pacing behavior, and prior dysrhythmia treatment should be documented. Many ICDs have ventricular-only (VVI) pacing capability, for the pt with intact atria and AV node, periop care must be directed to prevent the sinus rate from falling below the VVI pacing rate, since ventricular-only pacing will likely compromise hemodynamics. For the pt with hemodynamically advantageous pacing capability who is chronotropically incompetent or pacing dependent, consideration should be given to increasing the pacing rate for a significant procedure.
- For ventricular multisite pacing (called cardiac resynchronization therapy), assurance that the LV pacing lead is functioning. If central access is planned in a CRT pt, a recent CXR might be prudent to document the position of the LV lead.
- Alternate defibrillation (and pacing modalities [e.g., transvenous, transcutaneous] for the pacing-dependent pt) should be available. While transesophageal pacing might work as backup, it is contraindicated in atrial fibrillation, AV nodal block, and any pt with a permanently implanted pacing device.
- IV chronotropes (epinephrine, ephedrine)
- Discuss monopolar electrosurgery (ESU) precautions with surgeon and nursing staff. If monopolar ESU will be needed, the ICD should have the HVT disabled for the procedure. If magnet use is planned for this function, the interrogation should ensure that a magnet mode is present and enabled.
- Some ICDs allow demonstration of battery function without interrogation:
  - Boston Scientific ICDs will emit beeps if the magnet switch is enabled and the magnet is appropriately placed. A constant tone indicates that the ICD is disabled. No tone indicates magnet switch deactivation or a dead battery.
  - ELA (Sorin) ICDs will change pacing rate to 90 bpm if battery ok, 80 if elective replacement. But the patient rate must be less than 90 (80) to observe this function. No change indicates battery or other failure.
- Medtronic ICDs will emit a tone for at least 15 seconds when the magnet switch (nonprogrammable) is activated, even briefly, by a magnet. A warbling tone indicates a problem with the ICD, and no tone indicates a nonfunctioning device.
- MAGNET CAUTION: NO ICD provides asynchronous pacing to appropriate magnet placement. Only ICDs from ELA (Sorin) will change pacing rate (to 90 bpm if battery is ok) upon appropriate magnet placement. For many ICDs (Boston Scientific’s and St Jude Medical’s), the magnet switch can be programmed “OFF.” Only ICDs from Boston Scientific and its previous companies emit tones that identify correct placement of a magnet. Some older ICDs from Boston Scientific (with the “GDT” or “CPI” xray code) can undergo permanent disabling of tachy therapy by magnet placement.

- Regional technique offers CNS perfusion monitoring.
- Placement of defibrillation pads should be considered, esp. if a pt has been receiving HVT from the ICD.

Monitoring
- Mechanical pulse wave monitoring is required. It can be accomplished with the pulse oximeter plethysmogram, any invasive hemodynamic monitoring modality, or Doppler technique.
- Electrocardiographic (ECG) monitoring is required by ASA standards, but EMI perturbs the signal, and monitors frequently report incorrect heart rates (both too high and too low).
- For high risk cases with large potential fluid shifts, TEE might be indicated.

Induction
- Succinylcholine or etomidate might lead to inappropriate muscle activity, resulting in pacing inhibition, increased rates, or false VT/VF detection, but this has not been reported for ICDs. Succinylcholine-induced K+ fluxes theoretically can change pacing thresholds, but this has not been reported.

Maintenance
- Vigilant ECG/pulse monitoring
- Monopolar electrosurgical (ESU) cautery (i.e., the Bovie), which emits radiofrequency energy, has potential to cause inappropriate VT/VF detection (and HVT) as well as transient or permanent changes in ICD function. The most common problem is inhibition of pacing. Prevention includes: use of bipolar-only ESU, use of pure unblended monopolar ESU, and placement of the ESU current return pad so that the presumed current path of the ESU does not cross the chest. For all head and neck or contralateral breast surgery, the pad can be placed on the shoulder contralateral to the CIED. For ipsilateral breast surgery, the pad can be placed on the ipsilateral arm and the wire prepped into the field if needed.
- Electrophysiologic (EP) monitoring may be needed if ICD interrogation is not possible. The ICD should have the HVT disabled for the procedure. If magnet use is anticipated, the interrogation should be performed.
- For ELA (Sorin) ICDs, the magnet switch can be programmed “OFF.” Only ICDs from Boston Scientific and its previous companies emit tones that identify correct placement of a magnet. Some older ICDs from Boston Scientific (with the “GDT” or “CPI” xray code) can undergo permanent disabling of tachy therapy by magnet placement.

Postoperative Period
- Monitoring of mechanical pulse in the postoperative care unit
- ICD interrogation/reprogramming required if ICD was reprogrammed preop, advisable if monopolar ESU employed, any problems noted, or cardioversion/defibrillation has taken place.
- Some pts will require pacing changes to incl increased pacing rate, disabling of battery saving features, and adjustments to AV delays to optimize postop hemodynamics.
- Other risks related to associated medical problems

Anticipated Problems/Concerns
- Inappropriate delivery of HVT, which will occur without warning if due to EMI and will likely be missed by the intraop personnel.
- Intraop failure to pace, most likely related to monopolar electrosurgery.
- Periop pacing and sensing threshold changes.
- Risks related to associated medical problems.
- Iatrogenic misadventures resulting from misunderstandings of pacemaker system behavior.

*Some ICDs allow demonstration of battery function without interrogation:
- Boston Scientific ICDs will emit beeps if the magnet switch is enabled and the magnet is appropriately placed. A constant tone indicates that the ICD is disabled. No tone indicates magnet switch deactivation or a dead battery.
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*Boston Scientific owns the Guidant and CPI brands, and St Jude Medical owns the Pacesetter brand.
Infratentorial Tumors

Risk
• Highest incidence: Age 3–12 and 55–65 y
• 2/3 of children: 1.6–2.2/100,000 children – 1,300–2,000 new tumors in USA
• 1/3 of adult tumors: ~14,600 adults in the USA had new tumors in 2005

Perioperative Risks
• Very confined space, brain tolerates tumor poorly leading to symptoms and less forgiving with surgery than supratentorial
• CSF obstruction with hydrocephalus common, ICP tolerated poorly

Worry About
• Increasing ICP, hydrocephalus
• Impaired protective airway reflexes, aspiration
• Irregular resp due to brainstem compression, swallowing
• Impaired level of consciousness

Overview
• Survival 60% in children
• Prognosis is poor with glioblastoma, infiltrating brainstem glioma.
• Benign lesions such as meningioma, acoustic neuroma have low morbidity, mortality, but may recur if resection is incomplete.
• Degree of head elevation influences incidence, severity of air embolism (sitting > prone > park bench/lateral position).

ICD-9-CM Codes: 191.6; 191.7; 225.1; 225.2

Etiology
• Primary intra-axial lesions are generally malignant; extra-axial lesions are typically benign.
• Children primary most common: Astrocytoma, medulloblastoma, brainstem glioma are the most common posterior fossa tumors in children. Most common tumors in child age 3–12 y
• <1 y, astrocytoma, cerebellar PNET medulloblastoma common, epidermoid, brainstem glioma

• <2 y, 70% are medulloblastoma and low-grade glioma
• Pediatric cystic cerebellar astrocytoma is associated with 80% survival at 20 y
• Adult, most are metastases or acoustic tumors (most >60 y are acoustic)
• Acoustic neuroma (assoc neurofibromatosis NF II), metastases, meningioma are the most common posterior fossa tumors in adults. Metastases (lung and breast most common and vasogenic so inc ICP common). Metastases to cerebellum forms mass lesion.
• Differentiate from AVM and aneurysms
• Neurofibromatosis with some acoustic neuromas

Adjuvants
• Short-acting vasopressors or vasodilators for maintenance of CV stability
• Antiemetics

Postoperative Period
• Suspect brainstem compression or hematoma if postop Htn or profound bradycardia persists in previously normotensive pt.
• Suspect brainstem injury if persistent hypotension or apnea.
• Avoid potent narcotic analgesic drugs that may produce hypercarbia, decreased intracranial compliance.

Anticipated Problems/Concerns
• Intraop air embolism: Notify surgeon who should flood field, turn off N2O if on, acute CPAP may help find source, lay down supine if needed.
• Pts with higher-grade malignancy have greater likelihood of postop brain swelling.
• Postop inability to protect airway (loss of lower cranial nerves) watch for swallowing prior to extubation, use NG if question
• Loss resp drive in brainstem injury resp center
• Delayed awakening from pneumocephalus if sitting position (tension possible requiring relief), also supratentorial hemorrhage when sitting
• Massive tongue swelling, cervical spinal cord ischemia if sitting position
• Loss facial nerve (corneal ulceration from failed eye closing)
• Aseptic meningitis (blood irritating meninges)

ASSESSMENT POINTS

System       Effect                        Assessment by Hx          PE            Test
HEENT       Tonsillar herniation, cranial nerve VII compression     Dysphagia, change in voice, tinnitus, vertigo       Gag dysfunction, hyperthermia, ipsilateral hearing impairment               Indirect laryngoscopy, hearing exam
CARDIO, HEME       Progressive brainstem compression, ischemic cardiomyopathy     Cushing response: Htn, bradycardia, raised ICP, S, gallop, CHF               ECG, HCT, T&C
RESP       Progressive tonsillar herniation             Hyperventilation, irregular resp, apnea               CT exam, MRI
RENAL, GI  ↑ ICP                         N/V (esp. near 4th ventricle)                CT scan, MRI, glucose
CNS     ↑ ICP                          Listlessness, headache, nausea, drowsiness, diplopia         Papilledema, classic triad (headache, vomiting, ataxia)              CT scan, MRI
MS       Lesion in cerebellar hemisphere or midline                      Truncal ataxia                        Nystagmus hypotonia, limb ataxia intention tremor                     Extraocular movement abnormalities


Perioperative Implications

Preoperative Preparation
• Neuro exam: Cranial nerve deficits
• Presence and status of VP shunt
• Patent foramen ovale if sitting position
• Assess volume status from decreased intake, vomiting, diuresis (will increase risk of hypotension if sitting).
• Avoid narcotic premedication or any resp depressants if risk of ↑ ICP.

Monitoring
• Goals are maintenance of adequate CNS perfusion and cardiorespiratory stability, detection/treatment of air embolism, and surgical brainstem compression.
• Monitor CPP (MAP-ICP) measure BP at ear level; watch for hypotension when sitting.
• Capnography, precordial Doppler US, right atrial catheter for air embolism detection/retieval (TEE if available),
• Brainstem auditory evoked responses and cranial nerve VII stimulation may reduce morbidity from surgical manipulation. SSEP, MEP, and multiple cranial nerves often monitored.
• Watch for deep breath from brainstem compression resp center, watch for BP decreases and arrhythmias from brainstem compression.
• ECG, pulse oximetry to watch for arrhythmias (bradycardia common) from manipulation of brainstem cranial nuclei and dura (innervated by vagus n.). Avoid treating with anticholinergic as eliminate heart rate as monitor of brainstem
• If sitting position: Precordial Doppler and CVP with tip at right atrium needed.
• Watch eyes if prone for pressure and prep solutions

Airway
• Verify appropriate ETT position after final positioning; avoid large bite blocks and oral airways to minimize tongue and soft tissue compression, postop airway swelling.
• Watch for ETT kinking with neck flexion (armed tube if indicated).

Induction
• Hypotension on induction can be offset by pre-induction IV hydration.

Maintenance
• Positioning: Protect eyes, kink vertebral artery when truning head
• Preserve autonomic reflexes; avoid long-acting vasodilators.
• Monitor for changes in electrolyte balance due to loop and osmotic diuretics, replace diuresis if needed.
• Maintain normothermia, normovolemia, normotension, normoatremic fluids.
• Avoid hyperglycemia and hyperthermia.
• Controlled PPV, adequate hydration decreases risk of air embolism
• Avoid NMB with cranial nerve and MEP monitoring.
• Limited inhalational agents with SSEP and TIVA if MEP monitored.
• Dose, redose antibiotics
• Avoid anticholinergics and beta blockers to mask CV changes with brainstem compression

Extrusion
• Pt should be awake, following commands, and showing return of protective airway reflexes (swallow)
**Insulinoma**

**Risk**
- Most common functional islet cell tumor of pancreas
- Incidence 1–4/100,000 person-years
- Mean age of onset: 47 y
- Presentation earlier (mean age 25 y) if part of multiple endocrine neoplasia syndrome type I (MEN-1)
- More common in females

**Perioperative Risks**
- Hypoglycemia

**Worry About**
- Preop and intraop hypoglycemia
- Post-excision rebound hyperglycemia (not always present and not reliable to validate completeness of resection)
- Possibility of MEN-1 or multiple islet cell tumors

**Overview**
- 80–90% are <2cm, solitary, and benign
- Malignant lesions typically invade locally into the surrounding soft tissue or structures, to the lymph nodes, or to the liver
- Insulinomas are found equally distributed throughout the pancreas (i.e., head, body, and tail)
- 5–10% occur in the setting of MEN-1; increased risk of recurrence if associated with MEN-1
- Presentation: Post-absorptive hypoglycemia (fasting hypoglycemia), hypoglycemia after exercise, awakening at night to eat, wt gain due to frequent meals to avoid hypoglycemic symptoms
- Differential diagnosis: Factitious hypoglycemia, liver or metabolic disease, noninsulinoma pancreatic- atogenous hypoglycemia syndrome (NIPHS)
- NIPHS is associated with diffuse islet cell hyperplasia and presents with postprandial sympto- matology rather than postabsorptive
- Dx strongly suggested by Whipple's triad: (1) symptoms of hypoglycemia provoked by fasting; (2) blood glucose levels <50 mg/dL; and (3) relief of symptoms with glucose administration
- Typically, blood glucose <45 mg/dL, insulin level >6μU/mL, and C-peptide elevated to >200pmol/L
- Gold standard for Dx: Measurement of plasma glucose, insulin, C-peptide, and pro-insulin during a 72 hr fast with or without betahydroxybutyrate and absence of plasma levels of sulfonlurea
- Preop localization techniques incl.: CT, MRI, PET, endoscopic US, octreotide scintigraphy, selective mesenteric angiography with intra-arterial stimulation, and hepatic venous sampling for plasma insulin. Extensive preop imaging may not be helpful.

- 20–60% of insulinomas remain undetected at the time of surgery, likely due to small size and/or decreased density of somatostatin receptor subtypes compared to other neuroendocrine tumors.
- Recent data suggests intraop US along with surgical exposure and palpation is the most cost-effective approach.
- Gold standard is firm biochemical Dx and selected preop imaging along with thorough pan- creatic exploration and intraop US
- In absence of preop localization and intraop detection, blind pancreatic resection is not recommended.
- Neurogenic symptoms are 2nd to autonomic system discharge in response to hypoglycemia. Neuroglycopenic symptoms are 2nd to CNS glucose deprivation.
- ICD-9-CM: 211.7 (Benign neoplasm of islets of Langerhans)

**Etiology**
- Unknown: Most are solitary adenomas
- 5–10% of insulinomas assoc with the autosomal dominant MEN-1 syndrome

**Usual Treatment**
- Operative management only curative option
- Laparoscopic (14% conversion rate) versus open
- Most studies conclude laparoscopic approach feasible and safe, esp. for benign and distal pancreatic tumors (role for pancreatic stump and malignant lesions remains more controversial)
- Enucleation may be performed for lesions that are clearly localized preop, near or at the pancreatic surface, and easily defined intraop
- Resection recommended for lesions that are multiple, near the pancreatic duct or major ves- ses, MEN-1 cases, and suspected malignancy (infiltrating tumor, puckering of surrounding soft tissue, distal dilation of pancreatic duct, or lymph node involvement)
- Medical management reserved for unresectable malignant disease, high-risk surgical candidates, or unsuccessful operation with persistent symptoms
- Medical treatment of unresectable disease con- sists of small frequent meals, diazoxide, verapamil, and octreotide
- Octreotide is a somatostatin analogue that relieved symptoms in 50% of pts.
- Octreotide should be used with caution because many insulinomas lack octreotide receptors. Treatment may fail to suppress insulin production and blunt compensatory growth hormone and gluga- gon response leading to worsening hypoglycemia.
- Molecular targets are being investigated.

**Perioperative Implications**

**Preoperative Preparation**
- Maintain/optimze physiologic condition
- Evaluate for MEN-1
- Avoid severe hypoglycemia with frequent meals, avoidance of prolonged exercise
- Diazoxide, octreotide, and verapamil to control hypoglycemia if necessary
- Admit the night before and maintain on 10% dextrose infusion while NPO
- Remove dextrose from IV solution just prior to entering the operative room
- Monitor plasma glucose every 10–15 min

**Monitoring**
- Measure plasma glucose every 10–15 min
- Maintain plasma glucose >60 mg/dL
- Consider arterial line and/or CVP to facilitate sampling ease

**Airway**
- Nothing specific, although these pts may have significant wt gain

**Induction**
- Propofol has not been shown to significantly affect the release of insulin or glucose regulation

**Maintenance**
- Length of procedure highly variable
- Careful attention to fluid status
- Have dextrose solutions available to treat hypoglycemia

**Exubation**
- Nothing specific

**Anticipated Problems/Concerns**
- It has been proposed that glucose solutions be avoided intraop so that hyperglycemic rebound can be used to confirm tumor removal.
- More recent studies show that less than half of pts will have this rebound in the first 30 min fol- lowing tumor removal, and therefore, hyperglyce- mic rebound cannot be used as proof of complete tumor removal.
- Intraop insulin assays may be an alternative.
- Pts who have hyperglycemic rebound and/or successful tumor removal can still have hypo- glycemic episodes, so pts must be monitored for hypoglycemia in the postop period
- Most pts are discharged home with nml fasting glucose levels
- Postop complications incl pancreatic duct leak causing pseudocyst, abscess, and/or fistula (octreotide can be used to decrease fistula output)

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<td>Renal stones if MEN-1</td>
<td>Renal colic</td>
<td>Flank pain</td>
<td>Serum Ca++</td>
</tr>
<tr>
<td>ENDO</td>
<td>MEN-1</td>
<td>Pituitary tumors</td>
<td>Vision changes</td>
<td>Flank pain, Parathyroid hormone, serum Ca++</td>
</tr>
<tr>
<td></td>
<td>Insulinoma</td>
<td>Neurogenic symptoms: Hunger, sweating, and paresthesias (cholinergic) and anxiety, tremor, and palpitations (adrenergic)</td>
<td>Mental status exam</td>
<td>Skel x-rays and appropriate endocrine tests</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neuroglycopenic symptoms: Behavioral changes, death, confusion, vision changes, fatigue, seizure, loss of consciousness</td>
<td>Fasting glucose, insulin levels, C-peptide</td>
<td></td>
</tr>
</tbody>
</table>

**Intracranial Hypertension (ICH)**

**Risk**
- Incidence in USA: >50% of pts presenting with head trauma or other intracranial pathology (>600,000/y)
- Gender predominance: Depends on etiology

**Perioperative Risks**
- Increased risk of brain ischemia and herniation leading to brain infarction, disability, coma, and death
- Increased risk of permanent CNS dysfunction

**Worry About**
- Controlling intracranial pressure and preventing brain ischemia/herniation
- CV and resp instability
- Co-existing injuries in trauma pts (occult cervical spine and intra-abdominal injuries)

**Overview**
- Intracranial compartment has fixed volume with three components (brain = 85%, CSF = 10%, cerebral blood volume [CBV] = 5%)

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<tr>
<td>CV</td>
<td>Dysrhythmias, unstable vital signs Inferior wall myocardial ischemia</td>
<td>BP Pulse</td>
<td>S, gallop</td>
<td>Tachycardia, bradycardia, prolonged QT interval, ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Irregular breathing</td>
<td>Resp rate and pattern</td>
<td>Vomiting</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Reduced gut motility</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>SIADH Central DI</td>
<td>Oliguria Polyuria</td>
<td>Urinalysis, serum electrolytes</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Altered function</td>
<td>Headache, vomiting, unconsciousness</td>
<td>Neurologic deficits, papilledema</td>
<td>Direct ICP measurement (ventriculostomy, intracranial bolt, etc.)</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- **Judicious or no preop sedation due to risk of depressed ventilatory drive/hypoventilation/hypercapnia**
- **Assess volume status**

**Monitoring**
- **Consider arterial catheter for BP monitoring and for serial ABGs to properly manage mechanical ventilation**
- **Consider ICP monitor and CVP line**
- **Glucose**

**Airway**
- **Neutral cervical spine position for tracheal intubation if possible traumatic injury**
- **Possible aspiration risk (emergency procedure or severe ICH)**

**Preinduction/Induction**
- **Neutral neck position and head elevation**

- **Increased volume of one component (e.g., tumor, hydrocephalus, or hemorrhage) elevates ICP, leading to ICH (ICP >20 mmHg, >40 mmHg = severe life-threatening)**
- **ICH reduces CPP = MAP – ICP, causing brain ischemia and/or infarction**
- **ICH causes intracranial pressure gradients that may extrude brain parenchyma through dural or bony passages, resulting in herniation**
- **Some anesthetic agents, Htn, hypercapnia, and hypoxemia increase CBF, increasing CBV and ICP**

**ICD-9-CM Code: 348.2 (Benign)**

**Etiology**
- **Usually a 2° process accompanying other pathology (e.g., head injuries, hemorrhage, hydrocephalus, abscess, 1° and metastatic brain tumors, cerebral infarcts, hypertensive and metabolic encephalopathies, venous thrombosis, infection, burns, near-drowning, and status epilepticus) that increases brain, CSF, or cerebral blood volumes**

**Usual Treatment**
- **Treatment of 1° disease (e.g., removal of tumor, hematoma, or abscess)**
- **Control ventilation, avoid hypoxemia (PaO2 >90 torr), hyper- and hypocapnia**
- **Establish stable hemodynamics (normotension but estimated CPP >60 mmHg)**
- **Head elevation (head above heart) and neutral neck position to promote cerebral venous return**
- **Osmotic therapy (mannitol or hypertonic saline) to decrease brain size**
- **Corticosteroids (neoplasm or abscess)**
- **CSF drainage**
- **Sedation and NMB in responsive pts**

**Exubation**
- **Maintain tracheal intubation if concerns about postop resp function or persistent ICH; otherwise, prompt extubation for early neurologic evaluation**

**Adjuncts**
- Benzdiazepines, β-blockers, antihypertensives

**Postoperative Period**
- **If ICH persists, adequate ventilation and/or oxygenation, pain control, sedation essential**

**Anticipated Problems/Concerns**
- Use isometric crystalloid or colloid IV solutions to minimize brain H2O and cerebral edema. Avoid dextrose since it may exacerbate effects of brain ischemia.
- Renal dysfunction and severe hypovolemia are possible from preop osmotic therapy.
Intraoperative Recall

G. Richard Benzinger

Risk
- Incidence in USA: 20 million anesthetics annually

Perioperative Risks
- Incidence is approx 0.1% in general surgical population and increases to about 1% in high risk populations.
- Procedure risk factors incl OB surgery, cardiac surgery, trauma, and rigid bronchoscopy.
- Pt risk factors incl prior awareness, significant CV disease, COPD, substance abuse, chronic opioid use, and chronic benzodiazepine use.
- Anesthetic risk factors incl absent/low benzodiazepine premedication, absent/low halogenated agent, and dense NM blockade.

Worry About
- PTSD is a common sequela (up to 50% incidence).
- Awareness caused 1.9% of closed claims against anesthesia personnel.
- Many cases are preventable, and identified as attributable to lapses in technique.

Overview
- Explicit recall: Conscious, articulable recollection of events when intended to be unaware.
- Implicit recall: Change in behavior attributable to perception of intraop events, but no explicit awareness. Much harder to study.
- Hemodynamic changes are neither sensitive nor specific signs of awareness.
- Processed EEG monitoring (such as the bispectral index, BIS) may decrease incidence of awareness.
- Maintenance of adequate end-tidal halogenated agent (≥0.7 MAC, age-adjusted) may decrease incidence of awareness.

Etiology
- Inadvertent awake paralysis is usually due to drug labeling or administration error.
- Other awareness is frequently associated with light anesthesia: Intentional, unintentional, or equipment malfunction.

Usual Treatment
- Discuss incident with pt postop.
- Offer psychiatric referral to all pts with recall as screening or treatment for PTSD.
- Preliminary work suggests that beta blockers may reduce development of PTSD when administered shortly after a traumatic event; consider administration in PACU if explicit recall is reported there.
- Benzodiazepines are not effective in producing retrograde amnesia; can’t use for rescue of awareness.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Htn</td>
<td>Observation</td>
<td>BP</td>
</tr>
<tr>
<td>RESP</td>
<td>Tachypnea</td>
<td>Auscultation</td>
<td>EKG</td>
</tr>
<tr>
<td>CNS</td>
<td>Increased sympathetic tone</td>
<td>Lacrimation</td>
<td>Resp rate</td>
</tr>
<tr>
<td></td>
<td>Spontaneous movement</td>
<td>Observation</td>
<td>PIP</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preinduction/Induction/Maintenance
- Counsel all pts about risk of awareness as part of routine consent process.
- Consider benzodiazepine premedication in all pts without contraindication; titrate dose to clinical effect.
- Avoid muscle relaxant if not indicated. If needed, titrate to avoid dense paralysis.

Monitoring
- Consider use of bispectral index in high-risk pts.
- Keep inhaled agent ≥0.7 MAC with audible alarms in high-risk pts.
- Continue to monitor NM blockade.

General Anesthesia
- Consider redosing induction agent or using inhaled agents if time between induction and securing airway is prolonged.

Regional Anesthesia
- Counsel pts that awareness during regional anesthesia is expected, even with sedation.
- Limit incidental and alarming conversation during surgery with regional or any other anesthetic technique.

Postoperative Period
- Structured interview for recall
  - Last thing remembered before sleeping?
  - First thing remembered after awakening?
  - Anything in between?
  - Remember any dreams?
  - Worst thing about anesthetic?
- Many pts with awareness don’t report in recovery room. Serial interviews are necessary for complete surveillance.

Anticipated Problems/Concerns
- High risk of serious psychiatric sequelae.
Jaundice

Risk
- Chronic liver disease consistently ninth most common cause of death in USA
- M:F ratio: 2:1
- African-American to Caucasian 2:1

Perioperative Risks
- Jaundice per se poses no special risks
- Risks associated with co-existing or underlying conditions
- Use of regional anesthesia limited by coagulopathy and ascitis

Worry About
- Chronic liver disease
- Hepatopulmonary syndrome, hypoxemia
- Portopulmonary hypertension
- Hepatorenal syndrome
- CV dysfunction (cirrhotic; alcohol)
- Infection, protein-malnutrition
- Encephalopathy (hepatic and alcoholic; cerebral edema
- Esophageal varices (incompetent lower esophageal sphincter)
- Ascites; renal dysfunction
- Low systemic vascular resistance/hyperdynamic circulation
- Bleeding
- Inability to extubate at end of surgery
- Altered drug pharmacodynamics and pharmacokinetics
- Universal precautions
- Invasive monitoring

Overview
- Mostly unconjugated
- Excess production
- Hemolytic anemias (e.g., sickle cell anemia; β-thalassemia major)
- Extravascular hemolysis (tissue infarction; hemorrhage into tissue, postop jaundice)
- Ineffective erythropoiesis
- Decreased hepatic uptake
- Drugs (e.g., flavaspidic acid, novobiocin, some cholecyystographic dyes)
- Severe, prolonged fasting
- Decreased conjugation
- Neonate: Physiologic jaundice of the newborn; breast milk jaundice; hypothyroidism; galactosemia
- Sepsis
- Acquired transferase deficiency: Drug inhibition (e.g., pregnanediol, chloramphenicol); hepatocellular disease (cirrhosis, hepatitis)
- Gilbert’s disease: Decreased glucuronyl transferase
- Crigler-Najjar I (absent) and II (partial decrease) in glucuronyl transferase
- Mostly conjugated
- Decreased hepatic excretion
- Hereditary and/or familial: Dubin-Johnson, Rotor syndromes; recurrent intrahepatic cholestasis, benign; gestational cholestatic jaundice—(1:13,000 deliveries; third trimester; preclampsia, nulliparity; twin; decreased plt)
- Acquired: Sepsis; hepatocellular disease (drug- and viral-induced hepatitis); postop jaundice (pigment overload [transfusions, resorption of hematomas, hemolysis]; hepatocellular damage [drugs, incl halothane; shock]; benign postop jaundice; drug-induced cholestasis (e.g., oral contraceptives, methyltestosterone)
- Extrahepatic biliary obstruction (e.g., mechanical, from stones, stricture, tumor)
- Pseudojaundice
- Dietary carotenoids (primarily infants; excessive intake of vegetables, such as carrots, tomatoes)
- TPN–associated liver dysfunction
- Poisoning (picric acid)

ICD-9-CM Code: 782.4 (Jaundice, unspecified, non-newborn)

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Hyperdynamic</td>
<td>Duration</td>
<td>Yellow sclerae</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Cirrhotics have 6× increase in pulm Htn</td>
<td>General Sx</td>
<td>↑ HR; ↓ BP</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Severe dyspnea, hypoaxia, clubbing</td>
<td>Clubbing</td>
<td>Cyanosis</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Signs of chronic liver disease</td>
<td>LFTs</td>
<td>Coagulation time Hgb, plt</td>
<td></td>
</tr>
<tr>
<td>ENDO/METAB</td>
<td>↓ Synthetic function, ↑ enzymes, ↑ albumin, ↓ hepatic coag factors; ↓ clearance of toxins</td>
<td>General malaise Sx</td>
<td>Jaundice</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Easy bruising/bleeding</td>
<td>Ecchymoses, hematoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Duration; evidence of bleeding</td>
<td>Yellow color</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Edema</td>
<td>BUN, Cr; Cr may be spuriously lower with high bilirubin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Mental status</td>
<td>Normal to encephalopathy/comatose</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Perioperative Implications

- **Drug**: Decreased protein production leads to decreased albumin binding, more active drug
  - Cimetidine and/or ranitidine: Clearance reduced, esp. in pts with ascites, hypoproteinemia, encephalopathy
  - Benzodiazepines: Clearance of oxidative pathway markedly decreased; glucuronidation path (e.g., lorazepam) not greatly altered. Excessive sedation in severe liver disease.
  - Narcotics: Meperidine clearance is severely affected; succinylcholine activity may be prolonged somewhat because of decreased levels of pseudocholinesterase.
  - Miscellaneous: Phenobarbital and lidocaine have reduced clearance; diuretics may have reduced natriuretic efficacy.

- **Halogenated agents**: Halothane should be avoided; association of enflurane with hepatic toxicity is less clear; isoflurane, sevoflurane are preferred agents in setting of liver disease and best preserves liver hemodynamics.
- **Pregnancy**: Jaundice may signal HELLP syndrome and pregnancy-induced Htn.
- **Cardiac surgery**: Jaundice occurs in about 20% post-CPB pts; risk factor for mortality

Preoperative Preparation

- **NMB**: Dose muscle relaxants to effect and consider path of elimination

Induction

- **Airway**: May have bleeding disorder

Maintenance

- **Narcotics**: Meperidine clearance is severely affected; succinylcholine activity may be prolonged somewhat because of decreased levels of pseudocholinesterase.

Anticipated Problems/Concerns

- **Inability to extubate immediately postop due to prolonged action of NMB and sedative/hypnotic/narcotic medications**
Jehovah’s Witness Patient

Risk
- Incidence in USA: Approx 1.9 million members, 7.1 million worldwide
- Headquartered in Brooklyn, New York

Perioperative Risk
- Morbidity and/or mortality from massive hemorrhage 2^
- religious dogma banning members from accepting blood transfusions

Worry About
- Understanding the rights and duties of physician in regards to blood or blood product administration
- Trauma and emergency situations in which little time is available to discuss blood product transfusion
- Competent adults are those who know the nature and consequences of their actions; such adults have the right to refuse specific therapies
- Parens patriae ("parent of the nation"), refers to the public policy power of the state, represents the duty and interest of the state to preserve the health of minors. Medically, when a child’s right to live and parental religious beliefs collide, the courts have consistently ruled that the child’s welfare is paramount.

Overview
- Began as Bible study group in 1869; adopted the name Jehovah’s Witnesses (based on Isaiah 43:10–12) in 1931
- Strict interpretation and adherence to Biblical passages, which forbid eating of blood; interpreted as prohibition of acceptance of blood products to sustain life
- In 1942 the Watchtower Society, the governing body of Jehovah’s Witnesses, introduced the blood ban which forbids members from accepting allogeneic blood products: whole blood, red blood cells, white blood cells, platelets, and plasma
- There is variability among members to the interpretation of the prohibition regarding blood; Jehovah’s Witnesses may consider the use of one’s own blood in the course of a medical procedure or therapy provided there is no advanced storage. They may accept fractions of plasma such as albumin, recombinant human erythropoietin (rHuEPO), immunoglobulin, or factor concentrates

Usual Treatment
- Discuss and document preop the potential for life-threatening hemorrhage and therapies/interventions that would be acceptable to the pt.
- Seek evidence of advance directive, an affidavit that confirms the pt’s refusal to accept a transfusion (which forces discussion and releases physicians/hospitals of responsibility for outcome of the pt’s decision).
- Optimize Hct with rHuEPO prior to elective procedures in which risk for the need for a transfusion is high.
- Consider contacting a Jehovah’s Witness Hospital Liaison Committee, which consists of a group of individuals trained to work as intermediaries in avoiding conflict between pts and physicians.
- Contact legal counsel if pt is a minor, unconscious, or is an incompetent adult.

ASSESSMENT POINTS

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<thead>
<tr>
<th>System</th>
<th>Assessment by Hx</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEME</td>
<td>Evaluate for treatable forms of anemia</td>
<td>Hg/Hct, Folate, B12 Levels, Fe, Ferritin, Transferrin saturation</td>
</tr>
</tbody>
</table>


Perioperative Implications
Preoperative Preparation
- Iron therapy, esp. if evidence of decreased iron stores; ferrous sulfate 125 mg PO 23 × daily or
- Iron dextran 100–200 mg IV × 45 daily doses if unresponsive to oral medication or evidence of malabsorption
- Consider rHuEPO; 75100 U/kg SQ or IV, 3 wk × 34 wk
- Delay elective surgery until red cell mass is optimal

Monitoring
- Minimize phlebotomies; consider pediatric sampling tubes
- Consider oximetric pulm artery catheter if high possibility of hemorrhage

Intraoperative Therapeutic Options
Maintain Blood Volume
- Acceptable treatment
  - Nonblood volume expanders (saline, lactated Ringer’s, hydroxyethyl starches, dextrans)
  - Synthetic oxygen therapeutics (recombinant human or bovine hemoglobin, perfluorocarbons)
- Personal decision
  - Hyper- or normovolemic hemodilution (maintain continuous circuit with pt) in the absence of CAD or Hg <7gm/dL
  - Blood salvage techniques (equipment must be arranged in continuous series with the pt’s circulation)
  - Autotransfusion of shed mediastinal or wound blood
  - Plasma derived fractions (albumin, cryoprecipitate)

Maximze Oxygen Delivery
- Increase FIO
- Hyperbaric O
- Inotropic agents to augment cardiac index once volume resuscitated
- Synthetic O, carrying solutions

Prevention of Intraoperative Blood Loss
- Acceptable
  - Meticulous surgical technique, use of hemostatic surgical instruments
  - Laparoscopic, endovascular, or minimally invasive surgical techniques
  - Hypotensive anesthetic techniques

- Preop angiographic embolization
- Pharmacologic agents: Tranexamic acid, epsilon aminocaproic acid, desmopressin, recombinant factor VIIa. Careful consideration must given to the use of aprotinin.
- Personal decision
  - Hemostatic products containing blood fractions (fibrin glue/sealant, thrombin sealants)

Minimize O2 Consumption and Demand
- Hypothermia 30°C to 32°C (reduces O2 consumption 50%)
- Sedation and/or analgesia
- Paralysis

Postoperative Considerations
- Consider postop ventilation with paralysis, sedation, and hypothermia for severe anemia.
- Consider PA catheter to measure and follow CO and SvO2 to assess O2 delivery/consumption without resorting to phlebotomy.
- Supplement with IV hyperalimentation, rHuEPO, and iron dextran.
Jeune Syndrome (Asphyxiating Thoracic Dystrophy)

**Risk**
- Incidence in USA: 1:100,000–130,000 live births and prevalence of 2.6:100,000
- No race or sex predilection
- Skeletal survey by US after 14 wk gestation can detect defining deformities
- Four clinical forms: Lethal, severe, mild, latent

**Perioperative Risks**
- 70–80% mortality of homozygous carriers in infancy from restrictive lung disease
- Respiratory failure from small thoracic cage and hypoplastic lungs
- Progressive renal disease with cystic lesions and periglomerular fibrosis
- Liver and pancreatic involvement with fibrosis and cysts

**Worry About**
- Respiratory failure with hypoxia and hypercapnia
- Barotrauma with positive pressure ventilation

**Overview**
- Rare autosomal recessive disease with skeletal dysplasia and variable renal, hepatic, pancreatic and retinal abnormalities
- Poor survival beyond early infancy
- Narrow, rigid thoracic cage due to short horizontal ribs, short limbs, underdeveloped iliac wings and acetabula and occasional polydactyly
- Respiratory failure from restrictive thorax and hypoplastic lungs
- Changes in renal, hepatic, and pancreatic systems if survival past infancy
- Chronic renal failure can lead to transplantation
- Hepatic dysfunction can be controlled with ursodeoxycholic acid but those with severe portal Htn require liver transplantation

**Perioperative Implications**

**Preoperative Preparation**
- Assess ventilation
- Evaluate for possible pulm Htn
- Evaluate renal function and consider liver function testing

**Monitoring**
- Consider arterial catheter

**Airway**
- Small larynx requires smaller ETT size

**Induction**
- Agitation may make resp asynchronous (chest and/or abdomen), causing hypoxemia

**Maintenance**
- Lung hypoplasia makes barotrauma high risk; maintain low peak airway pressures

**Extubation**
- Document adequate ventilation before extubation; postop ventilation may be needed for a prolonged period, esp. after thoracoplasty

**Adjuvants**
- Renal function assessment guides selection of muscle relaxant and fluid management

**Etiology**
- Autosomal recessive inheritance
- Postulated involvement of chromosome 15q13 or IFT80 gene on chromosome 3

**Usual Treatment**
- Vertical expandable prosthetic titanium rib (VEPTR) thoracoplasty has been successful for Jeune syndrome but may require postop ventilation and has a high incidence of barotrauma
- Older children may require surgery related to renal failure (dialysis catheters, renal transplantation)

**ICD-9-CM Code:** 756.4

**ASSESSMENT POINTS**

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<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Cleft lip or palate</td>
<td>Assessment by Hx</td>
<td>Airway exam</td>
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</tr>
<tr>
<td></td>
<td>Small larynx</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Pulm Htn</td>
<td>Syncope</td>
<td>↑ 2nd heart sound</td>
<td>ECG (RVH)</td>
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<tr>
<td></td>
<td>Stiff, small rib cage</td>
<td>Pneumonia/respiratory failure</td>
<td>Small chest, horizontal ribs</td>
<td>ABGs</td>
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<tr>
<td></td>
<td>Hypoplastic lungs</td>
<td>Assisted ventilation</td>
<td>Cyanosis with crying</td>
<td>CXR</td>
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<tr>
<td></td>
<td>Hepatic fibrosis/cysts</td>
<td>Failure to gain wt</td>
<td>Hepatomegaly</td>
<td>Abdominal US</td>
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<tr>
<td></td>
<td>Pancreatic fibrosis/cysts</td>
<td></td>
<td></td>
<td>Bilirubin/LFTs</td>
</tr>
<tr>
<td></td>
<td>Foregut dysmotility/malrotation</td>
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<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Cysts</td>
<td>Polyuria, polydipsia</td>
<td>Increased OFC (head circumference)</td>
<td>BUN, Cr, lytes, Ca²⁺, PO₄⁻⁻</td>
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<tr>
<td></td>
<td>Nephritis</td>
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<td>Abdominal US</td>
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<tr>
<td>CNS</td>
<td>Occasional hydrocephalus</td>
<td>Polyuria, polydipsia</td>
<td>Increased OFC (head circumference)</td>
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<td></td>
<td>Retinal degeneration</td>
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<td></td>
</tr>
<tr>
<td>MS</td>
<td>Short limbs and stature</td>
<td>Polydactyly of hands and feet</td>
<td>X-ray of thorax, pelvis</td>
<td></td>
</tr>
</tbody>
</table>


**Anticipated Problems/Concerns**
- Asynchronous ventilation during crying with hypoxia
- Barotrauma during assisted mechanical ventilation
- Renal and/or liver disease and drug metabolism
- Postop resp failure requiring ventilatory support
Kartagener’s Syndrome

**Risk**
- Kartagener’s syndrome (KS), first described in 1933, is part of a larger family of diseases classified as Primary Ciliary Dyskinesia (PCD).
- The triad of KS consists of bronchiectasis, chronic sinusitis, and situs inversus, and it has an incidence estimated at 1:15,000–40,000 births.
- The disease is likely underdiagnosed, as a limited amount of centers have resources to provide an accurate diagnosis.
- No predilection for race or gender.
- Symptoms more prevalent in children in the first decade of life.

**Perioperative Risks**
- Morbidity: Lung infection, pulm edema, atelectasis, sinusitis.

**Worry About**
- Pulm function and anatomy.
- Airway obstruction due to ineffective clearance of secretions.
- Bronchiectasis, which can lead to cor pulmonale, amyloidosis, and pulm edema and is usually found in the middle or lower lobes in KS pts as opposed to the upper lobes in cystic fibrosis pts.

**Overview**
- Complete situs inversus (incl dextrocardia).
- PCD resulting in chronic respiratory tract infections, bronchiectasis and sinusitis.
- Approximately half of pts with PCD have situs inversus and, thus, are classified as having KS.

**ICD-9-CM Code: 759.3**

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Dextrocardia</td>
<td>Dyspnea</td>
<td>Right-sided heart tones</td>
<td>CXR, ECHO, ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchiectasis</td>
<td>Cough</td>
<td>Decreased breath sounds, ronchi, cracks, wheezes</td>
<td>CXR, bronchoscopy, spirometry, bronchography</td>
</tr>
<tr>
<td></td>
<td>Ciliary dyskinesia</td>
<td>Halitosis</td>
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<tr>
<td>IMMUNO</td>
<td>Chronic sinusitis</td>
<td>Nasal drainage</td>
<td>Frontal and maxillary tenderness</td>
<td>CT sinus</td>
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<tr>
<td></td>
<td>Bronchitis</td>
<td>Morning sore throat</td>
<td>Rhonchi</td>
<td>Sputum and tracheal aspirate for culture and Gram stain</td>
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<tr>
<td></td>
<td>Pneumonia</td>
<td>Cough</td>
<td>Rales</td>
<td>CXR</td>
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<tr>
<td></td>
<td>Otitis media</td>
<td>Fever</td>
<td>Rhonchi</td>
<td>SpO₂</td>
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<tr>
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<td></td>
<td>Earache</td>
<td>Erythematous tympanic membrane</td>
<td>Audiometry</td>
</tr>
</tbody>
</table>


**Perioperative Considerations**

**Preinduction**
- Consider omitting anticholinergics and cough suppressants from preanesthetic medication.
- Chest physiotherapy, bronchodilators, and incentive spirometry are often beneficial.
- Treat underlying pulm infections.
- Immunize against influenza A and pneumococcal organisms.

**Monitoring**
- In dextrocardia, position of ECG leads should be the mirror image of normal, as should that of paddles of external defibrillation, cardioversion, and pacing.
- Since the vessels and thoracic duct are likely to be reversed, consider cannulation of the internal jugular vein from the left.
- Pulm artery catheters should be oriented in anticipation of a clockwise direction of migration.
- Pregnant pts with KS should be positioned in right uterine displacement rather than left.

**Induction/General Anesthesia**
- Emphasize aseptic technique 2º to abn neutrophil chemotaxis.
- Aim for non-traumatic airway manipulation to avoid possible infection.
- Humidify inspired gases.
- Inhalation injury usually occurs in left lung, which is also larger lung.
- Bronchial intubation with a single-lumen ETT usually involves left side.
- Right bronchial suctioning will be more difficult to perform with nonangulated suction catheters.
- Left-sided double-lumen tubes may occlude orifice of left upper lobe.
- When lung isolation is needed, consider tracheal intubation first with a bronchial blocker in the appropriate bronchus.
- If a double-lumen tube is required, consider inserting a left-sided tube with the bronchial tube on the right; the endobronchial stent and the upper part of tube must be bent 180º from original orientation prior to insertion such that the normal curvature of the oropharynx is still followed. The same principles apply to use of a right-sided tube.
- Extubation of the trachea should occur as soon as possible after the pt meets common extubation criteria.

**Regional Anesthesia**
- Employ regional or local anesthetic techniques when possible to avoid airway manipulation/complications and to preserve resp muscle function intraop and postop.

**Postoperative Period**
- Consider nonopioid analgesia and/or epidural analgesia for postop pain.
- Avoid excessive sedation and encourage early ambulation to aid in clearance of airway secretions.
- Chest physiotherapy, bronchodilators, incentive spirometry may be beneficial.
- Oral airway preferred over nasal airway 2º to increased risk of sinusitis.

**Anticipated Problems/Concerns**
- Lung infection is common as result of ciliary dyskinesia.
- Fluid overload can precipitate cor pulmonale and pulm edema.
- Avoid nasal catheters and/or airways to minimize chances of paranasal sinusitis.
Klippel-Feil Syndrome

Ronald S. Litman

Risk
- Incidence estimated at 1:40,000 live births (probably underestimate, as milder cases go unrecognized)
- Slight female predilection (63%)

Perioperative Risks
- Cervical spine instability and cardiopulmonary complications
- Often occurs in association with other clinical syndromes (e.g., fetal alcohol, Goldenhar's)

Worry About
- Exacerbation of cervical spine instability during airway maneuvers, endotracheal intubation, and subsequent positioning

Overview
- Congenital abnormality consisting of the following triad of findings: Fusion of two or more cervical vertebrae; low posterior hairline; cervical immobility
- Severity ranges from mild (often not recognized until late in life) to severe (recognized at birth because of obvious deformity)

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Head and neck immobility</td>
<td>Decreased ROM of cervical spine, low posterior hairline, webbed neck, facial asymmetry, cleft palate, torticollis, vocal cord dysfunction</td>
<td>Flexion/extension radiographs of cervical spine</td>
<td>Consider MRI of cervical spine</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Bradyarrhythmias and AV conduction pathway abn (due to CNS malformations) Cardiac defects (most commonly VSD)</td>
<td>Syncope, Murmurs</td>
<td>ECG, ECHO</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Central alveolar hypoventilation, pulmonary agenesis or hypoplasia, restrictive lung disease (due to severe scoliosis)</td>
<td>Sleep apnea, snoring, difficulty breathing</td>
<td>ABGs, CXR (if symptomatic)</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Urinary tract abn, renal agenesis, ureteral duplication</td>
<td>Peripheral neurologic dysfunction (e.g., weakness, paresthesias, paraplegia, quadriplegia)</td>
<td>Neurologic exam</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Hindbrain abn (e.g., syringomyelia, Arnold-Chiari malformation) Mental retardation, deafness, strabismus</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Scoliosis, Sprengel's deformity (scapular elevation), hypermobility of C-spine, spondylosis/decreased mobility of C-spine</td>
<td>Exam of spine and shoulders</td>
<td>Radiographs if indicated</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Careful and complete evaluation of cervical spine anatomy and instability and of other major organ system abn

Monitoring
- Depends on pt's physical condition

Airway
- If indicated, awake intubation using maneuvers to stabilize cervical spine; complete immobility with use of fiberoptic intubating bronchoscope ideal

Preinduction/Induction
- Depends on pt's physical condition

Maintenance
- Careful positioning of head and neck with maintenance in neutral position

Extubation
- Depends on extent of cervical spine pathology and resp compromise

Adjuvants
- No special considerations

Anticipated Problems/Concerns
- Exacerbation of pre-existing cervical spine instability leading to neurologic deterioration
Latex Allergy

Robert H. Brown

**Risk**
- Myelomeningocele (25–50%)
- Congenital urologic anomalies (25–50%)
- Health care workers (3–17%)
- Atopic individuals (6–11%)
- General population (0–6%)

**Perioperative Risks**
- Anaphylactic reaction leading to hypotension, bronchospasm, CV collapse

**Worry About**
- A latex allergy is a Type I immediate hypersensitivity reaction. Life-threatening anaphylaxis can be the first manifestation of the reaction. Latex-containing medical products are common throughout most medical environments.

**Overview**
- Type I (immediate) hypersensitivity reaction: Immune mediated and involve IgE-specific latex proteins. Exposure can occur by either direct contact or through inhaled airborne particles. Symptoms can be localized or generalized, mild to life-threatening and incl pruritus, hives, angioedema, wheezing, hypotension, tachycardia, and CV collapse.
- Type IV (delayed or contact dermatitis) hypersensitivity reaction: Cell mediated, occurs 24–48 hr after exposure and is localized. Symptoms incl localized pruritus, swelling, and blisters.
- The increase in latex allergies coincided with the advent of universal precautions and the increased use of latex examination gloves, many with high allergen content.
- Exposure can occur both by contact and by inhalation of latex-containing powder.
- Considered to represent ~10% of all anaphylactic reactions reported for pts while under anesthesia.
- Increased risk with repeated exposures.
- Reaction caused by crosslinking latex specific IgE on mast cells leading to degranulation and release of both immediate and delayed inflammatory mediators.
- DX incl a Hx consistent with a latex reaction (e.g., time and exposure), nonspecific blood markers (e.g., serum mast cell tryptase), serology testing (radioallergosorbent [RAST] testing) and skin testing where available.

**ICD-9-CM Code: V15.07**

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypotension, tachycardia, CV collapse</td>
<td>Tachycardia, vasoconstriction</td>
<td>ECG, BP</td>
</tr>
<tr>
<td>PULM</td>
<td>SOB, stuffy nose, cough, elevated airway pressures</td>
<td>Wheezing, excessory muscle use</td>
<td>Spirometry, airway pressure</td>
</tr>
<tr>
<td>SKIN</td>
<td>Pruritus, edema</td>
<td>Hives, urticaria, erythema, swelling</td>
<td>Visual exam</td>
</tr>
<tr>
<td>EYES</td>
<td>Red, itching</td>
<td>Angioedema</td>
<td>Visual exam</td>
</tr>
<tr>
<td>GI</td>
<td>Cramps, N/V, diarrhea</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**
- Ask all pts about any Hx of an allergy or reactions to latex products.
- Do not attempt to prevent with premedications.
- Provide a latex-safe environment incl the pre-, intra- and postop environment.
- If latex allergic reaction suspected, make sure the postop environment is latex safe.

**Anticipated Problems/Concerns**
- Many latex-sensitized individuals are unaware of their allergic status.
- 10% of anaphylactic reactions under anesthesia are presumed due to a latex reaction.
- Maintain vigilance with regard to potential inadvertent latex exposures.
- Consider allergic reaction if hypotension is unresponsive to usual pressor agents.

**Etiology**
- Exposure with subsequent sensitization in at-risk individuals is the usual etiology of a latex allergy. At-risk individuals commonly have identified risk factors such as atopy, food allergies, and or a Hx of multiple surgeries.

**Usual Treatment**
- Avoidance of exposure should be the 1° consideration.
- There is no evidence any premedications can prevent or attenuate a Type I hypersensitivity reaction.
- In cases of an anaphylaxis reaction, treatment includes stopping the exposure, intravascular volume expansion, epinephrine as needed to support BP, bronchodilators to treat bronchospasm. Antihistamines and corticosteroids are distant 2° therapies.
- A latex-safe environment, one with minimal latex allergen, insufficient to elicit a latex allergic reaction, should be considered for all health care locations.
Lesch-Nyhan Syndrome

**Risk**
- X-linked recessive disorder (deficiency on the enzyme hypoxanthine-guanine-phosphoribosyltransferase (HGPRT), resulting in buildup of uric acid)
- Incidence ~5.2 per million male births (where symptoms appear)

**Perioperative Risks**
- Hyperuricemia and hyperuricosuria (gout)
- Airway problems 2° to scarification from self-mutilation (clip and finger biting)
- Involuntary writhing
- Repetitive movement of arms and legs
  - Impairment of renal function due to obstructive uropathy

**Worry About**
- Aspiration pneumonia (poor muscle control)
- May have associated megaloblastic anemia (poorly utilize Vitamin B₁₂)
- Drug metabolism and prolonged drug effects 2° to metabolic defect and impaired renal function

**Overview**
- Pts usually mentally subnormal
- Pts exhibit characteristic pattern of compulsive self-mutilation, spasticity, and choreoathetosis
- Primary biochemical defect is almost complete absence of HGPRT

**Enzyme defect leads to excessive purine production and elevated uric acid concentrations**

**ICD-9-CM Code:** 277.2

**Etiology**
- Genetic disease inherited as X-linked recessive trait (female carriers generally asymptomatic)

**Usual Treatment**
- No specific treatment of enzyme deficiency
- Benzodiazepines frequently used to control self-mutilation and spasticity (baclofen may be helpful)
- Gene therapy possibility
- Gabapentin
- Gout can be treated with allopurinol

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**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Distortion of airway structures due to self-mutilation</td>
<td>Examine airway</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Htn, CAD Adrenergic pressor response to stress is absent</td>
<td>Angina, angina-equivalent symptoms, PND</td>
<td>Displaced PMI S₃</td>
<td>ECG Pharmacologic stress testing Coronary angiography and ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Aspiration pneumonia</td>
<td>SOB following vomiting episode</td>
<td>Rales Wheezing</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Vomiting Athetoid dysphagia</td>
<td>Dysphagia</td>
<td>BUN Cr IVP</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Decreased renal function due to obstructive uropathy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Retardation Seizure disorders Decreased MAO activity</td>
<td>Mental status questioning EEG Mental function tests</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Spasticity, contractures ROM</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


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**Perioperative Implications**

**Preoperative Preparations**
- Antacids
- H₂ blockers
- Metoclopramide
- IV access may be difficult

**Monitoring**
- Routine
- ST-segment analysis if CAD present

**Airway**
- Rapid-sequence induction
- Avoid succinylcholine
- Awake fiberoptic intubation

**Preinduction/Induction**
- Premedication where appropriate to help with behavioral issues
- Avoid agents with renal metabolism (adjust dosing)

**Maintenance**
- Avoid agents with renal toxicity
- No one agent or technique shown superior
- Administer exogenous catecholamines with caution (due to assoc Htn)

**Exubation**
- Awake to avoid aspiration

**Adjuvants/Postoperative Period**
- Make some space accessible to avoid injury to child
- Benzodiazepines for spasticity

**Anticipated Problems/Concerns**
- Hx unavailable or inaccurate because of retardation
Leukemia

**Effect**
- Albumin
- Dysphagia
- Test

**Assessment by Hx**
- Renal failure from tumor lysis
- Airway assessment
- Ulceration, oral lesions
- Hepatosplenomegaly
- ↓ BUN/Cr

**Perioperative Risks**
- Neutropenic fever, opportunistic infection, sepsis, interstitial pneumonitis, acute resp failure and encephalopathy
- Hematoma and/or bleeding, diffuse alveolar hemorrhage from thrombocytopenia and splenic sequestration of pts

**Worry About**
- Bone marrow suppression with NO, potential for malignant hyperthermia in ALL, neuropathy, upper airway edema, anterior mediastinal mass, pleural effusion, and pulm fibrosis

**Overview**
- Hematologic malignancy with proliferation of cells may cause decrease in amino acids, causing fatigue and metabolic starvation
- Invasion possible in all organ systems
- Usually outpatient treatment, but may require several procedures incl bone marrow aspiration, central venous access placement, lumbar puncture, bronchoscopy, pericardiocentesis, external beam radiation

**ICD-9-CM Codes**
- 208.0 (Undifferentiated, acute, blastic); 204.0 (Lymphoblastic); 205.0 Myeloid leukemia

**Etiology**
- Unknown
- Strong suspicion that leukemia and lymphoma are virus-induced
- Chronic exposure to benzene (primarily from tobacco smoke), extraordinary doses of radiation, and certain cancer therapies, can be causes of the leukemia

**Treatment**
- Supportive treatment: Antimicrobial, blood transfusion, nutrition and pain control
- Newer approaches: Monoclonal antibody, experimental cancer vaccines, donor lymphocyte infusion, gene therapy, autologous and allogeneic transplantation, stem cell transplantation
- Treatment varies with type of leukemia, phase, age
- AML
  - Ara-C
  - Anthracyclines: Daunorubicin, idarubicin
- Gentuzumab ozogamicin: ATRA (All Trans Retinoic Acid)
- Arsenic trioxide: Vinca alkaloids: vincristine/vinblastine
- Bone marrow transplant
- CML
  - Imatinib mesylate (initial treatment of choice)
  - Nilotinib
  - Dasatinib
  - Busulfan
  - Hydroxyurea
  - Interferon alfa, allopurinol
  - Splenectomy, radiation, bone marrow transplant
- CLL
  - Cyclophosphamide
  - Corticosteroid
  - Fludarabine
  - Cytarabine
  - Bendamustine, rituximab
  - Alemtuzumab
- ALL
  - Imatinib, clofarabine, L-asparaginase, daunorubicin, vincristine, dexamethasone, doxorubicin, cytarabine (ara-C)
  - Radiation therapy, intrathecal chemotherapy

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRENT</td>
<td>Ulceration, oral lesions</td>
<td>Dysphagia</td>
<td>Airway assessment</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Rare: Pericardial effusion, conduction defects, murmurs, CHF</td>
<td>Dyspnea, fatigue</td>
<td>Narrow pulse pressure, pericardial friction rub, cardiomegaly</td>
<td>CXR, CT scan, ECG, ECHO</td>
</tr>
<tr>
<td>GI</td>
<td>Hepatosplenic enegia hypoalbuminemia</td>
<td>Loss of appetite wt loss</td>
<td>Hepatoplenomegaly</td>
<td>Albumin</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td>Weakness, easy fatigue, Pallor</td>
<td>Echymoses</td>
<td>CBC</td>
</tr>
<tr>
<td></td>
<td>Leukostasis</td>
<td>erosions, easy bruising, nosebleeds</td>
<td>Bone marrow aspirate results</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Thrombocytopenia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal failure from tumor lysis syndrome (acute loss of tumor)</td>
<td>↓ UO</td>
<td>↓ UO</td>
<td>BUN/Cr</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↑ Phosphate, ↑ or ↓ Ca↑↓ ↓ K↑</td>
</tr>
<tr>
<td>CNS</td>
<td>Cranial nerve infiltration (very rare), meningeal leukemia (less common in adults), vincristine neuropathy</td>
<td>Cranial nerve palsies, clouding of mental status, peripheral neuropathy</td>
<td>Weakness</td>
<td>EMG</td>
</tr>
<tr>
<td>MS</td>
<td>Infiltration of bony cortex and periosteum, synovial membranes</td>
<td>Bone pain</td>
<td>Bone swelling</td>
<td>X-ray</td>
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<td></td>
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<td>CT scan</td>
</tr>
</tbody>
</table>

Lipidemias

Overview
- Hypertriglyceridemia, hypercholesterolemia lipodystrophy: Köbberling-Dunnigan syndrome (familial lipodystrophy of limbs and trunk, autosomal dominant, may lead to macrosomia); familial generalized lipodystrophy (Berardinelli-Seip syndrome: Autosomal recessive, leads to macrosomia)
- Hypolipidemia: LDL deficiency (autosomal recessive abetalipoproteinemia, autosomal dominant familial hypobetalipoproteinemia); normotriglyceridemic abetalipoproteinemia (LDL absent); autosomal recessive Tangier disease (severe deficiency of HDL); 2° to cancer, myeloproliferative disorders, liver failure familial hypoalphalipoproteinemia (HDL deficiency)
- Transient ischemic attacks of CNS
- Worsening or new-onset CHF
- Peripheral atherosclerosis
- Angina of increasing frequency or severity and new-onset angina
- Family Hx of premature CHD in first degree relative (male <55 y or female <65 y)
- Hypothyroidism, nephrotic syndrome, and extrahepatic obstruction of bile

Perioperative Risks
- Pancreatitis with hypertriglyceridemia
- Stroke and transient ischemic attacks
- Myocardial ischemia, infarction, CHF

Perioperative Implications
Preoperative Preparation
- Assess for CAD and peripheral vascular disease
- 2° to systemic illness (i.e., primary hypothyroidism, nephrotic syndrome, and extrahepatic obstruction of bile)

Maintenance
- Avoid hypothermia and anemia
- Monitor for ischemia and cardiac failure
- Insulin increases activity of lipoprotein lipase and releases free fatty acids (FFAs)
- Sympathetic stimulation, stress, and catecholamines release FFAs
- Spinal or epidural anesthesia and β-blockers reduce FFA levels
- Heparin releases two triglyceride hydrolases: Lipoprotein lipase inhibited by protamine and hepatic lipase resistant to protamine

Extubation
- During noncardiac surgery, this may be period of greatest risk for ischemia
- Treat pain, hemodynamic, and biochemical abn

Adjuvants
- Depends on lipid-drug binding and end-organ disease

Postoperative Period
- High incidence of ischemia, tachycardia, and MI for several days after noncardiac surgery
- Concerns are related to issues associated with atherosclerosis

Ludwig’s Angina

Risk
- Odontogenic infections esp. of second and third molars (account for 90% of all cases). Dental and gingival disorders, bacterial infection of floor of the mouth, peritonsillar abscess, IV drug abuse, mandibular fracture, tongue piercing, sialadenitis, puncture wounds of floor of mouth. Predisposing factors: DM, alcoholism, acute glomerulonephritis, SLE, and aplastic anemia

Perioperative Risks
- Airway obstruction, aspiration pneumonia, sepsis, descending mediastinitis, subphrenic abscess, empyema, cervical or mandibular osteomyelitis

Worry About
- Airway obstruction, sepsis, jugular vein thrombosis, pneumothorax, pericardial/pleural effusion, infection of carotid sheath structures, descending, necrotizing mediastinitis occurring through the retropharyngeal space and carotid sheath

Overview
- Potentially lethal, rapidly spreading cellulitis of the sublingual and submandibular spaces. Five characteristics: Submandibular cellulitis; involvement of more than one space; progression of cellulites to gangrene; progression of cellulites to connective tissue, fascia, and muscles; and spread of cellulites by continuity and not via the lymphatics. Infection often starts as a periapical dental abscess of the third and fourth mandibular molars (the roots of these teeth penetrate the mylohyoid ridge such that any abscess or dental infection has direct access to the submaxillary space) usually with elevation and posterior displacement of the tongue.
- Presents with painful neck swelling, laryngeal edema, tooth pain, dysphagia, dyspnea, fever, and malaise. Neck swelling and protruding or elevated tongue are seen in the vast majority. Stridor, trismus, cyanosis, and tongue displacement suggest impending airway crisis.

ICD-9-CM Code: 641.2

Perioperative Implications

Preinduction/Induction
- Fully developed Ludwig’s: ET intubation is associated with high rate of failure with acute deterioration in resp status resulting in emergency slach tracheostomy.
- Elective, awake tracheostomy using local anesthesia is the preferred method of airway management in pts with fully developed Ludwig’s.
- In cases not fully advanced, awake nasal fiberoptic is the logical choice.

- The use of 10 mg of dexamethasone initially and 4 mg every 6 hours helps to decrease edema and cellulites. Nebulized adrenaline (1 mL of 1:1000 diluted to 5 mL of 0.9% saline) is also believed to help relieve upper airway obstruction. Pt must be maintained in sitting position and surgeon should be immediately available for tracheostomy.
- The first airway should be the definitive airway and induction should occur after airway has been secured. For mild cases that have not progressed, one may elect to do an inhalational induction but the majority of cases require awake intubation and then induction.

Monitoring
- Large-bore access should be obtained. Central line is not be advised with involvement in the neck.

Maintenance
- Avoid NO in case of pneumothorax.

Anticipated Problems/Concerns
- Blind nasotracheal intubation should not be attempted in Ludwig’s, given the potential for bleeding and abscess rupture.

DISEASES

## Lyme Disease

### Risk
- Accounts for >95% of all reported cases of vector-borne illness in USA; it is by far the most common arthropod-borne infection in USA.
- During 1992–2006, 93% of cases were reported from 10 states (Connecticut, Delaware, Massachusetts, Maryland, Minnesota, New Jersey, New York, Pennsylvania, Rhode Island, and Wisconsin).
- Incidence was highest among children aged 5–14 y; a disproportionate increasing trend was observed in children and in young males compared with other demographic groups. The majority of pts had onset in June, July, or August.
- Gender predilection: Male (53.4%)
- Children <15 y; adults 30–59 y

### Perioperative Risks
- Increased risk of dysrhythmias and CHF in pts with cardiac involvement

### Worry About
- CV: Volume overload, CHF, and AV block
- Neuro: Hyperkalemia from muscular weakness or paralysis, facial muscle paralysis (Bell's palsy), peripheral neuropathy and muscle weakness, meningitis, and confusion

### Overview
- Stage 1, Early localized infection: Chills, fever, headache, lethargy, muscle pain, erythema migrans (rash spreads centrifugally; lesion usually occurs at site of bite) in 68% of pts.
- Stage 2, Early disseminated infection: Arthritis, aseptic meningitis, cranial neuritis (Bell’s palsy), and peripheral radiculoneuritis are neurologic manifestations.
- Carditis occurs in 4–8% of pts during this stage of disease.
- Second and third degree AV block and myocardiitis may be documented by ECG and heart failure; symptoms resolve in days to weeks.
- Stage 3, Late persistent infection: Over years 60% of pts with untreated infection will begin to have intermittent bouts of arthritis, with severe joint pain and swelling. Large joints are most often affected, particularly the knees. In addition, up to 5% of untreated pts may develop chronic neurologic complaints months to years after infection.

### Etiology
- Lyme disease is caused by the spirochete *Borrelia burgdorferi*, which is transmitted by the tick *Ixodes dammini*.

### Usual Treatment
- Early doxycycline prevents infection in high percentages, amoxicillin, cefuroxime, and ceftriaxone. Pts with certain neurologic or cardiac forms of illness may require IV treatment with drugs such as ceftriaxone. Antibiotic therapy for 10–21 up to 28 d generally aborts stages 2 and 3. Pts may benefit from a second 4-wk course of therapy.
- Vaccine available for adults

### ASSESSMENT POINTS

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<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>AV node block</td>
<td>Palpitations</td>
<td>Bradycardia</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>CHF</td>
<td>Fatigue</td>
<td>Tachycardia</td>
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<tr>
<td></td>
<td></td>
<td>Dyspnea</td>
<td></td>
<td>ECHO</td>
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<tr>
<td></td>
<td></td>
<td>Dizziness with exercise</td>
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</tr>
<tr>
<td>RESP</td>
<td></td>
<td></td>
<td>Rales</td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Erythema chronic migrans</td>
<td>Erythematous annular lesions</td>
<td>Erythematous circular rash</td>
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<tr>
<td>CNS</td>
<td>Meningitis</td>
<td>Headache</td>
<td>Cranial nerve facial palsy</td>
<td>Serology</td>
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<tr>
<td></td>
<td>Bell's palsy</td>
<td>Cognitive impairment</td>
<td>Numbness, tingling</td>
<td>Lumbar puncture</td>
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<tr>
<td></td>
<td>Radiculoneuritis</td>
<td>Memory deficit</td>
<td>Muscular weakness</td>
<td>EMG</td>
</tr>
<tr>
<td>MS</td>
<td>Arthritis</td>
<td>Joint pain and swelling</td>
<td>Swelling of one or a few joints</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Musculoskeletal pain</td>
<td>Erythema of joints</td>
<td></td>
</tr>
</tbody>
</table>

### Perioperative Implications

#### Preoperative Concerns
- Ensure antibiotic Rx and cure of carditis prior to all but life-or-death emergency operations.

#### Monitoring
- Consider invasive monitoring with an arterial line based on cardiac manifestations.

#### Airway
- Routine

### Postoperative Period
- Depends on the cardiac and neurologic manifestations of the disease if present.

### Anticipated Problems/Concerns
- Pts may develop arrhythmias or CHF.
Lymphomas

Risk
- Hodgkin’s disease (HD) and non-Hodgkin’s lymphoma (NHL) are most common hematologic malignancies in the USA.
- HD and NHL represent 4–5% all new cancers
- Race with highest prevalence of HD and NHL: Caucasian
- In past 40 y, striking↑ in incidence of NHL partly due to lymphoma in AIDS pts

Perioperative Risks
- Morbidity and mortality related to compression of organs and chemotherapy
- Mediastinal mass
- Superior vena cava syndrome; anthracycline cardiac toxic effects
- Bleomycin pulm toxic effects
- Pericardial effusion
- Radiation pneumonitis

Worry About
- Tracheal or bronchial compression by large mediastinal mass
- Increased cardiac/pulm toxic effects with combination chemotherapy/radiation therapy (RT)

Overview
- Two major types of lymphoma: HD and NHL, many subtypes
- Seventh most common cause of cancer-related death in the USA
- Third most common childhood malignancy
- Average age at diagnosis: 42 y
- Often curable
- Accurate Dx and staging critical in determining Rx and prognosis

ICD-9-CM Codes: 200–202.8

Etiology
- HD: Pathogenesis remains obscure, genetic predisposition, increased risk with inherited immunodeficiency syndromes, EBV, increased educational level
- NHL: Pathogenesis involves clonal malignant expansion of B or T cells, increased risk with congenital and acquired immunodeficiency states, autoimmune disorders, infectious agents, phenoxyherbicides, organophosphates, ionizing radiation

Usual Treatment
- Diagnostic laparoscopy or laparotomy no longer routine, reserved for pts with limited disease, treated with RT alone.
- RT
- Chemotherapy with multiple agents
- Combination RT and chemotherapy
- Chemotherapy commonly incl: Bleomycin, doxorubicin, prednisone, etoposide, vincristine, cyclophosphamide
- RT commonly incl neck, chest
- Both chemotherapy and RT have cardiac and pulm toxic effects
- Advanced or recurrent HD: Treatment with biologies (e.g., radiolabeled immunoglobulin therapy)

ASSESSMENT POINTS

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<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Bulky nodal disease</td>
<td>SOB, DOE</td>
<td>Neck mass</td>
<td>Indirect laryngoscopy</td>
</tr>
<tr>
<td></td>
<td>Compression</td>
<td>Tracheal deviation</td>
<td>Wheeze, stridor</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>MRI</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Mediastinal mass</td>
<td>SOB, DOE</td>
<td>Facial swelling, wheeze, may be asymptomatic</td>
<td>CXR, CT/MRI</td>
</tr>
<tr>
<td></td>
<td>SVC syndrome (SVC obstruction)</td>
<td>Cough, orthopnea</td>
<td>Dilated veins upper half of body</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mental status change</td>
<td>Edema of head, neck, and upper extremities, cyanosis</td>
<td>CT of airway</td>
</tr>
<tr>
<td>RESP</td>
<td>Pericardial effusion</td>
<td>Frequently asymptomatic</td>
<td>↑ HR, ↓ BP, neck vein distention</td>
<td>CXR, ECHO</td>
</tr>
<tr>
<td></td>
<td>CHF due to anthracyclines</td>
<td>SOB, DOE</td>
<td>Rales, pedal edema</td>
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</tr>
<tr>
<td></td>
<td>Bronchial compression</td>
<td>Cough</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Obstructive pneumonia</td>
<td>Wheeze</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Pneumonitis due to bleomycin and/or RT</td>
<td>Sx worse in supine position</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Fever, cough</td>
<td></td>
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</tr>
<tr>
<td>GI</td>
<td>Abdominal mass</td>
<td>Abdominal pain, GI bleeding</td>
<td>Palpable mass</td>
<td>CT/MRI</td>
</tr>
<tr>
<td></td>
<td>Upper/lower GI bleed</td>
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<td>Perforated viscus</td>
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<td>HEME</td>
<td>Bone marrow involvement</td>
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<td>Alk phos, CBC, plt, bone marrow biopsy</td>
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<td>CNS</td>
<td>Leptomeningeal disease or single or multiple mass lesions</td>
<td>Headache</td>
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<td>Cranial nerve abn</td>
<td>Abnormal neuro exam</td>
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<td>Spinal tap</td>
<td>CT/MRI</td>
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<tr>
<td>RENAL</td>
<td>Ureteral compression</td>
<td></td>
<td></td>
<td>IVP, BUN/Cr</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Assess extent bulky nodal disease causing upper or lower airway and/or cardiac compression
- Assess LV function after anthracyclines
- Assess pulm function after bleomycin, RT
- If large mediastinal mass, use local anesthetic if possible

Monitoring
- Routine

Airway
- Routine, unless large anterior mediastinal mass calls for awake fiberoptic intubation
- Use armored ETI
- Rigid ventilating bronchoscope on hand

Induction
- If large mediastinal mass: Consider awake fiberoptic intubation, maintaining spontaneous ventilation, and semi-Fowler’s position

Maintenance
- Spontaneous ventilation as above; avoid muscle relaxants
- Choose shortest acting agents for rapid wakeup
- After bleomycin use lowest FIO2, possible

Exubation
- If mediastinal mass, extubate pt awake and breathing spontaneously and have rigid ventilation bronchoscope on hand

Adjuvants
- If asymptomatic with mediastinal mass, airway obstruction and/or cardiac compression may develop on induction

Postoperative Period
- Airway obstruction if mediastinal mass: Observe longer in intensive nursing setting
- Monitor fluid status if significant LV dysfunction

Anticipated Problems/Concerns
- If bulky nodal disease in neck and chest, at risk for SVC syndrome, difficult airway, and tracheobronchial compression on loss of spontaneous ventilation
- May have significant cardiac/pulm impairment due to combination chemotherapy and RT
Malignant Hyperthermia (MH) and Other Anesthetic-Induced
Myodystrophies (AIM)

**Risk**
- Incidence of MH: 1/15,000–20,000 anesthetics in children; 1/50,000–100,000 in adults depending on use of trigger agents, gene pool
- Male > female
- Family Hx of MH or unexplained death may predict MH susceptibility.
- Improved outcome by avoiding trigger agents in MH susceptible, availability of dantrolene, and using succinylcholine on indication

**Perioperative Risks**
- Mortality with MH in North America <10%, when event in a hospital; ~20% when pt transferred into a hospital; untreated, mortality >80%
- Mortality with other AIMs unknown
- Masseter muscle rigidity (MMR)—10–20% of pts experiencing MMR develop clinical MH;
  generalized rigidity predicts clinical MH in >60%
- Central core myopathy—very high risk for MH
- Multiminicore is also associated with MH susceptibility
- Hyperkalemia and cardiac arrest with Duchenne, Becker's dystrophy when succinylcholine used and sometimes with volatile agents only
- Certain forms of myotonia lead to risk for MH and/or hyperkalemia with succinylcholine
- Muscle rigidity common with all myotonia when succinylcholine used

**Worry About**
- Unexplained tachycardia, tachypnea elevated ET CO₂ during anesthesia
- Potent volatile anesthetics and succinylcholine contraindicated in MH and pts with AIM
- Availability of dantrolene
- Purge machine with 100% O₂ 15–20 min prior to case. Newer anesthesia workstations (e.g., Drager Fabius) require longer period of purging—up to 60 min.

**Recrudescence of MH in 25% of cases despite treatment**
- If working in ambulatory center, prearranged transfer protocol and blood gas analysis
- Counseling family regarding risk, muscle biopsy and genetic testing

**Overview**
- Malignant hyperthermia (MH)
- Autosomal dominant myopathy in humans
- No phenotypic signs predict MH other than previous Hx of MH or family Hx or unexplained elevated CK.
- Hypermetabolic disorder manifested by ↑ CO₂ production/O₂ consumption, acidosis, hyperkalemia, myoglobinuria/emia, tachycardia, tachypnea, increased ET CO₂
- Dantrolene is the only specific treatment
- Dx by halothane/caffeine contracture test of biopsied muscle is most sensitive and specific.
- DNA testing available in two laboratories in the USA and in many centers in Europe. Pts must be selected. Sensitivity is ~30%, specificity close to 100%.
- Pts with MD/myotonia may develop hyperkalemic arrest with succinylcholine and occasionally with potent volatile only
- Signs of dystrophy subtle or not apparent in young children
- Obtain muscle specimens for dystrophin analysis; genetic testing if cardiac arrest
- Test for CK elevation in suspicious cases
- Information for provider and pt available through MHAUS, the Malignant Hyperthermia Association of the US, Sherburne N.Y. www.mhaus.org, 607–674–7901
- MH hotline 1-800-MH-HYPER

**ICD-9-CM Code:** 995.86 (MH) Other AIMs

**Etiology**

**MH**
- Defect in intracellular calcium release/control in skeletal muscle leads to ↑ intracellular calcium
- Heterogenous predisposition
- Ryanodine receptor of skeletal muscle is defective in 70% of cases. Dihydropyridine receptor (DHPR, the CACNA1S locus) in about 1% of cases. Unknown loci, the remainder.
- RYR-1 gene on chromosome 19. Over 120 mutations of which 29 have been shown to be causal. The others are being clarified.

**Other AIMs**
- Muscular dystrophies: X-linked inheritance, several mutations
- Myotonia: Genetic abn of sodium, chloride channels, or protein kinase, linked to chromosomes 19, 17, others
- Central core disease: In most families, linked to ryanoide receptor
- Pts with CPT-2 deficiency may develop rhabdomyolysis with MH triggers.

**Usual Treatment**

**MH**
- D/C triggers
- Hyperventilate pt with 100% O₂
- Dantrolene 2.5 mg/kg IV; may use more to treat acute episode
- Treat metabolic acidosis; actively cool
- Increase fluids 1/2 to 2 × maintenance
- No calcium-channel blockers
- Maintain UO 1–2 mL/kg, diuretics if necessary
- Assess for hyperkalemia and treat appropriately
- Coagulation profile, DIC a problem
- Continue treatment for at least 36 hr at 1–2 mg/kg /4–6 hr

**Other AIMs**
- Treat for hyperkalemia

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx and PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Masseter muscle rigidity</td>
<td>Difficult intubation</td>
<td>ABGs/acidosis, Hypercarbia, Myoglobinuria</td>
</tr>
<tr>
<td>CARDIO</td>
<td>MH: Tachycardia, arrhythmias</td>
<td>Hyper/hypotension</td>
<td>Mixed venous and ABGs: ↑ ET CO₂, myoglobinuria, hyperkalemia</td>
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<tr>
<td>AIM: Sudden bradycardia VFIB, asystole</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Tachypnea</td>
<td>Tachypnea</td>
<td>↑ ET CO₂</td>
</tr>
<tr>
<td>MS</td>
<td>Generalized rigidity</td>
<td>Developmental delay</td>
<td>CK Muscle biopsy contracture test and histology DNA testing</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal failure</td>
<td>Low UO Dark urine</td>
<td>Myoglobin in serum and urine, serum potassium</td>
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<tr>
<td>SKIN</td>
<td>Vasconstriction</td>
<td>Mottled appearance (late)</td>
<td>Core temp</td>
</tr>
<tr>
<td>Heat</td>
<td></td>
<td>Hot skin Sweating</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** The caffeine/halothane contracture test is used to assess MH susceptibility.

Perioperative Implications

**Perioperative Preparation for Known MH**
- Avoid triggers (succinylcholine, all potent volatile agents)
- Use local anesthesia (amides and ester OK)
- Regional anesthesia (epidural, spinal, regional block)
- General: All following drugs are not triggers: pentothal (barbiturates), etomidate, ketamine, propofol, NO, all nondepolarizing muscle relaxants, narcotics, benzodiazepines. Suggest TIVA
- Anesthesia machine
  - Change circuit and bag
  - Remove and/or drain vaporizers
- \( \text{O}_2 \) flow at 10 L/min for 15–20 min prior to use
- Newer anesthesia workstations (e.g., Fabius) require up to 60 min of purging
- Dantrolene prophylaxis not necessary
- Dantrolene and calcium-channel blockers together produce hyperkalemia

**Monitoring**
- Routine incl ETCO\(_2\), core temp (e.g., esophageal, axillary, bladder, pulm artery)

**Perioperative Implications, Other AIMS**
- Some, not all, pts with DMD and Becker’s dystrophy will develop hyperkalemia with MH triggers
- Avoid succinylcholine in pts with myotonia and most other myopathies and neuromuscular disorders

**Anticipated Problems/Concerns**
- Sudden cardiac arrest in PACU
- Myoglobinuria, renal failure
- Postop rhabdomyolysis, follow CKs
- Hyperkalemia
- Postop muscle pain/weakness and persistently elevated CK
- Have pt enter the North American MH Registry of MHAUS. MHreg.org
Malnutrition

Risk
- Rate about 4% in general population, 10–20% in surgical pts, and rises to 40% or more in severely ill hospital admissions
- Risk increases with severity of underlying disease, presence of malignancy (esp. GI), and advancing age
- Hospitalized pts lose an average of 5% body wt during time of admission

Perioperative Risks
- Post complications are significantly higher in the malnourished.
- Severe undernutrition may result in CHF, resp failure, and immunologic dysfunction.

Worry About
- Need for early postop nutritional supplementation, particularly enteral if possible.
- Infection risk: Care should be taken with invasive procedures and sterile technique.
- Intraop problems may incl low cardiac output and resp failure.

Overview
- Results from inadequate intake of macronutrients (carbohydrate, protein, fat); referred to as protein-calorie malnutrition (PCM)
- There are two types of PCM:
  - Marasmus form (MF-PCM), which results in uniform loss of fat and muscle mass in all tissues and a concomitant loss of H2O in proportion to nonaqueous mass
  - Stress-induced hypoalbuminemic form of protein-calorie malnutrition (HAF-PCM), which results from neurohumoral modulation leading to depletion of visceral protein (in excess of muscle mass) and fat and is assoc with an expansion of extracellular fluid compartment. Stress may be surgery, infection, inflammation, trauma, neoplasia.
  - In hospitalized pts, marasmic-kwashiorkor type (i.e., wasting of muscle and fat with hypoalbuminemia) is most common.

ICD-9-CM Code: 263.9 (Unspecified protein-calorie malnutrition)

Etiology
- Decreased dietary intake: Advanced age, physical debilitation, GI-related illnesses, neck mass
- Increased metabolic demands and nutrient loss: Stress (physical and psychological), disease states (particularly GI and resp illness like emphysema), infections, burns, liver failure
- Conditions assoc with N/V
- Malignant conditions, esp. those involving the GI tract

Usual Treatment.
- Early PO intake postop is advantageous, esp. in GI malignancies; enteral intake reduces infections
- Enteral nutrition via g-tube or j-tube preferable to TPN if direct PO intake not possible but gut can still be utilized (e.g., esophageal surgery)
- TPN value is inconclusive, but probably indicated in severe malnutrition states. TPN reduces noninfectious complications but increases infectious complication rates in most studies.

Perioperative Considerations

Preinduction
- Use of a malnutrition risk assay (e.g., Nutrition Risk Score) for screening will help identify at-risk pts
- Nutritional and caloric supplementation in the days before surgery may be beneficial if possible
- Consider prophylaxis for aspiration of gastric contents if GI process is responsible for malnutrition (malignancy, obstruction, etc).

Monitoring
- Routine

Induction
- If resp muscle weakness/fatigue is suspected, avoidance of long-acting NMDBs may be prudent.

Maintenance
- Pts receiving TPN should continue to receive it in the OR as abrupt discontinuation may result in severe hypoglycemia. Many sources recommend a rate reduction of 30% intraop. Alternatively, TPN may be replaced with dextrose during surgery.
- Standard hydration and UOP monitoring

Extubation
- Resp muscle failure may preclude early extubation; careful attention to resp status warranted in PACU

Adjuvants
- Hepatic drug metabolism may be impaired
- Decrease binding (volume of distribution) of protein-bound drugs in hypoalbuminemic pts

Anticipated Problems/Concerns
- Because edema is prominent feature of HAF-PCM, interpretation of anthropometric measurements like arm circumference may be unreliable.
- Serum markers like albumin, transferring, and prealbumin can be unreliable in a wide array of disease states and do not correlate well with outcomes and complications.
- Pts with end-stage chronic obstructive lung disease usually have malnutrition, and sudden feeding periopt may precipitate acute resp failure and re-feeding syndrome.


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Marfan’s Syndrome

Overview
- Familial disorder of connective tissue (CT) underlying defect of collagen synthesis decreases tensile strength and elasticity of CT. Involves skeletal system, skin, fascia, lungs, skeletal muscle, CNS, and adipose tissue.
- Common causes of death are CV: Aortic dilatation, dissection, or rupture; aortic or mitral valvular regurgitation; coronary artery insufficiency
- Airway features: High-arched palate, facies dolichocephaly (long narrow skull), malar hypoplasia (an underdeveloped mid-face), retrognathia
- Skeletal features: Increased length of long bones, joint laxity, scoliosis, pectus excavatum and carinatum, possible laxity of cervical spine, hernia, lumbar sacral dural ectasia
- Pulm: Spontaneous pneumothorax, restrictive lung disease with thoracic deformity, and obstructive problems during sleep due to laxity of soft tissue
- Ocular: Lenticular subluxation or dislocation, flat cornea, increased globe axial length (myopia), hypoplastic iris or ciliary muscle, enophthalamos

ICD-9-CM Code: 759.82

Risk
- Prevalence is 2–3/10,000 population
- Inherited as autosomal dominant trait
- 25% of cases are sporadic due to de novo mutations

Perioperative Risks
- Aortic arch dissection, MVP, mitral or aortic valve regurgitation, coronary artery abn, cardiac arrhythmias, pneumothorax, restrictive lung disease

Worry About
- Symptoms referable to progressive dilatation or rupture of ascending thoracic aortic aneurysm (e.g., chest pain radiating to interscapular region)
- Symptoms of mitral (midysyctic click) or aortic valvular insufficiency
- Myocardial ischemia (angina) due to medial necrosis of coronary arterioles
- Arrhythmias and conduction disturbances (palpitations)
- SOB (dyspnea) due to restrictive lung disease

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by HX</th>
<th>PE</th>
<th>Test</th>
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<tbody>
<tr>
<td>HEENT</td>
<td>Lens dislocation</td>
<td>Myopia</td>
<td>Retinal detachment</td>
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<td>Aortic dissection</td>
<td>Chest pain</td>
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<td>CV</td>
<td>Myocardial ischemia</td>
<td>Palpitations</td>
<td>Pulse</td>
<td>ECG, Radionuclide studies</td>
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<td>RESP</td>
<td>Restrictive lung disease</td>
<td>Dyspnea</td>
<td>Pectus scoliosis</td>
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<td>Tall stature</td>
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<td>Arm span:height ratio &gt;1.05</td>
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<td>Joint hypermobility</td>
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<td>CXR</td>
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<td>Hernias</td>
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Perioperative Implications

Preoperative Preparation
- Consider antibiotics for SBE prophylaxis.
- Consider preop β-blockade Rx to mitigate increases in myocardial contractility and aortic wall tension (dP/dT).

Monitoring
- ST-segment analysis; QT interval analysis, consider TEE
- Invasive monitoring as appropriate for planned surgery

Airway
- High-arched palate
- Potential cervical laxity/instability
- Potential for TMJ dislocation with direct laryngoscopy

Preinduction/Induction
- Avoid sudden increases in aortic wall tension
- Careful positioning to avoid dislocations

Maintenance
- No one technique has demonstrated superiority

Exubation
- Avoid sudden increases in CO, BP as this may increase dP/dT
- High risk for developing myocardial ischemia

Adjuvants
- Adequate pain management is important
- May require ↑ doses of local anesthetic due to ↑ size and enlargement of the neural canal

Anticipated Problems/Concerns
- CV: Aortic dissection, MVP, mitral or aortic regurgitation, myocardial ischemia, cardiac arrhythmias
- Resp: Pneumothorax, restrictive lung disease with thoracic deformity

Perioperative Risks
- Potential for TMJ dislocation with direct laryngoscopy
- Potential cervical laxity/instability
- High-arched palate
- Harrington rod placement

Assessment by HX
- Restrictive lung disease with thoracic deformity, and obstructive problems during sleep due to laxity of soft tissue
- Ocular: Lenticular subluxation or dislocation, flat cornea, increased globe axial length (myopia), hypoplastic iris or ciliary muscle, enophthalamos

Anticipated Problems/Concerns
- CV: Aortic dissection, MVP, mitral or aortic regurgitation, myocardial ischemia, cardiac arrhythmias
- Resp: Pneumothorax, restrictive lung disease with thoracic deformity
Mastocytosis

Risk
• Cutaneous mastocytosis (CM) is primarily a disease of children and affects 1 in 1000 to 1 in 8000.
• Systemic mastocytosis (SM) is more prevalent in adults and affects between 1 in 10,000 and 1 in 80,000.
• Equal male and female prevalence

Perioperative Risks
• Increased risk of hypotension and bronchospasm as a consequence of paroxysmal release of mast cell mediators.
• Anesthetic drugs and procedures may induce mast cell degranulation.
• Mast cell degranulation may present as an anaphylactic shock with CV collapse. Fatal cases have been reported.

Worry About
• Increased risk of hypotensive shock and bronchospasm.
• Clotting factors may be disturbed as result of vitamin malabsorption, hepatic fibrosis, and massive heparin release from mast cells (uncommon).
• Profound CV collapse and death without signs of flushing or bronchospasm

Overview
• Group of rare mast cell proliferative disorders.
• CM is limited to the skin, SM may involve the bone marrow or other non-cutaneous organs, and may or may not affect the skin.
• CM usually presents in childhood and often resolves by adolescence.
• CM is classified as urticaria pigmentosa, diffuse cutaneous mastocytosis, or solitary mastocytoma.
• SM is more common in adults and is identified as indolent systemic mastocytosis, smoldering systemic mastocytosis, systemic mastocytosis with another associated non–mast-cell hematologic lineage disorder, aggressive systemic mastocytosis, and mast cell leukemia.
• Mast cells release histamine, heparin, leukotrienes and various cytokines.
• Degranulation can be caused by certain medications, physical pressure, extreme temp, spicy food, ingestion of hot beverages, alcohol, and emotional upset.
• Medications to be avoided: Morphine, atracurium, mivacurium, rocuronium, NSAIDs, vancomycin.
• Thorough pt trigger Hx is critical.
• Lower risk medications incl fentanyl, sulfentanil, remifentanil, acetaminophen, cisatracurium, midazolam.
• Avoid aspirin in those with no exposure Hx, but for those who can tolerate aspirin, its ability to block prostaglandin D, is a potential benefit in SM.
• Volatile anesthetics do not cause histamine release.
• For many the initial manifestation is a cutaneous eruption.
• Common symptoms incl episodic flushing, headaches, N/V, diarrhea, fatigue.
• May see vascular collapse with syncope and palpitations, abdominal pain, wheezing.
• Serum tryptase levels correlate with total body mast cell burden.
• Pts with systemic disease at greater risk for CV collapse.
• Main concern: To avoid mast cell degranulation.
• Rare: Mast cell lymphoma/leukemia; rule out carcinoid syndrome as cause of symptoms (elevated urinary 5-HIAA)

Perioperative Implications

Preoperative Preparation
• Refrain from ethanol, NSAIDs, aspirin (unless known tolerance for aspirin).
• Premedication with histamine-releasing drugs should be avoided.
• Diazepam, midazolam premedication—reported to be safe.
• Start prophylactic H2 and H2 blockers.
• May give diphenhydramine, 25–50 mg PO 1 hr prior, ranitidine 150 mg PO 1 hr prior, montelukast 10 mg PO 1 hr prior, prednisone 50 mg PO 24 hr and again 2 hr prior to procedure.
• Prophylactic cromolyn sodium: Yet to be confirmed (100 mg q6h).
• Predictive prick tests for drugs such as muscle relaxants are inconclusive because metabolites not seen in skin tests may cause degranulation.
• Serum tryptase levels correlate with mast cell burden.

Monitoring
• Routine monitors.
• Intra-arterial catheter (sudden BP changes).

Airway
• Intubation may be dangerous in the presence of mucosal lesions, as pressure can cause degranulation and bronchospasm or hypotension.

Preinduction/Induction
• Avoiding atropine, scopolamine, and sodium thiopental has been recommended.
• Midazolam and fentanyl both with good safety records.
• Muscle relaxants: Cisatracurium recommended.
• Avoid atracurium, mivacurium, rocuronium, dintubocurarine.
• Inhalational agents: Safe (may even increase mast cell stability).

Maintenance
• Maintain normothermia.

Assessment by Hx

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Rhinorrhea</td>
<td>Allergic rhinitis</td>
<td>Wheezing</td>
<td>Episodic elevations of plasma histamine levels, serum tryptase levels</td>
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<tr>
<td>CV</td>
<td>Episodic CV collapse</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Asthma</td>
<td>Wheezing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Malabsorption, GI bleeding</td>
<td>Abdominal pain, N/V, diarrhea</td>
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<tr>
<td>HEME</td>
<td>Anemia, thrombocytopenia, leukenia, mast cell leukemia, excess bone marrow blasts</td>
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<td>Mastocytoma/urticaria pigmentosa; Telangiectasia macularis eruptive perstans (TMEP)</td>
<td>Pruritis, urticaria</td>
<td>Skin biopsy</td>
<td>(+ Giemsa)</td>
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<td>CNS</td>
<td>Polyneuropathy</td>
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<td>CNS exam</td>
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<tr>
<td>MS</td>
<td>Bone pain</td>
<td></td>
<td>X-ray, 99mTc bone scan</td>
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</tbody>
</table>


Key Reference: Meenakshi Dogra, Jeremy L. Gibson

ICD-9-CM Codes 202.6 (Mastocytosis); 238.5 (Mastocytoma); 757.33 (Mastocytosis syndrome)

Etiology
• Pathogenesis incompletely understood but frequently involves mutations in the tyrosine kinase receptor c-kit. It is a mast cell proliferative disorder.

Usual Treatment
• H2 blockers, e.g., diphenhydramine, chlorpheniramine maleate, hydroxyzine, terfenadine.
• H1 blockers, e.g., cimetidine, ranitidine.
• Aspirin to control flushing (if pt has a past Hx of safe aspirin use) to block PGD2 synthesis.
• Proton pump inhibitors if H1 blockers ineffective for abdominal symptoms.
• Antileukotriene inhibitors, e.g., montelukast, zafirlukast.
• Cromolyn sodium for GI symptoms.
• Ketotifen mast cell stabilizing and H1 antihistamine (Canada and Europe).
• Shock: IV epinephrine (Adults: 2–10 μg/min infusion; children: 0.1 to 1 meg/kg/min infusion) and volume repletion.
• EpiPen and epinephrine inhalers should be carried by pts with known or suspected disease.
• PUVA (psoralens plus ultraviolet A) for cutaneous manifestations.
• Steroids: Topical and systemic.
• Chemotherapy.
• Splenectomy for hypersplenism with anemia and thrombocytopenia.
• Prostamide sulphate rarely necessary when endogenous heparin prolongs prothrombin time.

• Hypotension due to histamine release: IV epinephrine, (Adults: 2–10 μg/min infusion; children: 0.1 to 1 mcg/kg/min infusion)
• Dopamine: Not helpful
• Avoid dextran as colloidal solution

**Extubation**
- Should be smooth
- Keep pt warm

**Adjuvants**
- Blood transfusion—should be warmed and given only when essential

• Regional: Has been advocated but hypotension and bronchospasm reported to be even more common as well as urticaria and pruritus
• Antibiotics: Avoid polymyxin B sulfate, vancomycin. Used safely: Amikacin, cefazolin, metronidazole
• Miscellaneous drugs: Avoid dipyridamole, papaverine, quinine, thiamine
• Radiologic contrast dyes can induce acute episode.

**Postoperative Period**
- Continue with analgesics, H₁ and H₂ blockers

**Anticipated Problems/Concerns**
- Hypotensive and bronchospastic crisis due to mast cell degranulation induced by anesthetic or surgical procedures
- Anaphylaxis
Mediastinal Masses

Risk
- Usually a congenital lesion, occurring at 1:5000, no gender bias
- Benign or malignant; cysts or aneurysms that arise from the lung, pleura, or another structure of anterior mediastinum
- Lymphoma (Hodgkin’s or NHL), thymoma, germ cell tumor, granuloma, bronchogenic cancer, thyroid tumors (retrosternal goiter), bronchogenic cysts and cystic hygroma

Perioperative Risks
- Periop mortality is rare
- Sudden CV collapse from inability to ventilate or oxygenate
- Hypotension or tamponade
- Increased dyspnea (orthopnea) or cough when supine (increased risk of airway complications)
- Syncope or pericardial effusion (increased risk of CV complications)
- Major airway complications in these pts are now more likely to occur in the post-anesthetic care area rather than in the OR

Worry About
- Inability to get on cardiopulmonary bypass rapid enough to avoid permanent neurologic damage

Superior vena cava syndrome with airway edema and increased bleeding
- Recurrent laryngeal nerve injury
- Pts at risk with cough and pain, dyspnea and dysphagia, superior vena cava syndrome, tracheal deviation, Horner’s syndrome, cyanosis, mediastinal widening, and hoarseness

Overview
- Severity of symptoms does not predict intraop course
- Airway obstruction or hemodynamic compromise has occurred with induction of GA, intubation, muscle relaxation, position change, and after extubation
- Pts may present with Sx that incl chest pain or fullness, dyspnea, cough, sweats, superior vena cava obstruction, hoarseness, syncope, or dysphagia
- Pts can be asymptomatic and have a mass diagnosed on a screening chest radiograph or CT scan

ICD-9-CM Codes: 164.0 (Malignant thymoma) 201.9 (Hodgkin lymphoma); 202.8 (NHL)

Perioperative Implications

Preoperative Preparation
- Consider (incl pediatric pts) an IV prior to induction (lower extremity if SVC syndrome)
- All pts should have a CXR and a chest and neck CT scan prior to any surgical procedure to plan airway management.
- Those with PA or heart compression may need cardiopulmonary bypass (check availability prior to induction with cannulation sites prepared and draped)
- Studies of flow-volume loops have shown a poor correlation with the degree of clinical airway obstruction and have not demonstrated usefulness in managing these pts.
- Reserve use of premedication except for anticholinergic.

Monitoring
- Consider intra-arterial catheter, central venous, or pulm artery catheter.
- If SVC syndrome, insert central venous access or PA catheter via femoral vein.

Airway
- Tracheal or distal compression; may become obstructed with induction and muscle relaxation
- Maintain spontaneous ventilation throughout procedure unless ETT is below obstruction.
- Pts who are symptomatic in supine position are best induced sitting or semi-sitting.
- Awake fiberoptic intubation may be skipped if asymptomatic in supine position and CXR and/or CT scan do not reveal airway obstruction or compression.
- If in doubt, consider awake fiberoptic bronchoscopy to rule out obstruction or compression.
- If compression seen in thoracic trachea, consider a single lumen armored ETT with its tip distal to the compression.
- If compression is at level of carina or distal, endobronchial intubation or a double-lumen endobronchial tube is recommended.

Maintenance
- Consider local anesthesia, otherwise consider keeping pt breathing spontaneously.
- If obstruction occurs, consider altering pt’s position, attempt rigid bronchoscopy, median sternotomy, or femorofemoral cardiopulmonary bypass

Exubation
- Deep extubation during spontaneous breathing recommended; try to minimize straining, coughing, or bucking which would all increase intrathoracic pressure.
- Observe in a monitored bed for several hr after extubation to detect and treat delayed airway obstruction

Anticipated Problems/Concerns
- Airway obstruction with the inability to ventilate
- Vascular compression with hypotension, hypoxia, and arrest.
- Consider radiation and/or chemotherapy before surgery.
- If GA required, consider inspection of tracheobronchial tree with fiberoptic bronchoscopy.
- If GA required, maintaining spontaneous ventilation preferable.
- The most useful information for the anesthesiologist to guide management of these pts comes from the pt’s Hx and the chest imaging.

Perioperative Risks
- If SVC syndrome, insert central venous access or pulm artery catheter.
- Those with PA or heart compression may need airway management.
- Consider (incl pediatric pts) an IV prior to induction (lower extremity if SVC syndrome)
- If compression is at level of carina or distal, consider bronchoscopy to rule out obstruction or compression.
- If in doubt, consider awake fiberoptic bronchoscopy to rule out obstruction or compression.
- If compression seen in thoracic trachea, consider a single lumen armored ETT with its tip distal to the compression.
- If compression is at level of carina or distal, endobronchial intubation or a double-lumen endobronchial tube is recommended.

Preinduction/Induction
- May develop airway obstruction with inability to ventilate
- May develop hypoxia from obstruction of pulm artery and blood flow to lungs
- If muscle relaxants are required, assisted ventilation should first be gradually taken over manually to assure that positive-pressure ventilation is possible and only then can a short-acting muscle relaxant be administered.
- Development of airway or vascular collapse at induction demands immediate awakening.

System Effect Assessment by Hx PE Test
HEENT Possible tracheal compression by mass, bulky nodal disease Dysphonia, dysphagia, coughing paroxysms when supine or orthopnea Palpable neck mass, wheezing, stridor, Indirect laryngoscopy, CXR, CT scan, MRI, pulm flow volume studies
CARDIO SVC syndrome, compression of PA, cardiac failure Dyspnea, fatigue, syncope, peripheral edema, crackles, headache, chest pain, SOB Facial or neck swelling, upper body edema, cyanosis, increased JVP, hypotension CXR, ECHO, EKG, stress, ECHO, CT/MRI
CNS Recurrent laryngeal nerve compression, spinal cord compression Stridor, dysphonia, focal symptoms based on point of compression Anatomical distortion of neck or thorax CXR, CT
RESP Decreased lung volumes, bronchial compression, obstructive pneumonia SOB, ↑ respiratory rate, dyspnea, cough Wheezing, distant breath sounds, hypoxemia, pedal edema PFT, ABG, CXR, DLco

Special problems in pediatric populations: Anesthetic deaths have mainly been reported in children, possibly due to the more compressible cartilaginous structure of the airway or because of underestimation of the severity of the airway compression in children due to the difficulty in obtaining a clear Hx of positional symptoms. Even with proper management, children with tracheobronchial compression more than half cannot be safely given general anesthesia. Further increasing risk in pediatric pts, securing the distal airway with awake fiberoptic intubation and placement of an ETT distal to a tracheal obstruction, an option for some adults with masses compressing the mid-trachea, is not an option in most children.
**Mesothelioma**

**Overview**
- Diffuse malignant mesothelioma arises from the mesothelial surface of the pleura, peritoneum, and pericardium, and the tunica vaginalis of the testis
- 80–90% originate from the pleura
- Peak incidence 20–40y after asbestos exposure
- Usual onset of symptoms at age 55–70y
- Median survival after onset of symptoms is approx 18 mo

**ICD-9-CM Codes:** 162.9 (Lung neoplasm); 199.1 (Mesothelioma, malignant site unspecified); 162.9 (Malignant neoplasm of bronchus and lung unspecified)

**Etiology**
- Diffuse mesothelioma related to asbestos exposure in 12–93% of cases
- Also assoc with radiation therapy, erionite exposure, chronic inflammation and fibrosis, and other agents

**Usual Treatment**
- Treatment has been controversial and largely ineffective
- Therapy has consisted of combinations of radiation to hemithorax, chemotherapy, and sometimes surgery (parietal pleurectomy and decortication or extrapleural pneumonectomy)

**ASSESSMENT POINTS**

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<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Tracheal displacement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Superior vena cava syndrome</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumothorax</td>
<td>Cough, chest pain, increased SOB</td>
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<td></td>
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<tr>
<td></td>
<td>Restrictive lung disease</td>
<td>Dyspnea with exercise</td>
<td>Percussion and auscultation of chest</td>
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<tr>
<td>GI</td>
<td>Wt loss, debilitation, peritoneal tumors</td>
<td>Past body weights</td>
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</tr>
<tr>
<td>ENDO</td>
<td>Not associated with paraneoplastic syndromes</td>
<td></td>
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</tbody>
</table>


**Perioperative Implications**

**Perioperative Preparation**
- Usually come to surgery for lung biopsy via thoracoscopy or open-lung biopsy; some pts are scheduled for pleuropneumonectomy
- Assess pulse rate; size of effusion, no pneumothorax
- Pt often has one or more recent needle biopsies of lung or thoracotomies
- Review radiographic studies for size and location of tumor

**Monitoring**
- Routine monitors
- Resp system via stethoscope, SpO₂, and PETCO₂
- Intra-arterial catheter for complex surgical procedures

**Airway**
- Look for tracheal and mediastinal displacement on radiographic studies

**Induction**
- Propensity for hypoxia, particularly from restrictive lung disease

**Maintenance**
- High FIO₂ may be necessary
- One-lung ventilation
- Lateral positioning

**Extubation**
- Ensure pt meets extubation criteria

**Adjuvants**
- Pain control after thoracoscopy or thoracotomy
- No special considerations for muscle relaxants, reversal agents, local anesthetics, or special drug interactions

**Postoperative Period**
- Monitor ventilation and oxygenation
- Pain relief; consider epidural or spinal analgesia after thoracotomies
- May have air leak postop

**Anticipated Problems/Concerns**
- Anesthesia with one-lung ventilation for a geriatric pt with incurable malignancy
- Recent lung biopsy and thoracentesis prior to surgery and potential for complications from those procedures, incl pneumothorax and dehydration
- With extrapleural pneumonectomy a possibility of massive blood loss, dysrhythmias, and hemodynamic instability during pericardial window and patch
- Effective pain relief and monitoring of resp function postop
- Consider ICU stay for those undergoing complex procedures

---

**Risk**
- Incidence in USA: ~2000–3000 new cases annually, and decreasing. Increasing incidence in developing countries due to poor regulation of asbestos in mining and industrial use.
- Attributable mortality: 14 deaths per million USA population
- M:F ratio 3–6:1
- 0.16% of all malignancies

**Perioperative Risks**
- Usually discovered in geriatric male undergoing lung biopsy
- Pleural effusion
- General debilitation from malignancy

**Worry About**
- Previous needle biopsy of lung and thoracocentesis make pneumothorax a concern

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**Srinivasan Rajagopal**
**John R. Moyers**
Methemoglobinemia

H. Michael Marsh

### Risk
- Incidence within USA: Rare
- Gender prevalence: None
- Socioeconomic/ethnic prevalence: None

### Perioperative Risks
- Inadequate O$_2$ carriage and delivery to tissues
- Hemolysis may be induced by methylene blue, esp in pts with G6PD deficiency.

### Worry About
- Percent of methemoglobin or sulfhemoglobin. Acutely developing methemoglobinemia or sulfhemoglobinemia may become symptomatic at 1% with cyanosis; at 60%, acute CV collapse, coma, or death may occur.

### Overview
- Present when >1% of circulating hemoglobin is oxidized to ferric form
- Two hereditary forms: Due to NADH-diaphorase (cytochrome b5 reductase) deficiency, inherited as an autosomal recessive trait; and due to abn globins, hemoglobin M, which are inherited as autosomal dominant traits
- Toxic methemoglobinemia occurs from exposure to agents that directly oxidize hemoglobin or facilitate its oxidation by molecular O$_2$: nitrates ingested, nitroglycerin, isobutyl nitrite, and some local anesthetics

ICD-9-CM Code: 289.7

### Etiology/Pathogenesis
- Fe$^2+$ in hemoglobin is constantly oxidized in vivo, by NO and reactivity with O$_2$, to Fe$^3+$, methemoglobin. NADPH-diaphorase utilizes NADH generated by glyceraldehyde dehydrogenase, in the Embden-Meyerhof pathway, to reduce cytochrome b5, which in turn reduces Fe$^3+$ in methemoglobin to Fe$^2+$ in hemoglobin.

### Usual Treatment
- Medical therapy: Ascorbic acid 300–600 mg/d in divided doses. Methylene blue 1 mg/kg IV, repeated once provided that pt is not G6PD-deficient, since hemolysis will occur in this case.
- Methylene blue may also be taken orally as 60 mg tid.

### ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>SOB</td>
<td>Hx of cyanosis if hereditary form</td>
<td>RR</td>
<td>Co-oximetry</td>
</tr>
<tr>
<td></td>
<td>DOE</td>
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</tbody>
</table>

**HEME**
- Cyanosis if 1% methemoglobin is present or sulfhemoglobin seen
- Cyanosed
- Cyanosis
- Spectrometry at 630 nm


### Perioperative Implications

#### Preoperative Preparation
- Consider treatment if methemoglobin level is >1%. If sulfhemoglobin is present, may mean exchange transfusion.

#### Monitoring
- Use co-oximeter (IL282), since presence of methemoglobin will render pulse oximetry unreliable.

#### Airway
- Routine

#### Induction
- Routine

#### Maintenance
- Routine

#### Extubation
- Routine

#### Adjuvants
- Avoid nitrates and local anesthetics that act as oxidizing agents.

#### Postoperative Period
- See Monitoring

### Anticipated Problems/Concerns
- O$_2$ carriage is interfered with, proportional to concentration of altered hemoglobin present, and the interference with O$_2$ release and shift of tension-saturation curve from normal position.
- Pulse oximetry overestimates SaO$_2$, in presence of methemoglobin. Methylene blue will decrease the SaO$_2$ for about 30 min after injection.
Mitochondrial Myopathy

Risk
- More common than previously thought. Prevalence ranges from 1 in 7000 to 1 in 15,000
- Occurrence is usually sporadic or maternally inherited

Perioperative Risks
- Metabolic acidosis
- Resp and cardiac insufficiency/failure
- Delayed emergence

Worry About
- Resp failure following sedation
- Consider aspiration risk
- Metabolic acidosis
- Hypotension during induction

Overview
- Clinically heterogeneous collection of diseases with myopathy of mitochondrial origin as common trait
- Commonly assoc w/encephalopathy

Mitochondrial Myopathy

- Inc Kearn-Sayre syndrome (KSS); Pearson’s syndrome (PS); maternally inherited Leigh syndrome (MILS); late-onset Leigh syndrome; mitochondrial encephalomyopathy, lactic acidosis, and stroke-like syndromes (MELAS); myoclonic epilepsy with ragged-red fibers (MERRF); Leber hereditary optic neuropathy (LHON); chronic progressive external ophthalmoplegia (CPEO); neuropathy, ataxia, and retinitis pigmentosa (NARP)
- Onset is variable. Most severe phenotypes present in infancy
- Most common symptom is muscle weakness, and most common sign is lactic acidosis, resulting from the inefficient metabolism of pyruvate and shift to anaerobic resp
- Muscle biopsy often used for suspected cases. Hallmark is appearance of ragged red fibers
- Anesthetic sensitivity may manifest as decreased MAC of inhaled anesthetics (e.g., Complex I disorders), increased resp insufficiency from sedatives and narcotics, and decreased hepatic clearance or renal excretion of IV agents.

ICD-9-CM Code: 359.8

Etiology
- Genetic variation in either mitochondrial DNA (mtDNA) or nuclear DNA (nDNA)
- Large-scale mtDNA deletions (e.g., KSS, PS, PEO) most often acquired sporadically
- Single-base mtDNA changes (e.g., MELAS, MERRF, MILS, LHON) are often inherited maternally, and usually affect mitochondrial protein synthesis (via mRNA, tRNA, or rRNA) or components of the electron-transport chain (e.g., Complex I, III, IV, V)
- Single-base nDNA changes (e.g., late-onset Leigh syndrome) are often inherited in Mendelian patterns (autosomal dominant or recessive)

Usual Treatment
- Supportive measures
- Dietary supplements and coenzyme Q

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Dysphagia</td>
<td>Coughing, choking, aspiration with feeding</td>
<td>Salorrhea</td>
<td>CXR study</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cardiomyopathy Conduction defects (KSS)</td>
<td>Sx of CHF</td>
<td>Murmur, gallop, crackles</td>
<td>CXR, ECHO ECG, exercise testing (VO2 max)</td>
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<tr>
<td>RESP</td>
<td>Disorganized resp muscle effort</td>
<td>Hypoventilation, hypoxia following sedative use</td>
<td>Rhonchi</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Chronic diarrhea Exocrine pancreatic failure (PS)</td>
<td>Dehydration</td>
<td>Serum electrolytes</td>
<td>Serum electrolytes</td>
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<tr>
<td>ENDOMET</td>
<td>Lactic acidosis Hepatic insufficiency</td>
<td>N/V</td>
<td>Prolonged Rx effects</td>
<td>Hypermintilation</td>
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<tr>
<td>GU</td>
<td>Renal tubular defects (PS), nephropathy</td>
<td>Urinary changes</td>
<td>Urinalysis</td>
<td>Serum BUN, Cr, electrolytes</td>
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<tr>
<td>CNS</td>
<td>Encephalopathy (MILS) Ophthalmoplegia (CPEO, KSS) Stroke (MELAS) Seizure (MELAS, MERRF) Retinopathy, ataxia (NARP), blindness (LHON), deafness</td>
<td>Developmental delay</td>
<td>ROM of extraocular mm</td>
<td>Head CT or MRI Ophth exam</td>
</tr>
<tr>
<td>PNS</td>
<td>Peripheral neuropathy</td>
<td>Weakness, clumsiness</td>
<td>↓ Strength</td>
<td>Muscle biopsy-ragged red fibers</td>
</tr>
<tr>
<td>MS</td>
<td>Hypotonia, weakness Myoclonus (MERRF)</td>
<td></td>
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</tbody>
</table>

Perioperative Implications

Preoperative Preparation
- Assess cardiac involvement
- Preop anticholinergic for excessive oral secretions
- Avoid prolonged fasting and dehydration, which can worsen acidosis
- When possible, start IV fluid (IF) at NPO time, allow for late (2 hr prior) clear fluid intake, and book as first case

Airway
- Possible aspiration risk

Monitoring
- Routine, assuming no severe cardiomyopathy or CHF
- Consider bispectral index (BIS) monitor prior to induction, for possible increased anesthetic sensitivity

Induction
- Avoid lactate-containing IVF (e.g., lactated Ringer’s)

Maintenance
- Many techniques have been used safely
- Avoid prolonged infusion of IV anesthetics, esr propofol, which is a known electron-transport chain de-coupler, due to worsened acidosis and reduced ATP production
- Hepatic and renal insufficiency may increase IV anesthetic half-life and prolong elimination
- If NMB agent is required, consider careful titration with shorter-acting agents
- Implement aggressive temp control; recommend active warming techniques
- Avoid tourniquets

Extubation
- Muscle weakness and anesthetic sensitivity may delay extubation

Regional Anesthesia
- Used successfully, but caution in those with underlying cardiac conduction block
- Local anesthetics have potential to de-couple electron transport chain

Postoperative Period
- Close monitoring of resp function
- For cases of longer duration, consider serum electrolytes or blood gas to assess acidosis
- Some have reported increased incidence of PONV

Anticipated Problems/Concerns
- Generally not associated with malignant hyperthermia (MH); however, scenario of critical ATP depletion may lead to muscular contraction mimicking MH
- Although succinylcholine is not contraindicated as in Duchenne or Becker MD, acidosis and neuropathy may predispose to accentuation of hyperkalemia

Key Reference: Muravchick S, Levy RJ. Clinical implications of mitochondrial dysfunction. Anesthesiology. 2006;105:819–37. Clinical findings listed above may be characteristic of one or more mitochondrial myopathies. A specific disorder may follow in parentheses if the finding is a primary feature.
Mitral Regurgitation

Raj K. Modak

Mitral Regurgitation

Overview
- The mitral valve allows one-way blood flow through the left heart.
- During diastole, it acts as an open conduit for blood flow from the left atrium to the LV.
- Systole, it closes preventing back flow while the heart contracts.
- With mitral regurgitation, retrograde flow occurs from the LV to the LA during systole. This can occur as an acute or chronic process.
- The acute form results in sudden elevations in LA pressure. Elevated pressures in the pulm artery result in pulm edema and RV strain and possible failure.
- Chronic mitral regurgitation is tolerated well. LV hypertrophy is followed by dilatation and failure. Similar changes in the RV and pulm circulation occur, as in the acute form, but are better tolerated over the longer time period.
- As a general rule, the more precipitous the onset, the more significant the sequelae.

ICD–9–CM Code: 424.0

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<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Mitral regurgitation</td>
<td>Fatigue, exertional or nocturnal</td>
<td>Pansystolic and late systolic murmur, rales</td>
<td>Doppler ECHO</td>
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<td></td>
<td>LA enlargement</td>
<td>dyspnea</td>
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<td>2D-ECHO</td>
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<td></td>
<td>AFib</td>
<td>Palpitations, defibrillation,</td>
<td>Irregular rhythm, bruises</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>anticoagulation</td>
<td></td>
<td>PT/INR</td>
</tr>
<tr>
<td></td>
<td>RV failure</td>
<td>Peripheral swelling, RUQ</td>
<td>Ankle edema, hepatomegaly, hepatojugular reflex</td>
<td>Cardiac catheterization, 2D and Doppler ECHO</td>
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<td></td>
<td>Cardioemegaly</td>
<td>pain, tenderness</td>
<td></td>
<td>CXR, 2D-ECHO</td>
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<tr>
<td>RESP</td>
<td>CHF, pulm edema</td>
<td>Dyspnea, orthopnea</td>
<td>Gallop, rales</td>
<td>CXR</td>
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<tr>
<td>GI</td>
<td>Congestive hepatopathy</td>
<td>Bleeding with minor trauma</td>
<td>Bruises</td>
<td>PT, PTT, LFTs</td>
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<tr>
<td>RENAL</td>
<td>Perfusion</td>
<td>Oliguria</td>
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<td>▼ BUN, Cr</td>
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<tr>
<td></td>
<td>Diuretic-induced</td>
<td>Palpitations</td>
<td>Muscle weakness</td>
<td>Serum K^+ , Mg</td>
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<td></td>
<td>▼ K+, Mg^2+</td>
<td></td>
<td>▼ Reflexes</td>
<td>ECG</td>
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<tr>
<td>MS</td>
<td>Cachexia</td>
<td>Wt loss</td>
<td>Muscle wasting</td>
<td>▼ Wt</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Antibiotic prophylaxis
- Manage anticoagulation issues related to atrial fibrillation and the possible use of regional techniques
- Optimize HR issues related to atrial fibrillation
- Optimize symptoms related to CHF

Monitoring
- In procedures with expected wide variations in BP, direct arterial BP monitoring should be considered esp. with moderate or severe mitral regurgitation.
- In settings of LV failure, a pulm artery catheter or transesophageal echocardiogram may be useful in assessing changes and guiding pharmacologic therapy.

Airway
- Avoid hypoxemia and hypercarbia, which maintains the lowest pulm vascular resistance and reduces risk of RV failure

Preinduction/Induction
- “Faster, Fuller, Forward”
- Avoid bradycardia, maintain high-normal preload, reduce afterload
- Maintain stroke volume by avoiding myocardial depression and atrial fibrillation

Maintenance
- Cardiac and pulm goals, same as induction
- Avoid excessive PEEP, which reduces preload
- If possible, follow pulm output, utilizing pharmacology as needed
- Regional anesthetic techniques may be considered as they help reduce afterload, however, caution is recommended in the setting of impaired LV function

Extubation
- Airway management to avoid hypoxia and hypercarbia inducing right ventricular strain and failure
- Requires vigilance on BP management to avoid Htn

Adjuvants
- No known drug interaction problems

Postoperative Period
- Pain management critical to avoid hypertensive episodes
- Both pt-controlled analgesia and postop epidural useful for pain control
- Fluid shifts and intraop volume management may alter LV function and antiarrhythmic blood concentrations
- New onset atrial fibrillation from fluid shifts and electrolyte abn
- Consideration for restarting anticoagulation for chronic atrial fibrillation

Anticipated Problems/Concerns
- High periop risk is best predicted by impaired LV function, symptoms of both LV and RV dysfunction
- Htn can acutely worsen mitral regurgitation causing CHF and pulm edema
Mitral Stenosis

Risk
- Bimodal age distribution: 20–39 y and 50–60 y
- Mitral stenosis is 2–3 times more common in women and is the most common valve disease affecting pregnant women.
- Most common among immigrants to the USA from regions where rheumatic fever is prevalent (e.g., Middle East, Asia, Latin America)

Perioperative Risks
- Increased risk of periop cardiac complications that incl infectious endocarditis, pulm edema, resp failure, heart failure, tachyarrhythmias, new-onset AFIB or atrial flutter, embolic stroke of cardiac origin

Worry About
- Fluid status
- Paroxysmal AFIB or flutter
- Pregnancy
- Limited ability to increase cardiac output in response to increased metabolic demands or intravascular volume expansion
- Cardiomyopathy, pulm Htn, RV failure, hepatic dysfunction, tricuspid regurgitation, and assoc aortic valve disease
- Pulm edema

Overview
- The normal mitral valve has an area of 4–6 cm². Symptoms start when the mitral valve area is reduced to 1.5 cm². Diastolic emptying of blood from the left atrium into the left ventricle is impaired critically when the mitral valve area is <1 cm².
- Transmitral pressure gradient varies directly with blood flow across the valve; acute increases in cardiac output or venous return to the heart increases the mitral valve gradient and increases LA and pulm venous pressures. Pulm edema occurs when the pulm venous pressure > pulmonary capillary oncotnic pressure.
- Pulm venous Htn leads to left atrial dilation, left atrial thrombosis, AFIB, pulm Htn, RV failure, and tricuspid regurgitation.
- Symptoms of mitral stenosis can be elicited by conditions (fluid overload, exercise, pregnancy, sepsis, operation) that demand an ↑ in cardiac output or diastolic blood flow across the mitral valve (e.g., MR).
- Deformity of the mitral valve apparatus may cause mitral stenosis in combination with mitral regurgitation or left ventricular dysfunction.

ICD-9-CM Code: 394.0

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Mitral stenosis</td>
<td>DOE, NYHA class Chest pain or tightness</td>
<td>Diastolic murmur</td>
<td>ECHO</td>
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<td></td>
<td>AFIB</td>
<td>Palpitations</td>
<td>Irregular pulse</td>
<td>Cardiac cath</td>
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<td>Pulm Htn</td>
<td>DOE</td>
<td>Sternal heave</td>
<td>ECG</td>
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<td>Prominent S₂</td>
<td>CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>DOE Orthopnea Paroxysmal nocturnal dyspnea Hemoptyasis</td>
<td>Tachypnea Rales Wheezes</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Cardiac cirrhosis</td>
<td>Dependent edema</td>
<td>Hepatomegaly</td>
<td>Liver function tests</td>
</tr>
<tr>
<td>RENAL</td>
<td>Fluid retention Diuretic therapy</td>
<td>Pedal edema</td>
<td>Serum lytes</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Embolic stroke</td>
<td>Neurologic deficits, TIAs</td>
<td>Focal neurologic deficits</td>
<td>Head CT scan, TEE</td>
</tr>
<tr>
<td>HEME</td>
<td>Bleeding</td>
<td>Anticoagulation therapy</td>
<td>Echymosis</td>
<td>INR, PT, PTT</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Determine if pt is a candidate for percutaneous balloon valvotomy
- Optimize fluid status of pts in CHF
- Control ventricular rate in pts with AFIB
- Replete K⁺ in pts with hypokalemia on digoxin therapy
- Antibiotic prophylaxis for infectious endocarditis according to guidelines
- Keep pt calm using reassurance, anxiolytics, and analgesics
- Assess the risk of bleeding in anticoagulated pts and correct the prolonged PT (INR) with FFP if necessary

Monitoring
- ECG to detect paroxysmal AFIB or flutter
- Consider arterial catheter for continuous BP monitoring and ABG sampling

- Consider CVP line, PA catheter, or TEE to measure pulm artery pressure, assess RV function, and guide intravascular volume management when large fluid shifts are anticipated.

Preinduction/Induction
- Caution administration of drugs that decrease myocardial contractility, increase HR, or cause vasodilation.
- Hypoventilation and hypoxia may worsen pulm Htn and RV failure
- Positive inotropic drugs may precipitate pulm edema

Maintenance
- Control fluid administration

Exudation/Postoperative Period
- Provide adequate analgesia
- Increased risk of postop resp failure

Adjuvants
- Consider regional anesthesia or peripidural anesthesia and analgesia, esp. for labor and delivery in the pregnant pt with mitral stenosis
- Inhaled NO or epoprostenol for RV failure assoc with pulm Htn

Anticipated Problems/Concerns
- Pts have a limited ability to increase their cardiac output
- Acute pulm edema is precipitated by increased cardiac output, increased HR, pregnancy, anxiety, fluid overload, exercise, and postop mobilization of sequestered (third space) interstitial and extracellular fluid
- Bleeding in anticoagulated pts
Mitral Valve Prolapse

### Risk
- Believed to be most common form of valvular heart disease, with an incidence of 5% of the general population
- Based on strict ECHO criteria, the incidence of MVP is 2–3% with no predilection for gender or age
- The severity of disease varies widely in pts with the diagnosis of MVP depending on the severity of mitral regurgitation (MR), the degree of structural abn of the mitral valve apparatus, and LV function as a consequence of MR.

### Perioperative Risks
- Infectious endocarditis
- HF as a consequence of acute or chronic MR
- Embolic stroke
- Sudden cardiac death

### Worry About
- Severity of MR
- LV dysfunction, CHF, AFIB, sudden cardiac death, infective endocarditis, or embolic stroke as a consequence of MR
- Associated conditions: Marfan syndrome; Ehlers-Danlos syndrome, osteogenesis imperfecta, or pseudoxanthoma elasticum
- MVP syndrome: Atypical chest pain, palpitations, syncope, exertional dyspnea, or anxiety

### Overview
- Severity of disease in pts with MVP varies widely based on the clinical and ECHO criteria used to establish the diagnosis.
- MVP is defined as isolated prolapse of the mitral valve leaflets 22 mm beyond the mitral valve annular plane into the left atrium during systole by ECHO. The severity of myxomatous degeneration of the mitral valve apparatus causing MVP is characterized by leaflet thickening, leaflet redundancy, chordal elongation, or chordal rupture by ECHO.
- Structural abn in MVP lead to weakness and deformity of the valve apparatus. Annular dilation, stretching of valve leaflets, and chordal elongation impair leaflet coaptation causing progression of MR.
- A flail leaflet is caused by acute rupture of weakened chordae and produces severe MR.
- Chronic MR causes progressive LA dilation, eccentric left ventricular hypertrophy, HF, and AFIB.
- MVP syndrome is used to describe MVP assoc with a spectrum of nonspecific symptoms incl atypical chest pain, palpitations, exertional dyspnea, exercise intolerance, syncope, anxiety, lean body habitus, and electrocardiographic repolarization abn. A pathophysiologic basis establishing a link between these nonspecific symptoms and MVP has not been defined.

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Mitral valve prolapse</td>
<td>Atypical chest pain</td>
<td>Mid- to late-apical nonejection systolic clicks</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>Mitral regurgitation</td>
<td>DOE</td>
<td>Mid- to late-apical systolic murmur</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>AFIB</td>
<td>CHF</td>
<td>Irregular pulse</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>Infectious endocarditis</td>
<td>NYHA class</td>
<td>Embolic phenomena</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Palpitations</td>
<td></td>
<td>TEE, blood culture</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fever, chills</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Stroke</td>
<td>Neurologic deficits</td>
<td>Focal neurologic signs</td>
<td>Head CT scan</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TIAs</td>
<td></td>
<td>TEE</td>
</tr>
<tr>
<td>MS</td>
<td>Connective tissue disorders</td>
<td>Pectus excavatum</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Scoliosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lean stature</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Key Reference

### Perioperative Implications

#### Preoperative Preparation
- Assess existence and severity of MR
- Assess for signs and symptoms of HF
- Antibiotic prophylaxis for infectious endocarditis in pts with Hx of infectious endocarditis and procedure that may result in transient bacteremia

#### Monitoring
- Routine
- Consider invasive hemodynamic monitoring for major operations in symptomatic pts with severe MR and LV dysfunction.

#### Preinduction/Induction/Maintenance
- Avoid Htn and acute increases in sympathetic tone
- Consider regional anesthesia

#### Adjuvants
- Therapeutic interventions that increase BP, myocardial contractility, preload, or sympathetic tone may increase severity of MVP, MR, or the risk of chordal rupture.
- Antiarrhythmic agents and anticoagulation therapy in pts with AFIB (see atrial fibrillation)
- Mitral valve repair or replacement in patients with symptomatic MR or evidence of LV dilation, reduced LV ejection fraction, AFIB, pulm Htn, or severe MR by ECHO

#### Usual Treatment
- No treatment in asymptomatic pts or pts with MVP syndrome without significant MR by ECHO.
- ACE inhibitors, β-blockers, and diuretics in pts with significant MR or CHF (see mitral regurgitation)
- Antiarrhythmic agents and anticoagulation therapy in pts with AFIB (see atrial fibrillation)
- Mitral valve repair or replacement in patients with symptomatic MR or evidence of LV dilation, reduced LV ejection fraction, AFIB, pulm Htn, or severe MR by ECHO

#### Anticipated Problems/Concerns
- Presence of severe MR, LV dysfunction, or assoc connective tissue disorders may alter routine management of pts with isolated MVP (see Mitral Regurgitation and individual connective tissue disorders in Diseases section)
- MVP is assoc with a 3–8 fold higher risk of infective endocarditis. Traditionally, antibiotic prophylaxis was recommended for pts with MVP and significant MR undergoing procedures with a risk of transient bacteremia. The AHA revised recommendations for antibiotic prophylaxis in 2007 to incl only pts with Hx of infectious endocarditis or who have had mitral valve repair or replacement.

#### Risk factors for HF, sudden cardiac death, infective endocarditis, stroke, or need for mitral valve surgery in pts with MVP are LV dilation, depressed LV ejection fraction (<50%), severity of MR, AFIB, LA enlargement, flare leaflet (chordal rupture), leaflet thickening (>5 mm) and age >50 y.

### ICD-9-CM Code: 424.0

### Etiology
- Inherited connective tissue disorders
- Myxomatous degeneration caused by dysregulation of collagen and elastin matrix protein synthesis and degradation
- Inherited myxomatous mitral valve prolapse

### Assessment by Hx
- Consider regional anesthesia
- Avoid Htn and acute increases in sympathetic tone.
- The severity of disease varies widely in pts with the diagnosis of MVP depending on the severity of mitral regurgitation (MR), the degree of structural abn of the mitral valve apparatus, and LV function as a consequence of MR.

### Test
- ECHO
- CXR
- ECG
- TEE, blood culture

### Usual Treatment
- No treatment in asymptomatic pts or pts with MVP syndrome without significant MR by ECHO.
- ACE inhibitors, β-blockers, and diuretics in pts with significant MR or CHF (see mitral regurgitation)
- Antiarrhythmic agents and anticoagulation therapy in pts with AFIB (see atrial fibrillation)
- Mitral valve repair or replacement in patients with symptomatic MR or evidence of LV dilation, reduced LV ejection fraction, AFIB, pulm Htn, or severe MR by ECHO

### Testing
- ECHO
- CXR
- ECG
- TEE, blood culture

### Adjuvants
- Therapeutic interventions that increase BP, myocardial contractility, preload, or sympathetic tone may increase severity of MVP, MR, or the risk of chordal rupture.
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- Mitral valve repair or replacement in patients with symptomatic MR or evidence of LV dilation, reduced LV ejection fraction, AFIB, pulm Htn, or severe MR by ECHO

### Anticipated Problems/Concerns
- Presence of severe MR, LV dysfunction, or assoc connective tissue disorders may alter routine management of pts with isolated MVP (see Mitral Regurgitation and individual connective tissue disorders in Diseases section)
- MVP is assoc with a 3–8 fold higher risk of infective endocarditis. Traditionally, antibiotic prophylaxis was recommended for pts with MVP and significant MR undergoing procedures with a risk of transient bacteremia. The AHA revised recommendations for antibiotic prophylaxis in 2007 to incl only pts with Hx of infectious endocarditis or who have had mitral valve repair or replacement.
Mobitz I (Second-Degree Atrioventricular Block)

**Overview**
- Found usually in presence of CAD
- Block generally occurs in AV node, resulting in normal QRS complexes
- ECG reveals progressive lengthening P-R intervals at decreasing increments and progressively shortening R-R intervals leading to a regular atrial rhythm and an irregular ventricular rhythm
- Bradycardia usually responds to atropine

**ICD-9-CM Code:** 426.13 (Mobitz I)

**Etiology**
- Acquired, usually with MI
- Increased resting parasympathetic tone relative to resting sympathetic tone

**Risk**
- Occurs after inferior MI, or occasionally in trained athletes or in normal, sleeping people
- Incidence varies based on etiology.

**Perioperative Risks**
- Without assoc heart disease and without symptoms, should not present undue risk during anesthesia, for instance in trained athletes
- If occurs 2° to inferior myocardial infarction, the periop risk depends on extent of ischemic area

**Worry About**
- Advancing to a higher-degree block if ischemic zone extends to anterior wall
- Papillary muscle dysfunction may occur

**Usual Treatment**
- Specific therapy in absence of heart disease not necessary unless pt is symptomatic
- Treatment of an infarction-related Mobitz I block incl observation and medical therapy with atropine
- Temporary pacing is necessary only if a medically unresponsive pt is symptomatic
- Permanent pacing seldom required and considered only in persistently blocked, symptomatic pts

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Commonly no Sx</td>
<td>Bradycardia on occasion</td>
<td>Exercise tolerance, Angina, SOB</td>
<td>Signs of CHF and ↓ perfusion</td>
</tr>
<tr>
<td>RENAL</td>
<td>Likely normal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>No effect or ↓ perfusion of CNS</td>
<td>No Sx or only mild Sx: fainting, dizziness</td>
<td>Normal Bruits</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Consider availability of transcutaneous pacing

**Monitoring**
- Based on co-existing disease
- Observe for and prepare to treat 3° block when positioning PA catheter in pt with Mobitz I block.

**Airway**
- None

**Induction/Maintenance**
- Regional or general
- No contraindications to any standard anesthetic drugs
- Intraop processes and drugs that increase atrial rate could decrease ventricular rate.

**Exstubation**
- None

**Adjuvants**
- Cautious use of drugs that slow AV conduction

**Anticipated Problems/Concerns**
- Extension of infarcted area with higher degree block and CHF
### Mobitz II (Second-Degree Atrioventricular Block)

**Risk**
- Occurs after anterior infarction and can quickly proceed to a 3° heart block

**Perioperative Risks**
- Risk of developing 3° block

**Worry About**
- Rapid development into a 3° block, which requires temporary transvenous pacing

**Overview**
- Unlike Mobitz I block, Mobitz II block is located in bundle of His or bundle branches, resulting in lengthening QRS duration
- P-P and R-R intervals are constant, and P-R intervals are constant prior to the dropped QRS complex

**ICD-9-CM Code:** Mobitz II: 426.12

**Etiology**
- Acquired, usually associated with MI

**Usual Treatment**
- Temporary pacemaker insertion should be considered soon after onset of this block, because 3° block commonly occurs.
- Pacing does not improve survival.
- Atropine usually does not improve conduction.

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Bradycardia</td>
<td>Exercise tolerance, Angina, SOB</td>
<td>Signs of CHF and ↓ perfusion</td>
<td>ECG, CXR, Other tests as indicated</td>
</tr>
<tr>
<td>GU</td>
<td>Likely normal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>↓ Perfusion of CNS</td>
<td>Fainting, dizziness, Normal? Bruits</td>
<td></td>
<td>PE, Carotid US</td>
</tr>
</tbody>
</table>

**Preoperative Implications**

**Preoperative Preparation**
- Evaluation of CAD important
- Likely a transvenous pacemaker will be in place
- Transcutaneous pacing should be available if temporary transvenous pacing was not established prior to induction of anesthesia.

**Monitoring**
- Based on severity of heart disease and extent of infarcted area

- Prepare to treat 3° block when positioning a PA catheter

**Airway**
- None

**Induction/Mainteinance**
- No contraindications to any standard anesthetic drugs
- Any intraop process or drug increasing atrial rate could worsen block and decrease ventricular rate

**Adjuvants**
- Cautiously use drugs that slow conduction through AV node unless they also slow SA nodal rate and allow 1:1 AV conduction and increased ventricular rate.
- 1° AV block will persist if 1:1 conduction occurs.

**Morbid Obesity**

**Risk**
- Incidence in USA: ~5% morbidly obese

**Perioperative Risk**
- Increased morbidity/mortality versus normal BMI, from resp and cardiac issues

**Worry About**
- Challenging procedures: IV start, intubation, ventilation, epidural catheter placement
- Restrictive pattern of resp disease, hypoxemia, larger O2 demand, small FRC; obstructive sleep apnea (OSA) is common, with assoc cardiac issues

**Overview**
- Defined by BMI, (wt in kg/ht in m^2^), >30 obese, >35 morbidly obese
- Cardiac and resp issues mainly due to size. Large body mass to be perfused and oxygenated; increased cardiac strain and resp effort of breathing. OSA common, increased sensitivity to narcotics.
- Depression common

**ICD-9-CM Code:** 278.0

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Htn</td>
<td>Fatigue, dyspnea</td>
<td>Auscultation, increased heart size, ± rales</td>
<td>BP, EKG, CXR</td>
</tr>
<tr>
<td></td>
<td>Pulm</td>
<td>Dyspnea, fatigue, syncope inc JVP, peripheral edema, hepatomegaly, crackles</td>
<td>Auscultation, palpation, auscultation</td>
<td>CXR, EKG, ECHO</td>
</tr>
<tr>
<td></td>
<td>Htn</td>
<td>Chest pain, SOB</td>
<td></td>
<td>EKG, ECHO</td>
</tr>
<tr>
<td></td>
<td>Cardiac failure</td>
<td></td>
<td></td>
<td>EKG, stress ECHO</td>
</tr>
<tr>
<td></td>
<td>Coronary disease</td>
<td></td>
<td></td>
<td>Cor angiogram</td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive disease</td>
<td>SOB, inc resp rate, decreased exercise tolerance</td>
<td>Rapid shallow breathing, hypoxemia, large neck, redundant soft tissue in neck</td>
<td>PFT, ABG, CXR, Hg, pulse ox for room air saturation</td>
</tr>
<tr>
<td></td>
<td>OSA</td>
<td>Hx of snoring, periods of apnea in sleep, non-restful sleep, daytime somnolence and tiredness</td>
<td>Large neck, redundant soft tissue in neck</td>
<td>Overnight sleep study for apnea hypopnea index</td>
</tr>
<tr>
<td>NEURO</td>
<td>Depression</td>
<td>Hx</td>
<td>Question and answers, survey instruments</td>
<td>By psychologist and/or psychiatrist</td>
</tr>
<tr>
<td>AIRWAY</td>
<td>Potentially difficult intubation</td>
<td>Mallampati, upper lip bite test</td>
<td>Evaluation</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>NASH</td>
<td>Hepatomegaly, icterus, ascites Polyphagia, polyuria, polydipsia</td>
<td>Palpation</td>
<td>LFT, PT, PTT, BUN, Cr</td>
</tr>
<tr>
<td></td>
<td>NIDDM</td>
<td></td>
<td></td>
<td>UA, BS, GTT, HgA1c</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Perioperative Preparation**
- All medications except for DM
- Sedation titrated to effect f emerg
- Consider prophylactic preop IVC filter placement if risk of DVT is high

**Monitoring**
- Routine with arterial catheter if cardiac status dictates or ultra obese (BMI >70kg/m^2^ or weight >200kg)
- If severe cardiac or resp disease, ABGs
- UO
- Central venous access if peripheral access difficult, or CVP or pulm pressures need to be monitored for cardiac disease

**Airway**
- Position at 30° head elevated to improve probability of intubation with direct laryngoscopy
- Minority of pts may need awake FOI
- Prepare for difficulty; with multiple airway option like laryngeal masks and video laryngoscopes

**Induction**
- Pre-oxygenate with pressure support if possible, complete denitrogenation
- Rapid sequence with cricoid pressure preferable

**Maintenance**
- Drug dosing lipophilic dosed to real body wt; lipophilic to IBW or LBM
- Desflurane preferable due to complete and rapid recovery

**Etiology**
- Disputed role of genetics, mainly environmental and nutritional habits; essentially a form of severe malnourishment

**Usual Treatment**
- Medical treatment incl psychological counseling along with decreased calorie consumption with increased exercise, if physically able.
- Surgical treatment incl gastric banding, Roux-en-Y, sleeve gastrectomy, or intestinal bypass.

**Perioperative Preparation**
- Appropriate fluid infusion based on deficit, losses, and UO
- Ventilation: Start at TV 10–12 mL/kg IBW; RR 12–14/min; PEEP 8–10; adjust as needed

**Extubation**
- Wide awake, no residual volatile agent, normocapnic, responsive with appropriate resp effort and partially sitting up

**Postoperatively**
- Good analgesia with IV PCA, NSAIDs and local infiltration with LA and rapid mobilization helps resp function and decreases DVT
Diseases

Perioperative implications

Preoperative Preparation
- Assess for associated abn
- Avoid sedatives and narcotics that would result in hypocarbia

Monitoring
- Arterial line for BP monitoring and blood gas analysis

Induction/Maintenance
- Balanced anesthesia or total IV anesthesia
- Maintaining cerebral and systemic hemodynamics is paramount.

• Avoid cerebrovasodilators.
• Minimize increases in CMRO, with adequate levels of anesthesia during painful stimuli.
• Ensure adequate CBF by avoiding hypotension, hypocarbia, and hypercarbia.
• Maintain normothermia with warming blanket if needed.

Postoperative Period
- Monitor for hypoventilation to avoid hypercarbia-induced neurologic symptoms.
- Provide adequate analgesia.
- Maintain normotension, normocarbia, normovolemia, and normothermia.

• Stroke
• Subdural hematoma
• Intracerebral hemorrhage

Overview
- Moyamoya means “puff of smoke,” which describes the angiographic appearance
- Chronic progressive cerebrovascular disease consisting of concentric stenosis or occlusion in the distal internal carotid arteries and large vessels of the circle of Willis with prominent basal collateral vessels
- Adults present with intracerebral/intraventricular hemorrhages
- Children present with TIAs and strokes that lead to neurologic deficits. Symptoms may start from birth to age 5 y, with rapid deterioration in neurologic function over the next 2–3 y.
- Symptoms in children are precipitated by activities that involve hyperventilation, which results in hypocarbia. Changes in body temp may also precipitate attacks.
- Abn vessels have intimal thickening or deficiency of the internal elastic lamina.

ICD-9-CM Code: 437.5

Etiology
- Not clearly defined
- Moyamoya disease (congenital): Angiographic appearance with or without other risk factors, both cerebral and systemic vasculature involved
- Moyamoya syndrome: Present with other known associated conditions, e.g., meningitis, neurofibromatosis, connective tissue disease, sickle cell disease, SLE, trisomy 21, prior radiation therapy, brain tumors, and chronic inflammation in the neck region

Usual Treatment
- Medical
  * Aspirin in select pts such as asymptomatic, mild disease, increased risk for surgery. Not indicated in adult population with Hx of intracranial hemorrhage.
- Surgical
  * Direct: Superficial temporal artery or middle meningeal artery to middle cerebral artery bypass
  * Indirect: EDAS (encephalodural arterio-synangiosis). The scalp artery or temporal artery is placed onto the arachnoid surface of the brain. Collaterals to ischemic brain occur over time.

Assessment Points

<table>
<thead>
<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Decreased CBF</td>
<td>TIAs, strokes</td>
<td>Neuro deficits</td>
<td>CT/MRI/MRA EEG</td>
</tr>
</tbody>
</table>

Mucopolysaccharidoses

RISK

- Mucopolysaccharidosis type I (MPS I), Hurler syndrome, is inherited as an autosomal recessive disorder.
- MPS II, Hunter syndrome, is X-linked (only males affected)
- Incidence in USA: Estimated at 1/30,000

PERIOPERATIVE RISKS

- Estimated periop mortality: 20%
- Difficult intubation (25%), failed intubation (8%)

WORRY ABOUT

- Difficult airway, cardiac lesions, poor IV access, resp failure

OVERVIEW

- MPS is a group metabolic disorder caused by a lack of lysosomal enzymes required to break down glycosaminoglycans which, over time, build up in blood and connective tissue.
- The child may appear normal at birth but by age 1 y will often show signs of both growth and mental retardation. The dx is made by characteristic physical findings and increased urinary mucopolysaccharides (MPS).

- Hurler syndrome, considered the prototypic and most severe subtype of MPS I, is characterized by involvement of heart, liver, and bones. It is also assoc with corneal clouding, development delay, frequent resp infections, stiff joints and an abn airway.
- Scheie syndrome is a milder form of Hurler syndrome; pts have normal intelligence and life expectancy but may have stiff joints and aortic regurgitation.
- Hunter syndrome has diffuse joint limitations, short neck, short stature, and ischemic cardiomyopathy.
- Morquio syndrome (MPS IV) has severe kyphoscoliosis, possible cervical subluxation, and aortic regurgitation.
- Maroteaux-Lamy syndrome (MPS VI) has kyphoscoliosis, cardiac involvement, and mild joint stiffness.
- Recurrent hernias often occur in mucopolysaccharidoses.

ICD-9-CM Code: 277.5

ASSESSMENT POINTS

<table>
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<tr>
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<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Large tongue, small mouth, micrognathia; Difficult airway anticipated; Atlantoaxial subluxation possible; Corneal clouding; Chronic recurrent rhinitis and frequent ear infections</td>
<td>Exercise tolerance</td>
<td>Angina Hx</td>
<td>Neck ROM</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Frequent valvular lesions; Arrhythmias; Cardiomyopathy/CHF; Severe CAD (even at a young age); Difficult IV access</td>
<td></td>
<td></td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive lung disease; Propensity to develop pneumonia; Obstructive sleep apnea; Asthma; Bronchospasm</td>
<td></td>
<td></td>
<td>PFTs</td>
</tr>
<tr>
<td>GI</td>
<td>Frequent hepatomegaly; Hepatic function usually normal; Inguinal and umbilical hernias;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Mental retardation, deafness, minimal language skills; Hydrocephalus in Hurler and Hunter syndromes; Cervical myelopathy in Morquio syndrome</td>
<td></td>
<td></td>
<td>CT/MRI</td>
</tr>
<tr>
<td>MS</td>
<td>Short neck, severe progressive skeletal and joint disease; Characteristic gibbus deformity of lumbar spine; Defective ossification (dy sostosis multiplex); Anticipate difficulty in positioning</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


PERIOPERATIVE IMPLICATIONS

PREOPERATIVE

- May be resistant to sedative premedications
- Anticipate possible upper airway obstruction and/or cardiopulmonary difficulties
- Antisialagogue, such as glycopyrrolate
- Antibiotic prophylaxis for pts with Hx of endocarditis, prosthetic valves, or foreign material in the heart

MONITORING

- Routine

AIRWAY

- Anh airway and short neck predispose to complicated airway management, incl difficulty in performing a tracheotomy.
- Limited jaw movement, enlarged tongue, and thick secretions compound airway challenges.

- ETT may need to be smaller than anticipated for the age and size of the pt.
- Consider fiberoptic bronchoscopy.
- LMA may be useful.

PREINDUCTION/INDUCTION

- IV placement before induction
- Padding and positioning

MAINTENANCE

- Avoid myocardial ischemia.
Extubation
• Conscious with intact airway reflexes prior to extubation

Adjuvants
• Utilize local anesthetics and regional techniques when appropriate

Postoperative Period
• Delayed emergence
• Resp complications incl pneumonia, bronchospasm, and apnea

Anticipated Problems/Concerns
• Airway is likely to be difficult to manage
• Cardiac and pulm systems frequently affected
**Multiple Endocrine Neoplasia (MEN) Type I and II**

**Risk**
- Neoplastic syndromes, inherited in an autosomal dominant pattern, variable penetrance, rare incidence. Syndromes involve more than one endocrine gland.
- MEN tumors and their effects may be underdiagnosed, unrecognized when pt presents for unrelated surgery. (MEN2a and 2b assoc with pheochromocytoma)
- Medullary carcinoma of thyroid (MEN2a, 2b) is inherited with almost 100% penetrance, prophylactic thyroidectomy is recommended. Genetic screening tests are available.

**Perioperative Risks**
- See specific syndrome, risk related to functional components of tumors

**Overview**
- MEN I “Werner’s syndrome” incl: Parathyroid hyperplasia (95%), anterior pituitary tumors (30%), pancreas (insulinoma, glucagonoma) (50%), gastrinoma (“Zollinger Ellison”) (20-60%)

**MEN I**
- Parathyroid hyperplasia (assoc nephrolithiasis)
- Pancreatic tumors (insulinoma, glucagonoma), gastrinoma
- Ant pituitary tumor (prolactinoma, growth hormone (GH) tumor, ACTH/Cushings)

**MEN IIa & b**
- Pheochromocytoma
- Medullary cancer of thyroid

**MEN IIa**
- Parathyroid adenoma (see MEN I)

**MEN IIb**
- Extremely rare subtype, (5% of all)

**ICD-9-CM Code: 258.0**

**Etiology**
- MEN I/II: autosomal dominant, variable penetrance. MEN I caused by mutation in MEN-1 gene (tumor suppressor/regulatory), men and women equally affected. MEN II caused by oncogenic mutation in c-Ret gene (regulatory).
- Incidence of MEN2a > FMTC > MEN 2b

**Usual Treatment**
- MEN I: parathyroid hyperplasia: Treat hypercalcemia medically, surgical resection of hyperplastic tissue with parathyroid reimplantation.
- MEN II: Treatment for medullary carcinoma is total thyroidectomy; pheochromocytoma, same treatment as in MEN2-a.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>MEN I</td>
<td>Parathyroid hyperplasia</td>
<td>Family Hx of endocrine tumors</td>
<td>Htn</td>
<td>NIBP and EKG</td>
</tr>
<tr>
<td></td>
<td>Pancreatic tumors (insulinoma, glucagonoma), gastrinoma</td>
<td>Fatigue, muscle weakness, flank pain/renal stones/Hx pathological fractures</td>
<td>Neck nodule</td>
<td>Serum calcium</td>
</tr>
<tr>
<td></td>
<td>Ant pituitary tumor (prolactinoma, growth hormone (GH) tumor, ACTH/Cushings)</td>
<td>Diarrhea, reflu, dyspepsia</td>
<td>Altered mental status</td>
<td>Sestamibi scan, PTH level, neck CT, bone density</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Headache, visual changes</td>
<td>Tremor, mental status changes</td>
<td>BUN/creatinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(hypoglycemia)</td>
<td>pelvic x-ray</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Visual field defect</td>
<td>Serum glucose, electrolytes, CT/MRI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Acromegaly (GH)</td>
<td>Endoscopic US</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cushingoid habitus</td>
<td>Head CT/MRI metabolic panel, specific hormone level</td>
</tr>
</tbody>
</table>


**Perioperative Implications: Men I**

**Monitoring**
- Parathyroid surgery: ECG signs of hypercalcemia (arrhythmias, prolonged PR, short Q-T), consider using EMG ETG tube for monitoring recurrent laryngeal nerve intraop. Unpredictable response to muscle relaxants with hypercalcemia, monitor TOE. PTH levels; significant decrease expected post successful resection, monitor calcium level postop.
- Pituitary adenomas: Tight BP control; acromegaly may have impaired uN circulation to hand which increases risk morbidity from radial a-line; monitor urine output (risk diabetes insipidus, SIADH)
- Insulinoma surgery: Requires tight, careful blood glucose control, increased risk hypoglycemia periop, arterial line
- Gastrinomas: Arterial line, pts at risk for labile BP

**Airway**
- Acromegaly: Increased risk of difficult mask airway and intubation, also increased incidence of sleep apnea, have difficult airway equipment ready.
- Parathyroideotomy: Risk of surgical damage to recurrent laryngeal nerve, and vocal cord paresis periop (risk of hoarseness to stridor to complete airway obstruction if bilateral)

**Maintenance**
- Parathyroideotomy: Draw post resection PTH levels to confirm removal of tumor.
- Insulinomas, gastrinomas: Monitor volume status, glucose, BP control
- Pituitary adenomas: Usually transtemporal approach, tight BP control, watch O2

**Perioperative Implications: Men II**

**Monitoring**
- Pheochromocytoma: Standard ASA monitors, arterial line, CVP, UO

**Airway**
- Thyroidectomy: Standard ASA monitors. Consider use of EMG ETG to monitor recurrent laryngeal nerve intraop. Postop PTH levels to check for adequate parathyroid function
- Parathyroideotomy: See MEN-1 section

**Maintenance**
- Pheochromocytoma: Tight BP control before and during resection (anesthetics, nipride, phentolamine, esmolol, Ca channel blockers, epidural infusions); after adrenal ligation, BP support with fluid boluses, prn pressors (NE, phenylephrine). Monitor glucose.
Thyroidectomy: If using EMG ETT, avoid muscle relaxants.
Parathyroidectomy: See MEN I

Adjuvants
- Pheochromocytomas: Require adequate preop treatment to control BP, HR, and restore blood volume (10–14 d alpha adrenergic blockers (ex. phenoxybenzamine, or prazosin), hydration, then initiate beta blockade)

Hyperparathyroidism with symptomatic hypercalcemia: Preop hydration, diuresis with furosemide, consider bisphosphonates, calcitonin or glucocorticoids

Anticipated Problems/Concerns
- MEN I: Parathyroidectomy: Postop hypocalcemia, recurrent laryngeal nerve damage/VC paresis, neck hematoma/airway compromise.

MEN II: Pheochromocytoma; malignant Htn and labile BP, increased risk of CVA, MI.

Multiple Myeloma

Susheela Viswanathan
Alan Kaye
Alecia L. Sabartinelli

Risks

- Incidence: 4.3/100,000 white males; 3/100,000 black females
- Race: 1.1% of all malignancies in white population; 2.1% of all malignancies in black population
- M:F ratio: 3:2
- Age: Median age 68 y men, 70 y in females
- Survival: Median survival 3 y; 100% fatality rate

Overview

- MM is a part of a spectrum ranging from monoclonal gammopathy of unknown significance (MGUS) to plasma cell leukemia (malignancy of antibody forming cells)
- Also known as plasmacytosis or Kahler's disease; classified within non-Hodgkin's lymphomas
- Proliferation of plasma cells results in functioning peripheral blood cells and leads clinically to:
  * Impaired production of blood cells > pancytopenia (leucopenia anemia thrombocytopenia)
  * Formation of plasmacytoma (mass), leading to lytic lesions in bone
  * Impaired immunity (humoral) > infection

ICD-9-CM Code: 203.0

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Clinical Manifestations</th>
<th>Signs and Symptoms</th>
<th>Anesthetic Implication</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>Bone pain Pathological fracture</td>
<td>Usually lumbar 95% more than one side</td>
<td>Positioning to prevent fracture</td>
</tr>
<tr>
<td>HEME</td>
<td>Bleeding and bruising Coagulopathy Normochronic normocytic anemia</td>
<td>2° Thrombocytopenia Absorption of clotting factor Weakness Purpura Dark circles (raccoon like) around eye, 2° prolonged valsalva</td>
<td>Availability of FFP and plts ↑ Transfusion requirements, ventilator management</td>
</tr>
<tr>
<td>METAB</td>
<td>Hypercalcemia Infection</td>
<td>Confusion, somnolence, constipation, nausea, thirst, bone pain 2° humoral immunity of normality</td>
<td>↑ Fluid requirements, maintenance of adequate UO Antibiotic coverage</td>
</tr>
<tr>
<td></td>
<td>Hyperviscosity</td>
<td>Epistaxis Visual disturbance Carpal tunnel Headache Somnolence, bruisability</td>
<td>Preop: Plasmapheresis, ↑ fluid requirement intraop Temp maintenance to prevent microvascular sludging</td>
</tr>
<tr>
<td>CNS and PNS</td>
<td>Spinal cord compression Meningitis Carpal tunnel Peripheral neuropathies Stroke (hyperviscosity)</td>
<td>Signs of weakness and numbness of extremities</td>
<td>Positioning of pt Dilgent use of muscle relaxants Avoidance of depolarizing muscle relaxants</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficient/failure</td>
<td>2° Direct tubular injury Amyloidosis Involvement by plasmacytoma</td>
<td>Adequate hydration</td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumonia Resp insufficiency</td>
<td>2° rib fracture</td>
<td>Exubation problems Pneumothorax intraop</td>
</tr>
<tr>
<td>HEENT</td>
<td>Amyloidosis Macroglossia</td>
<td>Skin lesions of lips</td>
<td>Airway problems</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative

- Recombinant erythropoietin increased hemoglobin and decreased transfusion requirement
- Antibiotics and gammaglobulin prophylaxis

Airway

- May be difficult due to macroglossia

Maintenance

- Regional anesthesia is contraindicated due to bony lesions, coagulopathy, and neurologic deficit
- Unpredictable pharmacokinetic of protein-bound drugs

Post Operative

- Continue adequate hydration
- Aggressive pulm toilet
- Treat specific complication (refer to Treatment of Complication of Multiple Myeloma)

Anticipated Problems/Concerns

- Careful positioning to prevent fractures
Multiple Sclerosis

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Pseudobulbar palsy</td>
<td>Hx of swallowing difficulty</td>
<td>Cranial nerves IX, X</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>May have aspirated from bulbar dysfunction, seizure</td>
<td>Review with family</td>
<td>Auscultation</td>
<td>CXR, oximetry</td>
</tr>
<tr>
<td>GI</td>
<td>GI effects of steroids</td>
<td>Hx of pain, bleeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Cognitive dysfunction, optic neuritis, seizures, dysesthesias, ophthalmoplegia, autonomic dysfunction, monoplegia, transverse myelitis, quadriplegia</td>
<td>Hx of memory loss, emotional lability, “dropping things”, visual problems, Lhermitte’s sign (electric shock to legs); check with family for description of seizures</td>
<td>Mental status exam</td>
<td>Orthostatic vital sign changes</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Consider steroid supplementation; avoid anticholinergics
- May need benzodiazepine premedication to reduce risk of stress-induced exacerbation
- Carefully document preop neurologic status
- Adequate volume status
- Orthostatic intolerance and inappropriate heart-rate responses may be seen due to autonomic instability.

**Monitoring**
- Routine

**Airway**
- None

**Preinduction/Induction**
- Spinal anesthesia implicated in aggravating MS symptoms and considered contraindicated
- Caution with epidural anesthesia: Need clear indication, such as in obstetrics, pt informed of possible symptom exacerbation, use lower concentrations of local anesthetics, and epidural opioids are OK to use

**Maintenance**
- Careful maintenance of normothermia
- Continue steroid stress coverage
- Careful titration of nondepolarizing NMBs

**Exubation**
- Pts with brainstem involvement should be extubated awake
- Spasticity may diminish maximal insp effort

**Adjuvants**
- Duration of most NMBs shortened by phenytoin and carbamazepine

**Etiology**
- Cause unknown
- Autoimmune, genetic, environmental factors thought to combine to attack CNS myelin

**Usual Treatment**
- No treatment curative
- Steroids, interferon, azathioprine, cyclophosphamide ameliorate relapses; methotrexate may also be used.
- Intrathecal baclofen pumps are sometimes seen for control of spasticity.
- Carbamazepine used for paroxysmal Sx (incl pain); baclofen, and, occasionally, surgery for spasticity (thalamotomy)
- Interferon-β now a first line treatment, shown to decrease rate and severity of relapses, may delay onset of disability

**Postoperative Period**
- Exacerbations common in postop period regardless of anesthetic technique; perform neuro exam
- Continue supplemental steroid coverage
- Treat hyperthermia
- Spasticity may interfere with pulm toilet

**Anticipated Problems/Concerns**
- Unpredictable appearance of new neurologic deficits perop
- Exacerbation of MS symptoms with hyperthermia, stress, pain
- Hyperkalemia with succinylcholine, unpredictable blockade with NMBs
- Emotional lability and need for sedative premedication

**Risk**
- Prevalence: 10–90/100,000 in North America
- Occurs primarily in temperate climates, with a North to South gradient in the USA
- Female predominance up to 2:1
- Onset usually in third or fourth decade of life
- Racial predominance: Caucasian 6 x the incidence of all other races

**Perioperative Risks**
- Exacerbation of Sx with temp elevation, stress of surgery, infection, emotional trauma, postpartum state
- Risk of positioning injury (muscle wasting), hyperkalemia with succinylcholine, DVT
- Steroid therapy predisposes to adrenal suppression, gastric ulceration

**Worry About**
- Advisability of major conduction block; greater neurotoxicity of local anesthetics due to demyelination. Spinal anesthesia has been reported to exacerbate symptoms.

**Overview**
- Demyelinating disease of brain and spinal cord (peripheral nerves are not affected), with chronically remitting and relapsing or progressive course
- Assoc conditions incl seizures and uveitis; CNS components involved are cortex (cognitive dysfunction, memory loss, personality change, emotional lability) and spinal cord
- Chronic dysesthetic pain and spasticity contribute to disability
- Paroxysmal Sx may mimic cerebral ischemia, spinal cord compression, tic douloureux

**ICD-9-CM Code:** 340
Multisystem Organ Failure, Lung Dysfunction In

Risk Factors
- The incidences of adult respiratory distress syndrome (ARDS) and acute lung injury (ALI) are 8 and 50 cases per 100,000 person-years, respectively.
- ARDS/ALI are the manifestations of lung dysfunction in MODS.

Risk Factors
- Systemic sepsis, polytrauma, severe pancreatitis, multiple transfusions
- Pneumonia, lung contusion, near drowning, inhaled toxins, DIC
- ARDS affects 15% ICU pts and has a mortality rate of 40%.
- Refractory hypoxia is an uncommon cause of death in these pts.
- Majority of ARDS deaths due to MODS

Perioperative Risks
- Hypoxemia
- Systemic hypotension
- Pulm Htn, RV dysfunction/failure

Worry About
- High PEEP reducing preload causing hemodynamic instability
- Barotrauma and auto PEEP (esp in obstructive pulm diseases)

Overview
- Four overlapping disease states exist in a spectrum of severity, incl SIRS
  - Descending order of severity; MODS > severe sepsis > sepsis > SIRS
  - ALI is characterized by massive inflammation and leakage of protein rich fluid from pulm capillaries into the alveoli. This phase lasts a few days, inhibiting gas exchange (↓ PaO₂ / ↑ PaCO₂) and decreasing pulm compliance
  - ALI subsequently becomes a fibro-proliferative process, frequently permanent

Definition: ARDS and ALI, American and European 1994 Consensus Conference
- Acute onset hypoxemia and resp failure
- Bilateral diffuse infiltrates on chest radiography
- Pulm artery occlusion pressure <18 mm Hg (no left atrial Htn)
- PaO₂ / FiO₂ ratio of ≤300 for ARDS, ≤200 for ALI

ICD-9-CM Code: 995.92 (Severe sepsis)

Etiology
- ALI in MODS may have pulm or extra pulm causes

Usual Treatment
- Aggressive Dx, treatment of underlying cause with timely antibiotics
- General supportive therapy using well established sepsis bundles
- Lung protective strategies (LPS) to minimize ventilator-induced lung injury
  - Plateau <30 cm H₂O, TV 6 mL/kg, permissive hypercapnia

Perioperative Implications

Preoperative Preparation
- Transport from ICU to the OR, consider RRT/RN/perfusionist/intensivist help
- If not intubated, ensure equipment, medications, personnel, monitors present
- Consider ICU ventilator for case (high PEEP +/- inhaled NO), oscillator
- Consider arterial line, central line, PA catheter, TEE

Airway
- Ensure airway is secure. Decompensation may be rapid and hard to recover from.
  - Consider continuous suction catheter ET (potentially reduces incidence of ventilator-assoc pneumonia)

Preinduction/Induction
- Hypoxemia and/or hypercarbia may exacerbate pulm Htn and RV dysfunction
- Reduced FRC exacerbates ventilation/perfusion (V/Q) mismatch, worsening PaO₂

Maintenance
- Adequate PEEP restores FRC, decreasing shunt, improving V/Q and oxygenation
- Appropriate fluids and blood products for optimum preload
- Vasopressors and inotropic support when indicated
- Surgical manipulation of septic focus may cause adverse hemodynamic effects
- Consider use of spectral edge EEG analysis to guide mixed balance anestesia
- Consider cisatracurium for organ-independent elimination
- Maintain case-appropriate temp

Exubation
- Consider leaving pt intubated for continued postop ventilation
- Avoid shivering, which increases O₂ consumption dramatically

Adjuvants
- NO, inhaled prostaglandins, surfactant, bronchoscopy

Postoperative Period
- Close monitoring and treatment of volume status, PEEP, hemodynamic support
- Reduce FIO₂ as soon as reasonably possible, maintaining SpO₂ > 90%

Anticipated Problems/Concerns
- Ventilator-assoc pneumonia
- Barotrauma (pneumothorax/mediastinum/pericardium)

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Hx</th>
<th>Exam</th>
<th>Test</th>
<th>Rx</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO (septic shock)</td>
<td>Low CO</td>
<td>MAP</td>
<td>Edema</td>
<td>ECG, troponin</td>
<td>Euvolemia</td>
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<tr>
<td></td>
<td>RV dysfunction</td>
<td></td>
<td>Gallop</td>
<td>ECHO</td>
<td>Conservative fluids if no hyperfusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>S₁</td>
<td>PA catheter</td>
<td>Vasopressors</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>BNP</td>
<td></td>
</tr>
<tr>
<td>RESP (ALI/ARDS)</td>
<td>Hypoxemia</td>
<td>Respiratory distress</td>
<td>Crackles</td>
<td>ABG</td>
<td>Protective strategies</td>
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<tr>
<td></td>
<td>↓ Compliance</td>
<td>↑ P₂</td>
<td>P₂</td>
<td>CXR</td>
<td>TV – 6 mL/kg</td>
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<tr>
<td></td>
<td>Pulm Htn and edema</td>
<td></td>
<td></td>
<td>PA catheter</td>
<td>High PEEP</td>
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<tr>
<td>GI, HEPATIC (shock liver)</td>
<td>Ileus</td>
<td>Pain</td>
<td>Hydroactive</td>
<td>Lactate</td>
<td>NPO, NGT, IV fluid</td>
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<tr>
<td></td>
<td>Dysfunction</td>
<td>N/V</td>
<td>Ascites</td>
<td>KUB</td>
<td>+/- TPN</td>
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<tr>
<td></td>
<td>Hemorrhage</td>
<td>Distension</td>
<td>Tense abdomen</td>
<td>CT scan</td>
<td>GI stress ulcer Rx</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Guaic stool</td>
<td>Bladder pressures</td>
<td></td>
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<tr>
<td>RENAL (ATN)</td>
<td>Acute kidney injury</td>
<td>RIFLE criteria</td>
<td>Urine volume</td>
<td>Chem 7, FeNa</td>
<td>Renal replacement therapy</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Urinalysis</td>
<td>US</td>
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<tr>
<td>HEME</td>
<td>Anemia</td>
<td>↓ Hgb</td>
<td>Pallor</td>
<td>CBC</td>
<td>PRBC: Hgb 7–9 g/dL</td>
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<tr>
<td></td>
<td>Coagulopathy</td>
<td></td>
<td>Bleeding</td>
<td>Plts</td>
<td>Plts: 5–30 k</td>
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<tr>
<td></td>
<td>DIC</td>
<td></td>
<td></td>
<td>PT/PTT</td>
<td>FFP: High risk/IR/OR</td>
</tr>
<tr>
<td>CNS (↓ mental status)</td>
<td>Cerebral hypoxia</td>
<td>↓ GCS</td>
<td>Deficits: CNS/PNS</td>
<td>Fibrinogen</td>
<td>Cryo: ≤80 fibrinogen</td>
</tr>
<tr>
<td>ENDO</td>
<td>Insulin resistance</td>
<td>↑ BG</td>
<td>Goal &lt;150 mg/dL</td>
<td>CT brain, MRI</td>
<td>Maintain CPP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lumbar puncture</td>
<td>Avoid hypotension</td>
</tr>
</tbody>
</table>

Myasthenia Gravis

Overview
- Characterized by weakness and fatiguability of skeletal muscles
  - Insp muscle weakness from residual paralysis from nondepolarizing NM blocking agents
  - Exacerbation of underlying bulbar (airway) musculature weakness
  - Increased sensitivity to hypoventilation with narcotic analgesics
- Muscle strength improves similarly in both myasthenia gravis and nondepolarizing blockade after administration of anticholinesterase drugs.

ICD-9-CM Code: 358.0

Etiology
- Autoimmune disease of NM junction mediated by reduction in number of acetylcholine receptors at NM junction

Usual Treatment
- Anticholinesterase medications (pyridostigmine, Mestinon)
- Immunosuppression: Steroids, azathioprine
- Plasmapheresis
- Intravenous immunoglobulin (IVIG)
- Thymectomy

Risk
- Incidence in USA: 50–142 cases per million
- Affects all races
- M:F ratio: 2:1

Perioperative Risks
- Postop NM ventilatory failure
- Postop pneumonia due to poor cough and secretion clearance

Worry About
- Preop optimization of muscle strength
- Anticholinesterase medications, steroids, plasmapheresis

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>NM</td>
<td>Peripheral muscle weakness</td>
<td>Easy fatiguability</td>
<td>Arm adduction times &lt;1 min</td>
<td>Repetitive nerve stimulation</td>
</tr>
<tr>
<td>RESP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Airway</td>
<td>Bulbar weakness</td>
<td>Difficulty swallowing</td>
<td>Head lift &lt;5 sec</td>
<td>Formal swallowing evaluation</td>
</tr>
<tr>
<td>Ventilation</td>
<td>Insp muscle weakness</td>
<td>Orthopnea, breathlessness</td>
<td>Paradoxical insp motion</td>
<td>NIF &lt;30 cm H₂O</td>
</tr>
<tr>
<td>Ventilatory drive</td>
<td>CO₂ retention</td>
<td>Morning headache</td>
<td>↓ Ventilation of bases</td>
<td>FVC &lt;1000 mL</td>
</tr>
<tr>
<td>Secretion clearance</td>
<td>Weak cough</td>
<td>Recurrent pneumonia</td>
<td>ABGs</td>
<td>CXR</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Anticholinesterase medications
  - Hold 2–4 hr preop
  - Postop: May use IV neostigmine to replace pyridostigmine PO, 1 mg IV/60 mg PO or start IV neostigmine 1 hr before emergence at 1/30–1/60 daily pyridostigmine dose, infuse over 24 hr
- Steroid maintenance

Monitoring
- Routine
- Train-of-4 twitch monitor if short-active nondepolarizers used

Induction/Intubation
- Consider inhalational anesthetic breathe-down techniques

Intubation Without Muscle Relaxation, Using Propofol/Remifentanil Maintenance
- Minimize or avoid use of muscle relaxants
- TIVA or inhalational anesthesia

Extubation
- Check NIF (>30 cm H₂O), head lift, cough, gag, ensure full return of twitch

Adjuvants
- Avoid or minimize use of nondepolarizing muscle relaxants
- Depolarizing relaxants may have ↑ or ↓ efficacy
- Consider epidural analgesic, particularly for thymectomy.

Anticipated Problems/Concerns
- Postop ventilatory failure, pneumonia, aspiration
- Cholinergic crisis if excess anticholinesterase medications given
Mycoplasma pneumoniae Infection

Risks
- Endemic and/or pandemic worldwide every 3–5 y
- Outbreaks likely during summer, early fall
- Affects persons of all ages
- Long incubation periods 1–3 wk
- Transmitted person to person via aerosols
- Frequent in closed and semiclosed communities
- Common cause of upper and lower resp infections
  - Up to 40% of community-acquired pneumonias, walking pneumonia
  - Up to 5% of bronchiolitis in children
  - 3–10% of adults may develop broncho-pneumonia
- Clinical manifestations similar to C. pneumonia, S. pneumonia and resp viruses
  - Fulminant pneumonia may occur in children with sickle cell disease (functional asplenism), Down syndrome, and immunosuppressive conditions.
  - Extrapulmonary complications in 25% of pts infected with M. pneumoniae.

Perioperative Risks
- No periop risk data; hemolytic anemia, DIC, cross-reacting cold agglutinins are of concern esp if CPB is required.
- Hyperreactive airway disease

Worry About
- Multisystem organ dysfunction

Overview
- Clinical manifestations of resp involvement are mediated by activity of cytadherence on the airway epithelium and incl:
  - Sore throat, hoarseness, fever, cough (pertussis-like), may play a role in asthma, COPD.
  - Conjunctivitis, headache, chills, coryza, myalgias, earache, and generalized malaise are common.
- Extrapulmonary manifestations are the result of direct invasion or immune reaction.

Diagnosis
- Hx and clinical manifestations: Nonspecific
- CXR: Diffuse reticular infiltrates in perihilar and lower lobe regions. Bilateral in 20% of cases
- Pathology: Ulceration, edema, ciliary loss, bronchoalveolar inflammatory cell infiltration
- Culture: Incubation period of several weeks. Sensitivity around 60%. Not practical for routine Dxs.
- Serology: Current or recent infection likely if antibody titer increase ≥ 4-fold.
- Cold agglutinins: IgM within 1–2 wk after initial infection. Titters ≥ 1:32 correlate with severity of lung involvement
- Polymerase chain reaction: RNA-amplification techniques are highly sensitive and indicate viable bacterium.


ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Organ</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Otitis Retinitis conjunctivitis</td>
<td>Ear symptoms may affect 30%</td>
<td>Mucosal congestion</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Tracheobronchitis Pneumonia Asthma</td>
<td>Failure to respond to treatment with sulfonamide or penicillin</td>
<td>Persistent cough Expiratory wheezes</td>
<td>CXR Sputum</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Pericarditis Pericardial effusion Cardiac tamponade Myocarditis</td>
<td>Incidence: 1–8.5%</td>
<td>Distant heart sounds S3, JVD</td>
<td>ECG ECHO</td>
</tr>
<tr>
<td>CNS</td>
<td>Aseptic meningitis Meningoencephalitis Transverse myelitis Guillain-Barré Peripheral neuropathy Cerebellar syndrome</td>
<td>Incidence 7%, children more likely to die or have severe neurologic deficits</td>
<td>Focal or general neurologic symptoms, diplopia, coma</td>
<td>CSF Elevated cytokines IL-6, IL-8 MRI Serology</td>
</tr>
<tr>
<td>HEM</td>
<td>Hemolytic anemia Cold agglutinins DIC</td>
<td>More common in children Likely due to cross-reactive antibodies</td>
<td>Peripheral cyanosis</td>
<td>IgG-free Hgb D Coombs’</td>
</tr>
<tr>
<td>DERM</td>
<td>Maculopapular Vesicular rash Stevens-Johnson Syndrome</td>
<td>May affect up to 25%</td>
<td>Rash, but needs to rule out rash due to Abx</td>
<td>M. Pneumoniae has been detected in cutaneous lesions</td>
</tr>
<tr>
<td>RENAL</td>
<td>Glomerulonephritis Tubulointerstitial nephritis IgA nephropathy Paroxysmal cold Hemoglobinuria</td>
<td>Brisk hemolytic anemia</td>
<td>UA renal biopsy Ig G, M, A</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Evaluation
- Routine physical exam: Emphasis on, resp, CNS, CV, and HEENT systems.
- Resp-increased minute ventilation, low sat, prolonged ventilation may be required.
- CNS-document preexistent neuropathy
- CV-JVD, rule out tamponade physiology
- HEM-hemolysis and anemia. If cold agglutinins are suspected, determine temp range and titers.
- If surgery is non-urgent, consider postponing it until active issues resolved.

Monitoring
- Invasive monitoring necessary if resp and CV concerns.

Airway
- Desaturation is possible due to decreased FRC
- High incidence of hyperreactive airway disease

Maintenance
- Normothermia is essential to avoid cold agglutinins. Warm all fluids, humidify airway.
- If hemolysis develops; optimized UO, alkalinized urine and use diuretics.

Exubation
- Clear mental status, good resp mechanics, able to clear secretions.

Anticipated Problems/Concerns
- Resp distress 2° to asthma, COPD, high O2 requirements may result in prolonged intubation.
- Neurologic deficit may delay extubation.
- CPB and/or cold agglutinins may result in circuit obstruction and impair myocardial protection.
Myocardial Contusion (Blunt Cardiac Injury)

Risk
- Incidence unknown, in part due to absence of clear diagnostic criteria/test
- Two million motor vehicle accidents/y with ∼40% involving closed chest injury
- 20–70% incidence by clinical criteria
- 16–20% incidence by autopsy
- Motor vehicle > falls > crush injuries
- Males > females (5:1)
- Commotio cordis is a rare form of BCI
- Due to low impact chest injury (sports) causing sudden death

Perioperative Risks
- Abn ECG
- Nonspecific ST-T wave changes (70%) in trauma pts
- Q wave and ST elevation
- 7–17% false negative
- 60% false positive
- Ventricular arrhythmias most common in contusion
- Trifascicular conduction block
- Other cardiac conditions: Thrombosed, lacerated coronary arteries in spasm; ventricular hypofunction; pericardial effusion/tamponade; pericarditis; valvular insufficiency, left-sided > right-sided; ventricular wall rupture (incl septum)
- Possible increased risk of cardiac complications (arrhythmias, hypotension) with increased CK-MB troponins, and abn ECHO
- No evidence of increased mortality assoc with GA

Worry About
- Malignant ventricular arrhythmia (acute and delayed)
- Cardiac conduction blocks incl complete heart block
- Volume status
- Acute hypotension
- Delayed myocardial rupture
- Assoc injuries: Pulm contusion—hypoxemia, thoracic aorta injuries, flail chest

Overview
- Traumatic injury with hemorrhagic, well-circumscribed lesions of partial or full thickness from myocardial contusion
- Usually of RV but can be multichambered

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Ventricular contusion</td>
<td>Angina-like chest pain unrelieved by nitrates</td>
<td>Chest wall, sternal tenderness</td>
<td>ECG, serial</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dyspnea</td>
<td>Hypotension with severe dysfunction</td>
<td>Troponin I and T within 6 hr</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>S₃ Rales</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Palpitations, dizziness, syncope</td>
<td>Pulse</td>
<td>MRI</td>
</tr>
<tr>
<td></td>
<td>Arrhythmia</td>
<td>Dyspnea</td>
<td>Auscultatory murmurs</td>
<td>ECG monitoring</td>
</tr>
<tr>
<td></td>
<td>Valvular disruptions</td>
<td>Chest pain</td>
<td></td>
<td>Angio</td>
</tr>
<tr>
<td></td>
<td>Coronary artery injury: thrombosis, laceration, spasm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Effusion/tamponade</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>CHF</td>
<td>Dyspnea</td>
<td>Pericardial friction</td>
<td>TTE</td>
</tr>
<tr>
<td></td>
<td>Pulm contusion</td>
<td>Orthopnea</td>
<td>Diminished heart sounds</td>
<td>2-D cardiography</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chest tightness</td>
<td>Distended neck veins</td>
<td>PA catheter</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- 2-D ECHO or TEE abn predict periop hypotension
- Assess and ensure adequate volume replacement
- Assess and treat assoc concurrent injuries
- No evidence for benefit of prophylactic antiarrhythmic agents

Monitoring
- Continuous ECG for arrhythmias
- PA catheter for large fluid shift operations or pts with signs of LV dysfunction
- Increased risk of periop arrhythmias without increased mortality

Airway
- Evaluation for associ airway injury

Preinduction/Induction
- Adequate volume replacement

- Hypotension more likely with large contusions
- Extra attention to avoid hypoxia, hypovolemia

Maintenance
- No one agent or technique shown superior
- Avoid known pulm vasoconstrictors: catecholamine, hypoxia, acidosis, histamine-releasing agents (MgSO₄, mivacurium)
- Consider high inspired O₂ if contusion
- NO can aggravate pulm Htn
- Elevations in PVR may unmask RV failure
- Increased LV filling pressures and decreased cardiac output often reflect hypovolemia or are 2° to RV failure, not LV failure

Extubation
- May leave intubated if concerns for resp failure and hypoxia present

Adjuvants
- Combination of appropriate intravascular volume replenishment and vasodilators (nitroglycerin) for pulm Htn

Postoperative Period
- Delayed hypoxia from pulm injury common and can cause pulm Htn leading to hypotension if RV severely contused

Anticipated Problems/Concerns
- Variable diagnostic criteria, total CK-MB >50 U/L and ≥5% total CK
- Possible higher risk of cardiac complications with ↑ CK-MB
- Almost any arrhythmia reported, esp. conduc’ion delays; more severe contusion assoc with ↑ malignant ventricular arrhythmia
- Watch for RV failure leading to increase LV pressure but decrease LV diastolic filling.
Myocardial Ischemia (MIsch)

Risk
• Incidence in USA: 1.5 million/y develop acute myocardial infarction (MI); decreased rate of death in USA balanced by increased population has kept MI numbers constant since 1970 despite increased population; 7 million worldwide had MI/year
• 10 million in USA have ≥70% narrowing of 1 or more coronary arteries
• European, Indian, and African-American heritage > Japanese, but environment of North America equalizes risks
• Highest in pts with known other atherosclerotic disease (incl prior MI): smokers (3.5-fold increase); hypertensives (3-fold increase); diabetics (4-fold increase); hypercoagulable or chronic inflammatory diseases (3-fold increase); stressed, divorced, or unstable marriage (2.5-fold increase); with wt gain since age 20 y (1.5-fold increase for each 5 kg increase); increase LDL cholesterol in those who do not exercise (0.5% increase for each 1% increase than 100 mg/dL); who do not drink or take vitamin D or aspirin; whose parent died of CAD at < age 40 y (1.4 to 2.5-fold increase); age (3-fold increase per decade over 50), family Hx (1.1 to 2.4-fold increase)

Perioperative Risks
• Increases risk 9-fold of periop CV complications (MI, CHF, Rt HF, arrhythmia requiring Rx)

ASSESSMENT POINTS

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<thead>
<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Plaques in other areas</td>
<td>Risk factor search: Smoking stain; hypercholesterolemic lesions</td>
<td>McArule’s earlobe</td>
<td>ECG, CXR, stress ECHO or dipyridamole thallium or ambulatory Holter, troponins, and myeloperoxidase tests</td>
</tr>
<tr>
<td>CARDIO</td>
<td>↓ LV or RV compliance</td>
<td>SOB, DOE, Angina, ↓ Exercise tolerance, Palpitations</td>
<td>HR/BP prior to and after 2-stair climb; S; rales; JVD; use character and rhythm</td>
<td>BUN/Cr</td>
</tr>
<tr>
<td>RESP</td>
<td>Nocturnal cough, orthopnea</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Perfusion insufficiency</td>
<td>Nocturia, Erectile dysfunction (male), Loss of ability to achieve orgasm (female)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Autonomic pain syndromes</td>
<td>Pain in neck or left arm Stroke/TIA Hx</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Other atherosclerotic syndromes</td>
<td>CNS and cranial nerve exam Carotid Doppler</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Induction
• Without hemodynamic disturbance and esp. with HR control

Maintenance
• Tachycardia or hypovolemia and Hct <28 can precipitate ischemia.
• No agent with demonstrated outcome superiority.
• Intensively normalize hemodynamics and HR

Extubation
• In nonstressful fashion for pt without compromising supply of O2 to myocardium
• Aggressive stepped pain therapy recommended; alpha-2 adrenergic agonist recommended by some

Adjutants
• CHF decreases liver blood flow and clearance of drugs requiring hepatic metabolism (such as lidocaine)
• β-adrenergic receptor antagonists and nitrates can be assoc with profound hemodynamic disturbances if drug interactions or sudden preload, afterload, or contractility perturbations (such as rapid onset of spinal anesthesia) occur.

Anticipated Problems/Concerns
• Pre- and postop periods at least as great a cause of morbidity as intraop period
• Restart anti-anginal and antiplatelet therapies, i.e., statins, CO Q10, aspirin, DHA, etc., and physical activity rehab program as soon as possible postop if D/C preop
• Consider compassionate anxiety-relieving yet aggressive preop consultation and intensive stepped pain prophylaxis consultations postop.
Myotonia Dystrophica (Myotonic Dystrophy, Steinert’s Disease)

**Incidence**
- Myotonic dystrophy is a rare disease with an incidence of about 1 in 8000. The incidence of the congenital form is higher with an incidence of 1/100,000

**Perioperative Risks**
- Operative/anesthetic and postop morbidity/mortality are increased and not proportional to severity of disease
- High incidence of cardiopulmonary complications, incl sudden death, cardiac failure, cardiomyopathy

**Worry About**
- Increasing frequency of symptoms
- Signs of resp or cardiac decompensation

**Overview**
- Degenerative disease of skeletal muscles. Progressive distal muscle wasting. Triad of characteristic features described as frontal baldness, cataracts, and mental retardation.

- Extremely variable in presentation: Asymptomatic cases to congenital with mental retardation and resp insufficiency.
- Typically onset of symptoms in 2nd and 3rd decades of life with progressive muscular weakness and wasting most common in the cranial and distal limb muscles—temporalis and masseter muscle, atrophy known as hatchet face and limb muscles; initial affected result in footdrop and weak handshake. Deep tendon reflexes reduced and muscles of the vocal cord apparatus result in nasal speech. Proximal muscle variant recently recognized; death frequently in 5th or 6th decade of life, usually due to cardiopulmonary complications incl sudden death from conduction abn, cardiomyopathy, and CHF.
- Persistent contracture after cessation of stimulation or voluntary contraction of the muscle. This inability of the skeletal muscle to relax is diagnostic. EMG is corroborative and pathognomonic, showing continuous low-voltage activity with high-voltage, fibrillation-like potential bursts.

- Intrinsic disorder of skeletal muscle linked to myotonin-protein kinase gene on chromosome 19q13.2. Defect in Na⁺ and Ca²⁺ channel function produces electrical instability of the muscle membrane and self-sustaining runs of depolarization. May also have abn Ca²⁺ metabolism.

**ICD-9-CM Code: 359.2**

**Etiology**
- Inherited autosomal dominant trait; abn expansion of the nucleotide CTG on chromosome 19, which codes for a serine-threonine protein kinase. Variable gene expressivity as within same family can have minimally affected and severely affected individuals.

**Usual Treatment**
- Quinine, procainamide, phenytoin, tocainide, mexiletine (depress Na⁺ influx)

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE/Clinical Sequelae</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Visual disturbance</td>
<td></td>
<td>Presenile cataract, ptosis, strabismus, retinal pigmentation</td>
<td>Exam by ophthalmologist</td>
</tr>
<tr>
<td></td>
<td>Speech/swallowing impaired</td>
<td></td>
<td>Generalized weakness of pharyngeal, mandibular (and thoracic) musculature</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Dysarthria, facial weakness</td>
<td>ECHO, Holter</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Expressionless facies</td>
<td>Cardiology consult</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Dystrophias</td>
<td>CHF uncommon but may occur with pregnancy</td>
<td>Delayed intraventricular conduction</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>Cardiomyopathy</td>
<td></td>
<td>Heart block, hypotension</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Up to 20% with mitral valve prolapse, sudden death</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive lung disease</td>
<td>Weak cough</td>
<td>Wasting of sternocleidomastoid muscles; resp muscle weakness</td>
<td>PFTs</td>
</tr>
<tr>
<td></td>
<td>Chronic aspiration</td>
<td>Dyspnea</td>
<td>Lungs intrinsically normal; ↓ VC, ↓ ERV, ↑ CO₂</td>
<td>ABGs</td>
</tr>
<tr>
<td></td>
<td>Central hypoventilation</td>
<td>Hx of pneumonia</td>
<td></td>
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</tr>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>GI</td>
<td>High aspiration potential</td>
<td>Weak swallowing ability</td>
<td>Tensor paralysis, DM</td>
<td>Blood/urine glucose tests</td>
</tr>
<tr>
<td></td>
<td>Delayed esophageal and gastric emptying</td>
<td></td>
<td>↑ Thymus function</td>
<td>Thyroid function tests</td>
</tr>
<tr>
<td></td>
<td>Gastric dilation/atrophy ↑ Incidence of cholelithias</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>ENDO/IMMUNE</td>
<td>Testicular atrophy</td>
<td>Thyroid nodules</td>
<td></td>
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<tr>
<td></td>
<td>DM</td>
<td>↓ Immunoglobulins</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Thyroid function</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Adrenal insufficiency</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Frontal balding ?Malignant hypothyroidia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Mental retardation</td>
<td>Myotonic handgrip (delayed, incomplete release, ↑ CK in serum)</td>
<td>Myotonia can be initiated or worsened by exercise or cold temp, ↓ DTR</td>
<td>EMG, CK</td>
</tr>
<tr>
<td></td>
<td>Assoc with central sleep apnea and hypersomnolence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Emotional abn</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GYN</td>
<td>Pregnant pt is a challenge. Resp function threatened by ↓ FRC and myotonic weakness, which may be exacerbated by pregnancy. Seems to be added risk for uterine hemorrhage at delivery due to uterine atony and retained placenta. C-section may be safer.</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Ensuring NPO status (increased aspiration) and recent ECG
- No preop analgesics or sedatives and caution with benzodiazepines
- Warm ambient room air in OR may ↓ incidence and severity of myotonia

- Routine monitoring
- Airway
  - Propensity for frequent jaw dislocation
  - Potential inability to secure airway because of jaw muscle spasm
- Preinduction/Induction
  - Risk for aspiration of gastric contents
  - Induction: Gaseous; avoid slow metabolizing hypnotics; use lower doses on propofol
  - Relaxation: Avoid succinylcholine (link to malignant hyperthermia, severe extended contracts); use short-acting non-depolarizing agents at lower doses; recovery may be prolonged.
  - May be hard to differentiate from onset of MH.

- Induction: Gaseous; avoid slow metabolizing hypnotics; use lower doses on propofol
- Relaxation: Avoid succinylcholine (link to malignant hyperthermia, severe extended contracts); use short-acting non-depolarizing agents at lower doses; recovery may be prolonged.
Diseases

Maintenance
- Myotonia may be precipitated by drugs (propofol, succinylcholine, anticholinesterases, halothane, neuroleptics, liquid paraffin, etc.), physical factors (cold, shivering), surgical manipulation, or electrocautery.
- Avoid K+ containing fluids
- Regional or local anesthesia acceptable, but will not block myotonic response
- Regional +/- TIVA may be preferable when suitable

Extubation
- Beware of airway obstruction because of jaw muscle weakness.
- Delayed recovery from anesthetic common

Adjuvants
- Increased sensitivity to ventilatory depressant effects of all premedicants, sedatives, opioids
- Reversal agents can theoretically precipitate skeletal muscle contraction by facilitating depolarization of NMJ, but adverse responses do not predictably occur.

Postoperative Period
- Increased sensitivity to resp depressant effects of opioids or sedatives, incl epidural opioids; explore analgesic methods (e.g., regional or NSAIDs)
- Pulm complications due to poor cough possible
- Cardiac and resp monitoring and early chest physiotherapy

Anticipated Problems/Concerns
- If myotonia develops intraop, neither general or regional anesthesia, nor NMBs will attenuate it. Local infiltration of involved muscles may help. Even asymptomatic pts may have some degree of cardiomyopathy. Beware of premature extubation, consider postop ventilation.
- 57% of these of pts have conduction defects with one third have 1 block unresponsive to atropine, it is advisable to have antiarrhythmics and transthoracic pacing readily available as many anesthetic agents can increase vagal tone.
- For numerous reasons, it is advisable to avoid general anesthetics (myocardial depressants, conduction effects, link to malignant hyperthermia).
Risk

- Although primary cardiac tumors are rare (<0.01%) this is the most common (50%)
- 75% develop in LA
- Rarely develop in ventricle
- More common in females (70%)

Perioperative Risks

- May be friable and embolize
- Risk of LV or RV inflow obstruction
- May simulate pulm Htn and/or constrictive pericarditis

Worry About

- Hypotension due to obstruction of ventricular inflow and/or incompetence of tricuspid (right) or mitral (left) valve

ASSESSMENT POINTS

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<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Mitral stenosis or insufficiency syndrome</td>
<td>Edema, CHF</td>
<td>Left atrial enlargement</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm emboli (right)</td>
<td>DOE, cough</td>
<td>Systolic murmur (MI)</td>
<td>ECG</td>
</tr>
<tr>
<td>GI</td>
<td>Emboli (left)</td>
<td>CHF</td>
<td>Diastolic murmur (mitral stenosis)</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Stroke (left)</td>
<td>CNS dysfunction</td>
<td>Hepatic enlargement</td>
<td>Urinalysis</td>
</tr>
<tr>
<td>CNS</td>
<td>Constitutional symptoms</td>
<td>CNS dysfunction</td>
<td>Hepatic enzymes (if Sx of CHF)</td>
<td>ECHO</td>
</tr>
<tr>
<td>GENERAL</td>
<td></td>
<td>Fever, malaise</td>
<td>Weight loss</td>
<td>Cr clearance</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation

- Differential Dx incl mitral stenosis/insufficiency (left), tricuspid stenosis/insufficiency (right), constrictive pericarditis, pulm Htn, subacute bacterial endocarditis.
- Mitral stenosis: Hemodynamic aim is to keep in normal sinus rhythm with adequate preload and high normal afterload (see Mitral Stenosis in Diseases section).
- Mitral insufficiency (regurgitation): Hemodynamic aim is to keep HR normal or fast and vasodilate.
- Hemodynamics can mimic any or all of above depending on load-dependent variables prevailing in the cardiac cycle at the time (e.g., preload, afterload, HR).

Monitoring

- Routine monitors otherwise needed for cardiopulmonary bypass (e.g., temp, ECG, coagulation, Foley)
- Intra-arterial catheters

- Tumor flips on stalk across valves, causing stenotic or incompetent symptoms
- RV hypertrophy due to longstanding left inflow obstruction
- Rare pulm or systemic embolization

Overview

- Is a true neoplasm and distinct from a thrombus
- Usually polyoid with 1–2 cm stalk projecting into cavity; round with smooth margins
- Typically grows very slowly before symptomatic (10–20 y)

ICD-9-CM Code: D21.3 (Thorax myxoma)

Etiology

- Polyhedral cells with small nuclei are separated by an a fibrillar, eosinophilic myxomatous stroma that is predominantly a mucopolysaccharide
- Rarely extends deeper than endocardium
- Although benign, this tumor can undergo malignant degeneration

Usual Treatment

- Surgical
- Cardiopulmonary bypass required
- Atriotomy with transseptal approach through fossa ovalis

Anticipated Problems/Concerns

- Hypotension with inadequate preload when lesion obstructs inflow dynamics
Narcolepsy

Risk
- Prevalence: 1/2000, more common in males
- Race with highest prevalence: None

Perioperative Risks
- Risks related to treatment medications
- Possible increased sensitivity to anesthetic agents
- Possible increased incidence of delayed emergence and postop hypersomnia after GA

Worry About
- Tricyclic drugs increase incidence of periop hypotension
- Tricyclic drugs blunt pressor response to indirect-acting sympathomimetic agents (e.g., ephedrine) and exaggerate pressor response to direct-acting sympathomimetic agents (e.g., phenylephrine)
- Possible increased incidence of postop apneic episodes
- Possible increased sensitivity to anesthetic agents

Overview
- Lifelong disease. Peak age of onset 15–30 yr

Overview
- Initial symptom is excessive daytime sleepiness with irresistible sleep attacks
- 2° symptoms of cataplexy, hypnagogic hallucinations, disrupted nocturnal sleep, and automatic behavior have variable incidence and can occur later in the disease.
- Sleep attacks appear as clinically normal sleep lasting from seconds to minutes. Can be easily awakened by auditory or tactile stimulation.
- 80% incidence of cataplexy (sudden brief loss of voluntary muscle control), Usually precipitated by strong emotional response (e.g., laughter, anger, surprise). Pt remains conscious. Majority of pts develop a flat affect to suppress the emotional trigger.
- Diagnostic work-up incl nocturnal polysomnogram (documents adequacy of sleep and rules out obstructive sleep apnea) followed by a Multiple Sleep Latency Test (MSLT) to document hypersomnolence and REM onset sleep. Pts with narcolepsy fall asleep quickly (usually <5 min) and have early onset of sleep.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Conduction abn due to tricyclics</td>
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<td>ECG</td>
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<tr>
<td>CNS</td>
<td>Flat affect</td>
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<tr>
<td></td>
<td>Fatigue</td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Daytime sleep attacks</td>
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</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Avoid sedative premedication
- Continue all medical therapy related to narcolepsy on day of surgery.
- If antisialagogue needed use non-central acting agent

Monitoring
- May have conduction abn on ECG
- Pts with a Hx of chronic amphetamine therapy may benefit from direct arterial pressure monitoring.

Induction
- May have exaggerated hypotension if taking tricyclics
- Hydrate prior to induction

Maintenance
- Increased sensitivity to anesthetic agents. Consider use of propofol, desflurane, sevoflurane, and N₂O instead of longer acting intravenous/inhalation agents.
- Exaggerated pressor response to direct-acting sympathomimetics (e.g., phenylephrine) if taking tricyclics. Use small doses if clinically indicated.
- Blunted and/or unpredictable pressor response to indirect-acting sympathomimetics if taking tricyclics. Probably best to avoid.

Adjuvants
- Muscle relaxants: Life-threatening arrhythmias have been reported with the use of pancuronium in pts on tricyclics.
- Anesthetic agents: Life-threatening arrhythmias have been reported with the use of halothane in pts on tricyclics.

Postoperative Period
- May be prone to postop apneic episodes esp. with use of IV and/or neuraxial narcotics for postop analgesia.

Anticipated Problems/Concerns
- Pts often on tricyclic therapy and CNS stimulant therapy. Will often be sensitive to anesthetic agents. If on tricyclics need to be concerned about exaggerated pressor responses with direct-acting sympathomimetics. Indirect-acting sympathomimetics have a blunted pressor response and probably should be avoided. Postop apnea is of theoretical concern. Postop obstruction symptoms are extremely rare. Small retrospective study showed no increase in postop complications in pts receiving GA.
Necrotizing Enterocolitis

Overview
- Presents commonly with generalized signs of sepsis, incl glucose instability, hypothermia, apnea, feeding intolerance, and metabolic acidosis
- Terminal ileum most commonly involved, followed by the distal small bowel and ascending colon. Bowel ischemia may lead to gangrene of bowel with perforation as well as peritonitis, CV and resp collapse, shock, and death.
- Multisystem failure is commonly assoc involving the resp, CV, renal, and hepatic systems. Abn elevated inflammatory mediators, such as TNF, IL-6, and PAF, are assoc.

Risk
- Most common life-threatening intestinal surgical emergency in the newborn
- Occurs predominantly in premature infants, with 75% in infants <1500 g
- Increasing incidence in term and near-term neonates as well

Perioperative Risks
- CV instability, acidosis, shock, bowel ischemia, bacteremia, patent ductus arteriosus, polycythemia

Worry About
- Persistent metabolic acidosis and intestinal perforation are ominous signs

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Shock</td>
<td>Pulm edema, Hx</td>
<td>Murmur BP/HR</td>
<td>ABGs, BP UO</td>
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<tr>
<td>RESP</td>
<td>RDS</td>
<td>Apnea or tachynea</td>
<td></td>
<td>ABGs CXR</td>
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<tr>
<td>ID</td>
<td>Sepsis</td>
<td>Bacteremia</td>
<td>Peritonitis</td>
<td>Blood and peritoneal fluid cultures</td>
</tr>
<tr>
<td>GI</td>
<td>Peritonitis, bloody stools, malabsorption</td>
<td>Large feeding residuals, bilious emesis</td>
<td>Residuals, guaiac stools</td>
<td>Lytes, bowel sounds, KUB Temp instability</td>
</tr>
<tr>
<td>RENAL</td>
<td>Prerenal failure</td>
<td></td>
<td>UO, BP</td>
<td>BUN, Cr</td>
</tr>
<tr>
<td>HEME</td>
<td>DIC</td>
<td>Bleeding</td>
<td></td>
<td>Hct, plt count, fibrinogen PT/PTT</td>
</tr>
</tbody>
</table>


Preoperative Implications

**Perioperative Preparation**
- Most neonates may be treated medically with fluid resuscitation, antibiotics, ventilatory support, and hyperalimentation.
- Surgery indicated for pneumoperitoneum from intestinal wall perforation, intestinal gangrene (detected by abd paracentesis), and presence of portal vein gas. Other indications incl clinical deterioration, abd wall erythema, and an unresolved ileus.
- D/C enteral feeds and insert NG tube connected to suction for intestinal decompression.
- Therapeutic goals incl normalization of vital signs, ensuring adequate oxygenation and ventilation (e.g., trachal intubation, mechanical ventilation, adequate perfu-}

**Monitoring**
- Routine plus glucose and electrolytes

**Induction/Maintenance**
- Potent anesthetic agents are poorly tolerated.
- Carefully titrated narcotic and muscle relaxant technique is satisfactory.

Postoperative Period
- Closely monitor in NICU for ongoing fluid requirements as third space loss continues.
- Prolonged TPN is often required.
- Stricture formation leading to partial or total bowel obstruction is a common complication in both medically and surgically treated neonates.
- Short-bowel syndrome can occur, leading to long-term complications.

Anticipated Problems/Concerns

- Hypovolemia and bowel ischemia
- Acidosis, shock, and death

- Vigorous fluid resuscitation to keep up with third space losses from peritonitis and sepsis
- Correct metabolic acidosis—achieved through fluid resuscitation
- Inotropic agents such as dopamine and dobutamine may be required to optimize cardiac output
- Correct coagulopathy with FFP, pltls, and packed RBCs
- Administer broad-spectrum antibiotics, with anaerobic coverage highly considered as well

- In severe cases, abd wall may be erythematous, signifying intestinal perforation and peritonitis
- Pneumatosis intestinalis is evident as a linear collection of air and hydrogen gas in the wall of dilated loop of bowel; may extend into portal venous circulation

ICD-9-CM Code: 777.5

Etiology
- Assoc with bowel ischemia, enteral feeds, infection, and prematurity. Clearest link is with prematurity, leading to the theory that an underlying developmental immaturity of bowel is potentially the initiating problem leading to this life-threatening condition.
Necrotizing Fasciitis

Risk
- Incidence in USA: Approx 9 to 11.5 cases of invasive streptococcal infections/y, from which 1–1.8 die each year.
- Streptococcal toxic shock syndrome (STSS) and necrotizing fasciitis (NF) each comprise an average of 6–7% of these invasive cases, with an assoc mortality of 35–50% for the former and 29% for the latter.
- Predisposing risk factors: Diabetes, peripheral vascular disease, alcoholism, IV drug abuse, immunosuppression, obesity, or malnourishment

Perioperative Risks
- Shock, hypovolemic, and organ dysfunction
- Multiple organ dysfunction syndrome (MODS) and death

Worry About
- Making an early Dx and beginning treatment accordingly. This is the single most important factor to decrease morbidity and mortality and always includes surgery.
- STSS and septic shock.

Overview
- Necrotizing fasciitis constitutes one of the two severe manifestations of Group A streptococci, along with STSS, and often is asso with it during its initial presentation.
- NF is a common cause of CV collapse, shock, and hypoperfusion which could be aggravated by the anesthetics. High suspicion is important to ensure early detection and treatment of hypovolemia and hypoperfusion. A suitable anesthetic procedure should be planned. Aggressive and continuous assessment of the CV status is required to have a stable hemodynamic condition during the septic process.
- Despite the low incidence of the disease, prompt recognition is important given its devastating consequences, not only as a major cause of mortality but also morbidity, incl:
  - Organ failure with long-term requirement of support therapy (i.e., dialysis, home O.).

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Vasodilation, hypovolemia early after local symptoms (i.e., 24–48 hr)</td>
<td>Dizziness, alteration in mental status</td>
<td>Signs of dehydration, orthostatism, tachycardia, hypotension</td>
<td>Hemodynamic monitoring</td>
</tr>
<tr>
<td>HEME</td>
<td>DIC, hemorrage, leukocytosis</td>
<td>Petechiae, skin discoloration</td>
<td>Bleeding, poor coagulation, fever, chills, myalgias</td>
<td>Hemoglobin and Hct, clotting evaluation with platelets, PT/PTT, fibrinogen, fibrin split products, CBC</td>
</tr>
<tr>
<td>RENAL</td>
<td>Prerenal and acute renal failure</td>
<td>UO</td>
<td>Signs of hypovolemia</td>
<td>Urinalysis with specific gravity, Na excretion, serum creatinine and BUN</td>
</tr>
<tr>
<td>PULM</td>
<td>ALI/ARDS</td>
<td>None</td>
<td>Hypoxia, increased work of breathing</td>
<td>Arterial blood gas with low P/F ratios &lt;300</td>
</tr>
<tr>
<td>SKIN AND SOFT TISSUES</td>
<td>Inflammation, necrosis, blistering</td>
<td>Hx of skin/soft tissue injury (i.e., insect bite, comisation, ingrown nail)</td>
<td>Pain, erythema, edema, cellulitis with rapid progression to bluish discoloration, blisters, subcutaneous crepitus</td>
<td>Congelation biopsy, with fascia involvement</td>
</tr>
</tbody>
</table>


Periinduction/Induction/Maintenance
- Establish large bore venous access promptly and optimize CV status and perfusion. Anticipate additional fluid loss from exposed debrided areas and large fluid shifts.
- Type and cross.
- Consider ketamine 1–2 mg/kg vs. etomidate 0.2 mg/kg as induction agent. Watch for CV depression during induction. Anticipate the potential for adrenal insufficiency with etomidate.
- Provide adequate pain control.
- Consider procedure contaminated and use all recommended infection control guidelines.

Monitoring
- Establish invasive CV monitoring (CVC/PAC, A-line and UO), pulse-pressure wave form analysis (PPWF), ECHO, and temp monitor.
- Hx & H, clotting studies and CBC as above.
- Pulm pressures, tidal volumes, ABGs.
- Perfusion to end organs: ScVO2/ScVO2, lactate, base excess.
- General anesthesia

Preinduction/Induction:
- Hydrate aggressively with crystalloids/colloids.
- Aspiration prophylaxis
- Do not delay antibiotics if required at the time of surgery.

Maintenance
- Monitor volume status (CVP/SVV/PPWF/UO) and perfusion adequacy as above to optimize accordingly, incl volume responsiveness and consider requirement of vasopressors (such as norepinephrine).
- Monitor blood loss, coagulopathy, and electrolyte imbalance and replace accordingly.
- Avoid hypothermia, monitor for fever.
- Watch for the presentation of bacteremia during/after debridement (hypotension, tachycardia, fever).
- Extubation: Consider delaying extubation according to CV/pulm status.
- Regional anesthesia
- Do not use in the acute setting. Only use in the absence of shock, occult shock, hemorrhage, and significant coagulopathy. Usually adequate in later stages of the disease, while still requiring surgical management.
- Spinal versus epidural. Use dependent on affected region and length of the procedure.
- Anticipate important loss of sympathetic tone in the setting of potential hypovolemia.
- Do not use in anesthetic application implies puncture through potentially contaminated site.

Postoperative Period
- Potential requirement for continued intubation to maintain adequate oxygenation and to be admitted to ICU.
- Must be directed at obtaining adequate end-organ perfusion. Same strategies of monitoring.
- Optimize support according to requirement (i.e., CVVH, HD, mechanical ventilation, etc.).

Anticipated Problems/Concerns
- Pts often in septic shock, hypovolemic and hypoperfused to begin with. Optimize CV and perfusion status in pre-induction and be cautious during induction. Always consider associ co-morbidities.
• Surgical debridement and combined with antibiotic therapy is the only strategy to decrease poor outcome. Do not delay surgical intervention. Surgical procedures may incl amputation of limbs, which if delayed may cause uncontrolled systemic involvement and response.

• Complications such as organ failure (renal failure 80%, ARDS 50%), bacteremia (60%) are the rule, not the exception. Be prepared to support failing organs and troubleshoot acute destabilizations.

• Pts may require additional surgical interventions such as diverting colostomies or urinary diversions to avoid further contamination.

• Specific complications may arise depending on the location of NF.
Neurofibromatosis (NF)

Jane C. Ahn
Zeev N. Kain

Risk
• NF-1 birth incidence: 1/3000
• NF-2 birth incidence: 1/33,000–1/40,000

Perioperative Risks
• Risk depends on tumor and location

Worry About
• Difficult intubation
• Intraop Htn and tachycardia

Overview
• NF-1, also known as von Recklinghausen disease, is a genetic disorder with variable clinical presentation in which multiple organs, such as skin and peripheral nervous system, are site of tumors and hamartomas.

• Hallmarks incl café-au-lait spots (more than 6 that are >1.5 cm in diameter), Lisch nodules (benign iris hamartomas), axillary and groin freckling, and multiple neurofibromas
• Laryngeal and tracheal compression may occur 2° to assoc tumors
• Surgery may be indicated for pts with NF-1, esp. for removal of tumors (e.g., neurofibromas, pheochromocytoma), skeletal dysplasia (e.g., tibial pseudoarthrosis), scoliosis, and renovascular Htn
• NF-2, also known as central neurofibromatosis, is a genetic disorder characterized by bilateral vestibular schwannomas, hearing loss, and CNS tumors.

ICD-9-CM Codes: 237.71 (NF-1); 237.72 (NF-2)

Etiology
• NF-1 and NF-2 are both autosomal dominant, although about 50% of cases represent new mutations.
• The gene for NF-1 resides on the long arm of chromosome 17.
• The gene for NF-2 resides on chromosome 22.

Usual Treatment
• Radiation and surgical treatment for various tumors involved

ASSESSMENT POINTS

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<th>Assessment by Hx</th>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT*</td>
<td>Pharyngeal compression Laryngeal compression Vocal cord and arytenoid involvement Airway obstruction</td>
<td>Dyspnea, dysphonia, stridor, and voice changes Evaluation of airway</td>
<td>X-ray CT of neck</td>
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</tr>
<tr>
<td>CARDIO</td>
<td>Renovascular Htn Pheochromocytoma Autonomic dysfunction</td>
<td>Headache, perspiration</td>
<td>BP/HR Urinary catecholamines</td>
<td></td>
</tr>
<tr>
<td>RESP*</td>
<td>Restrictive lung disease Cor pulmonale Interstitial lung disease Hypoxemia</td>
<td>Exercise tolerance Cyanosis Clubbing</td>
<td>CXR ECG ABGs PFTs (rare)</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Carcinoid tumor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU*</td>
<td>Obstruction and uremia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Mental retardation Seizures Intracranial tumors and increased ICP Paraspinal tumors</td>
<td>MRI of brain and spine for neuraxial technique</td>
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<tr>
<td>MS*</td>
<td>Kyphoscoliosis Macrocephaly Craniofacial dysplasia Cervical dislocation Pectus excavatum</td>
<td>X-ray of neck</td>
<td></td>
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</tr>
</tbody>
</table>

* In severe cases


Perioperative Implications

Preoperative Preparation
• Evaluation of airway for possible laryngeal and pharyngeal tumors

Monitoring
• Routine
• Consider arterial line depending on resp status and presence of pheochromocytoma.

Airway
• Consider awake fiberoptic intubation or awake tracheotomy if laryngeal and pharyngeal involvement.

Preinduction/Induction
• No particular anesthetic drug or technique recommended
• Consider potential for increased ICP.
• Consider potential of pheochromocytoma (see Pheochromocytoma in Diseases section)
• Although there are case reports of abn response to NMBs, recent evidence suggests no or minimal effect.

Maintenance
• CV instability if pheochromocytoma present

Extubation
• Routine considerations

Postoperative Period
• Pain management may be critical

Regional Anesthesia
• Asymptomatic paraspinal neurofibromas can make identification and entry into epidural and subarachnoid spaces very difficult. A careful exam of the back is indicated before any regional technique is considered.
• Paraspinal and intracranial tumors are exacerbated during pregnancy.
• Consider potential for epidural hematoma, tumor trauma, brainstem herniation.
• Recommend MRI of brain and spine for tumor assessment prior to neuraxial technique.

Anticipated Problems/Concerns
• Difficult airway
• Presence of pheochromocytoma
• Potential for increased ICP with expanding intracranial tumor
• Difficult epidural or spinal placement. Potential for complications due to tumor involvement.
## Occlusive Cerebrovascular Disease

### Risk
- Prevalence: Approx 1 in 59, or 1.69%
- Annual incidence of stroke: 600,000 annually incl 500,000 new cases and 100,000 recurrences
- Incidence in the USA: Approx 1 in 453, or 0.22%
- Races with highest prevalence: Japanese and Eastern European (incidence 0.3%/y)

### Perioperative Risks
- Stroke
  - Major general surgery at age >50 y = 0.4%; at age >80 y = 2.5%
  - Major peripheral vascular reconstruction = 1%
  - CABG = 1–5%, carotid endarterectomy (CEA) = 3% or less

### Worry About
- Cerebral ischemia
- Myocardial ischemia (CAD, leading cause of morbidity following CEA)
- Control of co-existing CAD, DM, Htn

### Overview
- Two main clinical presentations
- Pts with known occlusive CVD undergoing CEA or carotid angioplasty. Risk factors incl CAD/CHF; stroke in evolution, frequent TIA; severe Htn; carotid siphon stenosis; COPD; poor cerebral collateral flow; age >70 y; intraluminal thrombus. Criteria for pt selection and acceptable periop morbidity and mortality rates are now well established for CEA.
- Pts with known or possible CVD defined poorly. For CEA.

### Perioperative Implications

#### Preoperative Preparation
- Neurologic assessment
- Optimize control of co-existing Htn, CAD, diabetes, COPD
- Evaluate normal BP range

#### Monitoring
- Arterial catheter and ST-segment monitoring
- For CEA, consider neurologic monitor: EEG or regional anesthetic with awake pt (if practical)
- Carotid angioplasty usually performed with the pt awake

#### Induction/Maintenance
- Have surgeon block carotid sinus nerve if bradycardic
- Maintain hemodynamic stability based on preop BP range
- Maintain normocapnia based on preop pH and Paco,
- Light IV sedation often administered during angioplasty
- Embolic stroke or severe, vagal-mediated, bradycardia can accompany carotid dilation during angioplasty

#### Extubation
- Be prepared to manage hemodynamic instability following CEA
- Avoid straining on ETT with fresh arteriotomy following CEA

#### Postoperative Period
- Hemodynamic instability due to baroreceptor dysfunction following CEA
- Adequate analgesia, supplemental O₂
- Awake pt allows early and frequent neurologic evaluation

### Anticipated Problems/Concerns
- Most pts with CVD also at high risk for CAD. Consistent approach to management of both problems incl hemodynamic stability, adequate oxygenation, normocapnia, adequate analgesia, normoglycemia.
- Caution regarding use of succinylcholine in pts with previous parietic CVA
- Angioplasty is not assoc with substantial discomfort. Only minimal sedation is typically needed for comfort.

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Possible positional cerebral ischemia</td>
<td>Sx of cerebral ischemia with head movements</td>
<td>Neck ROM</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Htn</td>
<td>Exercise tolerance</td>
<td>Arterial BP</td>
<td>ECG, CXR</td>
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<td>Vasculopathy</td>
<td>Angina, MI, CHF</td>
<td>S</td>
<td>ECHO</td>
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<td>LV dysfunction, CHF</td>
<td>Claudication</td>
<td>Peripheral pulses</td>
<td>Stress test</td>
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<td>RESP</td>
<td>COPD due to smoking</td>
<td>Dyspnea</td>
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<td>CXR</td>
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<td>Irritable airway</td>
<td>Chronic cough</td>
<td>Accessory muscles</td>
<td>?ABGs</td>
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<td>?PFTs</td>
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<td>Diabetes, Htn</td>
<td>Cr, urea</td>
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<td>RENAL</td>
<td>Possible nephropathy</td>
<td>Diabetes</td>
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<tr>
<td>CNS</td>
<td>Cerebral ischemia</td>
<td>TIA, stroke</td>
<td>Neurologic deficits</td>
<td>Cerebral angio prior to CEA</td>
</tr>
</tbody>
</table>

### Opitz-Frias Syndrome
(The G Syndrome)

#### Risk
- Overall incidence not reported
- Very rare congenital disorder

#### Preoperative Risks
- Very high risk of recurrent pulm aspiration; hypoplasia of both pulm and vascular components of one lung (pulm hypoplasia)
- High mortality rate in infancy

#### Worry About
- NM dysfunction of laryngoesophageal apparatus
- Laryngotracheoesophageal cleft or fistula
- Difficult tracheal intubation due to assoc craniofacial deformity(s)
- Assoc congenital anomalies (HtN, hypospadius, wide eyes, cleft lip, cleft palate, cryptorchidism, imperforate anus, cardiac deficits)
- Presence of one hypoplastic lung
- Laryngeal hypoplasia
- Laryngotracheoesophageal cleft or fistula
- Anticipate very difficult tracheal intubation
- Thorough preop cardiac evaluation need to access for cardiac abn (? ECHO)
- Any male infant presenting for TEF with genital defect should be suspected.
  - Classically—weak, hoarse cry

**ICD-9-CM Code: 759.9 (Congenital Anomaly, unspecified)**

#### ASSESSMENT POINTS

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<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Cleft lip–palate (35%)</td>
<td>Feeding difficulties, speech</td>
<td>Short lingual frenulum</td>
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</tr>
<tr>
<td></td>
<td>Ankyloglossia</td>
<td>anomalies</td>
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<tr>
<td></td>
<td>Micrognathia</td>
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<td></td>
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</tr>
<tr>
<td>CNS</td>
<td>Dolichocephaly (20%)</td>
<td>Mental dysfunction, prominent</td>
<td>“Cone-head” Palpation</td>
<td>CT (if indicated)</td>
</tr>
<tr>
<td></td>
<td>Large metopic sagittal suture and anterior</td>
<td>forehead</td>
<td></td>
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<td></td>
<td>fontanel</td>
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<tr>
<td>FACES</td>
<td>Hypertelorism/telecanthus (90%)</td>
<td>Mother-related disease</td>
<td>Large nasal bridge</td>
<td>Face X-ray</td>
</tr>
<tr>
<td></td>
<td>Mongoloid palpebral fissures</td>
<td></td>
<td>downsplaying</td>
<td></td>
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<tr>
<td></td>
<td>Strabismus</td>
<td></td>
<td></td>
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<tr>
<td>CARDIO</td>
<td>Congenital heart defects (40%)</td>
<td>Failure to thrive</td>
<td>Auscultation</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>(ASD, VSD, PDA, coarctation of aorta)</td>
<td></td>
<td></td>
<td>TEE, ABGs</td>
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<tr>
<td>RESP</td>
<td>Agenesis, hypoplasia of one lung</td>
<td>Polyhydramnios on delivery</td>
<td>Auscultation</td>
<td>CXR</td>
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<tr>
<td></td>
<td>Tracheoesophageal cleft, fissure</td>
<td>Coughing, choking, cyanosis</td>
<td></td>
<td>Bronchogram</td>
</tr>
<tr>
<td></td>
<td>Hypoplasia of vocal cord</td>
<td>Hoarse, weak cry</td>
<td>Tracheal stenosis</td>
<td>Esophagogram</td>
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<tr>
<td></td>
<td>Tracheomalacia</td>
<td>Stridorous resp</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Short trachea, high carina</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>GI</td>
<td>Achalasia of the cardia (70%)</td>
<td>Dysphagia</td>
<td></td>
<td>Esophagogram (if indicated)</td>
</tr>
<tr>
<td></td>
<td>NM dysfunction of esophagus</td>
<td></td>
<td></td>
<td>Cinеfluoroscopy of</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>swallowing (if indicated)</td>
</tr>
<tr>
<td>GU</td>
<td>Hypospadias with descended testis</td>
<td>Perineal or penoscrotal</td>
<td></td>
<td>Nephrogram</td>
</tr>
<tr>
<td></td>
<td>Ureteral stenosis or duplication</td>
<td></td>
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</tr>
</tbody>
</table>

#### Key Reference:

#### Perioperative Implications

**Preoperative Preparation**
- Evacuation of the stomach with NG tube (if pt has gastrectomy open to air)
- Feeding: Clear water or apple juice (standard NPO guidelines)
- Consider H₂ blocker
- No atropine IM or metoclopramide preop
- Give sodium citrate through NG tube
- Complete cardiac evaluation
- Assessment of renal function
- Not appropriate for outpatient or same day process
- IV access 24 hr before surgery to reduce stomach content

**Monitoring**
- All standard monitors
- Invasive arterial pressure if indicated due to procedure or unstable hemodynamics

**Airway**
- Tubes smaller than normal 2° (as assessed by age) to laryngeal hypoplasia

**Preinduction**
- Warm OR
- Decompress stomach with suction
- Atropine and suxamethonium backup

**Induction**
- Maintain spontaneous respiration
- Danger of regurgitation and aspiration requires careful inhalation induction
- Cricoid pressure should be applied
- Atropine 20 μg/kg of induction to prevent bradycardia during intubation

**Maintenance**
- Hand ventilation (low PPV)
- Avoid hypothermia

**Exubation**
- Based on pt’s lung condition and preop assessment and/or lung function

**Adjuvants**
- All medications can be used (no contraindication to IV or inhaled anesthesia)

**Anticipated Problems**
- Unanticipated cardiac issues
- Difficult to access recovery from anesthesia due to assoc mental conditions

### Etiology
- X-linked recessive inheritance
- Autosomal dominant inheritance or new mutation
- Partial male sex limitation
- Autosomal recessive inheritance, high parenteral consanguinity
- Females can be equally or nearly as severely affected as males

**Usual Treatment**
- Prophylactic gastrostomy
- Feeding jejunostomy
- Cervical esophagostomy if unable to swallow
- Prophylactic antibodies (pulm infection)
Osteoarthritis

Risk
- Osteoarthritis (OA) most common cause of impairment in the elderly
  - Incidence in USA: 63–85% >65 y have radiographic signs
  - 35–50% have pain, stiffness, or limitation of movement
    - 9–12% are significantly disabled
    - 46 million doctor visits and 68 million work lost per year
- Risk factors differ across joints: Knee, obesity, injury; hand, repetitive use; hip, congenital or developmental abn, male preponderance

Perioperative Risks
- Often assoc with obesity
- Common analgesics incl NSAIDs and intra-articular steroid injections
- Rarely affects neck or jaw
- Reported assoc with diabetes, hypothyroidism, hyperparathyroidism, gout

Worry About
- Anesthetic problems with assoc obesity
- Positioning may be difficult owing to joint pain and stiffness
- Possible assoc metabolic conditions
- Effect of medications on plt function and frequent steroid injections

Overview
- OA is age-related but not caused by aging
- Early radiographic findings incl joint space narrowing, osteophytes, subchondral stenosis
- With progression, osteophytes, subchondral cysts, intra-articular osseous bodies seen
- Subchondral bone collapse is a late finding
- Knees most common joint affected (41%), followed by hands (30%) and hips (19%)
- Risk factors for symptoms are obesity (knees) and severe radiographic findings

ICD-9-CM Code: 715.0 (Generalized)

Perioperative Risks
- Often assoc with obesity
- Common analgesics incl NSAIDs and intra-articular steroid injections
- Rarely affects neck or jaw
- Reported assoc with diabetes, hypothyroidism, hyperparathyroidism, gout

ASSESSMENT POINTS

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<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Rare C-spine involvement</td>
<td>Pain</td>
<td>Neck ROM</td>
<td>Usually not needed C-spine x-rays</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Age-related changes</td>
<td>Exercise tolerance may be limited by joint changes</td>
<td>HR and tolerance to 2-flight stair climb</td>
<td>ECG CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Nonspecific</td>
<td>Exercise tolerance</td>
<td>Gastric upset</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Sensitivity to NSAIDs</td>
<td>Gastric upset</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Associated diabetes</td>
<td>Fasting blood sugar</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Age-related changes</td>
<td>TIA's or stroke</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Multiple joint involvement</td>
<td>Joint pain</td>
<td>Joint ROM</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Age-related changes</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Assess joint involvement and ROM
- Question pt regarding nonprescription analgesics
- Consider regional anesthetic techniques
- Evaluate for steroid need

Monitoring
- Routine

Airway
- Assess neck ROM

Induction
- Age-related considerations: Elderly pts may have slow circulation times, CV disease, fluctuations in BP

Maintenance
- Position with consideration of other joint involvement

Exubation
- No special considerations

Adjuncts
- Elderly pts may be more sensitive to narcotics.

Postoperative Period
- Consider continuous regional technique with local anesthetic and/or narcotic for pain management

Anticipated Problems/Concerns
- Usually neck and airway normal
- Concomitant risk factors, esp. obesity
- Often several joints involved with pain and decreased ROM
- Regional anesthesia well suited
Osteoporosis

Risk

• Most common metabolic bone disease in USA
• All elderly pts of European descent considered at risk
• Non-Hispanic white women and Asian women at highest risk
• 10 million Americans have osteoporosis, 34 million have low bone mass and therefore at risk for osteoporosis
• Female > male: 3:1
• Postmenopausal female, small frame, low wt
• Risk factors for osteoporosis, such as advanced age and reduced bone density, have been established by virtue of their direct and strong relationship to the incidence of fractures; however, many other factors have been considered risk factors based on their relationship to bone density value as a surrogate indicator of osteoporosis. Risk factors include the following:
  • Advanced age, female sex, white or Asian ethnicity, family Hx of osteoporosis, body wt less than 127 pounds, amenorrhea, late menarche, early menopause, nulliparity, physical inactivity, alcohol and tobacco use, androgen or estrogen deficiency, calcium deficiency
  • 2\textsuperscript{nd} osteoporosis attributable to diseases (hyperparathyroidism, rheumatoid arthritis, sarcoidosis, thalassemia, idiopathic scoliosis, multiple myeloma, and thyrotoxicosis) and drugs (lithium, anticonvulsants, excessive alcohol use, excessive thyrine, prolonged unfractured heparin use (> 6 mo of >15,000 IU/day), glucocorticoids, and cytotoxic drugs).

Perioperative Risks

• Concomitant medical conditions in elderly
• Pneumonia
• Co-existing metabolic or endocrine disorders
• Fractures

Worry About

• Positioning because of increased risk of bone fractures
• Vertebral fractures: Vertebral compression fractures assoc with increased morbidity/mortality.
• Hip fractures: Significantly increased risk of morbidity/mortality in first year after fracture; men > female
• Pulm function/restrictive disease, esp if kyphosis present

Overview

• Osteoporosis is a systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue, with a consequent increase in bone fragility.
• Imbalance between bone resorption and formation causes loss of bone substance, resulting in bone fractures.
• Most common fracture sites: Vertebral body, neck of femur, distal radius, proximal humerus, pelvis
• 1.5 million fractures due to osteoporosis occur each year: Spine (700,000), hip (300,000), wrist (200,000)
• Severe kyphosis common
• Type I (postmenopausal) osteoporosis: Women 15–20 y after menopause; vertebral and Colles’ fractures most common

Etiology

• Insufficient accumulation of bone mass during skeletal growth
• Age-related factors: Decreased bone formation at cellular level begins in 4th decade and becomes more severe with age. Age-related ↑ in parathyroid function with age-related ↓ in calcium absorption.
• Menopause: Accelerated phase of bone loss is the result of estrogen deficiency
• Sporadic factors: Twofold increased risk with cigarettes and high alcohol consumption

Treatment

• Vitamin D and calcium
• Selective estrogen receptor modulators (SERMs):Raloxifene
• Bisphosphonates: Alendronate, risedronate
• Human recombinant PTH: Teriparatide
• Calcitonin
• D/C of glucocorticoid (if osteoporosis due to chronic use)
• Surgical stabilization of fractures: Kyphoplasty/vertebroplasty for spine fractures, ORIF (hip, wrist)

ASSESSMENT POINTS

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<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Osteoporosis of skull</td>
<td>Pain</td>
<td>Skull x-ray</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Kyphosis</td>
<td>Dyspnea</td>
<td>Neck x-ray</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Parathyroid function↓ in Type I ↑ in Type II Calcium absorption↓ Metabolic disorders of vitamin D</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Back pain</td>
<td>Acute back pain</td>
<td>Dowager’s hump</td>
<td>X-ray</td>
</tr>
<tr>
<td></td>
<td>Loss of ht</td>
<td>Remittance and recurrence until chronic</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Spinal deformity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fractures</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation

• Move and position carefully owing to risk of bone fractures
• Pulm function tests indicated in kyphoscoliosis present

• Detailed Hx to determine co-existing metabolic/endocrine disorders

Monitoring

• Routine
• Consider arterial line and frequent ABGs if pulm disease or pneumonia present

Airway

• Cervical fractures may require neck stabilization and fiberoptic intubation.
• Acromegaly may occur with osteoporosis.

Musculoskeletal

• Vertebral collapse may make spinal/epidural anesthesia more difficult

Anticipated Problems/Concerns

• Susceptible to fracture with routine positioning and moving
• Restrictive lung disease if scoliosis present may impair oxygenation

ICD-9-CM Code: 733.00

Overview

• Most common fracture sites: Vertebral body, neck of femur, distal radius, proximal humerus, pelvis
• 1.5 million fractures due to osteoporosis occur each year: Spine (700,000), hip (300,000), wrist (200,000)
• Severe kyphosis common
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• Calcitonin
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• Surgical stabilization of fractures: Kyphoplasty/vertebroplasty for spine fractures, ORIF (hip, wrist)
**Otitis Media**

### Risk

- **Age:** The highest incidence occurs between 6 and 24 mo of age. Incidence subsequently declines except for an increase at the time of school entry (between 5 and 6 yr of age)
  - Day care attendance
  - Tobacco smoke and air pollution
  - Other factors: Poor social and economic conditions, season (fall and winter), altered host defenses, and diseases with assoc craniofacial abn such as cleft palate and Down syndrome.

### Perioperative Risks

- Active or concurrent disease, such as upper and lower resp infections, may increase risk of airway reactivity, laryngospasm, bronchospasm, periop O₂ requirement, and postop mechanical ventilation.
- Inherent risks of assoc craniofacial abn may predispose to airway obstruction and/or difficult airway.
- N/V related to the infection, antibiotic therapy, and vestibular imbalance

### Worry About

- Chronic or recurrent otitis media can cause hearing loss (usually conductive) that may lead to problems in development of speech, language, and cognitive abilities in the child. This may impair communication in the periop period.
- Rare but important complications: Mastoiditis, petrositis, labyrinthitis, meningitis, epidual abscess, brain abscess, lateral sinus thrombosis, cavernous sinus thrombosis, subdural empyema, and carotid artery thrombosis
- Pts with fever ≥ 38 °C and/or concurrent disease, incl upper and lower resp infections with their potential risks
- Pts with assoc vestibular, balance, and motor dysfunctions

### Assessment Points

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENERAL</td>
<td>Patient age varies</td>
<td>Childhood vs. adult dz.</td>
<td>Find co-morbidities</td>
<td>As indicated</td>
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<tr>
<td>HEENT</td>
<td>Nasal secretions</td>
<td>Allergy vs. infection</td>
<td>Clear vs. green mucous</td>
<td>Eosinophil smear</td>
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<td></td>
<td>Middle ear fluid/drainage</td>
<td>Acute vs. chronic OM, ear pain, ear tugging</td>
<td>Fever vs. afebrile, inflamed tympanic membrane (red, opacified, bulging, immotile) vs. fluid level</td>
<td>Tympanogram</td>
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<tr>
<td></td>
<td>Hypertrophic T&amp;A</td>
<td>OSA, mouth breathing, snoring</td>
<td>Inspection</td>
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<tr>
<td>RESP</td>
<td>Cough</td>
<td>Dry vs. wet</td>
<td>Upper vs. lower tract Sx</td>
<td>Pulse ox</td>
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<td></td>
<td>Laryngo-tracheomalacia</td>
<td>OSA/feeding difficulty</td>
<td>Retractions, stridor</td>
<td>Bronchoscopy</td>
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<tr>
<td></td>
<td>Pneumonia</td>
<td>Fever, cough, dyspnea</td>
<td>Fever, tachypnea, crackles</td>
<td>Pulse ox, CXR, CBC</td>
</tr>
<tr>
<td>GI</td>
<td>NPO status, reflux Hx</td>
<td>Clear vs. fatty liquid</td>
<td>Non-fussy child</td>
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<tr>
<td>CNS</td>
<td>Developmental status</td>
<td>Developmental Hx</td>
<td>Congenital anomalies</td>
<td>Genetic testing Audiometry</td>
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<tr>
<td></td>
<td>Hearing (usually conductive loss)</td>
<td>Delayed speech and cognition</td>
<td>Fever, Brudzinski's signs and Kernig's signs, meningismus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Complications of untreated OM (such as meningitis)</td>
<td>Fever, headache, mental status changes, photophobia</td>
<td>MRI, lumbar puncture, cultures</td>
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</tr>
<tr>
<td>INTEG</td>
<td>Eczema</td>
<td>Allergy/steroid history</td>
<td>Allergic/non-allergic rash</td>
<td>Skin biopsy</td>
</tr>
</tbody>
</table>


### Preoperative Preparation

- Lower resp tract pathology or pneumonia may warrant further evaluation and case rescheduling; runny nose (rhinorrhea) is usually not an indication for case cancellation

### Usual Treatment

- Analgesics such as ibuprofen, acetaminophen, and auralgan (topical anesthetic) for symptomatic treatment of ear pain (otalgia)
- Antimicrobial therapy
  - Firstline therapy is amoxicillin (80 to 90 mg/kg orally per day divided into 2 doses). Other commonly used antibiotics incl cephalosporins (cefuroxime, ceftriaxone), macrolides (erythromycin, azithromycin), and trimethoprim sulfa sulfamethoxazole
  - Should be administered to any child younger than 6 mo
  - Should be administered to children 6 mo to 2 y in whom the Dx of acute otitis media is certain or if the Dx is uncertain but the illness is severe (moderate to severe otalgia or fever ≥39 °C). If the Dx is uncertain and illness is not severe, the child may be observed without treatment with antibiotics.
  - Should be administered to pts older than 2 y if the Dx is certain and illness is severe. When the Dx is certain but illness is not severe, observation alone is an option.
  - Acute otitis media usually resolves in 24–72 hr with appropriate antimicrobial therapy, however fluid may persist for weeks to months despite treatment. Placement of tympanostomy tubes is performed for pts with persistent middle ear effusion or severe and recurrent episodes of otitis media. Adenoidectomy may be indicated in selected pts.
  - Prevention is an important management strategy for otitis media: Minimize risk factors if possible (smaller day care groups and decrease smoke exposure), administer vaccines (influenza and pneumococcal), and encourage breastfeeding for at least 3 mo (diminishes colonization of the nasopharyx by bacterial pathogens and offers protective factors).

### Perioperative Implications

**Preoperative Preparation**

- Children: Avoid oral premed for myringotomy and pressure equalizing tubes (PETS) alone (short surgical time); consider parental presence for induction; allow comfort object in OR; develop medically appropriate review of procedures; consider preop oral acetaminophen to give analgesic regimen time to work
- Adult: IV midazolam or fentanyl before induction; topical local anesthetic drops in ear may be indicated

**Monitoring**

- Standard ASA monitors, skin temp probe
- Precordial stethoscope very helpful
**Airway**
- Children: Inhalation induction and mask airway maintenance for straightforward cases
- Adults: IV induction with mask airway or LMA maintenance
- Oral and/or nasal airways as indicated
- Be prepared to intubate if obstruction or as case direction changes
- Maintenance
- Volatile anesthetic in oxygen with NO usually sufficient
  - 70/30 \( \text{N}_2\text{O}/\text{O}_2 \) plus 8% Sevo for induction, followed by 50/50 \( \text{N}_2\text{O}/\text{O}_2 \) plus 4% Sevo for maintenance until first tube in place
- Turn off anesthetics at second myringotomy to avoid prolonged anesthesia for short operation.
- Consider IV propofol infusion to maintain spontaneous ventilation if laryngoscopy/bronchoscopy also planned
- Otherwise as required for additional operative procedures after PETs placed

**Extubation**
- Routine precautions and criteria

**Adjuvants**
- Determined by course and complexity of operation(s) to be performed
- PETs are frequently placed before other procedures (cleft lip/palate repair, auditory evoked potentials)

**Postoperative concerns**
- Postop analgesia: Multimodal approach
  - Children: Belly analgesia first (bottle, cup, soda); consider nasal fentanyl and/or oral acetaminophen if rectal not given intraop
  - Adults: IV / oral analgesics as needed; antiemetic may be needed more so than in children
- Emergence delirium: Nasal or IV clonidine or dexmedetomidine an option for children
- Slow introduction of PO fluids; limit volume if possible
- Plan to reunite child with parent and/or proxy after pt settled in PACU

**Anticipated Problem/Concerns**
- Separation of child and parent and/or proxy: have guardian present for induction
- Separation from child’s comfort object: Label with pt’s name
- Charting vital signs and maintaining anesthesia record in a short case with a lot to do: assistant or electronic medical record is helpful
- Difficulty maintaining mask airway: Use LMA, ET intubation
- Laryngospasm: Hold positive pressure, IM/IV succinylcholine and/or atropine, propofol if IV present, possible ET intubation
- Antibiotics: Start PIV if required
- Ear drops applied by surgeon: Can sting if pH is basic
- Unanticipated pathology: Cerumen impaction, cholesteatoma, other tumors, ossicular dislocation
- Excessive bleeding (ear canal trauma): Topical epinephrine application
- Small external ear canals: Change type of PE tube used
- Unable to place PE tube because of prior scarring: Abandon case
- PE tube falls into middle ear space: Surgical retrieval required
Pacemakers

Epidemiology

- Incidence in USA: Exceeds 3 million; 6 million worldwide have conventional pacing (but not high voltage) cardiac implantable electrical device (P-CIED)
- >600,000 pacemakers implanted per year in USA
- Some pts with cardiomyopathy have atrial, RV, and LV pacing
- Since all implanted cardioverter defibrillators (ICDs) provide pacing, this section also applies to these pts as well

Perioperative Risks

- No proven increase in risk due to CIED itself, although these pts might be at increased risk
- Risk related to associated medical problems
- Risk related to incorrect interpretation of events (pseudoaneurysm)

Worry About

- Intraop decrease in pacing rate due to inhibition of pacing output from electromagnetic interference (EMI) entering the P-CIED on the ventricular channel, esp. in the pacing dependent pt, which will result in asystole
- Intraop increase in ventricular pacing owing to EMI entering a dual chamber DDD P-CIED on the atrial channel with resultant tracking
- Intraop increases in pacing rates with misinterpretation as inadequate anesthesia owing to activation of the exercise sensor, whether due to direct mechanical stimulation (such as preparation of the chest), pressure on the device (peripheral leaching), or EMI interaction with a minute ventilation sensor.
- Failure to capture (i.e., pacing output without depolarization) due to inadequate pacing output (i.e., inadequate safety margin) or increase in pacing threshold, which can result from myocardial ischemia/infarction, drug administration, or electrolyte shifts. Note that any or all chambers can undergo failure to capture, with possible hemodynamic derangement but not apparent outright pacing failure.
- Hemodynamics can be degraded by magnet placement, which might produce asynchronous pacing 85–100 bpm (depending upon brand, model, and programming) with short (100 msec) AV delay.
- Careful attention to sterility if central access is placed. Central line placement in the chest is relatively contraindicated for the first 6 wk of implant for any new lead.

Overview

- Indications for permanent pacing: Symptomatic failure of impulse formation (sinoatrial disease), symptomatic failure of impulse conduction (AV block), hypertrophic or dilated cardiomyopathy, long QT syndrome
- Indications for temporary pacing (usually reversible issues): post-cardiac surgery, treatment of drug toxicity resulting in dysrhythmias, certain dysrhythmias complicating MI
- Codes: The NASPE/BPEG generic pacing code has 5 positions. The first position refers to the chamber(s) paced (A=atrium, V=ventricle, D=both, O=None). The second position refers to the chamber(s) sensed (A,V,D,O). The third identifies the response to sensed events (I=inhibit, D=dual chamber pacing and tracking). The fourth position will be “R” if the P-CIED will increase its rate in response to “exercise”; it will be “O” if rate responsiveness is programmed off and a fifth position is present. The fifth position identifies a multisite (A-biartrial, V-biventricular, or D-both) P-CIED.

ICD-9-CM Code: VS3.31 (Permanent pacemaker)

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<tbody>
<tr>
<td>CARDIO</td>
<td>Dysrhythmia</td>
<td>Pacemaker indication</td>
<td>ECG/pulse</td>
<td>Preop pacemaker check; CXR unnecessary to evaluate a properly working device except for multisite pacing device</td>
</tr>
<tr>
<td></td>
<td>Pacemaker</td>
<td></td>
<td></td>
<td>Pacemaker telemetry</td>
</tr>
<tr>
<td>Palpitations</td>
<td>Exacerbating cause(s), such as arm movement, body position, or exercise</td>
<td>Pacemaker pocket manipulation while monitoring pacemaker; arm movement, flexion/extension of shoulder</td>
<td>2-flight walk</td>
<td>Walk test to ensure correct settings of rate response sensor</td>
</tr>
<tr>
<td>Exercise intolerance</td>
<td>Exercise tolerance, angina, Sx CHF</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Atrial tachydysrhythmias</td>
<td>Hypo-, hyperthyroidism</td>
<td>TSH, free T4</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Other causes of syncope</td>
<td>TIAs, CVA, Bruits</td>
<td>Carotid Doppler exam</td>
<td></td>
</tr>
</tbody>
</table>

Code | Indication | Function | Perioperative Management |
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>VVI</td>
<td>Ventricular bradycardia without need for preserved AV conduction</td>
<td>Demand ventricular pacing</td>
<td>Magnet utilization might be helpful to produce asynchronous (VOO) pacing 85–100 bpm. Magnet effect can depend on programming.</td>
</tr>
<tr>
<td>VVIR</td>
<td>Ventricular bradycardia without need for preserved AV conduction, chronotropic incompetence</td>
<td>As above, but adjusts pacing rate to allow somewhat physiologic response to exercise</td>
<td>Pacemaker may sense periop changes (e.g., mechanical stimulus or resp rate) and increase pacing rate, misleading the anesthesia provider to treat “increased pain.”</td>
</tr>
<tr>
<td>DDD</td>
<td>Bradycardia when AV synchrony can be preserved</td>
<td>Provides more physiologic response, maintains AV concordance</td>
<td>Magnet utilization might be helpful to produce asynchronous (DOO) pacing 85–100 bpm. Magnet effect can depend on programming.</td>
</tr>
<tr>
<td>DDDR</td>
<td>Pts requiring AV synchrony and have chronotropic incompetence</td>
<td>Allows somewhat physiologic response to exercise, maintains AV concordance</td>
<td>Pacemaker may sense periop changes (e.g., mechanical stimulus or resp rate) and increase pacing rate, misleading the anesthesia provider to treat “increased pain.”</td>
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Perioperative Implications

Preoperative Preparation

- Comprehensive pacemaker evaluation and/or interrogation. A pacing consult might be needed. Consideration should be given to increasing the pacing rate for any pt undergoing a significant procedure who is chronotopically incompetent or pacing dependent in the atrium.
- For ventricular multisite pacing (called cardiac resynchronization therapy), assurance that the LV pacing lead is functioning. If central access is planned in a CRT pt, a recent chest x-ray might be prudent to document the position of the LV lead.
- Alternate pacing modality available (e.g., transvenous, transcutaneous). While transesoph-
- Regional technique offers CNS perfusion monitoring

**Monitoring**
- Mechanical pulse wave monitoring is required. It can be accomplished with the pulse oximeter plethysmogram, any invasive hemodynamic monitoring modality, or Doppler technique.
- Electrocardiographic (ECG) monitoring is required by ASA standards, but EMI perturbs the signal, and monitors frequently report incorrect heart rates (both too high and too low).

**Induction**
- Succinylcholine or etomidate might lead to inappropriate muscle activity, resulting in pacing inhibition or increased rates. Succinylcholine-induced K⁺ fluxes theoretically can change pacing thresholds, but this has not been reported.

**Maintenance**
- Vigilant ECG and/or pulse monitoring
- Monopolar electrosurgical (ESU) cautery (i.e., the “Bovie”), which emits radiofrequency energy, has potential to cause transient or permanent changes in pacemaker function. The most common problem is inhibition of pacing. Prevention includes use of bipolar only ESU, use of pure unblended monopolar ESU, and placement of the ESU current return pad so that the presumed current path of the ESU does not cross the chest. For all head and neck or contralateral breast surgery, the pad can be placed on the shoulder contralateral to the CIED. For ipsilateral breast surgery, the pad can be placed on the ipsilateral arm and the wire prepped into the field if needed.
- Magnet*: Assuming that the magnet converts the P-CIED to asynchronous pacing, it might be useful to prevent asystole from EMI-induced pacing inhibition. However, the asynchronous pacing rate must be greater than the pt’s own rate, or competition will result. Atrial competition usually just lowers the BP, but ventricular competition can lead to R-on-T pacing with triggering of ventricular tachycardia. Not all ICDs have a programmable asynchronous pacing mode, a possible concern where a pt might require pacing from their ICD.

**Adjuvants**
- K⁺ rapid fluctuations could affect capture.

**Postoperative Period**
- Monitoring of mechanical pulse in the postop care unit
- Pacemaker interrogation advisable if monopolar ESU employed, any problems noted, or cardioversion/defibrillation has taken place.
- Some pts will require pacing changes to incl increased pacing rate, disabling of battery saving features, and adjustments to AV delays to optimize postop hemodynamics
- Other risks related to assoc medical problems

**Anticipated Problems/Concerns**
- Intraop failure to pace, most likely related to monopolar electrosurgery
- Periop pacing and sensing threshold changes
- Risks related to assoc medical problems
- Iatrogenic misadventures resulting from misunderstanding of pacing system behavior

* CAUTION: If the pacing device is actually an implanted cardioverter defibrillator (ICD), then magnet application rarely affects brady pacing. For many ICDs (Boston Scientific [which owns Guidant and CPI brands] and St Jude [which owns Pacesetter]), the magnet switch can be programmed “OFF.” Only ICDs from Boston Scientific and its previous companies emit tones that identify correct placement of a magnet. Some older ICDs from Boston Scientific (with the “GDT” or “CPI” x-ray code) can undergo permanent disabling of tachy therapy by magnet placement. Only ICDs from ELA (Sorin) will change pacing rate (to 90 bpm if battery is ok) upon appropriate magnet placement.
Pancreatitis, Acute

Jeffrey J. Schwartz

Risk
- Incidence of 100–200/1 million in larger cities
- No racial predilection; gallstone more common in women; alcohol more common in men

Perioperative Risks
- Most mortality occurs with surgery for complications of severe pancreatitis: 10–30%
- Risk of nonpancreatic surgery probably dependent on severity of attack

Worry About
- Severe hypovolemia due to sequestration of fluid in retroperitoneal space
- Electrolyte abn, incl hypocalcemia, hyperglycemia, acidosis
- Systemic complications such as alcohol withdrawal, ARDS, acute renal failure, DIC, multi-system organ failure, sepsis (see these topics in Diseases section)

Overview
- Intense inflammatory response caused by release of activated pancreatic enzymes with resultant tissue destruction, fluid and electrolyte loss
- Most commonly a mild self-limited disease diagnosed by abd pain radiating to the back, elevated serum amylase and lipase, CT imaging
- Occasionally severe with renal, pulm, coagulation, septic complications

ICD-9-CM Code: S77.0

Etiology
- Many diverse causes
- Most commonly gallstones, alcohol, trauma, CPB, medications, hypertriglyceridemia, infection
- 10% of cases idiopathic

Usual Treatment
- In most cases, nonspecific and supportive only
- Adequate volume replacement and correction of electrolyte abn
- Intensive care of organ system failures
- Parenteral opioid analgesia
- Thromboprophylaxis
- Early nutritional support; enteral better than parenteral
- Rarely, judiciously timed open or endoscopic surgery to drain abscesses or debride necrotic tissue

Perioperative Implications

Preoperative Preparation
- Assess and correct volume status, hypocalcemia, hyperglycemia, acidosis

Monitoring
- Consider bladder catheter for monitoring of UO
- Consider arterial catheter if need for blood draws or hypovolemia
- Consider CVP or PA catheter for monitoring of volume status

Assessment Points

<table>
<thead>
<tr>
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<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Orthostatic dizziness</td>
<td>Lying and sitting BP and HR</td>
<td>BUN/Cr</td>
</tr>
<tr>
<td>RESP</td>
<td>ARDS</td>
<td>Dypsnea</td>
<td>Hypotension Oliguria</td>
<td>Hct (hemoconcentration)</td>
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<tr>
<td>GI</td>
<td>Ileus</td>
<td>Tachypnea</td>
<td>Chest exam may be nonspecific</td>
<td>ABGs</td>
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<tr>
<td>END</td>
<td>Hyperglycemia</td>
<td>N/V Hæmatemesis</td>
<td>Serum glucose</td>
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<tr>
<td>HEME</td>
<td>DIC</td>
<td></td>
<td>Bleeding</td>
<td>PT/PTT, plt FSR, fibrinogen</td>
</tr>
<tr>
<td>RENAL</td>
<td>Acute renal failure</td>
<td>Hypocalcemia</td>
<td>Tetany</td>
<td>BUN/Cr</td>
</tr>
<tr>
<td>CNS</td>
<td>Psychosis Encephalopathy</td>
<td></td>
<td>Mental status</td>
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Airway
- Routine

Induction
- Peritoneal irritation frequently leads to ileus and ↑ risk of aspiration
- Anticipate hypovolemia

Maintenance
- CV instability due to massive sequestration of fluid; depending on severity >10 L of isotonic fluid may be required over 24 hr

Exhalation
- Will likely require postop mechanical ventilation

Adjuncts
- Multiple interaction of protein-bound drugs, esp. if pt malnourished or undergoing alcohol withdrawal (see Malnutrition in Diseases section and Alcohol Abuse in Diseases section)

Anticipated Problems/Concerns
- Pts with pancreatic presentation for abd surgery are typically critically ill and require postop intensive care to manage hypovolemia, ARDS, DIC, acute renal failure, sepsis
- Hypoglycemia, hyperglycemia are life-threatening risks after pancreatectomy
- Alcohol withdrawal can be life-threatening.
Pancreatitis, Chronic

Overview
- A nonlethal condition characterized by fibrosis, inflammation, loss of exocrine pancreatic tissue
- Characterized by severe persistent or episodic abdominal pain
- Malabsorption and DM are consequences of loss of pancreatic tissue
- Endocrine insufficiency occurs later than exocrine insufficiency.

ICD-9-CM Code: 577.1

Etiology
- Most commonly chronic alcohol use leads to proteinaceous plugs in the ducts and atrophy of acinar tissue with fibrosis
- Other causes are pancreatic duct obstruction, cystic fibrosis, protein-calorie malnutrition
- Acute pancreatitis does not lead to chronic pancreatitis
- 30–40% of cases are idiopathic

Perioperative Implications

Preoperative Preparation
- If pt is receiving chronic opioids, the usual dose should be given on the day of surgery.
- Glucose/insulin management

Monitoring
- Routine

Airway
- Routine

Induction
- Consider full stomach if abdominal pain

Maintenance
- Consideration of opioid tolerance must be incorporated in plan.

Extubation
- Routine

Adjuvants
- Multiple interventions and adjustments needed for protein-bound drugs if pt is malnourished (see Malnutrition in Diseases section)

Anticipated Problems/Concerns
- Difficulty managing pain and opioids in pts on large doses of opioids for chronic pain
- Pancreatic endocrine insufficiency may lead to impaired glucose intolerance without chronic sequelae of DM.

Risk
- Unknown

Perioperative Risks
- Periop mortality directly related to chronic pancreatitis (rare)
- Assoc malnutrition may lead to difficulty with wound healing and infection.
- Endocrine insufficiency leads to glucose intolerance, but ketosis, coma, and chronic diabetic complications are rare.

Worry About
- Management of pain and opioids if pt is tolerant owing to chronic administration

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<td>GI</td>
<td>Malabsorption</td>
<td>Diarrhea</td>
<td>Orthostatic hypotension</td>
<td>BUN/Cr Albumin</td>
</tr>
<tr>
<td>ENDO</td>
<td>Glucose intolerance</td>
<td>Polyuria, polydipsia</td>
<td>Serum glucose</td>
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Parkinson’s Disease (Paralysis Agitans)

Overview
- Pathophysiology: Loss of dopaminergic fibers in substantia nigra in basal ganglia leads to dopamine deficiency.
- Dx based on clinical presentation: Resting tremor, muscle rigidity, bradykinesia, postural instability, facial immobility, with or without dementia or depression
- Aspiration pneumonia—most common cause of death

ICD-9-CM Code: 332.0

Etiology
- Etiology unknown
- Possible genetic link
- Exposure to environmental toxins (MPTP), pesticides may play a role.

Usual Treatment
- Pharmacologic
  - Dopamine precursors—L-Dopa (prodrug converted to dopamine in brain) mainstay of therapy, usually in combination with a Dopa decarboxylase inhibitor (carbidopa) to prevent peripheral conversion to dopamine in bloodstream.
  - Treatment with levodopa characterized by “on” periods of symptom amelioration and possible dyskinesias, followed by “off” periods with decreasing therapeutic levels of dopamine and return of Parkinsonism symptoms.
  - Dopamine agonists: Ergot alkaloids (bromocriptine, cabergoline, lisurid) and non-ergot alkaloids (pramipexole, ropinirole, rotigotine);
  - A D-1/D-2 receptor agonist, amantadine, is an effective treatment for “off” episodes. It is administered via subQ, intranasal, or sublingual route; high incidence of nausea and/or vomiting.
  - Antivirals—amantadine, useful for treatment of L-dopa induced dyskinesias
  - MAO-B inhibitors—selegiline, rasagiline
  - Catechol-O-Methyl Transferase Inhibitors (COMT): Entacapone and tolcapone inhibit breakdown of dopamine
  - Anticholinergics—trihexyphenidyl, benztpine
- Surgical
  - Deep brain stimulation: Inhibits neuronal activity at site of stimulation and therefore mimics an ablative lesion; stimulation to either subthalamic nucleus (most promising; treats tremor, bradykinesia and rigidity), internal globus pallidus, or unilateral thalamus (treats tremor)
  - Pallidotomy, thalamotomy: Rarely used today; replaced by DBS
  - Continuous infusion of levodopa via implantable intrathecal catheter
  - Experimental use of fetal stem cell implantation

ASSESSMENT POINTS

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<th>Assessment by Hx and PE</th>
<th>Test</th>
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</thead>
<tbody>
<tr>
<td>NEURO</td>
<td>Tremor, akinesia, depression, confusion, dementia, hallucination</td>
<td>Resting tremor that disappears with purposeful movement</td>
<td>Mini-mental status exam</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Muscle rigidity</td>
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<tr>
<td></td>
<td></td>
<td>Akinesia</td>
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<tr>
<td></td>
<td></td>
<td>Speech impairment</td>
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<tr>
<td></td>
<td></td>
<td>Confusion</td>
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<tr>
<td></td>
<td></td>
<td>Dementia</td>
<td></td>
</tr>
<tr>
<td>ENT</td>
<td>Pharyngeal muscle dysfunction, dysphagia, post extubation laryngospasm, siaorrrhea, papillary abn, ocuolytic crises</td>
<td>Dysphagia</td>
<td>Swallowing evaluation</td>
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<tr>
<td></td>
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<td>Sialorrhea</td>
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<td></td>
<td></td>
<td>Retained secretions</td>
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</tr>
<tr>
<td>PULM</td>
<td>Atelectasis, resp infections, aspiration pneumonia, diaphragmatic spasm, uncoordination of resp muscles, postop resp failure</td>
<td>Resp impairment</td>
<td>CXR</td>
</tr>
<tr>
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<td></td>
<td>Atelectasis</td>
<td>PFTs</td>
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<tr>
<td></td>
<td></td>
<td>Aspiration pneumonia</td>
<td>ABG</td>
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<tr>
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<td>Post extubation laryngospasm</td>
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</tr>
<tr>
<td>CARDIO</td>
<td>Hypovolemia, orthostatic hypotension, arrhythmias, depletion of myocardial norepinephrine stores</td>
<td>Orthostatic hypotension</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Arrhythmias</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypovolemia</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Wt loss, malnutrition, GERD</td>
<td>Wt loss</td>
<td>Swallow eval, serum albumin/transferrin</td>
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<tr>
<td>ENDO</td>
<td>Abnormal glucose metabolism (selegiline)</td>
<td></td>
<td>Blood glucose concentration</td>
</tr>
<tr>
<td>UROL</td>
<td>Bladder dysfunction from autonomic instability</td>
<td>Difficulty in urination</td>
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</tr>
<tr>
<td>DERM</td>
<td>Sebrrhoea</td>
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Perioperative Implications for DBS Surgery

Preoperative Implications
- Hold Parkinson medications the morning of surgery
- Avoid benzodiazepines that could interfere with cooperation or resting tremor

Monitoring
- Routine

Airway
- Poor airway access due to stereotactic headframe, hence always have plan to access airway, should need rise, incl having a key to the frame.

- Nasal cannula generally used, facemask, LMA,uffed opharyngeal airway and awake ET intubation with local anesthetic have also been used to secure the airway, when required.

Intraoperative
- DBS surgery requires the pts to be secured in a stereotactic headframe and be awake to allow identification of areas of brain, accurate electrophysiologic recording and for intraop neurologic assessment.
- Different techniques used are awake technique with local anesthesia, regional anesthesia using scalp nerve blocks or IV sedation, and general anesthesia.

- Dexmedetomidine ideal sedation agent as it does not interfere with Parkinsonian symptoms, generally used in doses of 0.3–0.6 mcg/kg/hr
- Propofol not ideal as may mask tremor but can be used during certain stages of procedure (CT and MRI studies) in a low dose infusion (up to 50 mcg/kg/min)
- Small doses of fentanyl or low dose remifentanil (less than 0.1 mcg/kg/min as a dose of 0.1 mcg/kg/min can cause rigidity) have been used.
- Ketamine may lead to exaggerated sympathetic response but recently has been reported to be used safely and successfully in a low dose for...
Preop sedation and dyskinesia attenuation during internal pulse generator placement
- Optimize pt comfort while positioning
- Minimize IV fluids (no Foley)
- Complications of DBS surgery incl intracranial hemorrhage, seizure, venous air embolism, infection

**General Anesthesia**
- Reserved for pts who cannot tolerate awake procedure
- Must decide on GA prior to procedure as difficult to access airway after headframe positioned
- Volatile anesthetics, IV anesthetics or combination used. Limit amount of anesthetic used. Using <1 MAC of volatile anesthetics, reportedly, does not drastically affect microelectrode mapping

**Perioperative Implications for Non-DBS Surgery**

**Preoperative Implications**
- Continue Parkinson medications the morning of surgery
- Administer L-dopa via OG/NG at regularly scheduled intervals during surgery to prevent Parkinson's exacerbation.

**Monitoring**
- **Routine**
- **Airway**
  - Aspiration risk
- **Induction**
  - Propofol ideal induction agent
  - Thiopental may cause parkinsonian episodes
- **Maintenance**
  - Exaggerated vasodilatation and cardiodepressant effects with volatile anesthetics
  - Nondepolarizing NMB drugs well tolerated, but mask tremor
  - Anecdotal reports of hyperkalemia with succinylycholine
  - Enhanced opioid-induced muscle rigidity following fentanyl administration
  - Increased risk of neostigmine-induced bronchoconstriction

**General Anesthesia**
- May see transient appearance of otherwise pathological neurologic reflexes (hyperreactive stretch reflexes, ankle clonus, Babinski reflex, decerebrate posturing) on emergence

**Regional Anesthesia**
- Advantageous
- Diphenhydramine useful for sedation

**Postoperative Period**
- Confusion, delirium, hallucinations common
- Shivering common

**Anticipated Problems/Concerns**
- Be aware of all Parkinson’s medications and possible drug interactions, particularly with MAO inhibitors.
- Avoid drugs that exacerbate parkinsonism (phenothiazines, butyrophenones, and metoclopramide).
- Use caution with airway management, esp. keeping in mind postextubation laryngospasm and resp failure.
Paroxysmal Atrial Tachycardia

**Risk**
- May be seen in ICU pts and indistinguishable from paroxysmal SVT
- Digitalis toxicity, acute electrolyte or acid-base imbalance
- Incidence of 2% in the periop period (excl AF or atrial flutter)
- No racial prevalence and all age groups
- May be seen with mitral valve prolapse esp in females

**Perioperative Risks**
- Rapid heart rate impairs LV filling and may adversely affect LV function in pts with LV failure, hypertrophic cardiomyopathy, aortic or mitral stenosis
- Cerebrovascular disease

**Worry About**
- Syncope: −15% on initiation or abrupt termination of rapid SVT
- Syncope may also indicate AF and rapid conduction over an accessory pathway
- Hypotension: In pts with systolic or diastolic dysfunction
- Chest pain: Pts with CAD
- ST-T segment changes: Common with rapid rates and reduced coronary filling even with normal coronaries
- VF: In Wolff-Parkinson-White (WPW) pts who develop AF
- Dig level, electrolyte and acid-base status

**Overview**
- Paroxysmal atrial tachycardia (PAT) is among a larger group of narrow (<120 ms) QRS-complex tachycardias defined by the ACC/AHA/ESC task force to incl: paroxysmal supraventricular tachycardia (PSVT), AF/flutter, permanent junctional tachycardia and focal atrial tachycardia and macro re-entrant tachycardia
- Rapid atrial arrhythmias occur after any major surgery in pts >60 y of age (3–4%) but with a greater incidence of cardiac (20–40%), thoracic (4–27%) and peak 2–3 d after surgery. Acute postop events such as pneumonia or ARDS may increase the incidence
- Causes poorly defined, probably multifactorial, and may incl catecholamine excess and pericardial inflammation
- Common mechanisms of narrow complex tachycardias in the periop period
- Re-entrant rhythms: AV nodal re-entrant tachycardia, AV reciprocating tachycardia through accessory pathway, AF/flutter (most common in over 90%)
- Unifocal or ectopic atrial tachycardia
- Multifocal atrial tachycardia in pts with chronic pulm disease

**ICD-9-CM Code: 427.0**

**ASSESSMENT POINTS**

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<tr>
<td>CARDIO</td>
<td>WPW AV nodal re-entry</td>
<td>Palpitations, diaphoresis Hypotension, chest pain</td>
<td>Prominent jugular venous pulsations</td>
<td>ECG (150-250 bpm, abn P waves preceding QRS, rarely discernible) Electrophysiologic studies, ECHO</td>
</tr>
<tr>
<td>NEURO</td>
<td>Rapid arrhythmia</td>
<td>Fatigue, presyncope or syncope</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Rapid arrhythmia</td>
<td>Dyspnea</td>
<td>Rales, wheezes</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Atrial dilation</td>
<td>Polyuria</td>
<td></td>
<td></td>
</tr>
</tbody>
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**Perioperative Implications**

**Preoperative Preparation**
- If possible, continue Ca²⁺-channel blockers and β-blockers periop to avoid withdrawal-associ arrhythmias.
- Correct hypoxemia and electrolyte imbalance
- Consider proven prophylactic regimens for high-risk pts undergoing cardiac or thoracic surgery.
- Pts with recurrent arrhythmias may be taking drugs such as flecainide, propafenone, amiodarone or dofetilide for prevention.

**Anticipated Problems/Concerns**
- Pts with refractory arrhythmias have usually had electrophysiologic studies and in some, catheter ablation procedure
- Continuous intraop ECG monitoring and postop ECG monitoring in high-risk pts
- Aim for effective postop analgesia.
- Consider β-blockers in hyperadrenergic postop pts with adequate cardiac output.

- IV adenosine or Ca²⁺-channel blockers (diltiazem or verapamil) are the drugs of choice but beta-blockers may also be used. Adenosine may provoke bronchospassin in pts with reactive airway disease, or excessive (prolonged) bradycardia in pts taking carbamazepine or in denervated heart transplant pts. Higher doses of adenosine may be needed in pts taking methylxanthines (i.e., theophylline). Adenosine may initiate AF in 1–15% which is usually transient.
- The goal of second-line therapy is to achieve ventricular rate control and possible conversion when PAT does not respond, or rapidly recurs, after adenosine. IV digoxin is not effective unless CHF is present.
- When AV nodal block is unsuccessful, electrical cardioversion is considered. If infeasible or unsuccessful, antiarrhythmic agents may also be used. When LV function is preserved, IV options incl procainamide and amiodarone. The proarhythmic potential of these agents makes them less desirable than AV nodal blockade. In pts with poor LV function, IV amiodarone is preferred.
- Pts with accessory pathway re-entrant rhythms who develop AF at risk for VFIB, and this scenario is exacerbated by agents that reduce the accessory bundle refractory period (digoxin, Ca²⁺-channel blockers, β-blockers, and adenosine). Hence, WPW pts who experience AF should not receive AV nodal blockers, and IV procainamide and amiodarone are preferred agents for slowing the rate and to achieve conversion.
- Multifocal and unifocal PAT: Correct underlying hypoxia, electrolyte imbalance. Therapy: Electrical cardioversion and procainamide are not effective. Effective IV agents available for use incl AV nodal blockers (Ca²⁺-channel blockers, β-blockers) and amiodarone. While digoxin slows the ventricular rate, toxicity may provoke automatic atrial tachycardia.

**Overview**
- Rapid atrial arrhythmias occur after any major surgery in pts >60 y of age (3–4%) but with a greater incidence of cardiac (20–40%), thoracic (4–27%) and peak 2–3 d after surgery. Acute postop events such as pneumonia or ARDS may increase the incidence
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<tr>
<td>RESP</td>
<td>Rapid arrhythmia</td>
<td>Dyspnea</td>
<td>Rales, wheezes</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Atrial dilation</td>
<td>Polyuria</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Patent Ductus Arteriosus**

Aris Sophocles
Mark Twite

### Overview
- **Preterm and low-birth-weight infants:** PDA may cause CHF and worsening of chronic lung disease which makes weaning from mechanical ventilation difficult.
- **Term and older infants:** PDA may be asymptomatic or occur with failure to thrive, recurrent resp infections, and CHF.
- **Silent duct is a small PDA detected with echocardiography with no murmur heard**
- **PDA leads to an increased risk of endocarditis**

**ICD-9-CM Code:** 747.0

### Etiology
- **Normal:** The arterial duct is the connection between the pulm artery and the aorta that shunts blood away from the lungs during in-utero fetal development. The duct normally constricts shortly after birth due to the postnatal drop in circulating prostaglandin levels as well as the rise in systemic O2 tension. Constriction is followed by permanent duct closure from endothelial and smooth muscle cell hypertrophy and eventual formation of the ductal ligament.
- **PDA:** In preterm infants the ductal muscle layer is thin and poorly contractile and has a poor constrictor response to changes in arterial oxygen tension.

### Perioperative Implications

#### Preoperative Preparation
- **Surgery:** Bedside unstable neonate: Cross-matched blood at bedside, adequate IV access with extension tubing, familiar with ventilator function and settings, check current running infusions (TPN, vasopressors)
- **OR stable child:** Cross-matched blood in the OR
- **Cardiac cath lab:** Routine setup for general ET anesthesia

#### Preinduction/Induction
- **Unstable neonate:** Induce with fentanyl (10–30 mcg/kg)
- **Cath lab/OR stable child:** Premedication and mask induction

#### Monitoring
- **Standard ASA monitors**
- **Unstable neonates require an arterial line for continuous BP measurement and blood gas analysis and central venous access for inotropic drug delivery.

### Anticipated Problems/Concerns
- **Stable older children do not require invasive monitoring.**
- **Airway:** Critically ill neonates are already intubated and ventilated. Check tube size for leak and confirm position on CXR.
- **OR cases:** Intubate for single lung ventilation (right main stem a single lumen ETT, bronchial blocker, or double lumen tube)
- **Cath lab cases:** Young children often require intubation, older cooperative children may be done with a natural airway.
- **Maintenance and extubation:** Bedside critically ill neonate: Fentanyl, paralytics, and remain intubated at the end of the procedure
- **Stable child in the OR:** Balanced anesthetic technique with the goal of early extubation and adequate analgesia (consider regional techniques)
- **Cath lab:** Balanced anesthetic technique and extubate at the end of the procedure. Analgesic requirements are minimal and related to the femoral vessel puncture sites

#### Adjuvants
- **Antibiotic prophylaxis for all cases (usually cefazolin 30–50 mg/kg)**

#### Postoperative Period
- **Adequate analgesia**

**Perioperative Risk**
- **Surgery:** Hemorrhage; hemodynamic instability esp in premature and low-birth-weight neonates; single lung ventilation resulting in hypoxia, atelectasis, and pneumothorax; recurrent laryngeal nerve injury; chylothorax; ligation of the incorrect vessel (aorta or pulm artery); thoracic scoliosis long term
- **Cardiac cath lab device closure:** Obstruction of the pulm artery and/or aorta from the occlusion device, arrhythmias, incomplete closure, and embolization of the device

**Worry About**
- **Premature infant:** Lung disease and high mechanical ventilator settings, hemodynamic instability after duct closure due to poor cardiac reserve

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CHF</td>
<td>FTT, difficulty feeding</td>
<td>‘Machinery’ murmur</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>Pulmonary Htn</td>
<td></td>
<td>Wide pulse pressure</td>
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<td></td>
<td></td>
<td></td>
<td>Pulsum biferiens</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Tachycardia</td>
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<td></td>
<td></td>
<td></td>
<td>Diaphoresis</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>Recurrent resp infections</td>
<td>Worsening mechanical ventilation</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>† O2 requirement</td>
<td>parameters</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Rales</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Necrotizing enterocolitis</td>
<td>Abd distention, Poor feeding, Blood in stool, Free air in peritoneum</td>
<td>Distended tense abdomen, Edema of abd wall, Tender abdomen</td>
<td>Abdominal XR</td>
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<tr>
<td>RENAL</td>
<td>Oliguria</td>
<td>† U/O due to ‡ renal blood flow</td>
<td>Serum chemistry</td>
<td></td>
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<tr>
<td>CNS</td>
<td>CNS hemorrhage</td>
<td>† Fontanel pressure † Hct</td>
<td>† Fontanel size and tension</td>
<td>Head USS</td>
</tr>
</tbody>
</table>

Pemphigus

**Risk**
- Incidence in USA: 0.1–0.5/100,000/y for pemphigus vulgaris (the most common form of pemphigus)
- Age: Most common from age 30–60 y; can occur in children or elderly
- Most common in people of Mediterranean descent

**Perioperative Risks**
- Infection
- Electrolyte abn with extensive lesions

**Worry About**
- Pharyngeal blisters, sloughing of mucosa, bleeding produced by airway manipulations
- Consequences of steroid treatment (e.g., Htn, hyperglycemia, gastric or duodenal ulceration, myopathy, infection, psychic disturbances, osteoporosis) or immunosuppressive therapy (bone marrow suppression)

**Overview**
- Autoimmune, intraepidermal blistering disease of skin and mucous membranes. Oral lesions most common. Blisters rupture easily, heal slowly, usually do not scar.
- Four types: Vulgaris (most common and severe form), vegetalis, foliaceus, erythematous
- 5 y mortality 5–15% for treated pemphigus vulgaris. Most common cause of death is infection, usually with Staphylococcus aureus.
- Occasionally co-exists with other autoimmune diseases, thymoma (with or without myasthenia gravis), or malignancies

**ICD-9-CM Code: 694.4**

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Oral and pharyngeal erosions and blisters</td>
<td>Painful oral lesions</td>
<td>BP</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↑ Salivation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Painful swallowing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Htn (due to steroids)</td>
<td>Fever, cough, sputum</td>
<td>Diminished breath sounds, dullness to percussion</td>
<td>CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>At risk for pneumonia</td>
<td>Epigastric pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dark stools</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Gastric or duodenal ulcer (due to steroids)</td>
<td>Fatiguability, weakness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Myopathy (due to steroids)</td>
<td>Blisters</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Blisters</td>
<td>Blisters</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Denuded areas</td>
<td>Denuded or crusted areas of skin</td>
<td>Electrolytes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lyte abn</td>
<td></td>
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</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Pts may require supplemental steroids.
- Avoid tape on skin because it can generate new lesions.
- Secure IV with loose cloth bandage or suture.

**Monitoring**
- Consider monitors that do not adhere to skin.
- Consider removing adhesive from ECG pads and oximeter probes and securing with loose bandage. Place soft padding (e.g., Webril) under BP cuff.

**Airway**
- Pts may have oral erosions or blisters; new blisters may form from airway manipulation. Risk is of airway obstruction or bleeding. Consider lubricating mask and laryngoscope blade to decrease friction, using small ETT, minimal cuff inflation; and suture or hold tube in place. Avoid LMA owing to quantitated risk of pharyngeal trauma.

**Premedication/Induction**
- Lubricate eyes, do not tape
- Allow pt to position self on well-padded OR table, to decrease risk of blister formation with positioning. Ensure all pressure points are padded once pt is on table.

**Maintenance**
- Neither general nor regional anesthesia clearly superior
- Local infiltration probably contraindicated owing to risk of blister formation

**Exubation**
- Minimize coughing during extubation

**Postoperative Period**
- New lesions of skin or mucous membranes may appear

**Adjuvants**
- Depends on agents and effects of agents used for chronic treatment
- Consider need for steroid supplementation

**Anticipated Problems/Concerns**
- Minor frictional trauma to skin or mucosa may generate new lesions. Airway must be instrumented gently and tape avoided anywhere on the skin.
- Pts are at risk of infection and lyte abn from pemphigus and of side effects of steroid and immunosuppressive therapy.
Pericardial Effusion

**Risk**
- Occurs rarely
- Postop open heart or PTCA: Blood and/or serous
- Infection: Viral, bacterial, fungal
- Neoplastic: Lymphoma, leukemia
- Post acute MI (esp transmural)
- Trauma
- Gender predominance: Male > female

**Perioperative Risks**
- If unknown, tamponade causing CV collapse possible with low probability of determining cause ante mortem
- If known, risk of CV collapse, esp with induction and institution of positive pressure ventilation

**Overview**
- Found in sac surrounding heart; if severe can restrict filling of heart
- Ventricular filling is depressed in both RV and LV
- Fluid bolus and inotropes do little to improve cardiac output
- Cardiac output becomes more dependent on heart rate
- Must have surgical drainage for proper treatment

**ICD-9-CM Code:** 423.9

**Worry About**
- Hypovolemia
- Limited filling of cardiac chambers

**Etiology**
- Postsurgical and catheterization procedures
- During or after viral, bacterial, or fungal infection
- Postinflammatory process: Acute transmural, SLE, rheumatoid arthritis
- Neoplastic
- Trauma

**Usual Treatment**
- Drainage either percutaneous or open
- Medical management is generally ineffective

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**ASSESSMENT POINTS**

<table>
<thead>
<tr>
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<th>Effect</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Tamponade limiting CO</td>
<td>Chest pain</td>
<td>Neck veins HR BP</td>
<td>Equalization of all pressures in heart (catheterization)</td>
</tr>
<tr>
<td></td>
<td>Hypotension</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Arrhythmias</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>↓ CO on institution of IPPB (mechanical ventilation)</td>
<td>Dyspnea</td>
<td>Change in BP on institution of mechanical ventilation</td>
<td>Pulm artery, RA, LA pressures</td>
</tr>
<tr>
<td>METAB</td>
<td>Metabolic acidosis</td>
<td></td>
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</tr>
</tbody>
</table>


**Induction/Maintenance**
- Do not decrease preload
- Slowly titrate small doses of barbiturates or propofol
- Monitor hemodynamics and use anesthetic, if tolerated, or etomidate
- Consider placing before induction invasive hemodynamic monitoring
- Ketamine and pancuronium have been advocated for new tamponade situations
- Initiation of positive pressure ventilation may cause severe CV compromise due to decreased filling of RV and LV

**Treatment Approach**
- Post open heart surgery hemorrhage—reopening sternum to explore for sites of hemorrhage—usually relieved by first few sutures released
- Infections and/or neoplasia: Subxyphoid pericardial window
- Small incision: Open pericardium under direct vision; chest tube placed behind heart

**Perioperative Implications**

**Preoperative Preparation**
- Appropriate monitoring before induction
- Preoxygenation is not always effective
- Support hemodynamics-catecholamines, keep acid-base balance full and fast
- Consider draining transthoracically if hemodynamic compromise severe
- Consider prep and drape prior to induction with surgeon ready
- Positive pressure ventilation may significantly worsen hypotension, resulting in shock and death
- Consider placing external defibrillator patches prior to anesthesia induction

**Monitoring**
- Arterial line indicated as BP may change suddenly; sampling of Hct for bleeding and acid-base status in low cardiac output state is useful
- Consider PA catheter, useful in making diagnosis and following surgical treatment. If pressures not relieved on surgical drainage, question original diagnosis.
- TEE, useful but less so than PA monitoring

**Adjuvants**
- Insertion of a pericardioscope provides the ability to visualize the pericardium and obtain biopsies

**Extubation**
- Consider awake extubation or postop mechanical ventilation, depending on etiology

**Anticipated Problems/Concerns**
- Many different causes, all with different sequelae
- Hypotension on induction of anesthesia or positive pressure ventilation
Pericarditis, Constrictive

Ribal Darwish

Overview

- CP is an inflammation of the pericardium, leading to an impaired filling of the ventricles and reduced ventricular function.
- Restriction of the pericardium results in increased ventricular interdependence and a reciprocal relation between the left and right heart filling.
- During spontaneous ventilation, the tricuspid blood flow is increased, resulting in increased filling of the RV. This will lead to the septum to shift to the left and the decrease in the LVEDV with subsequent hypotension, causing pulsus paradoxus.
- During expiration, the septum is shifted to the right. Opposite changes take place during mechanical ventilation.
- Pts present with dyspnea, fatigue, orthopnea, right heart failure with venous congestion and chest pain.

ICD-9-CM Code: 423.2

Etiology

- In developed countries, idiopathic or viral infections are the most common cause of CP followed by cardiac surgery and mediastinal irradiation.
- Bacterial infectious causes are more common in underdeveloped countries such as: Tuberculosis, staphylococci, group A and B streptococci and gram-negative rods.
- Less common causes are uremia, connective tissue disorders, and drug reactions.

Usual Treatment

- In advanced stages, the standard treatment is pericardectomy. Both median sternotomy and left thoracotomy approaches are used.

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Lymphadenopathy if the CP is caused by viral or bacterial infection, cyanosis</td>
<td>Hx of fever, chills, upper resp tract infections.</td>
<td>Enlarged cervical lymph nodes, jugular venous distention</td>
<td>Blood and sputum cultures, immunological assays for viral infections</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema if heart failure develops</td>
<td>Dyspnea, dry cough</td>
<td>Tachypnea, rales on auscultation</td>
<td>Chest x-ray, arterial blood gas analysis</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Right and left heart failure, arrhythmia, hypotension</td>
<td>Dyspnea, orthopnea, chest pain, peripheral edema, fatigue, palpitations, and hepatomegaly</td>
<td>Tachycardia, muffled and distant heart sounds, friction rub, apical pulse is not palpable</td>
<td>MRI and CT scans EKG -low voltage and ectopic AT. increased CVP (W shape). Left heart catheterization shows “square root sign” Doppler echo—restrictive LV diastolic filling, characterized by TMF E/A ratio &gt;&gt;1, short deceleration time (DT) of TMF E velocity, and PVF S/D ratio &lt;1</td>
</tr>
<tr>
<td>MS</td>
<td>Muscle atrophy, myositis if there is an underlying connective tissue disorder</td>
<td>Significant wt loss and muscle wasting</td>
<td>Clinical evidence of weakness</td>
<td>CPK to r/o myositis; specific tests if connective tissue disorder is suspected</td>
</tr>
</tbody>
</table>


Preoperative Implications

Preoperative Preparation
- Cardiac medications incl antidysrhythmics should be continued.

Monitoring
- Have invasive monitoring incl arterial line and pulm artery catheter.
- Intraop TEE is of significant help

Maintenance
- Conducted under general anesthesia
- Narcotic-based technique is preferred
- The intraop hemodynamic goals are adequate preload, maintenance of sinus rhythm, and rate control if sinus rhythm cannot be maintained.

Adjuvants
- Inotrop support is indicated if there is evidence of ventricular dysfunction.
- Most pericardioectomies are done without the need for CPB, but CPB should be on stand by.

Anticipated Problems/Concerns
- Myocardial infarction, major intraop hemorrhage, atrial and ventricular arrhythmias, and worsening of the heart failure.
Peripheral Vascular Disease

**Risk**
- 10–15% of those > age 50 y
- Long-term mortality increased 2–3x in those with overt CAD, large vessel arterial disease, DM
- 3-4 y mortality rate: 30–40%

**Perioperative Risks**
- High prevalence of co-existing CAD and carotid artery disease
- Presence of CAD increases operative mortality
- Pulm and renal insufficiency can cause prolonged recovery or morbidity

**Overview**
- Vascular abn involving extremities increase in frequency with age
- Co-existing diseases common (DM, COPD resulting from smoking, Htn, CAD)

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Htn Coronary artery stenoses MI</td>
<td>Usually asymptomatic Angina, may be asymptomatic</td>
<td>Normal if treated S, and/or S, cardiomegaly</td>
<td>Vital signs ECG Exercise ECG Treadmill Pharmacologic stress test Coronary angiography ECHO Radionuclide studies</td>
</tr>
<tr>
<td>PERIPHERAL VASC EXAM</td>
<td>Occlusive lesions Abd aortic aneurysm may co-exist</td>
<td>Claudication Abd pain, may be asymptomatic</td>
<td>↓ Pulses Pulsatile abd mass</td>
<td>Angio Aortogram MRI</td>
</tr>
<tr>
<td>RESP</td>
<td>COPD (many are smokers)</td>
<td>DOE</td>
<td>↓ Breath sounds Prolonged expiration Wheezes</td>
<td>ABGs PFTs</td>
</tr>
<tr>
<td>ENDO</td>
<td>DM and assoc effects such as angiopathy, peripheral and autonomic neuropathy, nephropathy</td>
<td>Attention to CV, PNS for ANS and other evaluation</td>
<td>Obesity (in DM type II) Cardiomegaly Foot ulcers</td>
<td>Fasting blood sugar</td>
</tr>
<tr>
<td>CNS</td>
<td>Ischemic CNS disease</td>
<td>Scotoma CNS and mental status evaluation Absence spells</td>
<td>CNS exam Search for carotid bruits</td>
<td>Doppler or angio (if indicated)</td>
</tr>
</tbody>
</table>

**Worry About**
- Aortic clamping: May induce myocardial ischemia or ventricular failure; hypotension with declamping
- Increased risk of periop myocardial ischemia and cardiac complications
- Postop thrombosis in arterial grafts
- Postop delirium, esp if >70 y

**Etiology**
- Chronic arterial occlusive disease
- Less common: Takayasu’s syndrome and thromboangitis obliterans

**Usual Treatment**
- Reconstitute pulsatile blood flow to distal vascular tree to allow healing of ulcerated or gangrenous tissue, relieve ischemic rest pain with the goal of salvaging a functional limb
- Most common surgical procedures are aortofemoral bypass, femoropopliteal bypass, femorotibial bypass or endovascular approach
- Angiogenesis gene therapy now being combined with percutaneous angioplasty techniques in experimental protocols

**Preoperative Preparation**
- Attention to and stabilization of concomitant medical conditions such as CAD, COPD, DM
- High-risk pts scheduled for noncardiac surgery may not benefit from a better periop outcome from preop coronary revascularization.
- Consider periop β-blockade in vascular surgery pts with CAD to decrease myocardial ischemia, but caution about acute preop implementation and the potential in increasing periop stroke rate. Ideally, office evaluation should be scheduled several weeks in advance to allow dose titration. Pts already on β-blockade should be have their medication continued.

**Monitoring**
- ST trending if available
- In aortic surgery, consider placement of CVP or TEE for monitoring preload
- Use of transesophageal ECHO may elucidate the mechanism(s) of declamping hypotension (hypovolemia versus ventricular dysfunction) and regional ventricular function (myocardial ischemia)

**Maintenance**
- See above

**Extubation**
- Same hemodynamic concerns as in induction
- Use of postop epidural analgesia decreases likelihood of arterial graft thrombosis

**Advantages**
- β-blockers and other antihypertensives useful in hyperdynamic situations
- Prophylactic nitroglycerin and Ca2+-channel blockers to treat myocardial ischemia not conclusively proven efficacious

**Anticipated Problems/Concerns**
- Periop myocardial ischemia and cardiac complications, thromboembolic events of grafts, CHF, renal failure

Pertussis (Whooping Cough) Raj K. Modak

Overview
• Pertussis is an acute resp infection caused by Bordetella pertussis
• Transmission occurs by resp droplet with a 7–10 d incubation
• Organism releases multiple toxins that damages the epithelial cells of the resp tract
• Characterized by 3 phases: Cattarral (cold symptoms), paroxysmal (cough symptoms), convalescent (persistent or episodic cough)
• Infectivity highest in cattarrhal and early paroxysmal phases
• Adolescents and adults display milder symptoms that may be indistinguishable from less serious causes of URI/LRI
• Immunization in childhood has decreased but increased in adolescence
• Vaccine estimated 80–85% effective after 3 exposures, usually given as combination tetanus, diphtheria, acellular pertussis (Tdap) vaccine
• Increased in incidence in adolescence (age 10–19) indicating a need for booster immunization
ICD–9–CM Code: 033.0

Etiology
• Bordetella pertussis, a fastidious, gram–negative, pleomorphic or rod bacillus

RISK
• Increased prevalence 1976 (lowest) vs. 2007:1010 vs. 10,454 cases
• Incidence highest for infants <1 y, 23% of all cases
• Adolescent group 10–19 y, 33% of all cases
• Incidence of death highest for infants <6 mo, 91% of all deaths
• Females > males (54%)
• Whites > minorities (90%)
• 90% susceptibility following exposure to index case, if immunized
• Only 2% of adult population protected against pertussis

PERIOPERATIVE RISKS
• Most common complications age <6 mo: Hospitalization (69%), pneumonia (13%), seizures (2%), encephalopathy (<2%)
• Common complications in adults: Cough-related incontinence (28%), syncope (6%), pneumonia (5%), rib fractures (4%), hospitalization (3%)

WORRY ABOUT
• Infectivity and contagion
• Secretions, pneumonia, altered mucociliary function, apnea, and decreased pulm reserves causing hypoxemia
• Postop complications related to coughing

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Upper airway obstruction</td>
<td>Difficulty feeding</td>
<td>Rhinorrhea</td>
<td>Nasal culture</td>
</tr>
<tr>
<td>CARDIO</td>
<td>High 0, consumption</td>
<td>Difficulty breathing</td>
<td>Laceration Conjunctivitis</td>
<td>Direct fluorescent antibody (DFA)</td>
</tr>
<tr>
<td>RESP</td>
<td>Cough</td>
<td>Irritability</td>
<td>Tachycardia</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>V/Q mismatch</td>
<td>Apnea, SOB</td>
<td>Inspiratory whoop</td>
<td>Culture + DFA</td>
</tr>
<tr>
<td></td>
<td>Hypoxemia</td>
<td>Tachypnea, rales</td>
<td>Cyanosis</td>
<td>Pulse oximetry, ABGs</td>
</tr>
<tr>
<td></td>
<td>Pneumonia</td>
<td>As above</td>
<td>Rales</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Poor oral intake</td>
<td>Dehydration</td>
<td>Altered turgor</td>
<td>Weigh on scale</td>
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<tr>
<td></td>
<td>Post–tussive emesis</td>
<td>Inability to retain food</td>
<td>Wt loss</td>
<td>LFTs</td>
</tr>
<tr>
<td></td>
<td>Fatty liver</td>
<td>Inguinal hernias</td>
<td>Hepatomegaly</td>
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<td></td>
<td>Cough–induced hernias</td>
<td>Reducible hernias</td>
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<tr>
<td>RENAL</td>
<td>Hypovolemia</td>
<td>Oliguria</td>
<td>Altered turgor</td>
<td>BUN, Cr, FeNa</td>
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<tr>
<td>CNS</td>
<td>Seizures</td>
<td>Seizure type</td>
<td>EEG, CT, MRI</td>
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</tr>
<tr>
<td></td>
<td>Encephalopathy</td>
<td>Altered neuro status</td>
<td>LFT, glucose, ammonia, BUN</td>
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</tr>
<tr>
<td>ID</td>
<td>Immunization Hx</td>
<td>Physical contacts</td>
<td>Culture + DFA</td>
<td></td>
</tr>
</tbody>
</table>


PERIOPERATIVE IMPLICATIONS

Preoperative Preparation
• Postpone elective surgery until noninfectious and symptom-free; uncomplicated disease resolves in 6–10 wk
• Emergency surgery based on risks and benefits
• Infectivity and contagion control with isolation precautions
• Usage of disposable anesthesia circuit system
• If possible, optimize resp function and nutrition prior to surgery
• If in early phases, consider premedication with topical or oral decongestants (epinephrine, pseudoephedrine, xylometazoline) to reduce upper airway secretions; β2 agonists (albuterol, metaproterenol) to minimize risk of bronchospasm
• Optimize prep volume status from dehydration
• Premedication with resp depressants may increase risk of hypoxemia

Monitoring
• Arterial catheterization may be useful in scenarios of impaired oxygenation for frequent blood gas sampling.

Airway
• Acute and chronic coughing increase the risk of upper and lower airway edema with possible obstruction.
• Nasal and tracheal secretions increase the risk of laryngospasm and bronchospasm.
• Inspissated secretions can cause hypoxemia by mucous plugging and atelectasis, barotrauma by airway obstruction, and an inability to ventilate by ET obstruction.

Preinduction/Induction
• In some scenarios, regional anesthesia may be favorable.
• Inhalational techniques with pungent agents should be avoided.
• Avoid agents assoc with coughing.

• A whooping cough syndrome also caused by B. parapertussis, Chlamydia trachomatis, and many adenoviruses

Treatment
• Infectivity and contagion control
• Most effective treatment occurs in cattarrhal and early paroxysmal phases
• Macrolides (erythromycin, azithromycin, clarithromycin) and trimethoprim-sulfamethoxazole
• Cough suppression: Dextromethorphan and codeine; expectorant: guaifenesin
• Corticosteroids and β2 agonists have an unclear role in paroxysmal stage.
• In some cases, hospitalization may be required to suppress cough, institute antibiotic treatment, monitor for apnea and hypoxemia, and general nutrition
• Intensive care treatment may be needed for severe sequelae of pneumonia, seizures, and encephalopathy
• Antibiotic therapy is not recommended in the convalescent phase.
• Emergence techniques using NO may carry an increased risk of post-extubation hypoxemia.
• An H$_2$-blocker should be considered with postop N/V prophylaxis to minimize risk of aspiration of acidic gastric contents.

Postoperative Period
• Infectivity and contagion control with isolation precautions should be maintained.

• Supplemental O$_2$ therapy should incl the use of a humidifier.
• Aggressive pulm toilet
• Monitoring for apnea and hypoxia are needed.
• Regional techniques for pain management may be useful in avoiding serious resp complications related to IV analgesics

Anticipated Problems/Concerns
• Infection risk to all contacts, incl family, other pts, and hospital personnel
• High risk of resp insufficiency causing hypoxemia from tissue damage, edema, and secretions
• Infants at higher risk for sequelae and death compared to adults
Pheochromocytoma

**Risk**
- Incidence in USA: 0.03–0.04% (~80,000) by autopsy of nonselected individuals; 0.1–0.3% of individuals with sustained Htn have pheochromocytoma. At least 20% are now diagnosed when the tumor is incidentally found during abd MRI or CT for other reasons.
- Race with highest prevalence: Caucasian

**Perioperative Risks**
- If emergency (life-threatening trauma, ruptured viscus), use α- and β-blockers and nitroprusside and keep in ICU until most painful time has passed or adrenergic control is attained.
- ↑ Risk of hypertensive crisis with bleeding into myocardium, brain, or kidney or ischemia
- Mortality rate of 0–3% even if appropriately prepared for tumor resection and in “good” hands for adrenalectomy—may be higher for undiscovered case undergoing nonadrenal surgery.
- 25–50% of those who die in hospitals of pheochromocytoma crisis do so during induction of anesthesia, during stressful periop periods, or during labor and delivery.
- Assoc with cholelithiasis and renal stones

**Worry About**
- Pheochromocytoma (catecholamine excess) crisis with hemorrhage/infarcts in vital organs
- Major goal is to avoid pheochromocytoma crisis; pre- and intraprop goals of management of extraadrenal surgery are same as for adrenal surgery. If adrenergic blockade not present prior to surgery, try to delay operation until pt has appropriate degree of α-blocker. Judge appropriate blockade by:
  - No BP readings >165/90 mm Hg for 48 hr.
  - Presence of orthostatic hypotension, but BP on standing should not be <80/45 mm Hg
  - ECG free of ST–T changes
  - Absence of other signs of catecholamine excess, and presence of signs of α-blocker

**Overview**
- Tumor of catecholamine-producing tissue (90% in adrenals). Painful (stressful) events cause exaggerated stress response if less than perfectly anesthetized or in daily living. Even small stresses can lead to blood catecholamine levels of 2000–20,000 pg/mL. However, infarction of tumor, with release of products onto retroperitoneal surfaces or pressure causing release of products, can result in blood levels of 200,000–1 million pg/mL—a situation that should be anticipated during tumor resection.
- Endocrinopathy assoc with CV disease—tachycardia, CHF, dysrhythmias (AFIB)

**Indications and Usual Treatment**
- 90% are spontaneously arising and 10% familial (autosomal dominant genetics involving chromosome 7 implicated)
- Assoc with MEA IIA (medullary thyroid carcinoma; primary hyperparathyroidism) and IIB (medullary thyroid carcinoma and mucosal neuromas) with mutation often at chromosome location 17q11.2
- Assoc with neurofibromatosis, von Hippel-Lindau disease (retinal and cerebellar hemangioblastoma), ataxia-telangiectasia syndrome, Sturge-Weber syndrome, with mutation often at VHL gene localized to chromosome 3p25–26

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td></td>
<td>Nasal stuffiness (from α-adrenergic blockade)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Htn; dysrhythmias; AFIB, sinus tachycardia, mitral valve prolapse; CHF, myoccardial fibral necrosis or myoccarditis</td>
<td>SOB, poor exercise tolerance, palpitations, Htn (50% sustained, 40% paroxysmal)</td>
<td>Standard exam + BP q 1 min in stressful environment + orthostatic maneuvers with BP/HR q 1 min</td>
<td>ECG, ECHO (if cardiomyopathy is suspected)</td>
</tr>
<tr>
<td>GI</td>
<td>90% of tumors adrenal or abd</td>
<td>Wt loss, diarrhea Dehydration</td>
<td>Palpating abdomen can trigger pheo crisis</td>
<td>No different from normal</td>
</tr>
<tr>
<td>HEME</td>
<td>Mild polycythemia, thrombocytopenia (2° to ↓ intravascular fluid)</td>
<td>Hgb (↓ polycythemia way to judge volume expansion by α-blocker)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Renal stones from dehydration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>↑ Catecholamine effects</td>
<td>Headache, tremor, anxiety, ↓ pain threshold, fatigue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Associated with hyperparathyroidism</td>
<td>Glucose intolerance from α-adrenergically induced glucogenesisis and ↓ insulin secretion</td>
<td>Insulin Rx often before Ds made; Ca²⁺</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Prehydrate liberally over 6–60 d if CV status will tolerate; expand with high salt/fluid diet while increasing α-adrenergic blockade over 7–60 d (some use calcium channel blockers, but increased complications assoc with this process epidemiologically)
- Monitoring
  - Temp
  - Art line placement prior to induction difficult and painful but desired because of variations in BP
  - PA catheterization or TEE if CV system severely affected; CVP used in minority of cases
- Anesthetic Technique
  - No technique/group of agents assoc with better outcome; use of droperidol controversial; agents that block reuptake (ketamine) or cause catecholamine release might be avoided.
- Early mobilization and deep breathing a must but fraught with difficulty owing to disturbed psyche that removal of catecholamines present for a long time often causes

**Adjutants**
- Drug interactions possible with chronic antiadrenergic agents such as between verapamil or diltiazem and β-blockers in depressing AV nodal conduction if pt chronically or acutely receiving a β-blocker or decreased clearance of phenytoin, barbiturates, rifampcin, chlorpromazine, and cinetidine.

**Anticipated Problems/Concerns**
- Important to interview family members and perhaps advise them to inform their future anesthesiologists about potential for such familial disease
Physiologic Anemia and the Anemia of Prematurity

**Risk**
- Physiologic anemia is a normal process in term infants.
- Anemia of prematurity is a pathologic anemia occurring in preterm infants. Extent of prematurity and co-morbidities correlates with extent of anemia.

**Perioperative Risk**
- Term infants with physiologic anemia tolerate minor surgery well.
- Premature infants must be evaluated for symptoms due to anemia that may contribute to increased risk of preop events.

**Worry About**
- Major surgery occurring at the physiologic nadir of anemia may require blood transfusion.
- Preterm infants with anemia undergoing physiologic stress due to surgery are at risk for tachycardia, tachypnea, lactic acidosis, and periop apnea and bradycardia.

**Overview**
- Physiologic anemia is normal response to extrauterine life. Nadir at 9th–12th wk of life, Hbg level varies 9 to 11 g/dL.
- In preterm infant, nadir occurs at 4–8 wk of life and may decrease to 8 g/dL.
- Anemia of prematurity may be asymptomatic or have non-specific symptoms such as tachycardia, tachypnea, lethargy, pallor, apnea and bradycardia, poor feeding, poor growth, and lactic acidosis.

**ICD-9-CM Code: 776.6 (Anemia, prematurity neonatal)**

**Etiology**
- Transition to extrauterine life incl increased O₂ available to bind to hemoglobin (HbO₂ saturation 50% in utero, 95% ex utero). Fetal hemoglobin with high O₂ affinity starts to be replaced with low O₂ affinity adult hemoglobin.
- Survival of neonatal erythrocytes is shorter than that of adult erythrocytes. Hemoglobin decreases until O₂ needs are greater than O₂ supply. Production of erythropoietin (EPO) is triggered and erythropoiesis increases.
- Rapid growth in infants causes a rapid increase in blood volume resulting in hemodilution. Growth is more rapid in preterm than term infants.

**Assessment Points (Apply to Preterm Infants Only)**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Tachycardia</td>
<td>Review of VS trends</td>
<td>Tachycardia</td>
<td>± ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Apnea/bradycardia</td>
<td>No. episodes, treatment required or spontaneous resolution</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Timing of elective blood-losing surgery depending on Hgb levels

**Monitoring**
- Routine

**Airway**
- None

**Preinduction/Induction**
- Routine

**Exubation**
- Recent Hx of apnea and bradycardia: Consider delaying extubation to allow metabolism of anesthetic agents and sedatives.

**Adjuvants**
- Spinal anesthesia, when appropriate, may be beneficial in preterm infant.

**Postoperative Care**
- Consider monitoring preterm infant for apnea and bradycardia for 24 hr.

**Anticipated Problems/Concerns**
- Anemia is significant risk factor for postop apnea in preterm infant undergoing surgery and anesthesia.

**Usual Treatment**
- No treatment required in term infants
- Preterm infants benefit from prevention: Reduction of blood draws, appropriate dietary supplementation, and erythropoietin therapy.
- Treatment of anemia of prematurity with blood transfusion occurs when symptoms of reduced O₂ supply are present. Symptoms incl continued need for mechanical ventilation, apnea and bradycardia, tachycardia (>180 bpm for 24 hr), inadequate wt gain, metabolic acidosis, or anticipation of major surgery.
Pickwickian Syndrome

Risk
- 5–10% of morbidly obese pts
- Usually assoc with long-standing obesity

Perioperative Risks
- Marked increased risk from normal body mass index (BMI) pts
- 40% serious morbidity in intra-abdominal or intrathoracic procedures of >2 hr duration

Worry About
- Hypoventilation
- Hypercarbia
- Hypoxemia
- Polycythemia, thrombophlebitis, and subsequent pulm embolism
- Pulm Htn
- Hypersomnolence
- Biventricular cardiac failure

Overview
- Obesity hypoventilation syndrome is defined as the combination of obesity (BMI above 30 kg/m²), hypoxia during sleep, and hypercapnia.
- Morbidly obese pts who hypoventilate due to sleep apnea and severe restrictive ventilatory disorder and have permanent pulm Htn, acidosis, and polycythemia because of their chronic hypoxemia and CO₂ retention.

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Difficult airway access</td>
<td>Snoring</td>
<td>Poor visualization</td>
<td>X-ray of neck may be helpful</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Biventricular failure</td>
<td>Dyspnea, poor exercise tolerance</td>
<td>Venous engorgement, S₃ and S₄, dyspnea</td>
<td>ECG, ECHO, CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoventilation</td>
<td>Dyspnea, sleeping upright</td>
<td>Rapid shallow breathing, cyanosis</td>
<td>ABGs, Hct, CXR</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Consider pulm function tests with bronchodilator to determine if reversible restrictive compo-
  nent exists.
- Assess for bronchitis/pneumonia that can be improved with pulm toilet and antibiotic therapy.
- Assess myocardial and volume status using a central venous catheter or pulm artery catheter (PAC).
- Consider maintaining semi-sitting position to avoid sudden shifts of volume to central circulation and pulm edema.

Monitoring
- Consider an arterial line to monitor frequent ABGs.
- Resp volumes and pressures
- Consider PAC or transesophageal ECHO to monitor filling volumes and wall motion.

Airway
- Awake intubation frequently required
- Shoulders and head elevated on bolster can sometimes facilitate laryngoscopy
- Usually assoc with systemic Htn and compensatory increased circulating blood volume, leading to right and left ventricular failure.
- Two subtypes are recognized, depending on the nature of disordered breathing detected on further investigations. The first is OHS in the context of obstructive sleep apnea; this is confirmed by the occurrence of 5 or more episodes of apnea, hypopnea or resp-related arousals per hr (high apnea-hypopnea index) during sleep. The second is OHS primarily due to "sleep hypoventilation syndrome"; this requires a rise of CO₂ levels by 10 mmHg (1.3 kPa) after sleep compared to awake measurements and overnight drops in O₂ levels without simultaneous apnea or hypopnea. Overall, 90% of all people with OHS fall into the first category and 10% in the second.
- On physical examination, characteristic find-
  ings are the presence of a raised jugular venous pressure, a palpable parasternal heave, a heart murmur due to tricuspid regurgitation, hepatomegaly, ascites, and leg edema.

ICD-9-CM Code: 278 (Obesity)

Etiology
- Work of breathing is increased as adipose tissue restricts the normal movement of the chest muscles and makes the chest wall less compliant caus-
  ing the diaphragm to move less effectively. Resp muscles are fatigued more easily, and airflow is impaired by excessive tissue in the head and neck area.
- Under normal circumstances, central chemore-
  ceptors in the brainstem detect decreased pH, and respond by increasing the resp rate; in OHS, the ventilatory response is blunted.
- Episodes of nighttime acidosis due to sleep apnea lead to renal compensation with retention of bicarbonate.
- Nighttime apnea leads to hypoxya causing hypoxic pulm vasoconstriction (HPV). HPV in turn leads to pulm Htn and right ventricular fail-
  ure and remodeling.
- The chronically low O₂ levels in the blood also lead to increased release of erythropoietin causing polycythemia.

Usual Treatment
- Wt loss through diet and exercise, which is rarely successful, or bariatric surgery
- Continuous positive airway pressure (CPAP)
- Uvulopalatopharyngoplasty
- Tracheostomy

Induction
- Do not expect to ventilate pt adequately by mask. Establish airway first.

Maintenance
- May have to remain in reverse Trendelenburg position to allow adequate ventilation.

Exsufflation
- Perform in sitting position without residual sedation.
- Ensure adequate tidal volume and consider preop levels of CO₂ retention when making deci-
  sion to extubate as normal CO₂ level may not be attainable in these pts.

Adjovants
- Regional anesthesia only if pt is able to main-
  tain ventilation
- Residual sedation or narcosis may preclude early extubation.

Postoperative Period
- Consider prophylaxis for thromboembolism—
  early ambulation may minimize pulm and thromboembolic complications.
- May be extremely sensitive to resp depressant effects of benzodiazepines and narcotics

Anticipated Problems/Concerns
- All those assoc with morbid obesity apply to Pickwickian pts.
- Early ambulation may minimize pulm and thromboembolic complications.
- Prepare the pt for a possible prolonged course of postop mechanical ventilation, esp. after upper abd procedures.
Pierre Robin Syndrome

Charles B. Cauldwell

Risk
• 1/8500 live births
• No known sex or race predilection

Perioperative Risks
• Assoc congenital anomalies, e.g., cardiac
• Pulm Htn, cor pulmonale, or pulm edema 2° to chronic airway obstruction
• Cachexia due to feeding difficulties

Worry About
• Airway obstruction
• Difficult intubation

Overview
• An anomaly consisting of micrognathia (or retrognathia), glossoptosis (posterior displacement of the tongue), and cleft palate, leading to varying degrees of airway obstruction and feeding difficulties. Some authors incl resp distress as necessary for diagnosis.
• Airway obstruction can lead to hypoxia, brain damage, or CHF.
• Feeding problems may cause malnutrition or aspiration.
• Obstruction often improves by several mo of age, 2° to mandibular growth, if hypoxia and malnutrition are avoided.

ICD-9-CM Code: 756.0

Etiology
• Congenital, found either as isolated syndrome or as part of multiple defect syndromes. In several series, about 50%-60% of cases are isolated, the rest are part of other syndromes, esp. Stickler and velo-cardio-facial.
• Also named Pierre Robin Sequence, reflecting the several disease processes that can lead to mandibular maldevelopment

Usual Treatment
• Prone positioning, lavage feeding
• Nasopharyngeal or oral airway, for short-term treatment
• Glossopexy or tracheotomy, if surgery necessary
• Mandibular distraction osterotomy for selected cases

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Airway obstruction</td>
<td>Sleep apnea</td>
<td>Micrognathia</td>
<td>Sleep study</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Feeding difficulties</td>
<td>Cleft palate</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Pulm Htn</td>
<td>Loud S2</td>
<td>Cyanotic episodes</td>
<td>EKG</td>
</tr>
<tr>
<td></td>
<td>Cor pulmonale</td>
<td>Murmurs</td>
<td></td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>Congenital defects</td>
<td>Tachypnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxia</td>
<td>Tachypnea</td>
<td>Cyanotic episodes</td>
<td>EKG</td>
</tr>
<tr>
<td></td>
<td>Pulm edema</td>
<td>Labored inspiration or stridor</td>
<td>Cyanotic episodes</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>Aspiration pneumonitis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Failure to thrive</td>
<td>Feeding problems</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Hypoxia</td>
<td>Seizures</td>
<td>Developmental delay</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
• Avoid sedative premedication.
• Consider atropine as antisialogogue and to maintain heart rate.

Monitoring
• Oximeter and precordial stethoscope particularly important

Airway
• Intubation may be very difficult.
• Consider awake intubation in neonates.

• Airway management and intubation tends to get easier with age, esp. in isolated Pierre Robin.

Preinduction/Induction
• May obstruct in supine position while awake or early during inhalation induction
• Consider oral or nasopharyngeal airway.
• Have difficult airway cart available.
• Consider use of LMA with fiberoptic bronchoscope and exchange catheter, or light wand.
• Have surgeon in OR capable of performing tracheotomy when induction begins.

Extubation
• Pt should be awake for extubation, may need to recover in ICU.

Adjuvants
• Do not use muscle relaxants unless absolutely sure pt can be intubated.

Anticipated Problems/Concerns
• Airway obstruction during all phases of anesthesia very common
### Overview
- Symptoms due to hormonal dysregulation or local mass effect
  - Microadenomas (secretion): Prolactinoma (increased PRL), Cushing's disease (increased ACTH), Acromegaly (increased GH)
  - Macroadenoma (mass lesion): Panhypopituitarism, Bitemporal hemianopsia

### Risk
- Surgical incidence in USA: 7500/y
- Incidental small adenomas occur in 17% adults
- M:F ratio: 1:8 in some histologies, gender neutral in others

### Perioperative Risks
- Risks due to 2° endocrine syndromes from secreting (functional) adenomas, e.g., acromegaly, Cushing's syndrome, DM, hyperthyroidism

### Worry About
- Angina, cardiomyopathy with evidence of CHF, electrolyte imbalance
- Difficult airway in acromegaly

### Perioperative Implications

#### Preoperative Preparation
- Replacement therapy for panhypopituitarism

#### Monitoring
- Invasive arterial pressure monitoring usually required if intercurrent disease
- Continuous ETCO$_2$ and N$_2$O to detect venous air embolism when incision >10 cm above right atrium
- Consider CVP in severe acromegaly

#### Airway
- Have variety of laryngoscope blades and small ETT available

- Consider awake, oral fiberoptic intubation if macroglossia is present

#### Maintenance
- Hts frequently associated with epinephrine infiltration of nasal mucosa and hammering of nasal speculum in classic approach. Anticipate and pretreat.

#### Extubation
- Despite pharyngeal packing and suctioning, pharynx and stomach may contain blood and irritant. Pt should be fully awake and capable of protecting airway to prevent aspiration following extubation.

### Usual Treatment
- Incidental (asymptomatic) microadenoma: Conservative
- Prolactin-secreting microadenoma: Bromocriptine or cabergoline (dopaminergic agonists)
- Somatostatin analogs (e.g., octreotide) usually effective in acromegaly
- Transsphenoidal resection is viewed as safe, and curative in ~90%
- Endoscopic approach generally less traumatic than traditional approach

### ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Acromegaly: Prognathism, lingual and laryngeal hyperplasia, mandibular enlargement</td>
<td>Mallampati class</td>
<td>Indirect laryngoscopy</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cushing's disease: ↑ BP Acromegaly: ↑ BP, cardiomyopathy</td>
<td>Exercise tolerance</td>
<td>Volume status, BP</td>
<td>ECG (± stress test) ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Sleep apnea</td>
<td>Truncal obesity, striae, moon facies</td>
<td>Serum cortisol; petrosal venous sampling of corticotropin; dexamethasone suppression test Serum GH and glucose suppression test Serum prolactin, glycoprotein, TSH</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Acromegaly: DM Cushing's disease: Hyperglycemia Prolactinoma: Infertility, amenorrhea, galactorrhea, impotence (male) Macroadenoma (usually due to a glycoprotein-secreting adenoma leading to panhypopituitarism by compression/atrophy)</td>
<td>Visual field cuts</td>
<td>Weakness</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Suprasellar compression of optic chiasm</td>
<td>Weakness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Acromegaly: Hypertrophy of facial bones and airway tissue Cushing's disease: Osteoporosis, truncal obesity, skin fragility</td>
<td>Formal visual field testing</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Key Reference

### Postoperative Period
- UO should be followed to detect onset of DI

### Adjuvants
- Esmolol and possibly phentolamine (during epinephrine infiltration)

### Anticipated Problems/Concerns
- Pts with hypersecretion of ACTH or GH at increased risk of myocardial injury if tight hemodynamic control not maintained during the transient, intense stimulations assoc with transsphenoidal surgery
Placenta Previa

**Risk**
- Incidence: 3.5–4.5 per 1000 births
- Highest incidence: Multiparous deliveries, repeat C-section, previous placenta previa, advanced maternal age

**Perioperative Risks**
- Maternal mortality: <1%
- Fetal mortality: ~20%
- Life-threatening hemorrhage of mother or fetus
- Fetal hypoxia

**Worry About**
- Blood loss, hypovolemia
- Full-stomach considerations due to pregnancy or recent oral intake

**Overview**
- Placenta accreta, increta, and percreta, predisposing to hemorrhage and possibly the need for hysterectomy
- Fetal compromise from inadequate intervillous blood flow
- Preterm labor

**Perioperative Implications**

### Preoperative Preparation
- Nonparticulate oral antacid premedication
- Assess volume status
- Crossmatch blood and consider transfusion
- Large-gauge IVs (2)
- Consider regional anesthesia if hemodynamically stable

### Monitoring
- Routine monitors
- Consider arterial and/or central venous catheter if hemodynamically unstable.

### Airway
- Airway edema may make intubation more difficult.
- Full stomach

### Preoxygenation/Induction
- Preoxygenate with four vital capacity breaths of O₂.
- Consider awake or rapid-sequence induction.
- Induction with thiopental or ketamine, depending on hemodynamics, plus succinylcholine

### Maintenance
- Low-concentration inhalational agent 0.5–0.75 MAC before delivery
- Use of NO before delivery of baby is controversial.
- Can use NO with IV opioid and consider benzodiazepine after delivery
- Restore intravascular volume

### Exutubation
- Exutubate awake; greatest risk is pulmonary aspiration of gastric contents

### Adjuvants
- Oxytocin, methylergonovine, prostaglandin F₂α to enhance uterine contraction and decrease bleeding after delivery

### Extubation
- None

### Anticipated Problems/Concerns
- Intrapartum and/or postpartum hemorrhage
- Full stomach
- Urgent induction of anesthesia
- Fetal distress

---

Pneumocystis Carinii Pneumonia (PCP)

**Risk**
- Resp infection in severely immunocompromised pts
- Pts with both acquired and congenital immunodeficiency syndromes
- Seen in all age groups
- Frequently assoc with pts with advanced AIDS, particularly if not treated with highly active antiretroviral therapy (HAART)

**Perioperative Risks**
- Resp failure often necessitating mechanical ventilatory support with high airway pressures
- Hemodynamic instability assoc with induction of anesthesia, positive pressure ventilation
- Pneumothorax
- Persistent expiratory airflow reduction after resolution of acute infection
- Bronchiectasis, lung cysts

**Worry About**
- Progressive resp failure
- Pneumothoraces, either spontaneous or assoc with positive pressure ventilation

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Oropharyngeal lesions</td>
<td>Fever, chills, sweats</td>
<td>Circumoral, acral, and mucous membrane lesions</td>
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</tr>
<tr>
<td>CARDIO</td>
<td>Intravascular volume deficits</td>
<td>Fluid intake, syncope, resp rate</td>
<td></td>
<td>Orthostatic BP changes</td>
</tr>
<tr>
<td></td>
<td>Cardiomyopathy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Cough, usually nonproductive</td>
<td>Tachypnea</td>
<td>ABGs</td>
<td></td>
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<tr>
<td></td>
<td>Progressive dyspnea</td>
<td>Breath sounds, prolonged expiratory phase</td>
<td>PFTs</td>
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<tr>
<td></td>
<td>Hemoptysis</td>
<td>Exam often normal</td>
<td>Gallium scan of lung</td>
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<tr>
<td></td>
<td></td>
<td>Heart sounds</td>
<td>LDH</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Hepatopathy</td>
<td>Often assoc with wt loss, other infections causing diarrhea, GI Sx</td>
<td>Hepatosplenomegaly</td>
<td></td>
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<tr>
<td>HEME</td>
<td>Anemia, leukopenia</td>
<td></td>
<td>CBC</td>
<td></td>
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<tr>
<td>RENAL</td>
<td>Nephropathy, oliguria</td>
<td>Oliguria</td>
<td>BUN, Cr</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>ENCEPHALITIS, meningitis</td>
<td>CNS changes</td>
<td>Abn mental status</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Ensure adequacy of oxygenation, ventilation, acid-base balance
- Assess pulm function, particularly expiratory phase of respiration
- Evaluate for evidence of other opportunistic infections
- Review CXR for evidence of infiltrates, abscesses, cystic lesions or cavitations, bullae, pneumothorax, effusions

**Monitoring**
- Confirm presence or absence of methemoglobinemia, if treated with sulfa drugs
- Interpret SpO2 with caution, if metHb present; measure SaO2 by co-oximeter

**Airway**
- Minimize airway pressures, TV
- Increased airway reactivity

**Induction**
- Maintain adequate PaO2
- Minimize airway pressures, risk of pneumothorax
- Hypotension assoc with myocardial depressants, vasodilators, positive pressure ventilation
- Ensure adequate intravascular volume

**Maintenance**
- Ensure adequate oxygenation, ventilation
- Minimize airway pressures
- Administer bronchodilators

**Extraventilation**
- May be delayed
- Prolonged ventilatory support often required

**Postoperative Period**
- Ensure adequate oxygenation, ventilation
- Minimize airway pressures using low TV ventilation

**Etiology**
- *Pneumocystis jiroveci* (previously *carinii*), originally characterized as a parasite, now classified as fungus
- Organisms reside in lungs, usually as latent infection; activated in immunosuppressed host
- High prevalence of antibodies to *Pneumocystis jiroveci* in immunosuppressed humans, suggesting that most are colonized early in life
- Human-to-human transmission has not been documented

**Usual Treatment**
- Trimethoprim/sulfamethoxazole (TMP-SMX)
- Pentamidine
- Primaquine
- Corticosteroids
- Prophylactic therapy with aerosolized pentamidine, oral TMP-SMX, or dapsone
- Supportive resp care

**ICD-9-CM Code:** 136.3

**Overview**
- Indolent disease that can progress to severe resp failure
- May be cause for non-productive cough in high risk pt
- High incidence of spontaneous pneumothoraces
- Extraventilation sites of infection rare, but should be considered in critically ill pt
- May be assoc with other infections (tuberculosis, bacterial, viral, fungal) and malignancies (Kaposi’s sarcoma, lymphoma) in immunosuppressed pts

**Perioperative Implications**

**Induction**
- Maintain intravascular volume; optimize myocardial function
- Continue anti-*Pneumocystis* therapy, consider other antiviral agents

**Anticipated Problems/Concerns**
- Deterioration of resp status, prolonged resp failure
- Pneumothorax; may require surgical repair if tube thoracotomy unsuccessful
- Nosocomial infections, assoc viral infections
- Difficulty monitoring oxygenation with pulse oximeter, if pt treated with dapsone, primaquine
- Drug resistance
Post Transplant Lymphoproliferative Disease

Risk
- 2–3% of all allograft organ transplants, highest risk in intestinal or multiorgan transplants
- Major risk factors are the degree and type of immunosuppression (OKT3, ATGAM induction or prolonged exposure to high doses of tacrolimus) as well as the EBV serostatus of the recipient (EBV negative recipients of EBV positive donor organs).
- Additional risk factors are the time after transplant (first year), recipient age (<25 y), and ethnicity (Caucasians).
- Overall survival rates ranging between 25–35%

Perioperative Risks
- Increased risk of airway or bowel obstruction and hematodinamic compromise
- Increased risk of dysfunction of the transplanted organs
- Increased risk for infection and CNS involvement

Worry About
- Enlarged tonsils and cervical adenopathy increasing difficulty of airway
- Thoracic adenopathy complicating intubation, ventilation, and cardiac output
- Pulm involvement causing decreased oxygenation and/or ventilation
- Dysfunction of the transplanted kidneys, liver, or heart
- GI involvement may manifest in N/V or bowel obstruction
- CNS involvement may manifest in mental status change or increased ICP
- Immunosuppression causing an increased rate of infection

Overview
- Lymphoproliferative disorders are among the most serious and potentially fatal complications of chronic immunosuppression in organ transplant recipients.
- These tumors are mostly B-cell type large-cell lymphomas. Extranodal involvement is occurring in 30–70% of the cases as a localized tumor in either the transplanted organ or another site, such as the GI system, lungs, skin, liver, and CNS.

ICD-9-CM Code: 202.8 (Non-Hodgkin’s lymphoma)

ASSESSMENT POINTS

<table>
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<th>Test</th>
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<td>Cervical adenopathy</td>
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<td>Lymphadenopathy</td>
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<td>Pharyngitis</td>
<td>Sore throat</td>
<td>Tonsillar enlargement</td>
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<td>Enlarged tonsils with pseudomembranous</td>
<td>Headache</td>
<td>Otitis media</td>
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<td>appearance</td>
<td>Facial pain, ear pain</td>
<td>Tenderness over sinuses</td>
<td>EBV</td>
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<td>Otitis media</td>
<td>Difficulty talking, breathing</td>
<td>Drooling, tripod position</td>
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<td>Sinusitis</td>
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<td>Difficulty of breathing</td>
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<td>Laryngeal edema</td>
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<td>RESP</td>
<td>Lung nodules</td>
<td>SOB</td>
<td>Decreased breath sounds</td>
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<td>Pleural effusions</td>
<td>Orthopnea</td>
<td>Crackles, egophony</td>
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<td>Hilar and mediastinal adenopathy</td>
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<tr>
<td>CARDIO</td>
<td>HF</td>
<td>SOB, tires easily Edema</td>
<td>New murmur, crackles</td>
<td>ECHO</td>
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<td>Liver dysfunction</td>
<td>N/V</td>
<td>Jaundice</td>
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<td>Bowel obstruction</td>
<td>Abd pain and discomfort</td>
<td>Abd distention</td>
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<tr>
<td></td>
<td>Bowel perforation</td>
<td>Distention</td>
<td>Tenderness over graft</td>
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<td>Tumors anywhere in GI tract</td>
<td>Swelling, tenderness over graft site</td>
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<tr>
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<td>Renal insufficiency or failure</td>
<td>Decreased UO</td>
<td>Pitting edema</td>
<td>BUN, Cr, electrolytes</td>
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<td>Crakles</td>
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<td>Mononucleosis syndrome</td>
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<td>Generalized lymphadenopathy</td>
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<td>EBV</td>
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<td>Sepsis</td>
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<tr>
<td>CNS</td>
<td>Brain tumors</td>
<td>Headache</td>
<td>Stupor, coma</td>
<td>CT, MRI</td>
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<tr>
<td></td>
<td></td>
<td>Loss of consciousness</td>
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<tr>
<td></td>
<td></td>
<td>Seizure</td>
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</table>


Perioperative Implications

Preoperative Preparation
- Difficult airway techniques and consider GE reflux precautions
- Evaluate the needs for blood products and specific antibiotics
- Evaluate the function of the transplanted organs
- Consider stress dose steroids if receiving steroids

Monitoring
- Consider invasive monitoring in organ failure or mediastinal mass.
- Consider ICP monitor as indicated for CNS involvement.

Airway
- Consider awake fiberoptic techniques if upper airway edema or masses or mediastinal masses

Preinduction/Induction
- Mediastinal mass can compress aorta and SVC, leading to significant hypotension if supine. Consider sitting or semi-sitting induction.
- Consider lower extremity for volume resuscitation if a large mediastinal mass

Maintenance
- Keep the pt breathing spontaneously in case of significant airway obstruction.
- If a mediastinal mass, keep in semi-sitting position and turn to lateral or prone position if hemodynamics become compromised.

Exubation
- Risk of airway obstruction if manipulated during surgery

Postoperative Period
- Airway edema can become a problem
- Continue stress dose steroids

Anticipated Problems/Concerns
- Airway obstruction and hemodynamic compromise
- Dysfunction of transplanted organs
- Mental status change or increased ICP in CNS involvement
Postoperative Encephalopathy, Metabolic

**Overview**
- Altered state of consciousness that becomes apparent in periop period
- Pts may fail to awaken after GA for these reasons: Anesthetic-induced: narcotics, inhalational anesthetics, benzodiazepines, hypnotics may impair consciousness; brain injury: direct surgical intervention (e.g., occlusion of major intracranial vessel, intracranial hemorrhage, edema) may result in impaired consciousness; or embolization to a major artery may occur (e.g., during or after cardiac surgery, interventional neuroradiology procedures)
- Metabolic abn: Circulatory failure, hypoxia, insulin use, hepatic and renal insufficiency; electrolyte abn in failure or slowness to awaken. In all cases, Dx should proceed quickly in order to treat underlying cause before severe brain injury results.

**ICD-9-CM Code: 348.3 (Encephalopathy)**

**Etiology**
- Anoxic-ischemic encephalopathy
- Hypercapnic encephalopathy (Paco, >70 mmHg)
- Hypoglycemic encephalopathy (glucose ≤30 mg/dL)
- Hyperglycemic coma (glucose ≥450 mg/dL; Osm ≥319 mOsm/mm³)
- Acute hepatic encephalopathy; Liver failure
- Uremic encephalopathy; Renal failure
- Other brain injuries: SIADH, seizures
- Lyte imbalance: Hypokalemia or hyponatremia, hypercalcemia
- Endocrine: Thyrotoxicosis, hypothyroidism
- Drug and/or toxin exposure (use a drug and/or toxicology screen)

**Usual Treatment**
- Depends upon the etiology—see Assessment Points

---

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Examples</th>
<th>Diagnosis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>ENDO</td>
<td>Hyperthyroid</td>
<td>Thyrotoxicosis</td>
<td>PTU Thyroid hormone replacement</td>
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<tr>
<td></td>
<td>Hypothyroid</td>
<td>Myxedema</td>
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<tr>
<td>ANOXIC-ISCHEMIC</td>
<td>Cardiac arrest</td>
<td>Obvious from clinical course</td>
<td>Reverse acute event</td>
</tr>
<tr>
<td></td>
<td>Prolonged shock</td>
<td></td>
<td>Then, ↓ cerebral edema, maintain BP, ↓, temp??, prevent seizures</td>
</tr>
<tr>
<td></td>
<td>Hypoxemia</td>
<td></td>
<td></td>
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<tr>
<td>HYPERCAPNIC</td>
<td>Narcotic-induced</td>
<td>† Heart rate and BP</td>
<td>Reverse narcotic</td>
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<td></td>
<td>Severe</td>
<td>† End-tidal or arterial Pco₂</td>
<td>Mechanical vent to ↓ Pco₂,</td>
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<td></td>
<td>COPD, sleep apnea</td>
<td></td>
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<tr>
<td>HYPOGLYCEMIC</td>
<td>Insulin overdose</td>
<td>No IVF or PO ingestion</td>
<td>IV glucose (D50)</td>
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<td></td>
<td>Ethanol ingestion</td>
<td>From Hx and alcohol level</td>
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<tr>
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<td>Neonatal (idiopathic)</td>
<td>↓ Blood glucose</td>
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<tr>
<td>HYPERGLYCEMIC</td>
<td>Hyperosmolar nonketotic coma</td>
<td>Suspect in known diabetic ketones in blood, urine Acidosis</td>
<td>Insulin, correct acidosis and fluid volume deficit</td>
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<td></td>
<td>Ketoacidosis</td>
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<td>ION DISTURBANCES</td>
<td>↓ Na⁺</td>
<td>Serum Na⁺ &lt;125 mmol/L (e.g., SIADH)</td>
<td>Hypertonic saline (caution)</td>
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<td>↓ K⁺</td>
<td>Serum K⁺ &lt;2.5 mEq/L</td>
<td>NaCl and diuretics</td>
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<td>RENAL</td>
<td>Renal failure</td>
<td>Severe muscle weakness</td>
<td>K⁺ replacement</td>
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<tr>
<td>HEPATIC</td>
<td>Hepatic encephalopathy</td>
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<td></td>
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</tbody>
</table>


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**Perioperative Implications**
- Correct ion and fluid disturbances
- Normalize blood glucose
- Optimize organ function (e.g., renal, hepatic)
- Adequate hormone replacement
- Search for drug/toxin exposure (sedative/hypnotics; ethanol and its street substitutes such as ethylene)
Prader-Willi Syndrome

Risk
- Prevalence: 1: 25,000.
- Incidence: 1:10,000–15,000
- Racial prevalence: None
- Gender predominance: Similar frequency in both sexes and all races
- Morbidity increases with 2° complications from obesity
- Annual death rate is 3% primarily due to resp arrest

Perioperative Risks
- Infantile hypotonia, hypoventilation, and breathing difficulty
- Potential difficult intubation and aspiration risk
- Worsening of obstructive/central sleep apnea and abn ventilatory responses to hypoxia and hypercapnia and bronchospasm
- Bradycardia, ventricular arrhythmias (PVCs)
- Postop resp insufficiency
- Potential risk of rhabdomyolysis with succinyl-choline
- Aberrant thermoregulation: Hyperthermia and MHS-like syndrome
- Glucose intolerance or DM

ASSESSMENT POINTS

System Effect Assessment by Hx PE Test
CRANIO-FACIAL ANOMALIES Facial dysmophasia, poor mask fit Snoring, nyustagmus, viscous and sticky saliva Dental crowding and caries Micrognathia, short neck with limited movement Imaging scans
CVS Htn Headache High diastolic BP EKG, CXR, renal function
Pulm Htn Exertional and at rest dyspnea Dyspnea, exertional intolerance Lung rales EKG, CXR
Cor pulmonale Dyspnea, exertional intolerance Tachypnea, orthopnea, systemic venous congestion, gallop sounds EKG, CXR, ECHO
Cardiomyopathy
RS Alveolar hypoventilation Increase airway responsiveness Increase work of breathing Snoring and interrupted sleep, day time somnolence, exertional dyspnea, wheezing Fatigue, limited upper airway access, short neck, limited mobility of neck PFT, room air ABG CXR Polysomnography for severe OSA
DIABETES TYPE I OR II Increased risk for CVS, CHF and autonomic dysfunction Hyperglycemia/hypoglycemia osmotic diuresis Dysfunction of CVS, renal and peripheral neuropathy Periop blood glucose Other test related to end-organ involvement


Perioperative Implications

Preoperative Preparation
- Only a well-supervised pt should be considered NPO
- Oral metoclopramide and cimetidine
- Assess airways difficulty, CV and pulm status
- Effective premedication to ensure a cooperative pt during awake/sedated intubation and induction of GA

Monitoring
- Standard ASA monitors. Consider direct intraarterial BP measurement if the non-invasive cuff does not fit. Continuous temp monitoring for instability
- Frequent ABGs, UO, and central venous or pulm artery pressure for major surgery

Airway
- Elective awake intubation if difficult airway is anticipated; increasing neck circumference, a Mallampati score of ≥3, micrognathia, and limited mouth opening

Worry About
- Abn of short and restricted neck mobility, limited mouth opening and difficult intubation
- Poor vascular access and intrapo positioning
- Systemic and pulm Htn, conduction defects RBBB and cor pulmonale. Dilated cardiomyopathy.
- Restrictive lung disease (obesity, kyphoscoliosis) and reactive airways

Overview
- Presents in two stages: Infantile central hypotonia, FTT, and delayed developmental milestones. Childhood stage presents with obesity (BMI > 97th percentile in a child and ≥30% in an adult), skeletal abn (dysmorphic, short stature, short hands and feet, scoliosis), hypogonadism, and hypothalamic dysfunction
- Restrictive pulm disease results from muscle weakness, obesity and kyphoscoliosis. It starts in early childhood and is present in 80–90% of pts over 30 y
- CV system: Htn in 17–32% and myocardial hypotrophic hypokinetic syndrome

Induction
- Gastric regurgitation due to delayed gastric emptying and hiatal hernia
- Be prepared to manage a situation where ventilation and/or intubation are not possible. The degree of obesity is one factor among others that makes glottis visualization problematic
- Semi-sitting position improves FRC and preoxygenation
- Slow IV induction with propofol and remifentanil or fentanyl with or cisatracurium to facilitate intubation

Maintenance
- Sevoflurane or desflurane with remifentanil infusion and cisatracurium. These inhaled agents are 1st line and allow rapid recovery from general anesthesia. No specific drug or combina tion is recommended; the aim is rapid emergence. Avoid long-acting opioid and substitute with IV NSAIDs.
- Regional anesthetic techniques are desirable alone or to supplement GA and provide postop analgesia to reduce the need for opioids

Exubation
- Decision is dictated by the severity of obesity, assoc risks such as OSA and the extent of surgical procedure. Early tracheal extubation is desirable

Adjutants
- Hydrophilic drugs (e.g., muscle relaxants are calculated by lean body mass). Lipophilic drugs (e.g., fentanyl) are calculated in mg/kg body weight

Postoperative Period
- Severe obesity is assoc with more atelectasis during, immediately, and for 24 hr longer after general anesthesia compared to non-obese pts. CPAP or BiPAP may be necessary to maintain patent airways, particularly during sleep and for those with severe OSA. High sensitivity to opioid-induced resp depression

Anticipated Problems/Concerns
- Monitor for OSA and alveolar hypoventilation in ICU/PACU. Monitor hyper- and/or hypoglycemia, hyperthermia, and arrhythmias
- Early ambulation and thromboembolic precautions

Central thermo dysregulation: May develop hyperpyrexia
- Cognitive problems: Mild–moderate mental retardation. Mean IQ 60s–70s; some have normal intelligence
- Behavior problems of oppositional behavior, emotional lability, aggressive and violent behavior, and obsession and compulsion to eat. Psychosis in 5–10% of adults
- High threshold for pain

ICD-9-CM Code: 759.81

Etiology
- A leading cause of genetic obesity, caused by paternally derived deletion of long arm of chromosome 15 at q1511–13. GH deficiency

Usual Treatment
- Early intervention and education: Physical, occupational, speech, and behavioral therapies
- Wt and dietary management; low calorie diet and regular physical therapy
- GH replacement therapy
- Nighttime CPAP ventilatory support for severe OSA

Preeclampsia

Risk
- 6–8% of all pregnancies
- Young, nulliparous, or multiparous with previous preeclampsia/eclampsia Hx, obesity
- May be increased with Hx of other microangiopathy (e.g., chronic Htn, diabetes, renal disease, SLE)
- Lower socioeconomic status; malnutrition; no prenatal care

Perioperative Risks
- Increased risk of fetoplacental or maternal deterioration necessitating (often operative) delivery
- Preeclampsia and eclampsia account for about 20% of maternal and perinatal deaths

Worry About
- Hypertensive crisis leading to intracerebral bleed or LV failure
- Increased interstitial volume leading to edema
- Maternal hypotension producing placental hypoperfusion
- Renal dysfunction progressing to acute renal failure
- Thrombocytopenia may contraindicate regional anesthetic
- Eclampsia (or seizure in a severely preeclamptic patient) necessitating difficult tracheal intubation

Perioperative Implications

Antepartum Management
- Optimize maternal perfusion while lowering systemic diastolic BP <110 mmHg
- Ensure therapeutic blood magnesium sulfate level
- Replenish intravascular volume
- Avoid aortocaval compression

Monitoring
- Consider intra-arterial catheter for extremes of BP
- Consider CVP or PA catheter for oliguria or puls edema
- Fetal heart monitoring

Airway
- Often difficult 2nd to edema
- Prepare for emergent airway

Postoperative Period
- Risk for developing pulm edema due to previous (appropriate) intravascular hydration
- Effective postcesarean analgesia beneficial in BP control

Anticipated Problems/Concerns
- Maternal Htn causes maternal morbidity/mortality; maternal hypotension causes fetoplacental hypoperfusion
- Eclampsia assoc with CNS residua

ASSESSMENT POINTS

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<td>Edema</td>
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<td>Systemic vasoconstriction</td>
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<td>Hepatic subcapsular edema</td>
<td>Epigastric/RUQ pain</td>
<td>Enlarged liver edge</td>
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<td>Thrombocytopenia</td>
<td>Easy bruising</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Petechiae, gingival bleed</td>
<td>LFT</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>↑ Capillary permeability</td>
<td>Wt gain</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Nondependent edema</td>
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<td></td>
<td></td>
<td>UO</td>
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<tr>
<td>CNS</td>
<td>Seizure</td>
<td>Headache</td>
<td></td>
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<tr>
<td></td>
<td>Intracerebral hemorrhage</td>
<td>Blurred vision</td>
<td></td>
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<tr>
<td></td>
<td>Cerebral edema</td>
<td>Seizure</td>
<td></td>
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</tr>
<tr>
<td>OB</td>
<td>↓ Placental perfusion</td>
<td>FHR—lack of variability or bradycardia</td>
<td>FHR monitoring</td>
<td>BPP</td>
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<tr>
<td></td>
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<tr>
<td></td>
<td>Placental abruption</td>
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<tr>
<td></td>
<td>Vaginal Bleeding</td>
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</tbody>
</table>


Overview
- Marked by Hm, proteinuria, edema
- Maternal vasoconstriction: Possibly leading to acute cardiorespiratory deterioration
- Proteinuria: Sign of deteriorating renal function and widespread endothelial damage
- Edema: Increasing total body water, proteinuria, Htn lead to increasing interstitial edema and decreasing intravascular volume
- Hematologic: Widespread endothelial damage often leads to thrombocytopenia,
- Epigastric/RUQ pain an ominous sign of liver subcapsular edema and possible rupture. Delivery should be urgently effected.
- HELLP (Hemolysis, Elevated Liver enzymes, Low Platelet count) a poor fetoplacental prognostic sign.
- Headache: seizure may be impending

ICD-9-CM Codes: 642.4 (Mild); 642.5 (Severe); 642.7 (With preexisting hypertension); 760.0 (Affecting fetus or newborn)

Etiology
- Acquired disease of unknown etiology
- Imbalance in circulating mediators of vascular tone and response (e.g., thromboxane vs. prosta-cyclin) from endothelial damage
- Pregnant pts who later manifest the disease have been shown to have demonstrated hyperdynamic CV response early in pregnancy compared with pts who do not go on to manifest disease
- Microangiopathy leading to endothelial change, plt consumption, hemolysis

Usual Treatment
- Prevention with daily low-dose aspirin beginning in 2nd trimester has had limited success
- Delivery becomes cure
- In-hospital therapy incl: Antihypertensives, seizure prophylaxis, and support of maternal perfusion, with magnesium sulfate (therapeutic blood levels = 5–7 mg/dL), and intravascular rehydration
- Analgesia, esp. epidural analgesia for labor, reduces catecholamine response to pain, increasing placental perfusion

Preinduction/Induction
- Epidural analgesia/anesthesia induces veno-dilation. Maintain maternal perfusion with judicious use of intravascular volume and (often) small, incremental prn doses of IV ephedrine, or low-dose phenylephrine, by bolus or infusion
- Rapid-sequence induction of anesthesia, titrating infusions of IV antihypertensive drugs or rapid-acting opioids to blunt pressure response to intubation

Maintenance
- Hemorrhage at delivery may lead to dramatic hypotension
- Titrte antihypertensive agents

Extubation
- Extubate awake, control pressure response

Adjuvants
- Magnesium sulfate for seizure prophylaxis and increased UBF; IV antihypertensive drugs (most often hydralazine, labetalol, nitroprusside, or nicardipine antepartum); rarely (but esp. in postpartum) dopamine to increase renal perfusion; finally, other inotropic support if demonstrable LV dysfunction

DISEASES

299
Pregnancy, Ectopic

**Risk**
- Implantation of fetus or blastocyst outside uterus
- Overall incidence, 1/90. More common in nonwhites in 35–44 y; ½ – ⅓ have no identifiable risk factors
- Risk factors incl pelvic inflammatory disease, IUD, tubal surgery, prior ectopic, tubal ligation, smoking.

**Perioperative Risks**
- Second leading cause of maternal mortality (leading cause in 1st trimester), accounting for 14.7% of all maternal deaths; nearly 2x greater in nonwhites
- 85% of deaths due to hemorrhage, 5% due to infection, 2% due to anesthetic complications
- Highest mortality assoc with intra-abdominal and interstitial tubal pregnancies, 2⁰ to larger size at time of Dx and therefore increased blood supply, and thus increased hemorrhage.

**Worry About**
- Hemorrhagic shock, decreasing intravascular volume
- Blood availability—that need type-specific or O neg blood
- Full-stomach/aspiration risk
- Consider physiologic changes of pregnancy if Dx made late in gestation, esp with intra-abdominal location (see Pregnancy, Intra-abdominal, in Diseases Section)

**Overview**
- Primary concerns with ruptured ectopic are intravascular volume, and airway management
- Approach similar to a trauma with profound hypovolemia
- Differential Dx of abd pain: Appendicitis, any intra-abdominal infection or process. Dx made by Hx and physical—95% have pelvic pain, 75% amenorrhea, 60–80% uterine bleeding.
- β-hCG—elevated in 100% of ectopics, US to rule out intrauterine pregnancy. Laparoscopy useful in Dxs of acute pelvic pain and to rule out ectopic.

**ICD-9-CM Code: 633**

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td></td>
<td>Snoring/difficult airway</td>
<td>Airway exam</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Hypovolemia 2⁰ to hemorrhage</td>
<td>Orthostatic dizziness</td>
<td>Vitals, neck veins, orthostatic vital signs</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Weak, thready pulse</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cold legs and arms of vasoconstriction</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Blood loss 2⁰ to rupture</td>
<td>Vaginal bleeding</td>
<td>Orthostatic vital signs</td>
<td>Hct</td>
</tr>
<tr>
<td></td>
<td>Hemoperitoneum/vaginal bleeding</td>
<td>Orthostatic dizziness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Hypoperfusion causing mental status changes and decreased urine production</td>
<td>CNS Hx</td>
<td>CNS exam</td>
<td>BUN/Cr</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>UA</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Assessment of volume status using clinical and laboratory measures
- 2 large bore peripheral IV’s
- Blood availability—at least O neg; type-specific preferable
- Consideration of full stomach

**Anesthetic Technique**
- GA: Preferable in unstable pt with ruptured ectopic; if laparoscopy to be used or contraindication to regional
- Regional anesthesia: Spinal or epidural T2–T4 level needed; consider in hemodynamically stable pts

**Monitoring**
- Routine; once ectopic bleeding stopped, fluid resuscitation for replacement only; too zealous replacement can lead to pulsw edema
- Consider arterial line placement in the hemodynamically unstable

**Airway**
- If difficult airway, awake fiberoptic; otherwise rapid-sequence induction and intubation

**Induction/Maintenance**
- If unstable, consider etomidate or ketamine, maintenance with O₂, inhalational, and narcotic with muscle relaxants
- Choice of drugs less important than anesthetic management

**Surgical Stages**

**Induction**
- Possible CV instability 2⁰ to uncorrected hypovolemia, as well as full-stomach/aspiration potential
- Skin incision
- Laparotomy for ruptured ectopic, hemoperitoneum and hypotension with uncontrolled bleeding. Upon opening abdomen, a release of tamponade may result in decreased BP.
- Incision: Pfannenstiel or low midline
- Laparoscopy: Infraumbilical and 1–4 supra-pubic incisions. Peritoneal insufflation: Monitor ETCO₂ and intraperitoneal pressures—should be <18 mm Hg. Potential for CO₂ embolus or intra-abdominal injury during introduction of the Veres needle.
- Dissection: Minimal to extensive depending on location of ectopic and degree of bleeding

**Definitive Surgery**
- Salpingectomy, ipsilateral oophorectomy—used for ruptured ectopic hysterectomy; may be necessary if interstitial implantation
- Salpingectomy
- Technique of fallopian tube conservation
- Can be performed via laparoscope; used to remove small ectopic <2 cm; preferred technique for unruptured ectopic
- Approximate duration: 1–2 hr
- Fluid shifts can be large with ruptured ectopic
- Closure: Minimal if laparoscopy; low midline or Pfannenstiel 15–20 min

**Exsufflation**
- Awake

**Worry About**
- Hemorrhagic shock, decreasing intravascular volume
- Blood availability—that need type-specific or O neg blood
- Full-stomach/aspiration risk
- Consider physiologic changes of pregnancy if Dx made late in gestation, esp with intra-abdominal location (see Pregnancy, Intra-abdominal, in Diseases Section)
Postoperative Period
- Blood loss may be extensive; check Hct
- Pain score: 4–6 laparoscopy, 5–8 laparotomy
- PCA or neuraxial narcotics; local anesthetics if regional ± neuraxial narcotics

Anticipated Problems/Concerns
- CV: Instability from massive hemorrhage from ruptured ectopic
- Potential for pulm edema, fluid overload in postop period due to massive crystalloid infusion and subsequent mobilization of third space fluid
- Postop shoulder and chest pain from unab sorbed gas and peritoneal irritation—30%
- Gastric dilation 3%, thrombophlebitis 3%, pulm embolism 2%, ureteral injury/stenosis 1% with laparotomy
- Postop infection, abscess
Pregnancy, Intra-Abdominal

**Risk**
- Incidence in USA: 11/100,000 live births
- Higher incidence in African-Americans, Asians, and immigrant populations from Third World countries
- Higher incidence following in vitro fertilization procedures
- Maternal mortality 100 times that of intrauterine pregnancy

**Perioperative Risks**
- Usually misdiagnosed at the time of laparoscopy or exploratory laparotomy
- Exsanguinating hemorrhage possible preop, intraop, or postop

**Worry About**
- Severe hemorrhage

---

**Overview**
- Pt usually has a normal early pregnancy and presents with midtrimester abdominal pain, N/V, shock, partial bowel obstruction, and vaginal bleeding
- Correct Dx is made preoperatively in approx 10% of cases
- Differential Dx includes abruptio placentae, placenta previa, pelvic inflammatory disease, and bowel obstruction. MRI is better than US diagnosis
- Exsanguinating intra-abdominal bleeding can occur at any time.
- Cases of twin fetuses, one intrauterine and one extrauterine, have been described. Perinatal survival 5–25%.

**ICD-9-CM Code**: 761.4

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**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV</td>
<td>Hemorrhage</td>
<td>Postural dizziness</td>
<td>Hypovolemia, hypotension</td>
<td>Hct</td>
</tr>
<tr>
<td>Gl</td>
<td>Bowel obstruction</td>
<td>N/V</td>
<td>GI bleed</td>
<td>Abd x-ray, CT, MRI, abd US falsely negative</td>
</tr>
<tr>
<td></td>
<td>GI bleed if bowel implantation</td>
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</table>

**CNS**
- Decreased consciousness if massive hemorrhage


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**Perioperative Implications**

**Preoperative Preparation**
- Assess volume status
- Fluid/blood resuscitation

**Monitoring**
- Arterial and central venous lines valuable if Dx known

**Airway**
- Rapid-sequence induction

**Induction**
- Rapid-sequence using ketamine or etomidate
- Two or three large venous access lines prior to induction

**Maintenance**
- Ensure vascular stability

**Extubation**
- May need to delay extubation for postoperative care
- Extubate awake

**Adjuvants**
- None

**Postoperative Period**
- May require intensive care if large fluid shifts or periop severe hypotension/hypoxia

**Anticipated Problems/Concerns**
- Hemorrhage, DIC
### Pregnancy, Maternal Physiology

**Risk**
- Incidence in USA: Estimated 6.4 million pregnancies resulting in 4.1 million live births per year
- Pregnancy rate of 103 pregnancies per 1000 women between the ages of 15 and 44 y

**Perioperative Risks**
- Maternal mortality rate: 15 deaths per 100,000 live births in USA, 400 deaths per 100,000 live births in the world
- Hemorrhage and embolic disorders are two leading causes of maternal deaths.
- Risks of maternal mortality incl advanced maternal age, obesity, multifetal pregnancies, cesarean delivery and African-American race.

**Worry About**
- Difficult airway incl inability to intubate and ventilate due to maternal wt gain, breast enlarge-
- and swelling of oropharyngeal tissues (inci-
ence of failed intubation 1:280 vs. 1:2230 in
nonpregnant patients)
- Hypoxemia occurs more quickly during periods
of apnea due to decreasing FRC and increasing \( O_2 \)
consumption
- Aortocaval compression causing decreased
uteroplaental perfusion and \( \Delta Hr \) late
decelerations
- Hypercoagulability causing DVT/PE
- Aspiration pneumonitis

### ASSESSMENT POINTS

<table>
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<th>Test</th>
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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Capillary engorgement/swelling of nasal and oral pharynx, larynx, trachea Vocal cords and arytenoid edema</td>
<td>Epistaxis, Voice changes, Difficult nasal breathing/congestion</td>
<td>Careful airway exam Temporomandibular distance Mallampati class Neck ROM</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>CO, SV, HR, ejection fraction ↑ SVR, BP ↓ 3rd and 4th heart sounds Systolic ejection murmur Tricuspid and pulmonic regurgitation Peripheral edema</td>
<td>Palpitations, Dizziness/pre-syncope</td>
<td>Auscultation of heart Pulse BP</td>
<td>EKG, ECHO, PA catheter (all rarely needed)</td>
</tr>
<tr>
<td>Resp</td>
<td>Tidal volume, resp rate ↑ FRC ↓ Minute and alveolar ventilation ↑ ( PaO_2 ), ( PaCO_2 ) ↓ Elevated diaphragm</td>
<td>Dyspnea</td>
<td>CXR, ABG, PFTs (all rarely needed)</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>↓ Lower esophageal sphincter tone ↓ Gastric emptying—only in labor ↓ Gallbladder emptying</td>
<td>GE reflux, Gallstones</td>
<td>RUQ US</td>
<td></td>
</tr>
<tr>
<td>renal</td>
<td>↑ RBF, GFR, Cr clearance ↓ Bicarbonate</td>
<td>↑ Drug clearance</td>
<td>J- BUN, Cr, bicarbonate</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>↑ ↑ Plasma volume, ↑ RBC volume ↑ Coagulation factors (I, VII, VIII, IX, X, XII), ↑ clotting ↑ Pt turnover, fibrinolysis ↓ Albumin, ( \alpha ), acid glycoprotein</td>
<td>Physiologic anemia, Leg pain, dyspnea Gestational thrombocytopenia Pale, nail beds Homan's sign for DVT</td>
<td>Hg, Hct PT/PTT, lower extremity Doppler, V/Q scan, spiral CT Pt count, TEG</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>↓ MAC, ↑ Pain threshold</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>↑ Insulin resistance Enlarged thyroid, ↓ TSH</td>
<td>Palpation of thyroid gland Glucose Normal free T3 and T4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>↑ Lumbar lordosis, ↑ Joint mobility</td>
<td>Back pain</td>
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</tbody>
</table>


**Perioperative Implications**

### Preoperative Preparation
- Large-bore IV, consider a second IV if pt is at increased risk for bleeding
- Consider use of nonparticulate antacids and metoclopramide to decrease gastric acid and volume
- Keep \( \Delta \) with left uterine displacement to relieve aortocaval compression
- Good oropharynx exam to assess likelihood of difficult intubation
- Recommend NPO 6–8 hr prior to elective surgery

**Monitoring**
- Routine

**Preinduction/Induction**
- Need access to difficult airway equipment incl FOB, LMA, and jet ventilation

**Maintenance**
- Use a short or stubby-handled laryngoscope, esp. in obese parturients
- Avoid nasal intubation due to increased risk of bleeding
- Preoxygenate with 100% \( O_2 \) at high-flow rates
- Use rapid sequence induction and cricoid pressure (Sellick maneuver) to decrease passive regur-
gitation of gastric acid into oropharynx
- ETT preferred to LMA to adequately protect against aspiration
- Pseudocholinesterase activity reduced, but recovery from succinylcholine usually not prolonged
- Decreased doses of induction agents needed
- Adjust ventilation to maintain \( PaCO_2 \) around 30 mmHg
- Decreased minimum alveolar concentration (MAC) for inhalation anesthetics.
- Avoid high dose inhalation agents due to uter-
- In atony.
- High doses of opioids and/or benzodiazepines given prior to delivery can cause resp depression in the neonate.

**Extubation**
- Awake without residual NM blockade

**Regional Anesthesia**
- Spinal, epidural, combined spinal-epidural all possible techniques and usually preferred over GA for surgical delivery, esp. in the obese pt or one with apparent difficult airway
- Decreased dose of spinal or epidural local anesthetic achieves same dermatomal level as higher doses in nonpregnant adults

**Overview**
- Physiologic changes occur during pregnancy to allow maternal adaptation to the demands of the growing fetus, supporting placental unit, and ult-
imately to facilitate labor and delivery.
- These changes affect almost every organ sys-
tem and influence the anesthetic and periop-
management of the pregnant woman.

**Etiology**
- Profound increases in hormonal concentra-
tions, esp. progesterone
- Mechanical effects of an enlarging uterus
- Increased metabolic demand
- Presence of the low resistance placental circulation

**Normal spontaneous vaginal delivery**
- C-section

**ICD-9-CM Code:** v22.2 (Pregnancy)

**DISEASES**
• Pharmacologic sympathectomy can cause severe hypotension at term
• Reduced response to vasopressors

**Postoperative Period**
• Pain can be treated with a combination of NSAIDs and preservative-free spinal or epidural morphine or PCA if GA is used
• Use compression stockings and early ambulation to lower the risk of DVT/PE
• Most physiologic changes of pregnancy resolve 6–8 wk postpartum

**Anticipated Problems/Concerns**
• Mallampati scores worsen during the progress of labor so the airway must be examined immediately prior to induction of GA
• Uterine artery blood flow 500mL/min at term so obstetric hemorrhage can become life-threatening very quickly
• Increased risk of C-section with obesity, a common and increasing problem
Pregnancy-Induced Hypertension

Susan K. Palmer

Risk
- (PIH) incidence not known because Dx may not be made in hospital discharge summary unless it progresses to preeclampsia (PE) and/or eclampsia (EC).
- PE may occur in 5% of all pregnancies, but is more frequent in some pt populations.

Perioperative Risks
- Htn remains a top cause of maternal mortality.
- Progression to PE and/or EC is unpredictable and may occur up to 7 d postpartum.

Worry About
- Decreasing IVF volume in pts with interstitial volume overload
- Hyperresponsive to endogenous and exogenous vasopressors
- Decreased uteroplacental perfusion despite raised maternal BP
- Edema in larynx and airway

Overview
- Blood-borne placental factors activate maternal vascular endothelium, which then directly affects vascular smooth muscle (VSM) tone/growth, causing Htn; endothelium also regulates the adherence and transmigration of WBCs (inflammation) and stimulates the aggregation of plts (coagulation cascade); endothelium controls access to the interstitium throughout the body (permeability, edema).
- PIH: BP >140/90 after 20 wk gestation in previously normotensive pt; BP must show this elevation at least twice >6 hr apart and not associ with uterine contraction
- PE: Above BP rise, plus evidence of other organ system involvement, e.g., proteinuria, nondependent edema, increased liver enzymes, decreased plt count, CNS dysfunction, low albumin
- Severe PE: Either BP >160/110 or proteinuria >5 g/24hr, or evidence of consumptive coagulopathy (DIC), or liver swelling and/or failure (epigastric or RUQ pain), or palm edema (desaturation), or evidence of CNS edema (severe headache)
- Eclampsia: PIH and/or PE plus seizure; can occur up to 1 wk postpartum

ICD-9-CM Code: 642.11

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Vasopasm, ↑ CO (usually) ↓ IVF</td>
<td>↓ Exercise tolerance ↓ UO</td>
<td>Is there dyspnea at rest? BP check frequently</td>
<td>UA, 24-hr output BUN, uric acid, Cr, albumin</td>
</tr>
<tr>
<td>RESP</td>
<td>Swelling, edema in airway “Hoarse” voice</td>
<td>Nasal obstruction?</td>
<td>Airway exam</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>↑ Hct, ↑ or ↓ plt ↓ Albumin ↑ BUN, Cr, uric acid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Oliguria Proteinuria</td>
<td>RUQ pain, jaundice</td>
<td>Oxytocin induction can cause IADHS ↓ UA, 24-hr quantified proteinuria</td>
<td></td>
</tr>
<tr>
<td>HEPATIC</td>
<td>↑ Enzymes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Cerebral edema</td>
<td>Anxiety, headache Hyperreflexia (DTR) Optic disc edema</td>
<td>Liver enzymes</td>
<td></td>
</tr>
</tbody>
</table>


Peripartum Implications

Preoperative Preparation
- PIH can go directly to EC, esp if BP is much higher than nonpregnant baseline. Before delivery, control BP using mostly vasodilators, since large doses of β-blockers raise intrauterine pressure, compromising fetoplacental circulation.
- Rapid-onset regional or general anesthesia may cause severe hypotension due to intravascular fluid deficit.

Monitoring
- Consider arterial line for BP management for severe PE or malignantly increasing BP
- Periop fluid challenge with plain balanced electrolyte solutions (with or without albumin) should increase renal output and improve fetal status. If no improvement, CVP/PA catheter may identify pts with low (<150% nonpregnant) cardiac output (CO) who need both vasodilation and cardiac contractility improvement.
- Mg2+ blood levels or repeated exam of DTRs necessary to prevent overdose in pts who may develop renal failure during labor

Airway
- Edema may obscure normal structures, making rapid-sequence intubation difficult or impossible
- Mask ventilation may be difficult if face, lips, tongue also swollen
- Awake, surface anesthetized, fiberoptic ETT tube placement recommended if airway is swollen or looks difficult. Avoid unopposed α-agonist in nasal spray because of systemic Htn.

Maintenance
- Pregnancy lowers MAC by 30% for all inhalation agents.

Post Delivery
- All inhalation anesthetics cause uterine relaxation. May require greater than normal oxytocin infusion to contract uterus after delivery.

Exubation
- Exubate only when awake and strong, because half of maternal aspirations occur during emergence. Ensure that the airway will not become obstructed by increased edema.

Adjuncts
- Nerve stimulator can be used to monitor Mg2+-potentiated effects of nondepolarizing muscle relaxants

Diuresis of interstitial fluid should occur
- Still at risk for progression to PE and EC for up to a wk

See also Eclampsia in Diseases section and Preeclampsia in Diseases section

Etiology
- Cauasion unknown, no animal models. Placental factors initiate maternal endothelial malfunction, which causes failure of normal CV adjustments needed for successful pregnancy and normal fetal growth.

Usual Treatment
- Control of BP, maintenance or improvement of uteroplacental perfusion, and prevention of seizures are primary goals.
- Epidural analgesia may relieve vasospasm and improve uteroplacental perfusion.
- Seizure prophylaxis can be accomplished with MgSO4, benzodiazepine, barbiturate, or phenytoin.
- Delivery of fetus and all of placenta usually, but not always, followed by amelioration of symptoms
Preterm Infant

Overview
- Incidence of prematurity continues to increase, but mortality has decreased due to use of surfactant, perinatal steroid administration, specialization of NICU, and improved mechanical ventilation strategies.
- Immature organ systems present many challenges to delivery of anesthesis care.
- Pulmonary: Low lung volumes, decreased compliance, lack of surfactant production. V/Q mismatches, barotrauma, O2 toxicity, and hypoxia.
- Long-term consequences: Bronchopulmonary dysplasia (BPD), chronic lung disease, reactive airway disease, O2 requirement.
- Immature resp control leads to poor response to hypoxia and hypercapria resulting in central apnea. Anatomic structures predispose to obstructive apnea. Increased risk of apnea with decreased gestational age and Hct <30%.
- Cardiac: CO dependent on HR. Structure of heart results in decreased ability to increase contractility, dependence on extracellular Ca2+, poor response to catecholamines. Small blood volumes, impaired autoregulation, and anesthetic blunting of baroreceptors increases risk of CV collapse. High frequency of PDA causing pulm Htn and CHF.
- CNS: Watershed regions (periventricular white matter) are prone to hypoxic-ischemic injury during hypotension, low CO, hypoxemia, and hypocapria. Incidence of IVH increased with pts with RDS, seizures, pneumothoraces, hypoxemia, acidosis, hypocapnia, use of vasopressor infusions. Long-term consequences of CNS injury include developmental delay, behavioral abn, hearing and visual deficits, and cerebral palsy. Management of stress responses and pain reduce physiologic stress.
- CNS: Recent animal data regarding neuronal cell apoptosis with exposure to anesthetic agents prompts considering avoidance or delay of elective surgeries in all infants.
- Retinopathy of prematurity (ROP) is common, incidence inversely proportional to birth wt and gestational age. Exposure to variations in arterial oxygenation and exposure to bright light play a role.

Risk
- Incidence in USA: <37 wk gestation, 12.8% of pregnancies; <34 wk gestation, 3.66%; <28wk gestation, 0.76% of all births in 2006.
- Prematurity frequently correlates with birth wt. As birth wt declines, mortality in the first year increases. Low birth wt births comprised 8.3% of total births.
- VLBW (<1500 g) represented 1.5% of births in 2006 in USA
- ELBW (1000–1500 g), make up 0.7% of U.S. births.
- Micropremie (500–750 g): 50% survival rate

Perioperative Risks
- Inadequate oxygenation and ventilation, atelectasis, pneumothorax, tube displacement, O2 toxicity, barotrauma.
- Hypotension, limited ability to compensate for hypovolemia, risk of CV collapse.
- Ductus arteriosus may shunt right to left with hypoxia, hypercapnia, hypervolemia, acidosis, hypothermia
- Hypoxic ischemic CNS injury due to poor perfusion and inadequate O2 supply
- Intraventricular hemorrhage (IVH) common
- Apnea and bradycardia increases after exposure to anesthetic gases and sedation.
- Bleeding due to inadequate coagulation factors, spontaneous liver hemorrhage, thrombocytopenia
- Immature liver function resulting in altered drug metabolism, reduced albumin
- Hypothermia
- Elevated stress response worsening co-morbidities
- Electrolyte disturbances due to immature renal function

Worry About
- Resp status: Reduced reserve, presence of chronic lung disease (CLD), postop apnea
- Volume status and/or presence of anemia
- Normalize electrolytes and glucose levels preop
- Coagulation status: Thrombocytopenia, coagulation factor levels
- Adequate vascular access and ability to place invasive monitors

ASSESSMENT POINTS

System	Effect	Assessment by Hx	PE	Test

HEENT	Difficult airway	Assoc with common difficult airway syndromes, Hx in NICU	Morphology of airway esp. jaw, evaluation of breathing mechanics (stridor)	Not typically required unless obvious abn, x-ray, head and neck CT, MRI

Intracranial bleed	Intracranial hemorrhage

ROP	Cranial US

REFSP	RDS/ BPD	Risk factors, use of supplemental O2 Hx/frequency of apnea	Intubated and/or ventilated Fever curves, increasing O2 requirement	Respir rate (<60 abn) Intercostal retractions Grunting Rales or rhonchi Absent/decreased breath sounds, SubQ emphysema	CXR Blood gases

Resp failure	Pneumonia	Pneumothorax

CVS	Hypovolemia	Hypervolemia	PDA	CHF	Viral signs chart Wk chart UO Inotropes infusing	HR (120–160), murmur Bounding pulses (PDA), BP normal Liver enlarged (CHF), edema: feet or eyelids	ECG, ECHO

Jessica Miller

DISEASES

ICD-9-CM Code: 765.x (Premature infant)

Etiology
- Multiple risk factors incl maternofetal endocrine activity, anatomic uterine factors, local or systemic inflammation, placental hemorrhage

Usual Treatment
- Optimization of care prior to surgery: Early surfactant use, antenatal glucocorticoids, appropriate nutrition, reduction of physiologic stress (handling, pain management).
- Minimizing inspired O2 concentration and peak insp pressures, utilization of PEEP, maintain oxygenation at lowest safe level (90-95%), maintain ventilation with reduced I:E to minimize air trapping and barotrauma.
- Judicious fluid and colloid administration. Transfusion when appropriate based on O2 supply and demand.
- Intracranial/intraventricular hemorrhage (IVH) incidence and severity reduced in pts receiving sedation with opioids, antenatal glucocorticoids, or indomethacin. Avoid rapid fluctuations in CBF, CBV, and cerebral venous pressure.

Continued
<table>
<thead>
<tr>
<th>Disease</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
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</thead>
<tbody>
<tr>
<td>Anemia</td>
<td></td>
<td>Precipitous delivery, placental bleeding</td>
<td>Tachycardia, hypotension, poor growth rate</td>
<td>CBC, retic count</td>
</tr>
<tr>
<td>Sepsis</td>
<td></td>
<td>Perinatal exposures</td>
<td>Recent change in physiologic status-activity, resp function, CV stability, peripheral perfusion</td>
<td>WBC and differential</td>
</tr>
<tr>
<td>Coagulopathy</td>
<td></td>
<td>Birth asphyxia (low factors)</td>
<td>Purpura, occult bleeding</td>
<td>Platelet count</td>
</tr>
</tbody>
</table>


### Perioperative Implications

#### Preoperative Preparation
- Reassess cardiopulmonary and volume status
- Correct metabolic status
- Treat coagulopathy (Vitamin K, FFP, cryo, plt)
- Warm the OR, prepare warming devices, maintain temp during transport
- NPO: 2-hr fast for clear fluids, 4-hr fast for breastfeeding and/or formula
- Assess IV access
- Determine location of surgery: ICU vs. OR
- Prepare appropriate dilute vasopressor solutions, IV calcium, epinephrine, atropine.

#### Monitoring
- Precordial or esophageal stethoscope
- Preductal and postductal SpO₂, ECG, BP cuff, temperature
- EtCO₂, arterial line for major procedures
- Make sure all monitors and/or lines are secure

#### Airway
- Large tongue, small mouth may lead to difficulty
- Anesthetize prior to intubation to avoid elevated ICP
- Prepare multiple sizes of ETT to account for possible subglottic stenosis

- With pre-existing ETT: Reassess position, suction to clear of secretions, position to minimize kinking

#### Induction
- Inhalational induction (uncommon except for outpatients with minor surgery): Careful titration of sevoflurane, monitor BP carefully, anticipate challenging IV access
- Alternatively, thiopental (1–2 mg/kg) plus fentanyl 5–10 μg/kg followed by relaxant
- In minor surgery: IV caffeine can reduce postop apnea

#### Maintenance
- Low concentrations of inhalational agent, fentanyl to control hemodynamics, paralytic to optimize surgical and ventilatory parameters, control ventilation but avoid hyperventilation
- BP is most reliable to indicate hypovolemia
- Infuse glucose at 4–6 mg/kg/min and monitor blood glucose levels with a glucometer
- Monitor hemoglobin if bleeding present. Administration of blood products frequently accompanied by IV Ca²⁺ due to myocardial dependence on extracellular Ca²⁺ and inability of liver to rapidly metabolize citrate. Monitor K⁺ levels carefully.

- Monitor acid-base status, lactate production. Lower threshold to treat metabolic acidosis due to myocardial sensitivity to acidosis, consider THAM versus sodium bicarbonate.

#### Extubation
- Longer recovery time due to drug metabolism. Likely to need slow weaning from resp support.

#### Postoperative Period
- Continue cardiorespiratory monitoring, temp maintenance, NICU care
- Minor surgery: Pts <56 wk PCA should be placed on apnea monitor and observed for 24 hr, need to be apnea free at least 12 hr prior to discharge. Anemia increases risk of apnea and/or bradycardia. Extreme caution in discharging formerly preterm infants <60 wk PCA to home if opioids required for pain control.

### Anticipated Problems/Concerns
- Risk of massive hemorrhage in major surgeries, can be difficult to obtain surgical control.
- Hypothermia
- Postop apnea
- Pain control necessary, monitor for affects on ventilation, hemodynamics
Protein C Deficiency

Risk
• Congenital deficiency: Homozygote is estimated at 1/500,000–1/750,000 live births. Occurs when gene coding for Protein C on both chromosomes #2 are affected.
• Heterozygote ~0.2–0.4% of healthy population; 2–5% of pts with DVT
• Acquired deficiency also seen

Perioperative Risks
• Pts with Protein C deficiency are at risk for venous thrombosis and pulm embolism (immobility, endothelial damage, and decreased blood flow during periop period may be triggers)

Worry About
• Increased incidence of thrombophlebitis and pulm embolism
• Thrombosis of other vessels, such as intracerebral and coronary arteries, can occur

Overview
• Protein C is a vitamin K–dependent protein found in blood and synthesized in liver.

ASSESSMENT POINTS

<table>
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<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Retinal vein thrombosis</td>
<td>Hx of vision problems</td>
<td>Ophthalmoscopic exam</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>MI Angina Peripheral arterial disease</td>
<td>Hx of MI, angina Peripheral vascular thrombosis</td>
<td>Peripheral pulses</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm embolism</td>
<td>Hx of previous pulm embolism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Mesenteric thrombosis</td>
<td>Hx of bowel infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombophlebitis</td>
<td>Hx (and family Hx) of thrombophlebitis, pulm embolism</td>
<td>Exam of veins in legs, evidence for lower extremity postthrombotic syndrome</td>
<td>Screen for hypercoagulable state: PTT, Protein C &amp; S; factor V Leiden; antiphospholipid antibody, antiithrombin 3</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal vein and artery thrombosis</td>
<td>Hx of renal problem</td>
<td>BUN/Cr</td>
<td>Urine protein</td>
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<tr>
<td>DERM</td>
<td>Necrosis</td>
<td>Cutaneous necrosis after warfarin is begun</td>
<td>Cutaneous necrosis</td>
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<tr>
<td>CNS</td>
<td>Intracerebral artery thrombosis</td>
<td>Hx of CVA, TIA</td>
<td>Neurologic exam</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
• Homozygotes and symptomatic heterozygotes: FFP and Protein C concentrates can be administered to increase Protein C levels.
• Warfarin can be stopped a few days before surgery to allow PT to return to normal range and heparin administered until surgery.
• Intermittent pneumatic compression stocking can be placed prior to induction of anesthesia.

Airway
• Some have suggested that the ETT cuff not be inflated to prevent tracheal venous thrombosis.
• In neonates, there should be an audible leak.

Preinduction/Induction
• Regional anesthesia may be preferable, if possible.

Maintenance
• Special attention can be paid to positioning to reduce venous and arterial stasis.
• FFP and/or Protein C concentrates should be given to pts with prior thrombotic manifestations and for prolonged operations.

Adjuvants
• Intermittent pneumatic compression stockings can be used.
• Postop heparinization should be started as soon as deemed safe.

Anticipated Problems/Concerns
• Increased risk of thrombosis, esp. thrombophlebitis and pulm embolism.
• When switching from heparin anticoagulation to warfarin, heparin should be continued until warfarin has achieved therapeutic effect to decrease risk of skin necrosis.

Charles Weissman
Pulmonary Atresia

**Risk**
- Pulm atresia with intact ventricular septum (PA/IVS) happens in 3% of all congenital heart diseases (CHD) and prevalence of 0.07/1000 live births.
- Pulmonary atresia with ventricular septal defect (PA/VSD) happens in 3.4% of all CHD and about 20% of all tetralogy of Fallots’ (TOF).
- Males are affected more than females.

**Perioperative Risks**
- RV failure (volume overload, pressure overload or both)
- Hypoxemia (metabolic acidosis)
- Myocardial ischemia

**Worry About**
- RV-dependent coronary circulation (rapid boluses of fluid through central line may precipitate myocardial ischemia)
- Maintaining a patent ductus arteriosus (continue prostaglandin infusion)
- Suicide RV (sudden release of PV obstruction leading to hyperdynamic RV and subpulmonic obstruction of RV outlet. Treatment: β-blockade)

**Etiology**
- Congenital

**ASSESSMENT POINTS**

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<tbody>
<tr>
<td>CARDIO</td>
<td>RV failure</td>
<td>SOB</td>
<td>Cyanosis</td>
<td>ECG—RA enlargement, ECG-QRS axis 30-90°</td>
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<tr>
<td></td>
<td>Hypoxemia</td>
<td></td>
<td>Metabolic acidosis</td>
<td>CXR—Left- or right-sided arch</td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis</td>
<td></td>
<td></td>
<td>CXR—↓ pulm vascular markings</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>ECHO—PV annulus size, flow across PV, TV size and function, infundibular atresia, ductus arteriosus, PA branches and size Cardiac cath—confirm ECHO findings and detect state of coronary blood flow, MAPCAs</td>
</tr>
<tr>
<td>RESP</td>
<td>↓ Pulm blood flow</td>
<td>SOB</td>
<td>Tachypnea</td>
<td>ECHO, cardiac catheterization</td>
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<tr>
<td>SYSTEMIC</td>
<td>Signs of RV failure</td>
<td>Hepatomegaly</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Key Reference: Lake CL; Booker PD. Pediatric cardiac anesthesia. 4th ed. 2003.

**Perioperative Implications**

**Preoperative Considerations**
- Catheterization suite intervention vs. surgical intervention
- Patenty of PDA
- Reduced pulm blood flow
- Avoid hypoxia and hyperventilation
- RV hypoplasia
- RV-dependent coronary circulation
- Type of shunt to be performed
- Which systemic artery is to be used for shunt? Don’t stick it during CVP attempts.
- Degree of hypoxemia, metabolic acidosis

**Monitoring**
- Standard ASA monitors
- A-Line
  - Umbilical artery if good trace
  - Radial artery: Opposite side of shunt
  - Could clamp (partially) subclavian artery: Same implication for pulse oximeter placement
- CVP for resuscitation drugs

**Usual Treatment**
- Prostaglandin E, infusion
- Systemic to pulm shunt
- Infective endocarditis prophylaxis
- Palliative therapy

**Surgical Treatment**
- PA with VSD (usually RV well developed):
  - Blalock-Taussig shunt (BTS) followed by VSD closure and valved conduit (Rastelli’s procedure)
  - If multiple MAPCAs present then unifocalization, followed by VSD closure and valved conduit (Rastelli’s procedure)
- PA with IVS (often under-developed RV and coronary artery obstruction):
  - Extent of coronary artery obstruction:
    - a) None: Radiofrequency perforation of inter-atrial septum and balloon dilatation, followed by staged BTS, Glenn shunt and total cavopulmonary connection (TCPC), and/or heart transplant.
    - b) Minimal: Staged surgery with BTS, Glenn shunt, TCPC, and/or heart transplant.
    - c) Severe: Heart transplant.

**Anticipated Problems/Concerns**
- Palliative surgery only
- Definitive procedure later, e.g., Fontan, Rastelli
- Hypoxemia progressing: Inadequate shunt/ductus closing
- Tachypnea worsening: Hypoxemia, laryngeal swelling, pneumothorax (small chest tube easily occluded)
Pulmonary Embolism

Risk
- Incidence in USA: 600,000/y
- No racial predilection
- Risk factors are those for deep venous thrombosis

Perioperative Risks
- Risk for hypoxemia and right heart failure
- Periop mortality of ~90% for acute thromboendarterectomy; of ~10% for chronic thromboendarterectomy
- Postop pulm embolism in up to 1% of surgical pts
- Pulm embolism accounts for 20–30% of deaths assoc with pregnancy

Worry About
- Recurrent pulm embolism (30% mortality if not treated)
- Right heart failure and CV collapse
- Hypoxemia
- Hemorrhage in pts on anticoagulants or thrombolytics

Overview
- Pulm embolism found in ~20% of autopsied pts
- Clinical presentation may range from asymptomatic to chest pain and hypoxemia to CV collapse depending on magnitude of the embolus
- Signs (tachycardia, tachypnea, calf swelling) and symptoms (dyspnea, pleuritic chest pain, calf pain) have low sensitivity and specificity
- Most pts have DVT (surgical pts may have pelvic vein thrombi).
- Dx involves a combination of clinical suspicion, ventilation-perfusion lung scan, evaluation of the deep venous system of the legs, and spiral lung CT scan; pulm angiogram rarely required
- Negative d-dimer test excludes Dx in selected pts

ICD-9-CM Code: 415.1

Etiology
- Acquired disease
- Risk factors present in almost all pts: age >40 y, obesity, malignancy, recent surgery, trauma, pregnancy, immobilization, estrogen use, prior Hx of DVT, hypercoagulable state (factor V Leiden, deficiency of Protein C, Protein S, or antithrombin III)

Usual Treatment
- Therapy decreases mortality from 30% to <5%
- Heparin (PTT 1.5–2.5 × normal) followed by warfarin sodium (INR 2–3) for most pts; LMWH and fondaparinux are at least as effective as dose-adjusted continuous IV unfractionated heparin
- Thrombolytic therapy for massive pulm embolism
- Vena caval filter if massive pulm embolism or if cannot receive anticoagulants
- Surgical or catheter thrombectomy in selected cases of acute massive pulm embolism
- Surgical thromboendarterectomy in selected cases of chronic thromboembolic pulm Htn

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>RV failure</td>
<td>Syncope</td>
<td>↑ JVP; RV heave</td>
<td>ECG, ECHO</td>
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<tr>
<td>RESP</td>
<td>Pulm infarction</td>
<td>Hemoptysis</td>
<td>Tachypnea</td>
<td>CXR, SaO₂, ABGs,</td>
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<td>V/Q ab</td>
<td>Chest pain</td>
<td>Pleural rub</td>
<td>Spiral CT scan,</td>
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<td>Pain from pleural irritation</td>
<td>Dyspnea</td>
<td>Wheezing</td>
<td>Pulm angio</td>
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<td></td>
<td></td>
<td>Orthopnea</td>
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<tr>
<td>CNS</td>
<td>Syncope</td>
<td>Syncope</td>
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<td>MS</td>
<td>Phlebitis</td>
<td>Hx DVT</td>
<td>Leg edema</td>
<td>Compression ultrasonography</td>
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<td>Leg pain</td>
<td>Inflammation</td>
<td>Impedance plethysmography</td>
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<td></td>
<td></td>
<td></td>
<td>Palpable cord</td>
<td>Venography</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Preop Rx with heparin/LMWH and sequential compression devices, which decrease incidence of periop DVT and PE
- If active DVT, consider preop vena caval filter.

Monitoring
- Consider PA catheter.
- TEE may demonstrate RV dysfunction and PA thromboembolism.

Airway
- None

Preinduction/Induction
- May develop hypotension due to RV failure

Maintenance
- Adequate preload essential to RV function
- Systemic vasoconstrictors for hypotension due to RV failure
- Inhaled vasodilators (NO, prostacyclin) for refractory RV failure

Extubation
- None

Regional Anesthesia
- Appropriate, esp. if compatible with continued anticoagulation

Postoperative Period
- Resume anticoagulation as soon as possible (or use IVC filter)

Anticipated Problems/Concerns
- RV failure with systemic hypotension may be initial presentation of PE or may develop with recurrent PE
- Consider PE in all postop pts with unexplained hypoxemia or hypotension
Purpura, Immune Thrombocytopenic (ITP)

Risk
- Rare
- Children, M:F ratio: 1:1
- Adults, M:F ratio: 2–4:1 (pregnancy 1/1000 deliveries, 5% of thrombocytopenia in pregnancy, esp. if present in first trimester)

Perioperative Risks
- Hemorrhage (mortality for splenectomy 1%, one-third of which related to bleeding)
- Infection and thrombocytosis postsplenectomy

Worry About
- Preop corticosteroids, immunosuppressives
- Splenectomy
- Hemorrhage (mucosal when plt <20,000 × 10^3/mm^3), severe risk (incl intracranial hemorrhage) with plt <10,000 × 10^3/mm^3).

Overview
- Acute, intermittent, or chronic immune-mediated thrombocytopenia (accelerated destruction with appropriate megakaryocyte response); dermal, mucosal, and CNS hemorrhage (most critical)
- Obstetric implications incl risk of transient neonatal thrombocytopenia

Etiology
- Antiplatelet IgG auto-antibodies target pH membrane glycoproteins. Leads to premature removal by spleen and RES.

ICD-9-CM Code: 287.3

evaluation
- Preop corticosteroids, immunosuppressives
- Splenectomy
- Hemorrhage (mucosal when plt <20,000 × 10^3/mm^3), severe risk (incl intracranial hemorrhage) with plt <10,000 × 10^3/mm^3).

Usual Treatment
- Corticosteroids: Initial therapy (1 mg/kg/d) with 30–60% response rates (up to 80% initially)
- IV immunoglobulin G (0.4–1 g/kg/d)
- Anti-D (if Rh+ pt) cheaper, easier than IV IgG
- Splenectomy: Defer as long as possible in children. Increasingly laparoscopic (requires disruption of spleen into bag before extraction to prevent splenosis). In chronic disease, indicated if steroids cannot be tapered or poor response to therapy. In acute disease, indicated for failed medical response and plt transfusion.
- Other second line: Azathioprine, vincristine, rituximab
- Platelets: Although have very short survival, may temporarily elevate pH count

Assessment Points

Perioperative Implications

Preoperative Preparation
- Consult with hematologist. Consider steroids, IV IgG ± anti-D to raise plt count
- Steroid supplement if already receiving
- Premedication: Avoid IM injections
- Pneumococcal, meningococcal vaccine (+ Haemophilus in children)

Monitoring
- Routine
- Protect pressure points and mucosal surfaces

Airway
- Avoid nasal ET intubation
- Careful instrumentation, esp. with plt count <50,000 × 10^3/mm^3

Induction
- Avoid hypertensive response to ET intubation, esp. with plt count <10-20,000 × 10^3/mm^3 (CNS risk)

Platelets
- If required, transfuse after splenic pedicle ligation. Intraop monitoring of plt function (e.g., thrombelastography) may be useful guide to replacement therapy (case reports).

Extubation
- As above: Care of mucous membranes and hemodynamic response

Adjuvants
- Although recombinant factor VIIa traditionally requires adequate plt count to facilitate generation of thrombin burst, case reports and in vitro data of successful use in pts with thrombocytopenia and ITP (20-30,000 × 10^3/mm^3).
- Individual analysis of risk-benefit for neuraxial technique, esp. in parturient. Authors recommend plt count range >50 – 100 × 10^3/mm^3 pt with reports of point-of-care testing (e.g., thrombelastography) in decision making. If time, consider steroids, IV IgG ± anti-D to raise plt count.

Postoperative Period
- Risks of thrombocytosis not as crucial as TTP

Anticipated Problems/Concerns
- Massive surgical hemorrhage
- CNS and airway hemorrhage

Purpura, Thrombotic Thrombocytopenic (TTP)

**Risk**
- Rare (1/1 million), adult, pregnancy may be predisposing factor, 80% 6-mo survival

**Perioperative Risks**
- Pt for splenectomy with failed medical therapy.
- Risks of microthrombi with CNS and renal dysfunction combined with thrombocytopenia

**Worry About**
- Preop drugs and therapies
- CNS, renal dysfunction
- Thrombocytopenia (although usual quantitative plt triggers do not apply)

**Overview**
- Severe microvascular occlusive disease characterized by thrombocytopenia, microangiopathic hemolytic anemia, multisystem organ involvement (particularly CNS and kidney), considered part of spectrum of disease with hemolytic uremic syndrome (HUS)

**ICD-9-CM Code: 446.6**

**Etiology**
- Assoc with ultra-large von Willebrand factor (vWF) proteins (congenital or acquired deficiency of protease to cleave large vWF), which promote plt aggregation and endothelial damage, all leading to thrombotic occlusion of microcirculation.
- May be assoc with ticlopidine, malignancy
- Has been described post-cardiac, vascular, and abd surgery

**ASSESSMENT POINTS**

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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Airway manipulation, potential hemorrhage</td>
<td>Baseline MAP for perfusion CNS/kidney Vascular access</td>
<td>ECG</td>
<td></td>
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<tr>
<td>CARDIO</td>
<td>Rare conduction pathway involvement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Rare infiltrates causing hypoxemia</td>
<td></td>
<td>CXR</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Proteinuria, hematuria, ARF &lt; common than HUS</td>
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<td>BUN, serum Cr, urine sediment</td>
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<tr>
<td>HEME</td>
<td>Thrombocytopenia</td>
<td>Hemorrhage Petechiae Jaundice</td>
<td>Plt 8000–44,000 × 10^3/mm^3; PT, PTT, fibrinogen, antithrombin usually normal. Fragmented RBCs (Hgb 8–9 g/dl); ↑ LDH, bilirubin</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Fluctuating course</td>
<td>Spectrum—headache, seizures, coma</td>
<td>Lumbar puncture, EEG, neuroradiology studies</td>
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</tr>
<tr>
<td>OB</td>
<td>May precipitate episode or relapse</td>
<td>Differentiate from HELLP/PIH (&gt; CNS, &lt; hepatic involvement, not improved post partum)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Key References:** Kam PCA, Thompson SA, Liew ACS. Thrombocytopenia in the parturient. *Anaesthesia*. 2004;59:255–264.


**Perioperative Implications**

**Preoperative Preparation**
- Steroid supplement if receiving
- Premedication: Not IM, caution with CNS involvement
- Pneumococcal, meningococcal (*Haemophilus* for children) if for splenectomy

**Monitoring**
- Protect skin, mucous membrane (NIBP cuff, esophageal probe, pressure points)
- Usually have central access for plasma exchange
- If required, avoid subclavian if possible (difficulty compressing hematoma).
- Theoretical risk of radial arterial line with thrombotic process

**Usual Treatment**
- Parity of RCTs, combination of therapies:
  - Plasmapheresis (exchange): More NB than plasma infusion primary therapy (plt-poor FFP) usually with rapid clinical response
  - Plt aggregator inhibitors once plt count >50 × 10^3/mm^3 (aspirin, dipyridamole)
  - Steroids: Adjutant (methylprednisolone 1 g/day)
  - Immunosuppression second line: Vincristine (risk of neurotoxicity, abn ADH secretion)
  - Splenectomy: For failed medical response, prevention relapse
  - Avoid plt transfusion

**Airway**
- Avoid nasal ETT. Careful instrumentation, esp. if plt count <50,000 × 10^3/mm^3.

**Induction**
- Avoid sympathetic intubation response (CNS disease spectrum), maintain MAP > CNS, renal autoregulatory thresholds (>50–60 mmHg)

**Maintenance**
- Theoretical advantage of inhibitory effect volatile anesthetics on plt aggregation

**Fluids**
- Do NOT transfuse plt unless life-threatening thrombocytopenia: Reports of deterioration due to further microthrombi
- Bleeding managed with RBCs (>48 hr old to avoid active plt) and FFP (plt-poor)

**Extubation**
- As above: Care of mucous membranes and hemodynamic response

**Adjuvants**
- Individual analysis of risk benefit for neuraxial technique in thrombocytopenic pt (if in remission)

**Postoperative Period**
- Mobilize early: Precipitous increase in plt count and viscosity with risk of thrombotic events

**Anticipated Problems/Concerns**
- Hemorrhage if life-threatening thrombocytopenia (no plt transfusion until then)
- Microthrombi with CNS dysfunction
**Pyloric Stenosis**

**Risk**
- Incidence: 1/300–1/1000 of all live births
- Children of affected parents have higher incidence (3–5%)
- Male predominance

**Perioperative Risks**
- Similar to other abd procedures in pts of same age
- Some association with GU anomalies
- Some have elevated unconjugated bilirubin related to decreased glucuronyl transferase activity; returns to normal after correction of stenosis

**Worry About**
- Full stomach. Recurrent emesis leads to dehydration, electrolyte imbalance, and alkalosis
- Typically hypochloremic, hyperkalemic metabolic alkalosis

**Overview**
- Reduced size of gastric outlet impedes emptying of contents, which can cause abn nutrition, gastric distension, repeated vomiting, and dehydration
- Onset of symptoms 3–6 wk of age
- Usually surgically cured

**ICD-9-CM Code: 750.5 (Congenital)**

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>Gastric outlet obstruction</td>
<td>Nonbilious projectile emesis</td>
<td>Pyloric &quot;olive&quot; palpable in upper abd</td>
<td>Contrast study Abd us</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Correct fluid and acid-base deficits
- Pyloric stenosis is not a surgical emergency

**Monitoring**
- Routine

**Airway**
- Full stomach

**Preinduction/Induction**
- IV atropine 0.02 mg/kg/min dose 0.15 mg
- Empty stomach with orogastric tube or suction catheter
- Consider awake intubation or IV rapid-sequence induction, esp. if pt received barium contrast.
- Hypoxemia is common during rapid-sequence induction; ventilate with cricoid pressure.

**Maintenance**
- No technique is absolutely contraindicated by pyloric stenosis alone.
- Inhalational agent in O₂ and air or N₂O, short or intermediate-acting muscle relaxant
- Avoid opioids
- Local infiltration with bupivacaine orropivacaine by surgeon
- IV fluids should be warmed
- Replacement fluids: LR 1–2 mL/kg/hr
- May consider using D5W, if the procedure lasts more than 1 hr

**Exubation**
- Potential of full stomach; suction stomach prior to exubation
- Reverse NM blocking agent
- Awake exubation
- Delayed awakening is common

**Adjuvants**
- Consider potential of assoc liver and GU abn

**Postoperative Period**
- Potential for central apnea and reactive hypoglycemia
- Pulse oximetry/apnea monitoring for the first 12–24 hr
- Continue IV glucose until adequate PO intake
- Pain score: 2–5, acetaminophen is usually sufficient

**Anticipated Problems/Concerns**
- Potential for full stomach
- Need to correct fluid and/or electrolyte imbalances preop
- Delayed awakening is common
Q Fever

Risk
- Greatest after direct or indirect exposure to infected cattle, sheep, or goats; particularly at parturition
- Less from a variety of other animals, rarely from blood products
- Abattoir workers, veterinarians, and other animal workers at greatest risk
- Patients with immune impairment are at a higher risk (e.g., HIV, steroids)
- Mortality 2.4% overall, chronic infection ~16%

Perioperative Risks
- Decreased resp reserve 2° to pneumonia
- Decreased myocardial reserve 2° to endocarditis
- Further increase in hepatic cellular injury if there is liver involvement

Worry About
- 2° resp complications
- Decreased myocardial performance and emboli with endocarditis
- Hepatic or neurologic involvement

Overview
- Acute infection: Asymptomatic (~50%) to moderate severity (2% hospitalized)
- Acute symptomatic disease presents as non-specific febrile syndrome ± pneumonitis (~50%), hepatitis (80%), pericarditis and/or myocarditis (~5%), neurologic disease (~5%)
- Chronic disease occurs in <1% of infections, usually without fever
- Chronic disease, primarily endocarditis (particularly abn or prosthetic valves) and occasionally bone

ICD-9-CM Code: 083.0

Perioperative Implications

Preoperative Preparation
- Continue or initiate antibiotic therapy and optimize any organ system dysfunction.
- Only emergency surgery should be performed.
- Assess resp and cardiac reserve and hepatic and neurologic status.
- With chronic Q fever, subacute endocarditis prophylaxis may be appropriate.
- Monitoring
- Arterial line may be necessary if pneumonia present.
- Myocardial valvar disease may require PA line or other invasive hemodynamic monitors.
- Increased arterial line complications due to vasculitis (rare)

Postoperative Period
- Monitor resp and/or myocardial status carefully, ICU monitoring may be required.
- Liver enzymes should be followed if hepatic involvement.

Anticipated Problems/Concerns
- Emergent surgical pts who present with an acute infection might require extended antibiotic therapy to prevent persistent C. burnetii infection.

Etiology
- Coxiella burnetii is a fastidious, obligate, intracellular bacterium
- Spore stage can withstand harsh environmental conditions for prolonged periods, facilitating indirect transmission
- Highly infectious (1–10 organisms) primarily by inhalation, unpasteurized milk, or tick bite
- Incubation period ~20 d (range, 3–40 d)
- Bacterium targets reticuloendothelial cells, and develops into granuloma

Usual Treatment
- DX: Epidemiologic circumstance and serology (positive in 2–4 wk)
- Acute disease: Doxycycline or quinolones for 2–3 wk hastens resolution
- Chronic disease: Doxycycline and rifampin for 1–3 yr, ± valve replacement with endocarditis


DISEASES

Preoperative Preparation

Airway
- None

Induction
- Pneumonia may cause rapid desaturation.
- Hypotension and CV instability if cardiac valvar injury present

Maintenance
- If acute hepatitis, avoid drugs that require hepatic metabolism or decrease blood flow to liver

Extubation
- Resp status and CV stability need to be considered.

Adjuvants
- Depends on hepatic or renal impairment
Raynaud’s Phenomenon

Stephan J. Cohn

Risk
- 1.9% of population (based on reporting of color changes on exposure to cold)
- Almost all with disease are 15–40 y; almost all with 2° phenomenon are over 40 y
- Often assoc with scleroderma, SLE, and/or primary pulm Htn

Perioperative Risks
- Rare morbidity

Worry About
- Arterial thrombosis
- Low blood flow states (e.g., prolonged hypotension or use of tourniquet) can lead to gangrene of extremities

Overview
- Abnormal sensitivity of small arteries and arterioles to vasoconstrictive stimuli
- Often manifested in a bilateral symmetric pattern, with hands being affected more often than feet
- Pts exhibit triphasic color pattern in affected areas: Pallor, then cyanosis due to small arterial occlusion, followed by erythema and edema as vessels suddenly reopen

ICD-9-CM Code: 443.0

Etiology
- Unknown
- Likely hypothesis: Hyperactive sympathetic nervous system with excess neurotransmitter and/or little or no inactivation of norepinephrine

Usual Treatment
- Prevention is most effective. Avoid prolonged exposure to cold, avoid cigarette smoking.
- IV regional blocks with lidocaine at regular intervals. Reserpine, bretylium, and guanethidine all used as additives to lidocaine in IV regional blocks.
- In severe cases, surgical sympathectomy an option but not always beneficial (see also Systemic Lupus Erythematosus in Diseases section)

Perioperative Implications

Preoperative Preparation
- Keep warm
- Assess for co-existing disease

Monitoring
- Assess risk-benefit ratio if considering arterial cannulation because of danger of thrombosis.

- Monitor pt’s temp and check pressure points and distal pulses frequently.
- Airway
- Reduced TMJ mobility if assoc with scleroderma

Induction
- General or regional anesthetic options acceptable

Maintenance
- Use of tourniquet controversial

Adjuvants
- When using regional anesthetic, consider avoiding epinephrine.

Postoperative Period
- Keep as warm as possible.
- Check pulses in all extremities.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Assoc with primary pulm Htn</td>
<td>Chest discomfort DOE Weakness</td>
<td>JVD Pulmonic ejection click</td>
<td>CXR—right cardiomegaly, dilated pulm artery, ECG—right atrial enlargement, renal vascular Htn</td>
</tr>
<tr>
<td>MS</td>
<td>Impaired joint mobility due to pain or scleroderma</td>
<td>Joint mobility</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VASC</td>
<td>Small arterial occlusion</td>
<td>Triphasic color pattern</td>
<td>Often assoc with numbness and diaphoresis</td>
<td></td>
</tr>
</tbody>
</table>

**Reflex Sympathetic Dystrophy (Complex Peripheral Pain Syndrome)**

**Risk**
- Incidence of 5.5 per 100,000 person years at risk
- Prevalence of 21 per 100,000 person years at risk
- M:F ratio; 2–4
- Mean peak ages 37–50 y
- CRPS I incidence 1–2% post fractures; 12% post brain lesions; 5% post MI
- CRPS II incidence 1–5% post peripheral nerve injury

**Perioperative Risks**
- Increased pain flare post if procedure on affected extremity
- Increased tolerance and/or requirements of opioids if managed with chronic opioids
- Increased incidence of co-morbid anxiety, depression

**Overview**
- Spontaneous, intractable, burning pain; allodynia; hyperalgesia
- Edema, autonomic (vasomotor/sudomotor) anhidrosis, trophic signs

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>DERM</td>
<td>Skin/hair/tail changes</td>
<td>Changes in limb appearance</td>
<td>Thickened/thin skin</td>
<td>Serial physical exams</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increased/decreased hair growth</td>
<td>Glossy, waxy skin</td>
<td>Comparative exam photos</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increased/decreased hair growth</td>
<td>Increased/decreased hair growth</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Thinened/brittle nails</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Muscle mass/strength change</td>
<td>Subjective weakness</td>
<td>Muscle atrophy</td>
<td>3 phase bone radiograph</td>
</tr>
<tr>
<td></td>
<td>Stiffened joints</td>
<td>Decreased ROM</td>
<td>Objective weakness</td>
<td></td>
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<tr>
<td></td>
<td>Bone changes/osteoporosis</td>
<td></td>
<td>Decreased active/passive</td>
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<td></td>
<td></td>
<td></td>
<td>ROM</td>
<td></td>
</tr>
<tr>
<td>PNS</td>
<td>Spontaneous pain</td>
<td>Spontaneous pain</td>
<td>Alloynia</td>
<td>Quantitative sensory testing</td>
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<tr>
<td></td>
<td>Alodynia/hyperalgies</td>
<td>Pain to non-noxious stimuli</td>
<td>Mechanical/thermal</td>
<td>(thermal/thermal/pain/vibratory)</td>
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<tr>
<td></td>
<td>Motor changes</td>
<td>Exaggerated pain to noxious stimuli</td>
<td>Hyperalgies</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Tremor/dystonia</td>
<td></td>
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<tr>
<td>ANS</td>
<td>Vasomotor/sudomotor abn</td>
<td>Hyper/hydrosis</td>
<td>Moist, clammy, cool skin</td>
<td>Qsart</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Temp changes</td>
<td>Edema, skin color changes</td>
<td>Infrared thermometry/thermography</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Swelling</td>
<td>Skin temp asymmetry of limbs</td>
<td>Thermoregulatry sweat test</td>
</tr>
</tbody>
</table>

**Etiology**
- Pathogenesis of CRPS is unknown
- Classically assoc with antecedent trauma, surgery, MI, stroke
- Likely involvement of peripheral, autonomic, and central nervous systems; myofascial dysfunction; altered psychological states
- Many proposed mechanisms
  - Abn sympathetic outflow and/or adrenergic receptor sensitivity
  - Abn spinal and/or central neuronal sensitization
- Abn and/or exaggerated inflammatory process
- Hypoxia
- Psychologic and/or psychogenic factors
- Genetic predilection with HLA-DR/DQ polymorphisms

**Perioperative Implications**

**Preoperative Preparations**
- Preop PE and notation of pain symptoms, location and neurologic and/or MS deficits
- Careful planning of pt positioning
- Consider combined regional/GA for periop and postop pain control
- Detailed plan for postop pain control strategies

**Monitoring**
- Standard ASA monitors
- Avoid BP cuff, pulse oximetry, other monitors on affected extremity

**Induction**
- Possible increased induction agent dosage (chronic opioid pts)
- Consider regional blockade and/or catheter infusion

**Maintenance**
- Possible increased anesthetic and periop opioid requirements (chronic opioids)
- Diligent assessment of affected limb position and temp

**Adjuncts**
- Consider pain medicine consultation if pt admitted postop

**Usual Treatment**
- Early DX and multidisciplinary treatment assoc with best outcomes
- Physiotherapy most important component of rehabilitation to achieve optimal functional restoration
- Psychological intervention, cognitive-behavioral therapy
- Typical first line oral medications incl:
  - Antidepressants
  - Antiepileptics
  - NSAIDs
- Long-term, chronic opioids controversal
- Oral corticosteroids employed if prominent inflammatory component
- Other adjuvant, second line therapies incl:
  - NMDA antagonists (ketamine, memantine)
  - GABA agonists (intrahecal baclofen)
  - Bisphosphonates
  - Free radical scavengers (DMSO, NAC)
  - Alpha-2 agonists (epidural clonidine)
  - Interventional therapies incl:
    - Sympathetic ganglion blockade
    - Chemical/surgical sympatholysis
    - Regional IV infusion therapy (lidocaine, reserpine, guanethidine)
    - Neurostimulation (SCS, TENS, deep brain stimulation)

Renal Failure, Acute (ARF)

**Risk**
- Incidence in USA: 1% of all hospital admissions (community-acquired), 5% of all general hospital pts (hospital-acquired), 10–30% of ICU pts
- Population with highest prevalence: Elderly (>65 y)

**Perioperative Risks**
- Overall mortality of periop ARF: 60–90%
- Hyperkalemia (and arrhythmias), metabolic acidosis, acute pulmonary edema
- Aspiration
- Bleeding (plt dysfunction)

**Worry About**
- Metabolic acidosis and hyperkalemia (pH decrease of 0.1 causes K⁺ increase of 0.5 mEq/L)
- Ventricular arrhythmias (may occur without warning)
- Encephalopathy (aspiration risk, increased sensitivity to all sedatives and anesthetics)
- GI symptoms and aspiration (N/V, bleeding and encephalopathy)

**Assessment by Hx**
- Elective surgery is contraindicated with new-onset ARF; procedures are urgent or emergency
- Regional anesthesia relatively contraindicated (plt dysfunction, encephalopathy)
- Repetitive hematologic insufficiency/markedly impairs renal recovery
- Dialysis partially controls thrombocytopenia and enteropathy, but does not decrease risk of sepsis and poor wound healing.

**ICD-9-CM Codes**: 584 (Acute); 977.5 (Due to procedure)

**Etiology**
- Ischemic ATN
  - Shock (hemorrhagic, cardiogenic, septic)
  - Nephrotoxic ATN
- Coagulopathy (plt dysfunction) and surgical bleeding
- Hemodynamic intolerance of hemodialysis, peritoneal dialysis compromises FRG

**Overview**
- Preoperative Preparation
  - Dialysis to control fluid overload, hyperkalemia, metabolic acidosis, acute uremia
  - Consider metoclopramide, H₂-blocker, rapid sequence induction to reduce refluid risk
  - Consider DDAVP 0.3 µg/kg to enhance plt function (effective 8–12 hr)
  - Regional techniques may be contraindicated by coagulopathy

**Monitoring**
- ECG for arrhythmia detection
- Consider PA catheter or TEE for large fluid shift operations with or without LV dysfunction

**Airway**
- Consider awake intubation with airway edema
- Avoid nasal intubation (epistaxis)
- Treat as for full stomach: Head up, cricoid pressure
- Succinylcholine is relatively contraindicated (avoid if K⁺ conc ≥ 5.0 mEq/L)

**Preinduction/Induction**
- Manage induction and/or replacement fluids as if renal function were normal (risk of hypovolemia)
- Anticipate enhanced pharmacodynamic effects of all sedative and/or anesthetic agents (encephalopathy)

**Maintenance**
- Restrict maintenance fluids; replace losses appropriately guided by hemodynamic monitoring
- Avoid morphine, meperidine, pancuronium
- Consider agents independent of renal elimination (volatile anesthetics, propofol, fentanyl, remifentanil, cisatracurium, esmolol, clevidipine)
- Increase minute ventilation to compensate for metabolic acidosis; sedative-hypnotic administration may lead to acidosis by eliminating compensatory resp alkalosis in spontaneously breathing pt
- Anticipate increased volume of distribution but decreased clearance of most drugs
- Check ABGs, serum K⁺

**Exubation**
- Anticipate delayed emergence, vomiting, aspiration
- Treat as for full stomach
- Neostigmine elimination is delayed in ARF

- Consider short period of postop mechanical ventilation if pt has intraop acidosis (will not be able to generate adequate spontaneous resp compensation)

**Postoperative Period**
- Careful assessment of CV, resp status; check ABGs, serum K⁺
- Morphine, meperidine have active metabolites that are renally excreted: Use with caution
- May require ultrafiltration and/or CVVHD for excess fluid and/or hyperkalemia in early postop period

**Anticipated Problems/Concerns**
- Major concerns are always hyperkalemia, acidosis, pulm edema
- Hyperkalemic arrhythmias may occur without premonitory ECG signs.
- Rapid K⁺ flux more ominous than stable high serum K⁺

**Usual Treatment**
- Treat underlying cause (shock, rhabdomyolysis, etc.)
- Medical therapy
  - Fluid and electrolyte restriction, loop diuretics
  - Hyperkalemia: beta-adrenergic agonists, hyperventilation, bicarbonate, Ca²⁺, insulin-glucose, kayexalate enema
  - Dialysis (renal replacement therapy [RRT]; periop continuous venovenous hemodialysis [CVVHD] is treatment of choice

**ASSESSMENT POINTS**

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Edema</td>
<td>Coagulopathy</td>
<td>Epistaxis, GI bleeding</td>
<td>Airway edema</td>
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<td></td>
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<td>Syncope, cardiac arrest</td>
<td>Muffled heart sounds</td>
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<td></td>
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<td>Dyspnea, pleuritic chest pain</td>
<td>Frothy sputum, crackles</td>
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<tr>
<td>CARDIO</td>
<td>Pulm edema</td>
<td>V̌tach, VFIB</td>
<td>Dyspnea, orthopnea</td>
<td>Absent bowel sounds, tympany</td>
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<tr>
<td></td>
<td></td>
<td>Pericardial effusion</td>
<td>Reflux</td>
<td>Tenderness, guarding</td>
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<td></td>
<td></td>
<td></td>
<td>Reflux</td>
<td>Stool guaiac, endoscopy</td>
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<tr>
<td>RESP</td>
<td></td>
<td></td>
<td>Abd discomfort</td>
<td>Same</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Acute abdomen</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td></td>
<td></td>
<td>Hematemesis, melena</td>
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<tr>
<td>RENAL</td>
<td>AKI</td>
<td></td>
<td>Oliguria, anuria</td>
<td>Edema</td>
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<td></td>
<td></td>
<td></td>
<td>Excessive bleeding</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td></td>
<td>Encephalopathy</td>
<td>Confusion, disorientation, coma</td>
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<td></td>
<td></td>
<td></td>
<td>Same + asterix</td>
<td>EEG</td>
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<td></td>
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<td></td>
<td>CT scan</td>
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<tr>
<td>MS</td>
<td></td>
<td>Rhabdomyolysis</td>
<td>Crush injury, limb ischemia</td>
<td>'Red urine'</td>
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</table>

Renal Failure, Chronic

**Risk**
- Incidence in USA and worldwide: >100 cases of end-stage renal disease (ESRD)/1 million population
- Racial prevalence: African-Americans ~200 cases/1 million; Hispanics ~100/1 million; Caucasians ~50/1 million

**Perioperative Risks**
- Overall periop mortality of pts with ESRD: 4%
- Overall periop morbidity of pts with ESRD: 50% (hyperkalemia, infections, hypotension/Htn, bleeding, dysrhythmias, clotted fistulas)
- Adjusted odds ratio assoc with renal failure was 3.56 in one study

**Worry About**
- Periop progression from chronic renal insufficiency (CRI), not requiring dialysis, to dialysis-dependent ESRD
- Hypovolemia and hypokalemia (esp if recently dialyzed)
- Hypervolemia, metabolic acidosis, and hyperkalemia (esp if not recently dialyzed)
- Autonomic dysfunction (excessive hypotensive responses)
- Exaggerated hypertensive responses to noxious stimuli
- Prolonged responses to renally excreted drugs and metabolites (e.g., vecuronium, pancuronium, narcotics)
- Impaired immune status
- Occult CAD

**Overview**
- Decreased excretory and other functions of kidneys related to long-standing disease; with dialysis, a disease that can persist for many years
- Assoc with multiple complications of failed renal excretory function, incl volume overload, accumulation of products of catabolism (e.g., K+ and hydrogen ions), plt dysfunction, and side effects of dialytic therapy; incl hypovolemia
- Associated with complications of concurrent diseases (e.g., DM, Htn)

**Perioperative Implications**

**Preoperative Preparation**
- Assess adequacy of dialytic therapy, volume and acid-base status, Hgb conc, CV status, serum K+.
- If not dialysis-dependent, assess renal reserve, CV status.
- Consider issues of vascular access.

**Monitoring**
- Temp, ECG (rhythm, rate, hyperkalemia)
- Pulse oximeter, capnometry, peripheral nerve stimulator
- Consider arterial catheter if chronically hypertensive; consider PA catheter for high-risk surgery in pts with cardiac dysfunction.

**Airway**
- Gastroparesis precautions if diabetic

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<tbody>
<tr>
<td>CARDIO</td>
<td>CHF</td>
<td>Exercise intolerance</td>
<td>Htn, Palpitations</td>
<td>Crackles, S3, S4</td>
</tr>
<tr>
<td></td>
<td>LVH</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dysrhythmias</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>GI</td>
<td>N/V, anorexia</td>
<td>N/V, anorexia</td>
<td>Melena, rectal bleeding</td>
<td>Malnutrition</td>
</tr>
<tr>
<td></td>
<td>GI bleeding</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Plt dysfunction</td>
<td>Easy bruising</td>
<td>Fatigability</td>
<td>Ecchymoses</td>
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<tr>
<td></td>
<td>Anemia</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>RENAL</td>
<td>↓ Concentrating ability (CRI)</td>
<td>Nocturia, frequency</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalopathy</td>
<td>↓ Mental acuity, disorientation</td>
<td>Postural hypotension</td>
<td>Mental status</td>
</tr>
<tr>
<td></td>
<td>Autonomic dysfunction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PNS</td>
<td>Peripheral neuropathy</td>
<td>Paresthesias, burning, itching of lower extremities</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Preinduction/Induction**
- Reduce dose of thiopental
- Higher doses of propofol required to achieve same level of BIS index
- Exaggerated response to benzodiazepines
- Consider avoiding renally excreted NM blockers (vecuronium, pancuronium)
- Use narcotics cautiously
- If not dialysis-dependent, theoretical concerns about sevoflurane, although do not appear clinically relevant
- Exaggerated BP swings with induction and intubation
- Reduce dose of local anesthetics if metabolic acidosis present or if sedatives will cause resp acidosis

**Extravasation**
- Ensure adequate reversal of NM blockers
- Evaluate airway reflexes

**Adjuvants**
- Avoid renally excreted NM blockers

**Postoperative Period**
- Use dialysis if necessary.
- Monitor for frequent causes of postop morbidity (see above).

**Anticipated Problems/Concerns**
- Hyperkalemia: Treatment with CaCl2, insulin/glucose, or NaHCO3 may be necessary; intraop dialysis occasionally required
- Balancing intraop volume requirements with need for postop fluid removal
- Exaggerated drug effects

Respiratory Distress Syndrome

Risk
- Mortality rates range from 10–90%, with an average of 50%
- Race and/or gender prominence: None

Perioperative Risks
- Frequent complication of trauma and surgery with a high morbidity and mortality
- Develops in 25% of pts with Gram-negative sepsis, 90% of those with Gram-negative septic shock, and 34% of pts who aspirate
- Large-volume crystalloid and blood product resuscitation

Worry About
- Decreased oxygenation
- Decreased pulmonary compliance

Overview
- Severe arterial hypoxemia
- Contributes to prolonged mechanical ventilation and lengthened stay in the ICU
- Reduced lung compliance
- Acute lung injury is defined as a PaO₂/FIO₂ below 300 mm Hg and a pulmonary artery occlusion pressure less than 18 mm Hg if measured or not.

ASSESSMENT POINTS

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<thead>
<tr>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Decreased O₂ delivery</td>
<td>SOB</td>
<td>Tachycardia</td>
<td>ECG</td>
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<tr>
<td></td>
<td></td>
<td>Decreased peripheral perfusion</td>
<td>Ischemia</td>
<td>Arterial line</td>
</tr>
<tr>
<td>RESP</td>
<td>Decreased oxygenation, CO₂ accumulation</td>
<td>Dyspnea and tachycardia</td>
<td>Rales</td>
<td>Cardiac output/SVO₂</td>
</tr>
<tr>
<td>CNS</td>
<td>Agitation, Confusion</td>
<td>Disorientation, Confusion</td>
<td></td>
<td>Level of consciousness</td>
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</tbody>
</table>


Perioperative Implications

Pre-induction/Induction/Maintenance
- Use of an ICU ventilator in the operating room
- Preop use of pressure control ventilation
- PIP >50 cm H₂O with high flows >50 L/min
- High preop PEEP values or an increased A-a gradient (PaO₂/FIO₂ <100)
- Coexisting expiratory obstruction (autoPEEP)
- Expecting large volume shifts

Monitoring
- Oxygenation monitored by pulse oximetry and blood gas analysis
- Peak and plateau inflation pressures
- Pulm catheter to manage fluid status, cardiac output and mixed venous saturation

Airway
- Maintain airway protection with intubation and mechanical ventilation
- High FIO₂ and PEEP
- Low airway pressures

Maintenance
- Intubation should be performed early and electively.
- If hypoperfusion is present, O₂ delivery may be compromised not only by hypoxemia but by inadequate cardiac output.
- Ventilator settings of low tidal volumes and PEEP with a goal of achieving maximal lung recruitment, and to achieve adequate oxygenation and avoid toxic levels of FIO₂

Extrication
- The pt should remain intubated at end of procedure in the majority of cases
- Maintain plateau pressure ≤50 cm H₂O

ICD-9-CM Code: 641.2

Etiology
- Direct lung injury: Pneumonia, aspiration, pulmonary contusion, fat emboli, near-drowning, inhalation injury
- Indirect lung injury: Sepsis, trauma, assoc shock, multiple blood transfusions, cardiopulmonary bypass, drug overdose, acute pancreatitis
- Increased intravascular volume and decreased colloid oncotic pressure can increase severity.

Usual Treatment
- Alleviation of hypoxemia utilizing lung protection strategy to ventilate
- Avoid large TVs that lead to alveolar overinflation (volutrauma assoc with high peak pressures >35 cm H₂O)
- Use PEEP so that functional residual capacity > closing capacity to reduce tendency towards alveolar collapse and worsening hypoxemia

ASSESSMENT POINTS

Postoperative Period
- Anticipate keeping pt intubated and mechanically ventilated
- Reduction of extravascular lung water if there is large volume shift
- If paralysis required, sedation is always necessary

Adjuvants
- It may be necessary to maintain paralysis in postop period.
- Muscle relaxation may improve gas exchange.

Anticipated Problems/Concerns
- Oxygenation will almost always worsen following surgery.
- CO₂ elimination may be a problem with worsening lung compliance.
# Rett Syndrome

### Risk
- Almost exclusively females
- Incidence 0.4–0.7 per 10,000

### Perioperative Risks
- Abn control of ventilation, with periods of apnea and hyperventilation
- May have GE reflux
- Multiple orthopedic and motor movement disorders

### Worry About
- Risk of periop apnea not known
- Risk of succinylcholine-induced hyperkalemia not known
- Aspiration due to GE reflux and swallowing disorder
- Cardiac: Prolonged QTc, abn autonomic regulation, increased incidence of sudden death
- Intraop positioning because of spasticity and contractures

### Overview
- DX based on clinical characteristics with inclusion and exclusion criteria, mutations in MECP2 gene
- Normal development for the first 5–6 mo of life followed by rapid loss of acquired cognitive, verbal, and motor skills with eventual severe impairment in all areas
- Abn EEG; nonspecific changes
- Pathognomonic stereotyped hand movements, tortuous hand-wrinking or other hand automatism
- Seizures very common
- Resp abn when awake, hyperventilation alternating with hypoventilation or apnea and hypoxemia
- Orthopedic and movement disorders such as scoliosis, spasticity, ataxia, loss of locomotion

### ASSESSMENT POINTS

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<tbody>
<tr>
<td>HEENT</td>
<td>Nonspecific Spasticity may make airway difficult</td>
<td>Neck ROM Airway exam</td>
<td>Neck X-rays if indicated</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Possible prolonged QTc Peripheral vasomotor disturbances</td>
<td>Extremities cool Trophic changes</td>
<td>EKG</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Abn control of ventilation when awake with hyperventilation, apnea, cyanosis Lung changes due to scoliosis or aspiration</td>
<td>Hx of apnea, cyanosis Hx of scoliosis, aspiration Observation Chest exam</td>
<td>O₂ saturation CXR</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>GE reflux possible, swallowing difficulties, constipation Growth failure</td>
<td>Hx of GE reflux, feeding difficulties</td>
<td>Thin, small for age Studies for GE reflux</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Severe developmental delay Seizures Ataxia, loss of locomotion</td>
<td>Developmental level, seizure activity Assessment of cognitive and movement disorders</td>
<td>EEG</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Hypotonia (early); spasticity (late); ataxia Secondary orthopedic manifestations: Scoliosis, joint contractures Progress and extent of MS abn</td>
<td>Chest exam for scoliosis Limb and joint positions X-rays</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Key Reference:

### Anticipated Problems/Concerns
- Resp control abn not well understood. Therefore, effect of anesthetic agents intra- and postop on respiration not known. Need for postop monitoring for apnea unknown.

### Perioperative Implications

#### Preoperative Preparation
- As for any pt with developmental delay
- Optimize resp status
- Assess resp control
- Minimize aspiration risk

#### Monitoring
- Routine
- More invasive depending on procedure

#### Airway
- Normal face
- Spasticity may make positioning difficult

#### Preinduction/Induction
- Risk of hyperkalemia following succinylcholine unknown
- Possible aspiration risk due to GE reflux

#### Maintenance
- Resp control abn; unknown if spontaneous ventilation under anesthesia assoc with significant apnea
- Attention to body temp because of thin body habitus and peripheral vasomotor disturbances

#### Exubation
- Possible aspiration risk
- Assess resp control

#### Postoperative Period
- Resp control abn
  - Effect of anesthetic agents
  - Duration of resp monitoring
  - Effect of narcotics versus local anesthetics for pain control

#### Adjuvants
- None
Reye’s Syndrome

Mary A. Keyes

<table>
<thead>
<tr>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Incidence prior to 1990: 0.3–0.6/100,000</td>
</tr>
<tr>
<td>• 1987-1993: 0.03–0.06/100,000; 2 cases reported/y since 1994</td>
</tr>
<tr>
<td>• During early 1980s, association between aspirin and Reye syndrome was recognized and incidence dramatically declined. In 1986, a warning label on all aspirin containing products was mandated in the USA.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Perioperative Risks</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Surgery (all but life-and-death emergencies) contraindicated during Reye syndrome</td>
</tr>
<tr>
<td>• Following recovery, repeat liver function tests</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Worry About</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Unrecognized inborn errors of metabolism that produce Reye-like syndromes such as fatty-acid oxidation defects, carnitine deficiency, and amino and organic acidopathies</td>
</tr>
<tr>
<td>• Recurrent liver dysfunction</td>
</tr>
<tr>
<td>• Permanent neurologic sequelae</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Overview</th>
</tr>
</thead>
<tbody>
<tr>
<td>• An acute, noninflammatory encephalopathy with hepatic dysfunction predominantly in children; typically starts several days after viral illness, usually influenza or varicella</td>
</tr>
<tr>
<td>• Encephalopathy heralded by protracted, severe vomiting, with abn behavior and combativeness that may progress to coma</td>
</tr>
<tr>
<td>• Dx made by unexplained encephalopathy with one or more of following: Serum transaminases elevated to at least 3× normal; blood ammonia levels at least 3× normal; or hepatic microvesicular fatty infiltration on liver biopsy. There should be no other reasonable explanation for the cerebral or hepatic abnormalities.</td>
</tr>
<tr>
<td>• The CDC uses a Stage 1–6 classification of progressive disease severity. Mortality has decreased from 50% to 20% as a result of recognition in early phases and aggressive treatment.</td>
</tr>
<tr>
<td>• Prognosis depends on severity and duration of cerebral dysfunction. Severe disease may lead to subtle neuropsychological defects.</td>
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<table>
<thead>
<tr>
<th></th>
<th>ICD-9-CM Code: 331.81</th>
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<tbody>
<tr>
<td><strong>System</strong></td>
</tr>
<tr>
<td>GI</td>
</tr>
<tr>
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<tr>
<th>Perioperative Implications</th>
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<tr>
<td>• Surgery not undertaken except in life-and-death emergencies</td>
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<tbody>
<tr>
<td>• Ondansetron to decrease vomiting</td>
</tr>
<tr>
<td>• Treat seizures with phenytoin</td>
</tr>
<tr>
<td>• Correct hyperammonemia with sodium phenylacetate/sodium benzoate</td>
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</table>

<table>
<thead>
<tr>
<th>Anticipated Problems/Concerns</th>
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</thead>
<tbody>
<tr>
<td>• Hepatic dysfunction</td>
</tr>
<tr>
<td>• Neurologic sequelae</td>
</tr>
<tr>
<td>• Underlying inborn errors of metabolism, particularly in children under 5 y</td>
</tr>
</tbody>
</table>
Rheumatoid Arthritis

**DISEASES**

**Risk**
- Incidence in USA: 1% of population
- M:F ratio: 1:2

**Perioperative Risks**
- Risk of neurologic injury is increased due to occult cervical spine damage
- Assoc cardiac disease may be present but is not clinically apparent
- Pulm complications arise 2° to possible pulm fibrosis and restrictive lung disease

**Worry About**
- Visualization of glottis and tracheal intubation may be difficult 2° to rheumatoid-assoc damage of the cervical spine
- Former successful ET intubation does not reliably eliminate existing airway abn

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<td>ECG</td>
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**Perioperative implications**

**Preoperative Evaluation**
- Thorough airway evaluation is a priority. If atlantoaxial instability exists, flexion of neck can compress the spinal cord. Radiating pain to occiput may be indication of cervical cord involvement. Imaging such as X-ray, CT, MRI may be indicated if amount of cervical involvement is not known.
- Cardiopulmonary status needs to be evaluated. If severe restrictive lung disease is suspected, preop pulm function tests may be indicated. Anticipation of postop ventilatory support to be considered.
- Adequate knowledge of pt's current medications. Stress dose corticosteroid supplementation may be indicated for pts being treated chronically with these drugs. Antiinflammatory medications, aspirin, and other rheumatoid drugs can interfere with pt function, clotting, as well as RBC formation.
- Joint mobility and restrictions should be assessed to determine appropriate intraop positioning.

**Monitoring**
- Standard monitors
- Further invasive monitoring dependent on pt's disease state and anticipated procedure
- Occult pericardial effusion, pericardial thickening, rheumatoid nodules in the cardiac conduction pathway, valvular fibrosis
- Iatrogenic injury to the cervical spinal cord during laryngoscopy and tracheal intubation
- Chronic corticosteroid use may necessitate intraop steroid administration

**Overview**
- Chronic inflammatory disease involving diffuse joints and organ systems
- Systemic effects incl pericardial effusion, tamponade, pleural effusion, pulm fibrosis, anemia, and keratoconjunctivitis, and renal failure

**ICD-9-CM Code: 714.0**

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**Etiology**
- Autoimmune disorder triggered by an antigen in genetically predisposed persons
- Clinical variability may stem from differences in triggering antigens and immune response
- Pathology: Synovial cellular hyperplasia, synovial infiltration by lymphocytes, plasma cells, and fibroblasts leading to degeneration of cartilage and articular surfaces

**Treatment**
- Aspirin and NSAIDs: Ibuprofen, indomethacin, naproxen, piroxicam, sulindac, and tolmetin
- Immuno-regulating drugs: Methotrexate, hydroxychloroquine, sulfasalazine, azathioprine, penicillamine, and gold

**Exubation**
- Post extubation laryngeal obstruction 2° to edema and erythema possible from cricoarytenoid involvement
- Postop ventilatory support anticipated with severe restrictive lung disease

**Adjuvants**
- Regional and neuraxial techniques can be utilized assuming no significant thoracic, lumbar, and sacral spine involvement as well as normal coagulation studies

**Anticipated Problems/Concerns**
- Tracheal intubation difficulties 2° to cervical spine and TMJ involvement
- Intraop CV instability and restrictive pulm disease issues
- Assoc side effects of current drug therapy, e.g., anticoagulation, anemia, poor wound healing, etc.
- Multi-organ system involvement
- Intraop positioning concerns 2° to advanced joint involvement and decreased ROM
- Potential for postop ventilatory support

**Radiography**
- Neck pain
- Numbness
- Motor deficits
- ROM of neck

**Radiography**
- Joint pain
- Swelling
- Pain with motion
- Restricted motion

**Direct laryngoscopy**
- Epistaxis
- Hx of voice change
- Friable mucosa
- Voice, airway exam

**ECG**
- Dyspnea
- Orthopnea
- Reduced exercise
- Reduced exercise
- Dyspnea
- Friction rub

**ECHO**
- S
- Rales
- Diastolic mumur (A1)
- Distant heart sounds
- Friction rub

**CXR, PFTs**
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- Numbness
- Sensory deficits
- Motor deficits
- ROM of neck

**Radiography**
- Joint pain
- Swelling
- Pain with motion
- Restricted motion
Rocky Mountain Spotted Fever

Risk
- Incidence in USA: In every state, most common in southeast and south central, ~250–1200 cases/y
- Exposure to tick-infested terrain or dogs
- Severe infection, young and healthy, men over 20 and G6PD deficiency at risk for death
- Mortality 20% untreated, 3–9% even with early treatment (within first 5 d)
- Mortality increases with delay in DX, older age (over 60), male sex, very young (<4 y), black race, chronic alcohol abuse, G6PD deficiency

Perioperative Risks
- Increased mortality 2° to CV instability and noncardiogenic pulm edema
- Increased risk of organ injury due to compounded insults
- Increased bleeding tendency

Worry About
- Severe intravascular volume depletion leading to shock
- Electrolyte disturbances

- Cardiac arrhythmias
- Microvascular hemorrhage
- Consumptive coagulopathy
- Intraop resp and renal failure

Overview
- Uncommon but severe, pathophysiology primarily due to endothelial cell infection resulting in vascular permeability, edema, hypovolemia, and ischemia
- Initial symptoms in 1–3 d: Nonspecific, mimicking a viral syndrome, specific symptoms in 2–14 d, most in 5–7 d
- Rash appears in 3–5 d, after the onset of fever, initially as maculopapular progressing to petechiae; usually centripetal progression, rash absent in 10–15%
- Disease progression (more likely with delay in treatment) results in multi-organ involvement: noncardiac pulm edema, CNS signs, myocardiitis, hepatisis, bleeding (2° to thrombocytopenia and direct vessel damage), and acute renal failure

ICD-9-CM Code: 082.0 (Spotted fevers)

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<tr>
<td>CARDIO</td>
<td>Extensive microvascular leak; interstitial myocarditis</td>
<td>Rash, swelling</td>
<td>Rash, edema, arrhythmias</td>
<td>ECG, CXR, Lytes, BP</td>
</tr>
<tr>
<td>RESP</td>
<td>Noncardiac pulm edema; interstitial pneumonitis</td>
<td>↓ Exercise tolerance, dyspnea, cough</td>
<td>Rales by auscultation</td>
<td>CXR, spirometry</td>
</tr>
<tr>
<td>GI</td>
<td>Gastroenteritis; liver, spleen, and pancreatic microvascular hemorrhage and edema</td>
<td>N/V, abdominal pain, diarrhea</td>
<td>Abd tenderness</td>
<td>Hepatosplenomegaly, SGOT, bilirubin</td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenia, anemia</td>
<td>Easy bleeding, malaise</td>
<td>Rash</td>
<td>Hct/Hgb, plt/PT, PTT</td>
</tr>
<tr>
<td>RENAL</td>
<td>Microvascular hemorrhage and edema, interstitial nephritis, prerenal azotemia</td>
<td>Lumbar pain</td>
<td>UO, Cr Lytes</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Meningoencephalitis</td>
<td>Focal defects, deafness, confusion, meningismus, photophobia, seizures</td>
<td>CSF: ± ↑ WBC, ↑ protein</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Microvascular hemorrhage and edema</td>
<td>Myalgia, arthralgia</td>
<td>↓ ROM</td>
<td></td>
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</table>


Perioperative Implications

Preoperative Preparation
- Antibiotic therapy and correction of underlying organ system dysfunction
- Surgery only for emergency
- Assess volume, resp, renal status

Monitoring
- Consider PA catheter, arterial line, UO
- Intraop ABGs and lytes
- Plt and other coagulation variables

Airway
- Severe edema of oropharynx and increasing bleeding tendency can lead to difficult intubation

Induction
- Hypovolemia can cause hypotension

- Microvascular leak in lung can cause rapid desaturation
- Increased cardiac arrhythmias

Maintenance
- Owing to CV instability, volume status is key
- Possibility of resp failure and constant volume resuscitation should be anticipated when selecting anesthetic technique

Exubation
- Oropharyngeal edema and ↑ bleeding tendency may make reintubation very difficult

Adjuvants
- Vasodepressor drugs used in acute resuscitation should be readily available
- Lidocaine for treatment of cardiac arrhythmias

Postoperative Period
- Intravascular volume shifts; coagulation defects, resp failure, CV instability, renal failure

Anticipated Problems/Concerns
- Owing to the possibility of multisystem failure, prolonged postop ICU management may be required
- Because early treatment with antibiotics is curative and highly successful in preventing complications, high index of suspicion, e.g., after tick exposure in endemic areas, is needed

Etiology
- Rickettsia rickettsii transmitted in saliva of ticks after 6–10 hr of attachment and feeding or by exposure to infected tick hemolymph, during the removal of ticks
- Incubation period ~7 d (2–14 d)
- Obligatory intracellular bacterium that replicates in the vascular endothelial cells, causing direct cell injury with loss of vascular integrity

Usual Treatment
- Dx difficult, primarily clinical and epidemiologic (potential tick exposure), biopsy of skin lesion to confirm or serologic testing
- Doxycycline, chloramphenicol (pregnant women), importance of early therapy within first 5 d (mortality 6.5 vs. 22.9)
- Correct hypovolemia, coagulation defects, thrombocytopenia, provide intensive, supportive care for various organ system failures
Sarcoidosis

Risk
- Varies: ≤1–80/100,000 with highest incidence in Sweden; in USA 30/100,000
- Presenting ages 20–40 y in USA
- More common in African-Americans than Caucasians in USA
- Females > males

Perioperative Risks
- Severity depends on degree of airway, lung, cardiac, and CNS involvement

Worry About
- Airway granulomas distorting and obstructing anatomy risking obstruction with sedation and making intubation potentially difficult

Overview
- Multisystem granulomatous disorder with widespread noncaseating epithelioid cell granulomas
- Lung most frequently affected organ
- Airway abn 2° to granulomas
- Local organ distortion can result in symptoms
- Mononuclear inflammatory cells: T-helper cells + mononuclear phagocytes lead to formation of granulomas

ICD-9-CM Code: 135.0

Etiology
- Unknown disease due to exaggerated cellular immune response involving mononuclear phagocytes and T lymphocytes

Usual Treatment
- Steroids: Oral prednisone (inhaled steroids have not been shown to be consistently effective)
- NSAIDs incl salicylates
- Chloroquine or hydroxychloroquine for mucocutaneous sarcoidosis
- If steroids ineffective: Methotrexate or immunosuppressive agents

Assessment Points

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Involvement of nares, polyps with distorted anatomy; larynx granulomas, epiglottis, arytenoid involvement</td>
<td>Dyspnea Breathing difficulty</td>
<td>Nasal stuffiness, wheezing, hoarseness, stridor</td>
<td>Laryngoscopy</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Heart block</td>
<td>Palpitations</td>
<td>Arrhythmia Rales</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm granulomas, airway obstruction Bilateral hilar lymphadenopathy (eggshell calcifications of hilar nodes); pulm fibrosis; interstitial disease</td>
<td>Dyspnea Wheezing, cough</td>
<td>Dry rales Wheezes</td>
<td>CXR PFTs (↓ vital and diffusing capacities) ABGs CT if airway obstruction considered an issue</td>
</tr>
<tr>
<td>GI</td>
<td>Liver involvement</td>
<td>Thirst</td>
<td>Space-occupying lesions</td>
<td>Focal nerve deficits Psychiatric examination</td>
</tr>
<tr>
<td>ENDO</td>
<td>DI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Di Ca²⁺ resorption</td>
<td></td>
<td></td>
<td>BUN/Cr</td>
</tr>
<tr>
<td>CNS</td>
<td>Nerve involvement</td>
<td>Space-occupying lesions</td>
<td>Focal nerve deficits</td>
<td>Psychiatric examination</td>
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<td>DI</td>
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</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Adequate steroid coverage
- For pulm and airway, determine if airway obstruction exists and degree of pulm involvement. Evaluate for Hx of SOB and dyspnea. Obtain CXR, consider PFTs and preop ABG based on symptoms and Hx. If airway obstruction suspected, obtain CT to better define issues.

Airway
- Distortion or obstruction 2° to granulomas
- Hypoxia 2° to lung disease

Monitoring
- Observe for heart block
- Arrhythmia

Anticipated Problems/Concerns
- Airway problems 2° to distorted anatomy
- Pulm problems 2° to lung involvement
Perioperative Implications

**Preoperative Preparation**
- Metoclopramide, sodium citrate, ranitidine in pts with gastroparesis
- Assess end-organ impairment 2° to antineoplastic chemotherapeutic agents

**Monitoring**
- Arterial line and CVP or PA catheter for resection of large tumors

**Airway**
- Risk of aspiration with large abd mass, or brainstem compression

**Induction**
- Caution: With cardiac involvement, caval compression may have hemodynamic instability

**Maintenance**
- Potential CV instability

**Extubation**
- Awake, if at risk for aspiration

**Adjuvants**
- Altered pharmacokinetics with hepatic or renal involvement

**Postoperative Period**
- Pulm embolism, coagulopathy

**Etiology**
- Genetic factors, high-dose radiation, carcinogens (dibenzanthracene, methylcholanthrene), Maloney sarcoma virus may predispose to sarcoma
- von Recklinghausen's disease: 10–12% develop neurofibrosarcomas
- Paget's disease: 0.9% develop osteosarcoma
- Kaposi's sarcoma in AIDS pts and immunodeficient pts

**Risk**
- Incidence in USA
- Osteosarcoma: 1:100,000; 2000 new cases/y; second most decade (mean age 15)
- Soft tissue sarcoma: >20 types, 5500 new cases/y in USA, peak incidence in children and adults age 45–50 y
- Equal in male and female, all races

**Overview**
- Malignant tumors derived from embryonic mesoderm
- Multiple types in connective tissue, muscle, fat, vasculature, neural and other tissues
- Spread aggressively by local invasion and early hematogenous spread, esp to lung

**ICD-9-CM Code:** 171 (Depends on type)

**Worry About**
- Adriamycin-induced cardiotoxicity (global LV hypokinesis)
- Mitomycin-induced acute pulm toxicity, pulm fibrosis, ARDS with increased FIO
- Immunosuppression, hemorrhagic cystitis, renal failure induced by antineoplastic chemotherapeutic agents

**Anticipated Problems/Concerns**
- Adverse effects of chemotherapeutic agents (see Drugs section)
- Resp compromise due to pulm metastases
- Mass effect and/or organ compression and functional impairment
- Effects of prolonged anesthesia
- In prolonged abd cases, hypothermia, complications of massive transfusion

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Atrial myxoma—ball-valve effect</td>
<td>Sx CHF, pulm edema</td>
<td>Rales</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>Vena caval obstruction</td>
<td>RV failure, CV collapse</td>
<td></td>
<td>ECHO</td>
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<tr>
<td></td>
<td>SVC syndrome</td>
<td>Head, airway edema ↑ ICP</td>
<td>Venous congestion of head and neck</td>
<td>Angio</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm embolus</td>
<td>Dyspnea</td>
<td></td>
<td>V/Q scan</td>
</tr>
<tr>
<td>GI</td>
<td>Gastroparesis</td>
<td>Early satiety</td>
<td></td>
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<tr>
<td></td>
<td>Bowel obstruction</td>
<td>Vomiting</td>
<td></td>
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<td></td>
<td>Hepatic metastases</td>
<td>Obstructive jaundice</td>
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<tr>
<td></td>
<td>Sarcoma of ampulla of Vater</td>
<td>Hepatic dysfunction</td>
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<tr>
<td>HEME</td>
<td>Hypercoagulable</td>
<td>Alopecia</td>
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<td></td>
<td>Immunosuppressive chemotherapy</td>
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<tr>
<td></td>
<td>Anemia, due to GI hemorrhage</td>
<td></td>
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<tr>
<td>RENAL</td>
<td>Compression of ureters by retroperitoneal tumor</td>
<td>Sx uremia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>CN compression</td>
<td>Various symptoms</td>
<td>Neurologic exam</td>
<td>EMG</td>
</tr>
<tr>
<td>MS</td>
<td>Bone sarcomas</td>
<td>Hypercalcemia</td>
<td>Chvostek's sign</td>
<td>Blood Ca^2+</td>
</tr>
<tr>
<td></td>
<td>Limb loss</td>
<td></td>
<td></td>
<td>Albumin</td>
</tr>
</tbody>
</table>

Schizophrenia

Risk
- Most common psychotic disorder with a lifetime worldwide prevalence of 1%.
- Increased risk of suicide 5–10%

Perioperative Risks
- Marked deterioration of function and self care
- Exacerbation of psychosis with abrupt discontinuation of medications

Worry About
- Uncooperative, combative, or catatonic pt
- Increased morbidity and mortality due to poorly controlled co-existing systemic disease and increased incidence of alcohol and substance abuse
- Drug interactions and side effects

Overview
- Schizophrenia is a psychiatric disorder that may be characterized by thought disorders, hallucinations, and fixed false beliefs.
- Antipsychotic medications are the mainstay treatment for schizophrenia.
- Antipsychotics have anticholinergic effects (dry mouth, blurry vision, urinary retention, constipation, tachycardia), histamine antagonism (sedation), and α1 antagonism (orthostatic hypotension).
- First generation antipsychotics have strong dopamine antagonism leading to extrapyramidal side effects (EPS) such as tardive dyskinesia.
- Second generation or atypical antipsychotics have serotonin antagonism and less dopamine antagonism leading to less EPS.
- EPS can be treated with anticholinergics such as benztropine 2 mg or diphenhydramine 50–100 mg.
- Neuroleptic malignant syndrome (NMS) is a rare but potentially fatal syndrome occurring after exposure or abrupt D/C of antipsychotic medications. The syndrome is marked by muscle rigidity, hyperthermia, and autonomic instability. It is clinically similar to malignant hyperthermia and may be related to dopamine blockade.
- Treatment of NMS includes hydration and cooling measures, IV dantrolene, and dopamine agonists.
- Avoid dopamine antagonists, such as metoclopramide, if NMS is suspected.

ICD-9-CM Code: 295

Etiology
- Functional hyperactivity of dopamine transmission may play a role
- Genetic and environmental factors are unclear and controversial.

Usual Treatment
- Antipsychotic medications
- Psychotherpay
- ECT

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>QT, PR prolongation</td>
<td>Dizziness, Palpitations</td>
<td>Orthostatic hypotension, Arrhythmia</td>
<td>EKG</td>
</tr>
<tr>
<td>RESP</td>
<td>Significant increased incidence of smoking</td>
<td>SOB, wheezing</td>
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<td></td>
</tr>
<tr>
<td>GI</td>
<td>Paralytic ileus (Postop)</td>
<td></td>
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</tr>
<tr>
<td>HEME</td>
<td>Agranulocytosis due to meds</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>DM due to meds, Hyperlipidemia due to meds</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEURO</td>
<td>Sedation, Extrapyramidal side effects</td>
<td>Somnulence</td>
<td>1. Choreathoid movements of head, limbs, trunk</td>
<td>WBC, body temp monitoring, creatine kinase, UA (myoglobinuria)</td>
</tr>
<tr>
<td></td>
<td>1. Tardive dyskinesia</td>
<td></td>
<td>2. Subjective discomfort causing agitation and restlessness</td>
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<td></td>
<td>2. Akithisia</td>
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<td>3. Slow sustained bodily contractions</td>
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<tr>
<td></td>
<td>3. Dystonia</td>
<td></td>
<td>4. Catatonia, rigidity, akinesia</td>
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<td></td>
<td>4. Parkinsonism</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>GENERAL</td>
<td>Neuroleptic malignant syndrome (NMS)</td>
<td>Antipsychotic use (usually increase in dose) or abrupt discontinuation</td>
<td>Hyperthermia, rigidity, autonomic instability, cardiac arrhythmia</td>
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Perioperative Implications

Preoperative Preparation
- Hx may be unreliable or unattainable
- Continue antipsychotic medications preop

Monitoring
- Routine

Airway
- Routine considerations

Preinduction/Induction
- No specific technique clearly superior

Maintenance
- Hypotension
- Tachycardia, arrhythmia
- Increased risk of thermodynamics and hypothermia. Monitor temp and warm pt appropriately.

Extubation
- Usual criteria

Postoperative Period
- Decreased reports of pain
- Increased incidence of severe postop ileus
- Increased risk of postop confusion
- Increased postop mortality

Regional Anesthesia
- Contraversial, but epidural analgesia may decrease incidence of postop ileus

Anticipated Problems/Concerns
- Cardiac arrhythmia
- Hemodynamic instability and hypotension
- Hypothermia
- Potential for neurolytic malignant syndrome
Scleroderma

Risk
- Incidence: 9/1 million/y
- Prevalence: 240/million
- More severe in Native Americans and African-Americans
- 10-y survival: 55–60%, presence of pulm Htn is major prognostic predictor

Perioperative Risks
- Severe hypotension 2° to hypovolemia
- Hypoxia 2° to pulm Htn and restrictive disease
- Failed intubation

Worry About
- GI reflux
- Obliterative vasculopathy leading to pulm Htn
- Restrictive lung disease

Overview
- Onset 35–50 y
- Chronic, systemic disease that targets skin, lungs, heart, GI, kidneys, and MS system
- Three features: Tissue fibrosis, small blood vessel vasculopathy, autoimmune response
- Two major classifications: Limited and diffuse cutaneous scleroderma
- May have overlap syndromes with other rheumatic diseases

ICD-9-CM Code: 710.1 (Diffuse), 701.0 (Localized)

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Cutaneous fibrosis</td>
<td>Masked facies</td>
<td>Small oral aperture</td>
<td>Hyperpigmentation</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Pericardial disease</td>
<td>DOE</td>
<td>CHF</td>
<td>ECHO, Holter</td>
</tr>
<tr>
<td></td>
<td>Myocardial fibrosis</td>
<td>CHF, Arrhythmia</td>
<td>Syncope</td>
<td></td>
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<tr>
<td></td>
<td>Conduction abn</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>RESP</td>
<td>Fibrosing alveolitis</td>
<td>Dyspnea</td>
<td>Nonproductive cough</td>
<td>CXR, PFT</td>
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<tr>
<td></td>
<td>Obliterative vasculopathy</td>
<td></td>
<td></td>
<td>Bronchoalveolar lavage</td>
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<tr>
<td></td>
<td>Pulm Htn</td>
<td></td>
<td></td>
<td>ECHO</td>
</tr>
<tr>
<td>GI</td>
<td>Esophageal fibrosis/colonic dysmotility</td>
<td>Difficulty chewing Dysphagia Bleating Bruddhe</td>
<td>Weight loss</td>
<td>UGI/endoscopy</td>
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<tr>
<td>RENAL</td>
<td>Intrinsic renal vessel disease</td>
<td></td>
<td>Malignant Htn</td>
<td>Proteinuria, Hematuria</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>BUN or Cr</td>
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<tr>
<td>DERM</td>
<td>Cutaneous fibrosis</td>
<td>Fibrosis of limbs</td>
<td>Sweating</td>
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<tr>
<td></td>
<td></td>
<td>Atrophy and contractures</td>
<td>Telangiectasis</td>
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<tr>
<td>MS</td>
<td>Raynaud’s disease</td>
<td>Excessive cold sensitivity</td>
<td>Pain</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Cyanosis of digits</td>
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</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Proton pump inhibitors to reduce gastric acid
- Consider metoclopramide for early disease
- Less effective for late disease

Monitoring
- Invasive arterial monitoring relatively contraindicated in pts with Raynaud’s disease because of risk of digit ischemia, but ABG may be indicated
- BP may be difficult because of reduced forearm blood flow
- Consider PA catheter if pulm Htn
- Skin temp may be significantly lower (1.5°C) than core temp

Anesthetic Technique
- Regional anesthesia may be preferable considering pulm problems
- Regional technique may be assoc with prolonged block in the presence of epinephrine because of severe vasoconstriction
- May see vasomotor instability

Airway
- Pt may have severe decrease in oral aperture
- Consider awake FOB intubation
- May require nasal intubation

Preinduction/Induction
- Pt may be hypovolemic due to vasoconstriction
- Consider volume expansion
- May initially observe Htn followed by vasodilation and hypotension

Maintenance
- Usually requires mechanical ventilation because of restrictive lung disease
- Intraop hypoxemia may develop 2° to pulm Htn

Extubation
- May require postop ventilation if significant pulm compromise
- Pain control important for pulm status

Anticipated Problems/Concerns
- Difficult airway
- Hypoxemia
- Hypotension
Overview
- Scoliosis is the deformity of the lateral curvature. Idiopathic scoliosis involves the thoracic and lumbar spine with rotation of the vertebrae and rib cage deformity. With increased curvature, rotation progresses and the chest cavity becomes more narrowed causing restrictive lung disease. With severe curves, V/Q mismatch occurs and pulm Htn causes cor pulmonale over time.
- Kyphosis is the posterior curvature deformity, which can be assoc with scoliosis.
- Scoliosis assoc with NM disorders usually involves the thoracolumbar spine and the underlying disease may compromise the resp function due to cough dysfunction, aspiration risk, and reduced ventilation capacity. NM disorders may also be assoc with cardiomyopathy.
- Cobb method is used to measure the severity of the curvature. A parallel line is drawn to the superior border of the causal most vertebral body which tilts to the concavity of the curve, then a second line is drawn parallel to the inferior border of the cephalad most vertebral body that tilts to the concavity of the curve. Perpendicular lines are drawn from these two lines and the angle made by the intersection is measured as the Cobb angle.
- Surgery is indicated in idiopathic scoliosis with curve progression >40–45 degrees in pts of skeletal immaturity, and curves >50 degrees in skeletal mature pts. Severe resp compromise occurs in pts with curves >100 degrees (See Overview in Scoliosis and Kyphosis Surgery).

ICD-9-CM Codes: 737.30 Scoliotic (idiopathic); 754.2 (congenital); (acquired) Kyphosis 737.10 (postural); 756.19 (congenital)

Perioperative Implications
- Major blood loss is assoc with scoliosis correction. Preparation includes large-bore IV access, strategies for transfusion reduction (hypotension, antifibrinolytic agents, preop erythropoietin, cell saver, preop autologous donation, and hemodilution).

Monitoring
- Routine
- Consider arterial line and CVP line
- SSEPs, MEPs and EMG
- Consider BIS monitor
- Estimate blood loss from suction canister contents, cell-saver device and sponges
- Urinary catheter


Prophylactic measures
- Consider O₂ sat pulse oximetry on big toes during anterior exposure of the lower lumbar spine to assess amount of iliac artery compression.

General Anesthesia
- Positioning for posterior fusion is prone with the abdomen free and in reverse Trendelenburg to reduce venous pressures at the surgical site and bleeding. Special care should be taken to ensure peripheral nerves are padded to prevent compression neuropathies, and eyes should be protected to avoid vision loss/corneal abrasions.
- Thoracic approaches may require a lateral position with a double-lumen ETT for OLV. DLT position should be verified by fiberoptic bronchoscopy.
- Wake up test (WUT) provides intraop testing of the lower limb motor for early detection of neuroprotection injury after instrumentation. This test requires the cooperation of the pt and carries the risk of unintentional extubation or IV access loss.

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<tbody>
<tr>
<td>AIRWAY</td>
<td>Potential for difficult airway management</td>
<td>Prior difficult intubations, neck movement restrictions, glossal hypertrophy or aspiration risk 2+ to DMD</td>
<td>Airway and neck exam</td>
<td>Cervical lateral spine, CT scan</td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulation disorders</td>
<td>Hx of easy bruising or bleeding disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Pulm Htn, cardiomyopathy 2+ to underlying muscular dystrophies or mediastinal distortion</td>
<td>Functional status by exercise tolerance</td>
<td>ECG, ECHO, pulm arterial pressure</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive pulm defect, severity of functional impairment related to curve severity</td>
<td>Functional status by exercise tolerance</td>
<td>CXR, ABG, spirometry, PFT with bronchodilator reversibility</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Poor nutrition</td>
<td>Feeding difficulty</td>
<td>Albumin, serum protein</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications
- Premedication with bronchodilator may help to optimize the pt's resp status.
- The use of succinylcholine should be avoided in pts with muscular dystrophies due to the risk of hyperkalemia causing cardiac arrest.
- Boluses of IV induction agents cause a reduction of amplitude of evoked potential responses (cortical responses), but do not interfere with somatosensory potential (SSEP) or transcranial electrical motor evoked potentials (TcMEP).
- Surgery is indicated in idiopathic scoliosis with curve progression >40 degrees with nonop treatment, curves with >40–45 degrees in pts of skeletal immaturity, and curves >50 degrees in skeletal mature pts. Severe resp compromise occurs in pts with curves >100 degrees (See Overview in Scoliosis and Kyphosis Surgery).

ICD-9-CM Codes: 737.30 Scoliotic (idiopathic); 754.2 (congenital); (acquired) Kyphosis 737.10 (postural); 756.19 (congenital)
WUT requires the lighting of sedation and having the pt complete motor activity on command.

**Postoperative Period**

- Postop ventilation made necessary for some pts. Factors that suggest the need for postop ventilation incl NM disorder, severe restrictive pulm defect, congenital cardiac abn, RHF, obesity, prolonged surgical procedure, thoracotomy, and significant blood loss.
- Pain management requires a multimodal technique incl spinal/systemic opioids, local anesthetics, and NSAIDs.
- Ileus can be minimized with utilization of a multimodal pain management regimen.
- Fluid management with UO monitoring and replacement is necessary. ADH levels may be high postop requiring treatment/monitoring.
- Atelectasis, pneumonia, and decreased pulm function may occur postop.

**Anticipated Problems/Concerns**

- Resp complications (atelectasis and pneumonia)
- Neurologic injuries (spinal cord, nerve roots and peripheral nerves)
- Blood loss
- Wound infection
- Continued pain
- Vision loss
- Pulm embolism (PE)
Seizures, Epileptic

**Risk**
- Incidence of epilepsy estimated to be 0.5–2%
- 20–40% of pts with epilepsy will develop intractable seizures (greater than 1 per mo, unsponsive to two or more medications)
- Approx 400,000 people in USA have medically uncontrolled epilepsy

**Perioperative Risks**
- Many rare syndromes are assoc with epilepsy, which can involve disturbances in major organ systems
- Various psychiatric disorders are assoc with epilepsy; anti-epileptic drugs may cause mood, behavior, or cognition disturbances
- Sudden unexpected death reported with epilepsy, poorly understood, incidence is estimated at 2 cases per 1000 pt-years
- Many anti-epileptic drugs are hepatic enzyme inducers or inhibitors which may affect blood levels of drugs such as warfarin, tricyclic antidepressants, statins, chemotherapeutic agents, and antivirals.

**Perioperative Implications**

**Preoperative Preparation**
- Assess neuropsychiatric status.
- Determine antiepileptic drug Hx and review potential drug interactions.
- Assess for signs of concurrent disease, such as murmur suggestive of myocardial tumor (tuberous sclerosis) or stigmata of neurofibromatosis.

**Monitoring**
- For seizure surgery EEG may be placed intraop.

**Maintenance**
- If intraop EEG not planned, use an anticonvulsant anesthetic maintenance regimen such as desflurane with or without NO, propofol, or moderate-dose opioid.
- For GA with intraop EEG, low dose (less than 0.5 MAC) inhalational agent with opioid, low dose propofol. Methohexital, 25–50 mg, alfentanil, 50–100 µg, and remifentanil, 2.5 µg/kg, have been used as activating agents.
- For conscious analgesia continued titrated of sedation/analgesia during painful parts of procedure.
- Consider “asleep-awake-asleep” anesthetic plan

**Anticipated Problems/Concerns**
- Blood levels of antiepileptic drugs can be significantly affected by anesthetics, changes in body physiology, and prolonged NPO status.

**Etiology**
- Congenital often assoc with other syndromes such as tuberous sclerosis, neurofibromatosis, multiple endocrine adenomatosis, Jervell-Lange-Nielsen syndrome
- Acquired assoc with traumatic brain injury, stroke, brain tumor, Alzheimer’s, or idiopathic causes

**Usual Treatment**
- Antiepileptic drugs as monotherapy or in combination, incl phenytoin, phenobarbital, benzodiazepines, carbamazepine, and newer agents such aslevitiracetam, lamotrigine, topiramate, oxcarbazepine, and many others
- 15% of epileptic pts are thought to be candidates for epilepsy surgery, but only about 1% actually undergo surgery.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Gingival hyperplasia</td>
<td>Phenytoin use</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cardiac tumors with tuberous sclerosis</td>
<td>Tuberous sclerosis</td>
<td>Murmur possible</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm involvement with neurofibromatosis</td>
<td>Neurofibromatosis</td>
<td>Cor pulmonale</td>
<td>CXR ECG</td>
</tr>
<tr>
<td>GI</td>
<td>Anticonvulsant-induced hepatitis</td>
<td>Anticonvulsant use</td>
<td>Jaundice, tender RUQ</td>
<td>LFTs if symptomatic</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyponatremia</td>
<td>Carbamazepine use (rise)</td>
<td></td>
<td>Na+</td>
</tr>
<tr>
<td>CNS</td>
<td>Tolerance to fentanyl, psychiatric disturbances</td>
<td>Anticonvulsant use</td>
<td></td>
<td>Assess effects of preop sedatives</td>
</tr>
<tr>
<td>MS</td>
<td>Tolerance to NMBs</td>
<td>Anticonvulsant use</td>
<td></td>
<td>Train-of-four monitoring in OR</td>
</tr>
</tbody>
</table>

**Key Reference:** Kofke WA. Anesthetic management of the patient with epilepsy or prior seizures. Curr Opin Anaesthesiol. 2010;23(3):391–399.
Seizures, Grand Mal (Tonic-Clonic)

**Risk**
- Incidence in USA: 500,000–1,000,000 with recurrent tonic-clonic seizures
- 10–20 million at risk to have one tonic-clonic seizure 2° to alcohol withdrawal, febrile convulsions (in children), CNS pathology, metabolic disturbances
- Benzodiazepines useful adjunct
- IV levetiracetam (Keppra) often used in intraop settings owing to lack of hemodynamic disturbance and limited drug-drug interactions
- Prudent to avoid drugs that may lower seizure threshold: Tricyclics, etomidate, ketamine

**Perioperative Implications**

**Preoperative Preparation**
- Ensure therapeutic anticonvulsant levels
- Provide protection from injury should seizure occur

**Monitoring**
- Routine
- EEG postop if poor emergence observed

**Airway**
- Evaluate for past seizure-induced oral trauma
- Gingival hyperplasia (phenytoin)

**Induction**
- Standard induction drugs provide anticonvul-sant action
- Benzodiazepines useful adjunct

**Maintenance**
- CV changes may be indicative of seizure

**Extrication**
- Exsufflate awake if possible
- Delayed emergence could signal postictal state or status epilepticus, EEG suggested

**Adjutants**
- Anticonvulsants for acute seizure: IV benzodi-azepines, propofol, barbiturates
- Load with phenytoin or barbiturate; oral drugs less reliable absorption
- Muscle relaxant doses altered by some anticon- vulsants

**Perioperative Risks**
- Intraop and postop seizures
- Status epilepticus (unrecognized intraop)
- Physical injury
- Delayed awakening
- Todd’s paralysis
- Pulm aspiration
- Transient hypoxemia, tachycardia, Htn
- Elevated intracranial pressure
- Caution with intraop IV phenytoin or fosphenytoin (hypotension, 50 μg/min and 150 μg/min limit respectively) or phenobarbital

**Worry About**
- Check serum anticonvulsant levels preop, consider free versus total serum phenytoin equivalent levels (pt receiving fosphenytoin) in nutritionally depleted pts. May be best to normalize if serum levels are low preop.
- Somnolence
- Projectile vomiting
- Acute and immediate intervention to termi-nate attack before cerebral injury results (30–60 min). Subtherapeutic anticonvulsant serum levels and alcohol withdrawal most commonly provoke status epilepticus.
- During seizures and postictally, airway reflexes are typically preserved—intubation not indicated unless aspiration is strongly suspected.
- Postictally, enhancement of previous neurologic motor deficit is common (Todd’s paralysis) for hours after seizure.

**Overview**
- Although typically a benign event, trauma to head or extremities is common if precautions not taken (padded hospital bed). May lead to status epilepticus, a life-threatening condition requiring active and immediate intervention to termi-nate attack before cerebral injury results (30–60 min). Subtherapeutic anticonvulsant serum levels and alcohol withdrawal most commonly provoke status epilepticus.
- During seizures and postictally, airway reflexes are typically preserved—intubation not indicated unless aspiration is strongly suspected.
- Postictally, enhancement of previous neurologic motor deficit is common (Todd’s paralysis) for hours after seizure.

**ICD-9-CM Code**: 345.3

**Etiology**
- Leading cause (30%) is idiopathic; undeter-mined fraction have genetic predisposition
- Acquired: 2° to congenital defects, perinatal asphyxia, trauma, hypoglycemia, CNS infection, drug withdrawal (alcohol most common), metabolic pathology resulting in low Na+, Ca++, or Mg++ or increased BUN

**Usual Treatment**
- For one seizure, no therapy necessarily required. Check serum anticonvulsant levels if there is a Hx of epilepsy determines underlying cause.
- Rule out hypoxia, STAI determination of serum glucose, electrolytes, and serum Ca++.
- For recurrent or prolonged seizures: IV lorazepam 1–5 mg, midazolam 5–10 mg, diazepam 5–10 mg. Alternatively, thiopental 1–2 mg/kg, propofol 1 mg/kg. Ventilatory assistance should be avail-able for greater dosage requirements.
- To prevent recurrence, IV anticonvulsants (fosphenytoin, levetiracetam, sodium valproate) should be considered to reach appropriate serum or dosage (levetiracetam) target level.
- STAT EEG is mandated in pts who do not emerge within 10–5 min to expected presure neurologic state in order to rule out continued seizures (nonconvulsive status epilepticus).

**ASSESSMENT POINTS**

<table>
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<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Gingival hyperplasia (phenytoin)</td>
<td>Seizure-induced oral trauma</td>
<td>Oral exam</td>
<td>CBC, electrolytes</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Drug-induced SIADH (carbamazepine)</td>
<td>Hypoglycemia, hypocalcemia</td>
<td></td>
<td>Drug levels</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxia</td>
<td>Sternal dullness, SO2, fever, supplemental</td>
<td>Auscultation</td>
<td>CBC, O2, sat</td>
</tr>
<tr>
<td></td>
<td>Postictal somnolence</td>
<td>Poor absorption of anticonvulsant</td>
<td>Low serum levels</td>
<td>Drug levels</td>
</tr>
<tr>
<td></td>
<td>Nonconvulsive status epilepticus</td>
<td>Drug-induced increase of hepatic P450</td>
<td>Increase dosage requirement of various drugs</td>
<td>Drug levels</td>
</tr>
<tr>
<td></td>
<td>Possible multiple CNS abn</td>
<td>Drug-induced transaminase elevation</td>
<td></td>
<td>Drug levels</td>
</tr>
<tr>
<td>CNS</td>
<td>Disturbed sensorium</td>
<td>Disturbed sensorium</td>
<td>Disturbed sensorium</td>
<td>Drug levels</td>
</tr>
<tr>
<td></td>
<td>Cognitive, motor deficits</td>
<td>Delayed emergence</td>
<td>Cognitive, motor deficits</td>
<td>EEG</td>
</tr>
<tr>
<td></td>
<td>Seizure-induced focal injury</td>
<td>Developmental Hx</td>
<td></td>
<td>EEG</td>
</tr>
</tbody>
</table>

Seizures, Intractable

René Tempelhoff

Overview
- Neurologic disease assoc with birth, congenital malformation, trauma, CNS pathology, idiopathic
- Periop risks increased for acquired seizure disorder, but furthermore some epilepsy and/or congenital malformations carry their own anesthetic risks
- Check type of seizures, clinical manifestations, duration, frequency
- Anticonvulsant therapy and side effects (liver function, level of consciousness)

ICD-9-CM Code: 780.3 (Seizure, recurrent)

Etiology
- Congenital (e.g., tuberous sclerosis and/or infantile seizure)
- Idiopathic
- CNS pathology: Trauma, tumor, hemorrhage

Usual Treatment
- Anticonvulsant and diet
- Surgery for ablation of foci
- GA regarded as a last resort for seizure that is unresponsive to sedative-hypnotics and resulting in decrease in consciousness or significant (<7.28) metabolic acidosis


Perioperative Implications

Preoperative Preparation
- Usual anticonvulsant regimen

Monitoring
- Routine monitors
- ETCO₂: Increase in CO₂ production could be indirect sign of seizure
- Consider EEG monitoring

Induction
- Have sodium thiopental and/or benzodiazepines to treat possible seizures
- Significantly higher requirement for nondepolarizing muscle relaxants and narcotics

Maintenance
- Avoid proconvulsants (ketamine, etomidate, enflurane, and probably sevoflurane)
- Continue scheduled anticonvulsants
- GA is sometimes used as treatment for status epilepticus

Extrusion
- To be delayed in case of doubt or situation such as:
  - High ETCO₂ despite adequate ventilation can be a sign of active seizure
  - Pt nonresponsive
  - Obvious convulsions
- Consider adding anticonvulsant (benzodiazepines) and ordering EEG

Adjuvants
- See specific anticonvulsant used

Postoperative Period
- Watch ETCO₂ on awakening, as high production may indicate seizure activity
- Resume anticonvulsants
- Treat seizures ad lib

Anticipated Problems/Concerns
- Seizures on induction and awakening are treated with first-line benzodiazepine Rx (e.g., lorazepam load) rather than long-acting anticonvulsants. The latter (e.g., phenytoin +/-leviteracetam) to be used after the seizure has been controlled.
- Evolution to status epilepticus: GA
- Sudden death (ventricular arrhythmias)
Seizures, Petit Mal (Absence)

Risk
- Incidence in USA: Approx 75,000–100,000
- Pure cases almost exclusively a risk in children, with age at onset 4–10 y

Perioperative Risks
- Risk of transition of petit mal absence seizures into tonic-clonic seizures or status epilepticus is exceedingly low.

Worry About
- Maintenance of serum anticonvulsant levels
- Inducing seizures with hyperventilation

Overview
- Relatively common seizure of childhood
- Seizure typified by brief absence (5–20 sec) with impairment of consciousness, 3/sec spike-wave EEG, mild facial motor manifestations
- Attacks may be few or occur >100/d
- Return to pre-seizure state without confusion and/or somnolence as typical with either temporal lobe or grand mal seizures
- Hyperventilation and bright flickering lights are common triggers.
- Atypical absence seizures may have more motor features and be of longer duration.
- Trauma from seizures rare, axial posture almost never affected
- No postictal sequelae; EEG and level of awareness return immediately
- Spontaneous resolution frequent in adolescence (25–30%); ~50% go on to develop tonic-clonic seizures

ICD-9-CM Code: 345.2

Etiology
- Strong genetic predisposition in otherwise normal children
- Structural lesions in adults

Usual Treatment
- Valproic acid (VPA) or ethosuximide (ESM) is drug of choice
- No emergent therapy required unless other seizure type present

ASSESMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Mild thrombocytopenia (VPA)</td>
<td>CBC with platelet count</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Hyperventilation may induce seizure</td>
<td>PE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>↑Liver enzymes (ESM, VPA)</td>
<td>GI Sx</td>
<td>SGPT, SGOT</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>EEG typically normal between seizures</td>
<td>EEG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Mild myoclonic movements</td>
<td>Movements</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Continue anticonvulsant therapy
- Verify adequate anticonvulsant levels: ESM 40–100 µg/mL, VPA >50 µg/mL (variable)

Monitoring
- No issues

Airway
- No issues

Preinduction/Induction
- Avoid bright flashing lights and hyperventilation

Maintenance
- Normocarbia unless otherwise indicated

Extubation
- Normocarbia

Adjuvants
- Muscle relaxant action is affected by some agents used to treat petit mal seizures

Postoperative Period
- Pain management beneficial if it results in avoidance of stress-induced hyperventilation

Anticipated Problems/Concerns
- Major periop morbidity rare
- Major concern is to document if other seizure types, such as tonic-clonic seizures, occur. Other seizure types would affect periop risk.
Septic Shock, Hyperdynamic; Systemic Inflammatory Response Syndrome (SIRS)

Risk
- Incidence in USA: Approx 750,000. Severe sepsis and septic shock is the tenth leading cause of death, and most common cause of death among critically ill pts in non-coronary ICUs
- Mortality rates are approx 25% for severe sepsis, 50% for septic shock
- Increased prevalence with advanced age, male gender, non-white ethnic origin, co-morbid diseases (COPD, cancer, chronic renal and liver disease, DM)

Perioperative Risks
- Hemodynamic and respiration instability
- Thrombocytopenia and DIC
- End-organ ischemia and worsening multisystem organ dysfunction

Worry About
- Rapid hemodynamic deterioration following induction of anesthesia 2° to limited physiologic reserve
- Blunted response to vasopressors and inotropes
- Early and appropriate initiation of antibiotics (initial choice is wrong in up to 20% of cases, and mortality increases with each hour of ineffective antimicrobial therapy)
- Multidrug-resistant bacteria (up to 25% of cases of severe sepsis and septic shock)
- Multisystem organ failure (mortality increases with each successive organ failure)

Overview
- Syndrome is a continuum from SIRS to Sepsis to Severe Sepsis to Septic Shock resulting in worsening inflammation and widespread tissue injury and ultimately leading to multisystem organ dysfunction

ICD-9-CM Code: 785.52

Etiology
- Environmental factors (exposure to infecting pathogen) plus possible genetic predisposition result in abn immune, coagulation, and inflammatory responses
- Gram-positive bacteria (MRSA, VRE, streptococcus) have become the most common causative pathogens. Other causative pathogens are gram-negative bacilli (Escherichia coli, Pseudomonas), and fungi (Candida)
- Most common site of infection is resp tract (pneumonia). Other common sites are genitourinary, abd, skin and soft tissue, device related (central lines), CNS, endocarditis.
- Also consider noninfectious causes of SIRS (burns, acute pancreatitis, trauma, thromboembolism, surgery)

Usual Treatment
- Speed and appropriateness of treatment administered affects outcome.
- General approach is trial of broad spectrum antimicrobial therapy (ideally within 1 hr of Dx), hemodynamic resuscitation (EGDT during first 6 hr) to maintain adequate perfusion pressure and optimize O, balance, and source control.
- Key considerations
  - Blood cultures ideally before antibiotic therapy
  - Imaging studies if warranted to confirm potential source of infection
  - Reassessment of antibiotic therapy to narrow coverage when appropriate
  - Vasopressors to maintain MAP>65 if fluid resuscitation is inadequate
  - Target Hb 7–9 in absence of tissue hypoperfusion, CAD, or acute hemorrhage
  - Lung protective ventilation strategy for ALI or ARDS
  - Other considerations
    - Stress dose steroids (hydrocortisone preferred) in septic shock only if BP has been poorly responsive to fluid and vasopressor therapy
    - Activated protein C (Caution: risk of bleeding) in severe sepsis or septic shock and clinical assessment of high risk of death
    - Addition of low-dose vasopressin infusion in refractory septic shock

Perioperative Implications

Preoperative Preparation
- Septic pts are often extremely unstable and have limited physiologic reserve.
- Surgery should be postponed until sepsis is treated unless underlying cause requires surgical intervention (source control).
- If surgery is urgent consider whether pt’s condition may be optimized before proceeding to the OR.

Intraoperative
- Goal for induction is hemodynamic stability
- Invasive monitoring is generally indicated
- Inotropes and vasopressors should be readily available
  - Target goal directed resuscitation to MAP >65, CVP 8–12, adequate UO, Normal pH, SVO2 >70
  - Consider steroids for refractory shock
  - Consider need for pt to remain intubated postprocedure

Postoperative Period
- Need for ICU care and possible prolonged mechanical ventilation

Anticipated Problems/Concerns
- Hemodynamic and resp instability
- Worsening metabolic acidosis, low central or mixed venous O, sat
- Multisystem organ dysfunction
- Prolonged ICU stay
- High morbidity and mortality

ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE | Test
---|---|---|---|---
NEURO | Altered mental status | Level of consciousness, delirium | Somnolent, obtunded, confused | Head CT if focal deficit
CARDIO | Vasodilation, hypovolemia, acidosis, hypocontractility, circulatory failure | Signs of end-organ hypoperfusion | Tachycardia, hypotension, wide pulse pressure, warm (or cold) extremities, low SVR, high CI, low SVO2 | Invasive hemodynamic monitoring, ECHO
PULM | Hypoxemia, hyperventilation, resp failure | Tachypnea, dyspnea | Use of accessory muscles, rapid and shallow breathing, cyanosis | CXR, ABG
RENAL | Oliguria, acute kidney injury, ATN | UO | Signs of hypovolemia, rising Cr, BUN | UO, Cr, BUN, urine I&O, UA
ID | Infection | Fever, chills, rigor | Hyper- or hypothermia | WBC with differential, cultures, radiographic imaging
HEME | Hemolysis, thrombocytopenia, DIC | Bleeding | | CBC, D-Dimer, INR, PT, fibrinogen


Peter Schulman
Shy-Drager Disease

Brad J. Hymel
Don D. Doussan

Risk
- More common in men than women
- Symptoms begin in 5th–7th decades

Perioperative Risks
- Autonomic dysfunction with CV collapse due to decreased sympathetic outflow
- Aspiration risk

Worry About
- Orthostatic hypotension and intraop BP fluctuations particularly during induction
- Obstructive sleep apnea—found in advanced stages
- Vocal cord paralysis—found in advanced stages
- Response to sympathomimetic drugs is unpredictable and may be extreme due to denervation hypersensitivity

Overview
- A parkinsonism-plus syndrome. 80% of pts present with Parkinson’s Disease.

ASSESSMENT POINTS

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<tr>
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<tbody>
<tr>
<td>HEENT</td>
<td>Vocal cord paralysis</td>
<td>Obstruction; apnea episodes; stridor particularly during sleep</td>
<td>Midline position of the cords after induction</td>
<td>Direct laryngoscopy</td>
</tr>
<tr>
<td></td>
<td>Obstructive sleep apnea</td>
<td>Snoring; dysphonia; dysarthria</td>
<td>Neck circumference</td>
<td>Polysomnograph</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Orthostatic hypotension</td>
<td>Syncope; dizziness; supine Htn with positional hypotension</td>
<td>Postural changes in BP</td>
<td>Tilt table test ECG</td>
</tr>
<tr>
<td></td>
<td>Fixed HR</td>
<td></td>
<td>Palpation</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Irregular resp</td>
<td></td>
<td>Ascultlation; visualization</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Gastroparesis</td>
<td>Early satiety, dysphagia</td>
<td>Loss of rectal sphincter tone</td>
<td>Electrolytes/BMP</td>
</tr>
<tr>
<td></td>
<td>Fecal incontinence, diarrhea, constipation, sodium loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Urinary incontinence</td>
<td>Nocturia; stress/overflow incontinence</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Atonic bladder</td>
<td>Sexual dysfunction</td>
<td></td>
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</tr>
<tr>
<td>CNS</td>
<td>Parkinsonian symptoms</td>
<td>Cogwheel rigidity</td>
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<tr>
<td></td>
<td>Anhidrosis</td>
<td>Shuffling gait</td>
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<tr>
<td></td>
<td>Heat intolerance</td>
<td>Anisocoria</td>
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<tr>
<td></td>
<td></td>
<td>Horner’s syndrome</td>
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</tr>
<tr>
<td>MS</td>
<td>Osteoporosis and aseptic necrosis (may be assoc with autonomic dysfunction)</td>
<td>Muscle atrophy</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Reduce venous pooling; increase peripheral vascular resistance; increase plasma volume. Care must by taken using these techniques in the attempt to decrease postural hypotension, as fluid overload can occur. Avoid drugs that cause excessive decreases in peripheral vascular resistance and sympatholytics during induction.

Monitoring
- Arterial and central venous catheters if fluid shifts likely to guide fluid replacement
- Temp—reduced sweating may lead to elevations in temp

Airway
- Vocal cord paralysis and dysautonomia with gastroparesis may make awake intubation the more desirable choice. Direct laryngoscopy allows the anesthesiologist to diagnose subclinical vocal cord abductor paralysis by observing the position of the cords.

Preinduction/Induction
- Consider steroid supplementation if on fluorinated corticosterone
- Consider effects of MAO inhibitors
- Avoid agents that may cause a decrease in cardiac output, decrease in HR, or vasodilatation, as profound hypotension may occur due to decreased sympathetic outflow.

Maintenance
- IPPV may cause a decrease in venous return and exaggerate hypotension.
- Norepinephrine stores at the nerve endings may be reduced. Therefore, the response to adrenergic drugs may be reduced or exaggerated: Use direct-acting drugs in small doses titrated to effect. Vasopressin has been used successfully in case reports when hypotension is not responsive to other sympathomimetics.
- Atropine may not increase the HR owing to parasympathetic deficiency

Extubation
- Awake

Postoperative Period
- Autonomic dysfunction. Continue invasive monitoring.

Anticipated Problems/Concerns
- Autonomic dysfunction with CV collapse
- Aspiration risk
**Sick Sinus Syndrome (SSS)**

**Risk**
- Highest incidence in patients older than 60 y
- Common in pts who have had congenital heart defect repair surgery
- Sinus node dysfunction (SNDS) is the most common indication for pacemaker implantation in North America.

**Perioperative Risks**
- Syncope, cardiac arrest
- Angina, CHF exacerbation, or acute onset HF

**Worry About**
- Sinus bradycardia that can be poorly responsive to atropine, and require a pacer intraop
- Tachy-brady event where an atrial tachycardia occurs, and its termination leads to a prolonged bradycardia or asystole.
- Tachy-brady event can lead to demand myocardial ischemia, and can precipitate heart failure in pt with related co-morbidities

**Overview**
- A significant portion of elderly pts with SSS frequently have other cardiac co-morbidities such as CAD.

**ASSESSMENT POINTS**

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<tbody>
<tr>
<td>CARDIO</td>
<td>Low cardiac output Tachy-brady event</td>
<td>Syncope, presyncope, lightheadedness, decreased exercise capacity, dyspnea on exertion, fatigue, confusion, memory loss, CVA (esp. in elderly)</td>
<td>Bradycardia, tachycardia</td>
<td>ECG</td>
</tr>
<tr>
<td>RENAL</td>
<td>Accentuate SSS</td>
<td>--</td>
<td>--</td>
<td>Potassium (hypokalemia)</td>
</tr>
</tbody>
</table>

**Etiology**
- Numerous and not clearly defined
- Elderly: Likely due to fibrosis of SA node, and hypersensitivity to autonomic changes
- Adults with congenital heart disease (esp. with ASD repair, or extensive atrial reconstruction): Likely due to surgical (direct or inflammatory) trauma to SA node.

**Usual Treatment**
- Undiagnosed until an episode of bradycardia in the OR: Atropine (0.5 mg–2 mg) and ephedrine (2–10 µg/min); pacing (external or transvenous). Pts with SSS often have a poor response to atropine.
- Diagnosed preop (i.e., ECG changes with symptoms): Pacemaker. Theophylline has been used to raise the resting heart rate. Pacemaker does not control tachyarrhythmia, but instead it allows antiarrhythmic therapy for tachycardia by pacing during bradycardia caused by the therapy.
- Anticoagulation with warfarin is used if continuous ECG monitor detects paroxysms of atrial tachyarrhythmia since this subset of pts has an increased thromboembolic risk.

**Perioperative Implications**

**Preinduction/Induction**
- Factors that alter autonomic balance can produce sinus bradycardia: Eye surgery, increased ICP, severe hypoxia, cervical/mediastinal tumors
- Optimize extrinsic factors that can decrease heart rate: Hyperkalemia, hypoxia, hypothermia, ICH, hypothyroidism
- Volatile anesthetics, propofol, vecuronium all decrease sinus node activity in a dose dependant fashion
- Standard monitors (pulse oximetry, ECG)
- Pt population that is dependent on their atrial systole for sufficient cardiac output (such as those with ischemic cardiomyopathy, aortic stenosis, or diastolic dysfunction), an atrial tachyarrhythmia (as part of the tachy-brady phenomenon) can lead to hypotension.
- MAC or elective general anesthesia in a pt with known SSS (i.e., with pacemaker)
- Consider using fentanyl, propofol, dexmedetomidine instead of inhaled anesthetics.

**Regional Anesthesia**
- Case reports show that SSS may manifest from several types of blocks incl a regional block that leads to sympathectomy, stellate ganglion blockade, thoracic epidural, and spinal anesthesia.
- Standard monitoring is recommended.

**Postoperative Period**
- If SSS manifests in pt without pacemaker intraop, consult cardiology for evaluation for pacemaker.

**Anticipated Problems/Concerns**
- An episode of atrial tachyarrhythmia that terminates in asystole or significant bradycardia
- Atrial tachyarrhythmia in a susceptible population (aortic stenosis, diastolic dysfunction) may itself compromise cardiac output.
- Symptomatic sinus bradycardia may not respond robustly (or at all) to atropine. It may need a pacing if a second agent (isoproterenol, epinephrine, ephedrine, or dopamine) does not work.

Sickle Cell Disease

Risk
- Affects persons with ancestors from areas endemic for P. falciparum malaria: Greece, Turkey, Italy, Arab Peninsula, India, Africa
- Incidence in USA: 1/500 African-Americans (0.2%) have sickle cell anemia
- Early mortality: Median age of death in men is 42 y and in women is 48 y

Perioperative Risks
- Pts have 30% overall complication rate; risk decreases with increased levels of fetal Hgb
- Complications incl anemia, stroke, acute chest syndrome, myonecrosis, heart failure, MI, hepatic or splenic sequestration, retinal hemorrhage, hematuria, renal failure, atelectasis and pneumonia, new-onset tonic-clonic seizure, intraop stasis and hypotension, wound infection, UTI, unexplained death

Worry About
- Degree of anemia, dehydration, sepsis, stress, acid-base status, hypoxemia
- Percentage of HbsS-containing cells
- Postop atelectasis and pneumonia
- Previous renal or heart failure
- Precipitation of vaso-occlusive crisis

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Hypoxemia due to sleep apnea</td>
<td>Snoring or sleep apnea Hx</td>
<td>Tonsillar hypertrophy</td>
<td>ABGs</td>
</tr>
<tr>
<td>CARDIO</td>
<td>MI; LV and RV dysfunction; CHF</td>
<td>Angina Sx; poor exercise tolerance; dyspnea</td>
<td>Displaced PMI S, S1</td>
<td>ECG, exercise ECG; ECHO, Hct</td>
</tr>
<tr>
<td>RESP</td>
<td>Acute chest syndrome; lung and rib infarction; pneumonia</td>
<td>Previous acute chest syndrome; dyspnea</td>
<td>Point tenderness over rib; rales; crackles</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Gallstones; sickle girdle syndrome (mesenteric ischemia); hepatic sequestration crisis</td>
<td>RUQ pain; abd pain</td>
<td>Jaundice; RUQ tenderness</td>
<td>Bilirubin</td>
</tr>
<tr>
<td>HEME</td>
<td>Sickle pain crisis; asplenia or splenic sequestration crisis; anemia; infection</td>
<td>Pain in affected areas; fatigue; sepsis</td>
<td>Pallor; splenic enlargement; flank tenderness; fever</td>
<td>Hgb, Hct, WBC, % HbsS Electrophoresis</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal failure and insufficiency</td>
<td>Hematuria; hemodialysis Hx</td>
<td></td>
<td></td>
</tr>
<tr>
<td>REPROD</td>
<td>Preterm labor and delivery; perinatal mortality; placenta previa; abruptio placenta</td>
<td>Vaginal bleeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Stroke; intracranial hemorrhage; pneumococcal meningitis; retinopathy and hyphema; seizure</td>
<td>Previous CNS Sx (weakness, TIA, or neurologic dysfunction); headache; vomiting or altered mental status</td>
<td>Focal deficits, stupor or coma; nuchal rigidity</td>
<td>Head CT; EEG</td>
</tr>
<tr>
<td>MS</td>
<td>Leg ulcers; myonecrosis; myofibrosis; infant hand-foot syndrome; shoulder or hip avascular necrosis; osteomyelitis</td>
<td>Pain in affected areas</td>
<td>ROM; skin changes; fever</td>
<td>WBC, UA, x-ray</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Latest data suggest there is no benefit in exchange transfusion preop. Rather, transfuse to an Hgb of 10 g/dL, independent of HbsS percent, with HbsA2 erythrocytes using extended matched transfusions (minor group E, K, C, Fya).
- Alkalization has no benefit.
- Autotransfusion: Predonated units and Hgb-based O2 carriers remain of unestablished efficacy
- Venous access may be difficult and a central line or implantable reservoir is useful.
- Preop hydration for 12 hr preceding surgery is controversial and many have abandoned excessive hydration.

Monitoring
- Routine
- Avoid central lines unless absolutely indicated given increased risk of infection

- If PA catheter indicated due to co-morbidity and surgical setting, an osmotic catheter is useful in providing continuous mixed venous blood O2 sat for assessment of O2 utilization and delivery.

- Airway
  - None

- Induction
  - Avoid oversedation, which may decrease respiration and lead to hypoxemia
  - Avoid hypovolemia
  - Retrobulbar blocks appear safe
  - No differences in morbidity or mortality shown among various anesthetic agents or between regional and GA techniques

- Maintenance
  - Cardiopulmonary bypass presents special problems causing dilutional anemia, mechanical hemolysis, hypothermia, low-flow state, and plt activation

- Tourniquet use is relatively contraindicated, but unproven to show increased risk for sickle pts

Extubation
- Analgesic-induced resp depression at extubation may contribute to atelectasis, pulm infections, and hypoxemia

Postoperative Period
- Adequate hydration; analgesia; pulm toilet, incl incentive spirometry; supplemental O2 therapy for 12–48 hr postop

Anticipated Problems/Concerns
- All blood transfusions in these pts carry high risk for hemolytic reaction due to previous exposure
- Avoid all situations leading to hypoxemia, hypovolemia, or stasis
- Organ damage is due to vaso-occlusive ischemia, which occurs because the sickled cells are unable to traverse narrow capillary beds, leading to distal blood flow impairment. Also, there is an enhanced tendency for sickle cells to adhere to the endothelium and cause release of vasoactive substances.

ICD-9-CM Code: 282.60
- See also Sickle Cell Trait in Diseases section

Etiology
- Molecular lesion is on β-chain of Hgb at position 6 Gln → Val
- Sickle erythrocytes are more fragile with shortened life span, which leads to chronic hemolysis and anemia

Usual Treatment
- Vaccines against pneumococcus and Haemophilus influenzae type b, and prophylactic penicillin therapy effective in autopsplenectomized pts
- Palliative care for painful crisis
- Simple and exchange transfusions
- Hydroxyurea to increase fetal Hgb
Sickle Cell Trait

Risk
- Incidence in USA: 2.5 million; 300 million in world
- Race with highest prevalence: African-Americans

Perioperative Risks
- Increased risk of complications following CABG
- Periop mortality rate in published cases of SA trait is 0.8%
- Some increased risk of CVA and pulm infection but not well quantified

Worry About
- Increased risk of vaso-occlusive phenomenon with hypoxia and stress
- Sudden death with stresses such as vigorous exercise may make one worry as recovery from surgery may be considered a similar stress as vigorous exercise

Overview
- Is not a disease
- Is not a cause of an in blood count
- Does not produce vaso-occlusive symptoms under physiologic conditions—painful crisis not a hallmark or concomitant of condition

- Does not adversely affect individual’s life expectancy
- Dx established by Hgb electrophoresis

ICD-9-CM Code: 282.5
- See also Sickle Cell Disease in Diseases section

Etiology
- Heterozygous in which individual has one beta S and beta A globin gene (SA disease)

Usual Treatment
- None, except iron supplementation (debated)

- See also Sickle Cell Disease in Diseases section

Overview
- Is not a disease
- Is not a cause of an in blood count
- Does not produce vaso-occlusive symptoms under physiologic conditions—painful crisis not a hallmark or concomitant of condition

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ICD-9-CM Code: 282.5
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- Heterozygous in which individual has one beta S and beta A globin gene (SA disease)

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- None, except iron supplementation (debated)

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- Is not a disease
- Is not a cause of an in blood count
- Does not produce vaso-occlusive symptoms under physiologic conditions—painful crisis not a hallmark or concomitant of condition

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ICD-9-CM Code: 282.5
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Etiology
- Heterozygous in which individual has one beta S and beta A globin gene (SA disease)

Usual Treatment
- None, except iron supplementation (debated)

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<tbody>
<tr>
<td>RESP</td>
<td>Pulm embolism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Hgb level usually 13–15 g/dL</td>
<td>Hx SOB: poor exercise tolerance 10–40% of Hgb S—same cells as Hgb A</td>
<td>Hgb</td>
</tr>
<tr>
<td>GU</td>
<td>Painless hematuria and bacteriuria; pyelonephritis (esp. with pregnancy) RO polycystic kidney disease</td>
<td>UA (culture if prosthesis planned)</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Stroke</td>
<td>Migraine headache</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Warm room
- Consider prehydration

Monitoring
- Temp

Airway
- Occasionally distorted anatomy 2° to extramedullary erythropoiesis
- Sinusitis possible
- Prehydrate liberally if CV status will tolerate

Induction
- Routine

Maintenance
- Keep warm
- Keep vasodilated
- Keep without stasis
- High O₂ content

Extubation
- Keep warm

Adjuvants
- Vary if hepatic or renal insufficiency exists

Postoperative Period
- Aggressively prevent pain, hypovolemia, and hypothermia

Anticipated Problems/Concerns
- Stroke and/or pulm emboli or infection have been reported after CPB. Five of 544 pts in literature of sickle trait disease died periop.
Silicosis

Karen B. Domino

Overview
- Silicosis-pulm fibrosis commonly occurs after 4–5 y (acute, very rare), 5–10 y (accelerated), or >10 y (chronic) of occupational exposure
- In advanced stage, both obstructive (graduated loss of FEV1, FVC and decrease of FEV1/FVC ratio) and restrictive ventilatory defects, as well as decreases in diffusing capacity, are common; ejection dysplasia is the predominant symptom. 
- CO2 retention, pulm Htn, cor pulmonale late in the course
- Assoc TB, lung cancer, connective tissue diseases (scleroderma, rheumatoid arthritis, Sjogren’s syndrome), nephritic syndrome, and renal failure

ICD-9-CM Codes: 502 (Nodular); 503 (Non-nodular)

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Pulmonary Htn</td>
<td>Dyspnea</td>
<td>S1, Peripheral edema</td>
<td>ECG</td>
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<tr>
<td></td>
<td>Cor pulmonale</td>
<td>Exercise tolerance</td>
<td>Distended neck veins</td>
<td>CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm fibrosis</td>
<td>Cough</td>
<td>Rales, rhonchi</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>Bulla/bleb formation</td>
<td>Sputum production</td>
<td>Wheezing</td>
<td>ABGs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dyspnea</td>
<td>Cyanosis</td>
<td>PFTs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Exercise tolerance</td>
<td>Use of accessory resp</td>
<td>Inspir force</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>muscles</td>
<td>Diffusing capacity</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>RR</td>
<td>Lung biopsy/lavage fluid</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>microscopy</td>
</tr>
<tr>
<td>GI/ENDO</td>
<td>Wt loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hyperglycemia (in chronic steroid treatment)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Generalized weakness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td></td>
<td>Htn</td>
<td>Serum creatinine, BUN, K+</td>
</tr>
<tr>
<td>IMMUNO</td>
<td>Hilar adenopathy (eggshell calcification)</td>
<td></td>
<td>Oliguria</td>
<td>Creatinine clearance</td>
</tr>
<tr>
<td></td>
<td>Increased susceptibility to infection, esp. pulm</td>
<td></td>
<td>Cough</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fever</td>
<td>Sputum culture and sensitivity</td>
</tr>
</tbody>
</table>


Preoperative implications

Preoperative Preparation
- Lung condition optimization: Treat bronchospasm (if present), bronchitis, other pulm infections, possible lung laveage
- Consider steroids (short course)
- Stop smoking at least 24 hr before surgery

Monitoring
- Pre- and postop: Consider repetitive ABGs, lung mechanics (RR, TV, MV, FVC, NIP, etc.)
- Intraop: Arterial line; CVP is controversal. Consider PA catheter if pulm Htn is present and/or significant fluid shifts are expected

Preinduction/Induction
- Caution with IV agents that depress ventilation and regional techniques that affect accessory muscles of respiration (e.g., high epidural and inter-scalene blocks)
- Maintain adequate preload, optimize cardiac output. Avoid hypoxemia, hypercapnia, and acidosis (both resp and metabolic), as these may increase PA pressures and worsen cor pulmonale

Airway
- In case of difficult airway, consider techniques with spontaneous respiration preservation (e.g., awake FOI)

Maintenance
- Consider pressure-controlled mode of ventilation, for poor lung compliance may require increased airway pressures to reach the adequate TV. Observe for spontaneous pneumothorax, esp. in severe disease
- Optimize volume status, while avoiding crystalloids’ overload; rather, use colloids. If possible, minimize blood products use to avoid lung injury.
- Avoid hypotension. Treatment may incl low doses of vasopressin that decrease PA pressures while maintaining systemic BP; rather than nor-epinephrine, which increases PAP and promotes acidosis
- For severe metabolic acidosis treatment, consider THAM. Bicarbonate should be avoided because of excessive CO2 production and hypernatremia.

Perioperative Risks
- Hypoxemia, bronchospasm, pneumothorax, atelectasis, mycobacterium (30-fold increased risk for TB) and fungal infection, bacterial pneumonia, chronic bronchitis exacerbation
- Periop resp failure, esp. following thoracic and upper-abd surgery
- Pulm Htn, cor pulmonale
- Renal insufficiency (tubular nephropathy)
- Steroid-induced diabetes (in cases of chronic steroid treatment)

Worry about
- In cases of assoc scleroderma and /or rheuma- toid arthritis, possible difficult intubation
- Bronchospasm, chronic bronchitis exacerbation
- Resp failure
- Cor pulmonale

General Risk
- Silicosis is irreversible fibronodular lung disease caused by inhalation of dust, containing crystal-line silica alpha-quartz or silicon dioxide, during occupational exposure
- Currently, >1,000,000 workers exposed; 200–300 deaths/y; protection devices decrease incidence
- Mostly older than 65 y
- Males>females
- No racial predilection

Perioperative Risks
- Hypoxemia, bronchospasm, pneumothorax, atelectasis, mycobacterium (30-fold increased risk for TB) and fungal infection, bacterial pneumonia, chronic bronchitis exacerbation
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- For severe metabolic acidosis treatment, consider THAM. Bicarbonate should be avoided because of excessive CO2 production and hypernatremia.

- Consider use of remifentanil. Caution with long-acting opioids.
- For muscle relaxation, short- and medium-acting agents titrated to effect may be preferred. Consider cisatracurium or rocuronium.
- Any of inhalational agents are adequate options.

Extubation
- Consider temporary postop mechanical ventilation, esp. for upper abd and thoracic surgery, until stringent criteria are met.

Postoperative Period
- Pain management is critical for adequate respiration and to avoid pulm Htn

Adjuvants
- Bronchodilators, supplemental O₂, incentive spirometry may improve ability to wean

Anticipated Problems/Concerns
- Increased risk of resp failure and complications esp. after upper abd and thoracic surgery
- Pts with cor pulmonale at increased risk of cardiac complications
Single (Including Common) Ventri

**Risk**
- Hypoplastic left heart syndrome (HLHS) is the most common single ventricle (SV) congenital cardiac malformation.
- HLHS accounts for 7.5% of newborns with CHD.
- Male predominance for HLHS

**Perioperative Risks**
- Risk of paradoxical emboli
- Risk of complications of chronic hypoxemia: Hyperviscosity, coagulation factors and plts
- Risk of surgical shunts—narrowing of vessels anastomosed, obstructed shunts
- Hypoxemia-induced poor pulm blood flow or shunt occlusion
- Additional risk specific to anatomy and planned procedure

**Worry About**
- Impact of changes in PVR, SVR, and cardiac function on blood flow, cardiac output and O₂ sat
- Diastolic pressure and coronary perfusion
- Artioventricular (AV) valve regurgitation
- Systolic and diastolic dysfunction
- Assoc anomalies

**Overview**
- Wide variety of lesions usually assoc with atresia of the ipsilateral AV or semilunar valve resulting in SV physiology
  - Tricuspid atresia (TA) is the prototypic single left ventricle. See TA in Diseases section.
  - HLHS with mitral and aortic stenosis/atresia is the prototypic single right ventricle.
  - Other anatomic incl unbalanced AV canal, some double inlet or double outlet ventricles, some heterotaxies.
  - Initial lesion requires mixing of systemic and pulm venous return at atrial (ASD) or ventricular (VSD) level. The SV output is divided between pulm and systemic circulations.

- Single ventricle anatomy may be assoc with hypoplasia of a great vessel (pulm artery or aorta) and prior to initial palliation; systemic or pulm blood flow may be dependent on duc tus arteriosus patency.
- Balance of blood flow in each circulation (Qp:Qs) is governed by the relative resistance to flow as determined by both anatomic and vascular resistance considerations.
- Goal throughout all stages is to balance the Qp:Qs at 1:1
  - With complete mixing, Qp:Qs at 1:1 results in sat of 75-80% at FIO₂ 0.21
  - FIO₂, CO₂, and pH management can be used to manipulate the Qp:Qs.
  - Qp:Qs > > 1 results in pulm overcirculation/pulm vascular congestion and potentially hypoperfusion to end organs.
  - Qp:Qs < < 1 results in hypoxemia

**ICD-9-CM Code: 746.7 (HLHS)**

**Etiology**
- Congenital defect, unclear etiology

**Usual Treatment**
- Series of palliative procedures with following goal: Creation of reliable systemic and pulm blood flow
- Staged connection of systemic venous return directly to pulm artery, dedicating the SV to systemic circulation
- First, stable blood flow to systemic and pulm circulations are established and balanced.
  - For TA, a Ballock-Tausig (BT) shunt is placed (See BT Shunt in Procedures section)
  - For HLHS, a stage I Norwood procedure is performed
  - For other SV lesions, BT shunt or PA banding as dictated by anatomy
- Complete intracardiac mixing of blood is imperative.

**Risk**
- Stage I Norwood
  1. A neoaoa is created from hypoplastic aortic arch and native PA tissue, connecting the SV to systemic circulation.
  2. A BT shunt provides pulm blood flow, connecting branch of neoaoa to ipsilateral pulm artery.
  3. An atrial septectomy is performed for to ensure complete intracardiac mixing of systemic and pulm venous blood.
- At completion of the stage I Norwood, the SV provides cardiac output to the systemic circulation via the neoaoa and the pulm circulation via the BT shunt.
- The second stage is the first of two procedures to direct systemic venous return to the pulm
- The SVC is connected to the ipsilateral PA, which remains connected to the PA confluence.
  - This procedure is referred to as a cavopulmonary connection, Bi-directional Glenn or hemi-Fontan and is commonly performed around 6 mo of age.
  - Low PVR is necessary to promote pulm blood flow, which is passive.
  - The final stage, Fontan completion, is typically done 18 mo-5 y.
  - The IVC blood is directed to the ipsilateral PA, either intracardiac via lateral tunnel or extracardic via graft.
  - This effectively separates the circulations and reduces volume workload on SV; systemic venous return now flows passively to the PA without interposed pumping chamber.
  - A small fenestration from the IVC-PA conduit to the atrium is sometimes created. The fenestration ensures preload to the systemic circulation when PA pressures fluctuate, maintaining cardiac output but at the expense of decreased O₂ sat via right-left shunt.

**Perioperative Implications**

**Preoperative Preparation**
- Depending on the stage of the palliative process (Norwood stage I, Glenn/hemi-Fontan, completion Fontan), optimize hemodynamics.
- Cardiac catherization is typically performed prior to Glenn/hemi-Fontan to measure PVR and coil any collateral venous vessels.
- Higher O₂ sat can ↓ O₂ delivery to the tissues by facilitating overcirculation to the lungs, particularly when pulm blood flow is via BT shunt.

**Monitoring**
- Arterial BP
- CVP monitoring via IJ is controversial due to SVC thrombosis risk and implications for subsequent staging, which requires patency of these vessels.
- Consider TEE

**ASSESSMENT POINTS**

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<tbody>
<tr>
<td>CARDIO</td>
<td>CHF</td>
<td>Dyspnea, tachypnea, feeding difficulties</td>
<td>S₉, rales, wheeze, enlarged liver, metabolic acidosis</td>
</tr>
<tr>
<td>HEME</td>
<td>Polycythemia</td>
<td>See above</td>
<td>See above</td>
</tr>
</tbody>
</table>

**Preinduction/Induction**
- Dependent on exact anatomy and stage of palliation
- Induction technique should consider impact of PVR and SVR changes on myocardial, systemic, and pulm blood flow.

**Airway**
- ET intubation and PPV
- Minimize intrathoracic pressures where possible to encourage pulm blood flow.

**Maintenance**
- IV or inhalational agents are acceptable.
- Body temp as dictated by potential use of cardiopulmonary bypass

**Extubation**
- Following stage I Norwood, pt requires mechanical ventilation for >2 d.
- Early extubation is recommended to facilitate pulm blood flow after stage II (Glenn or hemi-Fontan) or the completion Fontan. High intrathoracic pressure from PPV impedes venous flow to the pulm circulation, while negative intrathoracic pressure (spontaneous respiration) enhances flow.

**Anticipated Problems/Concerns**
- Overcirculation
- Hypoxemia
- New anatomy post-procedure will necessitate a reassessment of desired PVR and SVR to optimize flow to both circulations.
- Postop low cardiac output syndrome

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Sleep Apnea, Central and Mixed

Risk
- Incidence in USA: 2 to 9% of middle-aged adults (increased 3-fold in last 10 y, presumably due to increase in obesity); M:F ratio: 2:1; obstructive or mixed
- Risk increases with male sex, upper middle age (55 to 64), obesity, Hx of snoring with impaired daytime performance
- In elderly, risk is 2x higher for African-Americans

Perioperative Risks
- Increased risk of central and mixed (central and obstructive) apnea. In mixed SAS, obstructive apnea component can mask central apnea.
- Risk for resp depression also in intubated, tracheotomized, and awake pts
- Increased risk with sedative-hypnotic narcotics, postop with any form of pain relief

Worry About
- See medical records for previous problems.

ASSESSMENT POINTS

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<td>Dyspnea at rest, DOE</td>
<td>Cardiomegaly</td>
<td>ECG, ECHO</td>
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<td>RESP</td>
<td>Right-sided heart dysfunction, snoring, resp dysfunction, DOE</td>
<td>Awakening at night with grunts</td>
<td>Venous engorgement, Rapid resp rate</td>
<td>SaO, supine</td>
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<tr>
<td>GI</td>
<td>Hepatic dysfunction Full stomach NIDDM</td>
<td>Jaundice, bleeding disorders, ascites, heartburn, hiatus hernia, polydipsia, polypolia</td>
<td>Hepatomegaly, asites, spider nevi, jaundice</td>
<td>LFTs, PT, TTT</td>
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<td></td>
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<td>Polycythemia</td>
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<tr>
<td>CNS</td>
<td>Disturbed sleep, impaired daytime performance, morning headache, memory problems, irritability</td>
<td>Daytime sleepiness, complaints of disrupted sleep Ask for encephalitis, autonomic neuropathy, brainstem damage</td>
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<td></td>
<td></td>
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<td>Plethora, clubbing, cyanosis</td>
<td>O2, sat, Hct</td>
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Perioperative Implications

Preoperative Preparation
- Take sleep Hx, if possible from bed partner
- If a question of sleep apnea, can use home sleep apnea tests (helmet, or wrist—two distinct types now exist) as a screen—do not need sleep lab for this screen. If positive refer to sleep lab preop.
- Avoid preop sedation with benzodiazepines and narcotics
- Examine airway carefully
- Consider metoclopramide 10 mg, metoclopramide 300 mg PO the night before and IV preop.
- Assess myocardial and volume status.
- Initiate CPAP therapy over periop period, and in recovery room.

Monitoring
- Routine; consider arterial line
- UO, possible CVP or PA catheter if volume status likely to be significantly altered

Airway
- Airway control necessary if prominent central component and sedation mandatory
- Awake, siting, fiberoptic intubation may be indicated if difficulty anticipated.

Induction
- Pt may need to remain semi-sitting if SaO2 drops when supine. Preoxygenation should be complete throughout lung.

Maintenance
- Oxygenation may deteriorate with upper abl surgery or increased intra-abdominal pressure
- Consider the use of short-acting substances (e.g., propofol, remifentanil)
- Minimize postop sedation

Exubation
- Extubate as soon as pt maintains normocapnia and responds to command.
- Consider close monitoring after extubation.

Adjuvants
- Initial dose of induction agent and narcotics calculated on a kg/kg basis and muscle relaxants calculated on estimated lean body mass
- Subsequent doses of sedatives, hypnotics, relaxants, and narcotics calculated on estimated lean body mass

- Regional anesthesia if physically possible and if pt can use accessory muscles to help with breathing

Postoperative Period
- Pain control with opioids only when NSAIDs and/or regional anesthesia is contraindicated and/or insufficient, as (sudden) complete pain relief may increase risk of resp arrest
- Some think epidural or narcotic indicated and others think relatively contraindicated
- Extended resp monitoring
- Stabilize ABGs to adequate levels
- Pain control necessary. PCA acceptable in sleep apnea, but not in continuous mode.

Anticipated Problems/Concerns
- Resp insufficiency and pneumonia postop; use devices and/or CPAP in immediate and long-term pre- and postop periods
- Postop thromboembolic phenomena
- If problems occur, inform pt before discharge with written instructions, esp. for further anesthetic interventions.
Sleep Apnea, Obstructive

Risk
- Incidence in USA: 2–9% of whole population (increased 3-fold in last 10 y, presumably due to increase in obesity)
- M:F ratio: 2:1
- Race with highest prevalence: Unknown

Perioperative Risks
- Increased risk of pulm Htn, RV failure, systemic Htn
- Some pts may be polycythemic and have an increased risk of CVA
- Complications assoc with obesity, and craniofacial and upper airway soft tissue abn
- Increased risk in supine position of sudden arrest postop

Worry About
- Airway obstruction with sedating drugs: Need for awake, sitting intubation without sedation if obstructs when supine
- Increased sensitivity to sedating drugs
- Difficult airway management: Mask ventilation and intubation
- Aspiration risk in morbidly obese
- Postop airway obstruction or resp depression
- Nasal obstruction from NG tubes, e.g., may lead to resp compromise
- Have pt bring CPAP or other apparatus with them to hospital and to OR/PACU

Overview
- Apnea refers to cessation of airflow at the mouth for >10 sec
- Sleep apnea: Repetitive episodes of upper airway occlusion during sleep, often with O₂ sat to 85%, nearly always assoc with loud snoring. Episodes of apnea often terminate with a snort or gasp.
- Upper airway obstruction from relaxation of muscles of oropharynx
- Frequent periods of apnea lead to hypoxia and hypercarbia, which could lead to cor pulmonary.
- Polycythemia may result from chronic hypoxia.
- Nocturnal cardiac arrhythmias are common.
- Monitoring depth and quality of sleep along with cardiopulmonary variables in those with severe symptoms
- Other name is Pickwickian syndrome assoc with morbid obesity (see also Morbid Obesity in Diseases section)

ICD-9-CM Codes: 780.57, 278.0 (Morbid obesity)

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<td>CARDIO</td>
<td>Htn</td>
<td>Dyspnea at rest and on exertion Poor exercise tolerance</td>
<td>Rapid resp rate ↑ BP, cardiomegaly</td>
<td>ECG; ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Right-sided heart dysfunction Restrictive dysfunction</td>
<td>Snoring; partner gives Hx of pt's awakening with grunts at night DOE</td>
<td>Venous engorgement, rales, S₁ and S₂ cardiomegaly</td>
<td>Pulse oximetry on room air while supine ECG, CXR, ABGs, Hct, polysomnogram</td>
</tr>
<tr>
<td>GI</td>
<td>Hepatic dysfunction Full stomach NIDDM</td>
<td>Angina Jaundice, bleeding disorders, ascites Heartburn; hiatus hernia Polydipsia, polyuria</td>
<td>Hepatomegaly, asites, spider angiomas, jaundice</td>
<td>LFTs, PT, PTT Fasting glucose</td>
</tr>
<tr>
<td>ENDO</td>
<td>Obesity Hypothyroidism Acromegaly</td>
<td>Mental function Reflexes BMI</td>
<td>Free T₁, estimate TSH level; GH level</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Polycythemia</td>
<td>Plethoric clubbing; cyanosis</td>
<td>Hypoxemia</td>
<td>Hct</td>
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<tr>
<td>CNS</td>
<td>Disturbed sleep Memory problems Irritability</td>
<td>Daytime sleepiness Complaints of disrupted sleep</td>
<td></td>
<td>Polysomnogram</td>
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Preoperative Preparation
- Avoid sedatives
- Assess CV status
- Histamine H₁ blockers, metoclopramide, and antacids for morbidly obese pts
- Have pt bring CPAP or other apparatus with them to hospital and to OR/PACU

Monitoring
- Routine
- Volume status if RV dysfunction present
- Consider arterial catheter if BP cuff doesn’t fit or takes too long to inflate

Airway
- Airway obstruction with induction—see HEENT
- Awake intubation in those with potentially difficult airway
- Consider elevating shoulders on bolsters
- Airway obstruction from pulm Htn by hypoxemia and hypercarbia
- Volume status may change precipitously with position change
- Oxygenation may deteriorate with upper abd surgery or increased abd pressure

Anticipated Problems/Concerns
- Airway obstruction at induction and after extubation
- 13% risk of periop complications esp. of pneumonia; avoided by minimal sedation, appropriate pain control, early ambulation
- Worsening pulm Htn and right-sided heart failure
- Aspiration risk in morbidly obese
- Postop thromboembolism
- Poor motivation resulting in poor ambulation. Avoided by intensive preop teaching and postop coaching.

Induction
- Airway obstruction
- Exacerbation of pulm Htn by hypoxemia and hypercarbia

Maintenance
- Airway obstruction from residual anesthetics
- Avoid opioids and sedatives
- Monitor for airway obstruction and apnea

Adjutants
- Very sensitive to CNS-depressant drugs
- CPAP or other apparatus for use in PACU and hospital or home recovery periods

Etiology
- Cessation of airflow due to complete obstruction of upper airway
- Narrowing due to enlarged tonsils, adenoids, uvula, low soft palate, or craniofacial abn superimposed on co-existent abn of upper airway muscle tone and/or neurologic control
- Obesity exacerbates upper airway obstruction
- Structural abn such as tonsillar hypertrophy, enlarged tongue, and micrognathia may contribute to airway obstruction

Usual Treatment
- Wt loss in overweight pts
- Avoidance of alcohol and sedatives before sleep
- Nasal CPAP
- Physical aids such as devices to detect and prevent snoring, keep pt off back while sleeping (e.g., tennis ball sewn on nightshirt)
- Nasopharyngeal or oropharyngeal airway
- Uvulopalatopharyngoplasty
- Tracheotomy in extreme cases
- Electrohydric pacing for central sleep apnea

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- Nasal CPAP
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Nasopharyngeal or oropharyngeal airway
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- Electrohydric pacing for central sleep apnea
Spasmodic Torticollis

Risk
- Estimated prevalence of 9 cases per 100,000
- Spasmodic torticollis (ST), also known as cervical dystonia, is the most common form of focal dystonia
- Peak incidence in the fifth decade
- Two times more common in females
- 80% of cases are sporadic or primary
- 20% of cases are 2° to an underlying brain lesion or trauma

Perioperative Risks
- Dysphagia
- Aspiration
- Consider co-morbid neurologic problems such as seizures, cranial nerve palsies, hemiplegia, etc.

Worry About
- Difficult pt positioning 2° to sustained muscle contractions
- Difficult intubation as a result of poor extension of the cervical spine and diminished mouth opening

Overview
- ST is defined as twisting of the neck caused by involuntary muscle contractions.
- Idiopathic ST is a slowly progressive disease that manifests between the third and fifth decades. Idiopathic ST is likely caused by abn of the basal ganglia circuitry.
- The dystonia typically progresses over 3 to 5 y before it plateaus.
- Pain occurs in 75% and contributes to disease disability.
- If ST occurs acutely, must rule out causes related to trauma, medications (metoclopramide, halol, phenothiazines), intracranial abn (tumors, AVMs, hemorrhages), and neck pathology (retropharyngeal abscess).
- The sternocleidomastoid and trapezius muscles are most commonly involved, but extracervical dystonia may occur in 20%.
- Jerking of the head and head tremors are common features.
- Head positioning determines the type of torticollis
  - Rotational torticollis: Rotation of the head around the long axis of the neck with the chin turned toward a particular side
  - Anterocollis: Head tilts forward with neck flexion
  - Retrocollis: Head tilts backward with neck extension

Monitoring
- Routine

General Anesthesia
- Propofol is likely to be safe with all dystonias.
- General anesthesia with thiopental, succinylcholine, atracurium, isoflurane, and fentanyl are thought to be safe in spasmodic torticollis.

Regional Anesthesia
- Limited reports available but thought to be safe
- Postop period
- Risk of aspiration

Anticipated Problems/Concerns
- Anticipate difficult intubation 2° to fixed head turning from muscle contractures that do not respond to muscle relaxants.
- Be aware of cervical spine pathology that may result from prolonged torticollis.
- Neurologic conditions such as cranial nerve dysfunction and seizure disorders may accompany 2° spasmodic torticollis caused by an underlying intracranial lesion.
- Spasmodic torticollis can cause head tremors, which should not be confused with hyperkinetic movement disorders.

ICD-9-CM Code: 333.83 (Spasmodic torticollis)

Etiology
- Likely a genetic component that contributes to the development of spasmodic torticollis.
- Trauma, medications, and intracranial pathology can cause focal dystonic reactions such as torticollis.

Usual Treatment
- Chemical denervation with botulinum toxin is the mainstay of therapy. Botulinum toxin is injected into overactive muscles in the neck that are responsible for the dystonia. It usually takes a week to take effect and lasts up to 3 mo before a repeat injection must be performed.
- Pharmacological therapy with anticholinergics, benzodiazepines, and baclofen are used as adjuncts to botulinum toxin.
- Surgical options incl mechanical denervation of affected muscles, deep brain stimulation, and intrathecal baclofen if spasticity is prominent.

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Perioperative Implications

Preinduction/Induction/Maintenance
- Routine considerations
- Consider the use of nondepolarizing muscle relaxants.
- NMB may have no effect on muscle contractures, which are permanently shortened muscles that result from structural muscle changes.
- Anticipate the use of fiberoptic intubation.

Preoperative Considerations
- Consider preop Botox injections at least 1 wk prior to anesthesia.
- It is imperative to preop evaluate the range of cervical spine extension, maximal mouth opening, and integrity of the temporomandibular joint.
- Estimated prevalence of 9 cases per 100,000
- Spasmodic torticollis (ST), also known as cervical dystonia, is the most common form of focal dystonia
- Peak incidence in the fifth decade
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- Risk of aspiration

Anticipated Problems/Concerns
- Anticipate difficult intubation 2° to fixed head turning from muscle contractures that do not respond to muscle relaxants.
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Subclavian Steal Syndrome

Dolores B. Njoku
Sapna R. Kudchadkar

Risk
- Uncommon entity with a variably reported clinical significance
- M:F ratio: 2:1

Perioperative Risks
- Stroke from a plaque originating from vertebral artery system

Worry About
- Worsening neurologic symptoms

Overview
- Retrograde blood flow from vertebral artery to distal subclavian 2° to proximal ipsilateral subclavian or innominate artery stenosis or occlusion
- Presence of other extracranial arterial disease is a prerequisite to development of symptoms
- Criteria for diagnosis: Must be symptomatic
  - Cerebral ischemia causing neurologic symptoms assoc with ipsilateral arm exercise

Etiology
- Most commonly atherosclerosis
- Other causes incl Takayasu’s arteritis, tumor, Hx of aortic stenting/grafting for aneurysm, and previous surgery
- Rare causes incl congenital atresia of first portion of left subclavian, hypoplastic arch with severe coarctation, or stenosis of left subclavian at old suture site of a coarctation repair

Usual Treatment
- Surgical
  - Common carotid to subclavian artery bypass graft
  - Subclavian-to-subclavian artery bypass graft
  - Axillary-to-axillary artery bypass graft
- Nonsurgical
  - Percutaneous transluminal angioplasty and stent placement

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| CARDIO | Claudication      | Bruit | Difference in brachial systolic BP of at least 20 mm Hg
Bruit at base of neck or supraclavicular area on affected side (proximal subclavian artery)
Reactive hyperemia: Temporary cuff inflation causes peripheral vasodilation distal to cuff,
when released results in increased demand leading to neurologic symptoms
Color Doppler ultrasound: Ipsilateral vertebral artery flow reversal with a parvus tardus
waveform in the ipsilateral subclavian artery confirms the diagnosis of SSP
Vascular structures well demonstrated by contrast-enhanced MRA
Flow reversal well demonstrated by flow-encoded MRI |
| CNS    | Vertigo          | Retrograde catheter | Angiogram |
|        | Rarely cortical visual disturbances, ataxia, syncope, dysarthria | |
| MS     | Paresis/paresthesias | See CV |


Perioperative Implications

Preoperative Preparation
- Bilateral upper extremity BP in pts undergoing surgery characterized by large variations in hemodynamic status or in pts with previous internal mammary-coronary bypass grafts

Monitoring
- Consider arterial catheterization since BP maintenance may be essential for cerebral perfusion

- Consider CVP monitoring and/or PA catheterization if contributing factors in pt

Maintenance
- Consider maintaining arterial BP and heart rate near preop levels to facilitate cerebral perfusion

Extubation
- None

Postoperative Period
- Neurologic evaluation at end of surgery

Anticipated Problems/Concerns
- Pts with internal mammary grafts may experience a similar syndrome of coronary-subclavian steal: There is a gradient in systolic brachial blood pressure of 60 mm Hg. In such situations myocardial ischemia that is refractory to medical management may occur.
## Subphrenic Abscess

### Overview
- **Primary:** Associated with perforated viscus such as duodenal ulcer, diverticulitis, appendicitis, primary liver abscess, immunocompromised state.
- **Secondary:** Following surgical intervention, critical illness, or blunt abdominal trauma. (Pathogens include *Candida* spp., *Enterococcus* spp., *Enterobacter* spp., *Staphylococcus epidermidis*, *E. coli* and are often polymicrobial with anaerobic bacteria outnumbering or equal to aerobic bacteria in all but postbiliary surgeries.)

### ICD-9-CM Code: 567.22

### Clinical Implications
- **Usual Treatment**
  - Broad-spectrum antibiotics +/- antifungals.
  - Narrow coverage after cultures obtained based on culture and sensitivity.
  - Supportive therapy: Appropriate monitoring, nutrition, oxygenation, hydration, vasopressors as indicated using the surviving sepsis recommendations.

### Key Reference:

### Perioperative Implications

#### Preoperative Preparation
- Appropriate broad spectrum antibiotics
- Restore intravascular volume
- Optimize resp function: PEEP, bronchodila-
tors, rarely thoracentesis
- NG tube for ileus and/or obstruction
- Tenacious CV status may require central venous access for monitoring/access or vasopressor and/or intropine infusion
- Assess coagulation status

#### Monitoring
- Tailor to severity of illness

#### Anticipated Problems/Concerns
- Drainage will need to be prolonged (often greater than 10 days)
- Recurrent abscess formation or sepsis (57% in high-risk pts)
- At risk for MODS (resp/ARDS, renal, hepatic, GI bleed)
- High mortality rate (23–50%) in the presence of multiple organ dysfunction
- Periop pneumonia/empyema/pleural effusion
- Fistula formation

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<td>Early: Hyperdynamic state, high cardiac output assoc with low SVR</td>
<td>Tachycardia Bounding pulses Warm, rubberous skin Tachycardia Diminished pulses Cool integument Peripheral cyanosis ECG CVP or PA catheter</td>
<td>ECHO</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Atelectasis, elevated diaphragm, pleural effusion, abd distention, pain, or ARDS</td>
<td>Dyspnea Ipsilateral shoulder pain ↓ Or abnormal breath sounds, dullness to percussion</td>
<td>CXR, fluoroscopy ARGs CT scan</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia due to suppressed marrow Coagulopathy assoc with sepsis</td>
<td>Fatigue Pallor Oozing around old incisions or IV sites Petechiae Ecchymoses</td>
<td>Hgb, Hct Plt count PT/APTT Fibrinogen, FSPs, d-dimer Thromboelastogram</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>↓ Perfusion due to hypovolemia or sepsis</td>
<td>↓ UO</td>
<td>BUN, Cr Lytes Acid-base balance</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Mental status changes assoc with sepsis</td>
<td>Range from mild confusion to coma</td>
<td>Must exclude other possible causes (e.g., CVA, CNS infection)</td>
<td></td>
</tr>
</tbody>
</table>

### Key Points
- **Risk**
  - Prior abd surgery, either open or laparoscopic
  - Blunt or penetrating trauma
  - GI perforation (malignancy, appendicitis, diverticulitis)
  - Inflammatory bowel disease
  - Immunocompromised pt

- **Perioperative Risks**
  - Developing sepsis
  - New onset cough
  - Tachypnea
  - Fevers

- **Worry About**
  - Resp compromise (pleurueffusion, aletectasis, V/Q mismatching, ARDS)
  - Preop ileus/bowel obstruction; aspiration risk
  - Sepsis incl septic shock and assoc renal failure and/or coagulopathy
  - Increased capillary permeability (hypovolemia)
  - High-output cardiac failure and/or VF

- **Electrolyte and acid-base disturbances**
  - Fluid shifts

- **Increased capillary permeability (hypovolemia)**
  - High-output cardiac failure

- **Sepsis incl septic shock and assoc renal failure and/or coagulopathy**
  - Increased capillary permeability (hypovolemia)
  - High-output cardiac failure

- **Diaphragm excursion**
  - Usual Treatment
  - Broad-spectrum antibiotics +/- antifungals.
  - Narrow coverage after cultures obtained based on culture and sensitivity.
  - Supportive therapy: Appropriate monitoring, nutrition, oxygenation, hydration, vasopressors as indicated using the surviving sepsis recommendations.

### Key Reference:
Supratentorial Brain Tumors

Tod B. Sloan

Risk
- Highest incidence age 3–12 and 55–65 y
- 7% of adult tumors: ~29,000 adults (USA) new tumors in 2005
- 1/3 of childhood tumors: ~0.8–1/100,000 children 600–1000 new tumors in in USA.

Perioperative Risks
- Increased ICP: Headache, seizures, neurologic deficit/dementia, visual and hearing changes, focal neurologic changes (hemiparesis, numbness, ataxia)
- Endocrinopathy and/or visual deficits if pituitary tumor

Worry About
- Seizure medications: Dilantin, keppra, tegretol
  - Need adequate levels to avoid postop seizures
- Raised ICP and brain edema: May lead to herniation (transientorial [dilate ipsilateral pupil], subfalcine [leg weakness], tonsillar [neck stiffness, spasticity, extensor-plantar response], upward transtentorial [small pupils, extensor rigidity])
- Dexamethasone Rx may lead to hyperglycermia
- Hyperglyceremia may cause more retractor-induced ischemic injury to adjacent brain tissues

Overview
- Endocrinopathy, particularly diabetes insipidus if near pituitary
- Brain edema surrounding malignant tumors causes initial Sx; often improve initially after corticosteroids

Assessment
- Consider arterial line: BP control, frequent ABGs, glucose, avoid dec. PaO₂

Assessment by Rx
- Acromegalic features
  - 3rd nerve palsy, papilledema, vision changes, hearing changes, macrocrania, bulging fontanel (infant)
- Gallop, rales, jugular distention, Htn, bradycardia
- Signs of COPD, altered breathing pattern
- CXR, ECG, ECHO
- FEV₁, FVC (if indicated) ABGs
- Cushingoid appearance
- Glucose levels
- Pale conjunctiva, positive occult fecal blood
- Hct, Hgb, T&C
- Altered consciousness, personality changes, memory loss, speech changes
- MRI, CT
- Nerve conduction velocity

PE
- Monitor CPP (MAP-ICP), (MAP at ear level)
- ETCO₂ as rough guide only, rely on Paco₂, avoid inc. CO₂
- ICP: If lumbar CSF drains are used, connect to transducer, leave closed until head open and surgeon ready, then drain slowly as need by surgeon
  - ICP monitors for postop measurement
  - Optimize hyperventilation (25–30 mm Hg PaCO₂), mannitol Rx
- Diagnostic if pt slow to awaken from anesthetic
- NMB: Increased receptor density in parietal extremities gives false twitch data: Use nonparetic arm/leg
- EEG used if surgery to treat seizure disorder
- Positioning occipital and pineal tumors: mild head up, if sitting then monitor for air embolism (precordial Doppler, CVP, etc.)

Airway
- Cushingoid facies may result in difficult mask ventilation
- Acromegaly causes laryngeal compromise by cartilaginous overgrowth. Anticipate difficult intubation. Consider lateral neck x-ray for airway abs such as enlarged epiglottis, narrowed cricoid ring.

Preinduction/Induction
- Induction with agents that act to ↓ cerebral blood flow (avoid ketamine)
- Avoid ↓ BP with intubation/head pins
- Avoid brain swelling due to venous outflow occlusion: Do not permit overflexion or excessive rotation of neck
- Eye protection from prep solution and pressure while face covered by drapes, instruments
- Use soft bite block (esp. with MEP)

Maintenance
- Goal normovolemia, normotension
- Normotensive fluids, replace diuresis if needed
- Avoid hypoglycemia, hypo-osmolality (<290 mOsm/kg)
- Low intrathoracic pressure
- Monitor PaCO₂, esp. with COPD
  - Mannitol: 0.5–1 gm/kg per surgeron


ASSESSMENT POINTS

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<td>Cartilaginous overgrowth in acromegaly</td>
<td>Acromegalic features</td>
<td>Lat neck x-ray</td>
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<td>CARDIO</td>
<td>Age effect: CHF, ASCVD, chemotherapy cardiomyopathy, inc ICP</td>
<td>DOE, edema, angina</td>
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<td>Mannitol, diuretics, decreased intake, vomiting (esp. children)</td>
<td>Dry mucous membranes</td>
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<td>ENDO</td>
<td>Iatrogenic Cushing’s syndrome due to decadron, infertility</td>
<td>Improved level of consciousness with decadron</td>
<td>Cushingoid appearance</td>
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<td>HEME</td>
<td>Anemia, paraneoplastic syndrome, increased thromboembolism</td>
<td>Occult GI bleeding caused by primary tumor</td>
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Risk Assessment

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• No painful structures below dura: Minimal anesthetic requirement with brain manipulation, low-dose inhalation agent and/or propofol infusion
• Avoid hyperthermia, hyperglycemia
• Avoid N₂O, use inhalational agent <1 MAC <½ MAC if SSÉP, TIVA if MEP monitored
• Avoid NMB with MEP monitoring
• Maintain good cerebral perfusion but not Htn
  * Antibiotics, redose at appropriate interval

**Extubation**
  * Extubate in head up position to dec bleeding
    * Awake: Normocarbia, early neuro assessment (risk of coughing, straining, possible hematoma formation. Risk ↑ postop Htn)
    * Deep: AVOIDS COUHING, may be Htn (transient PaCO₂ about 50 mm Hg until pt awakens, use deep ext. only if no brain edema during craniotomy and no anticipated airway problems)
  * Consider postop intubation/ventilation with preop poor mental status

**Adjutants**
  * Decadron, mannitol, lasix
  * Muscle relaxants
  * Profound paralysis: May minimize need for inhalation agents
  * Expect nondepolarizing NMB will be shorter acting if pt taking dilantin, most other antiseizure medications (except keppra)
  * Regional and local drugs
  * Expect hemodynamic effects from epinephrine in local infiltrated into scalp incision site
  * Antiemetics (differentiate PONV vs. Inc ICP)
  * Vasoactive compounds

  * Consider treatment with labetalol
  * Consider cerebral vasodilators: Hydralazine, sodium nitroprusside if severe Htn

**Anticipated Problems/Concerns**
  * Intraop brain swelling (head up, dec. CO₂, inc. venous drainage, dec. inhaled anesthesia, propofol/barbiturates
  * Air embolism with tumors near dural sinuses and sitting position
  * Intracranial bleeding (dural sinuses, vascular tumors)
  * Postop inc. ICP due to loss autoregulation
  * Delayed awakening, esp. with depressed consciousness preop
  * Postexcision brain swelling; seizures
  * Postop arterial Htn/bleeding
Supraventricular Tachycardia
(Tachyarrhythmias)

Overview
- Tachycardia (HR>100 in adults) with origin above the bundle of His in sinus node, atrial or junctional tissue. It may be re-entrant, automatic or triggered in origin.
- SVT may be paroxysmal (PSVT) or gradual in onset (sinus tachycardia, atrial tachycardia, or multiform atrial tachycardia). Tachycardia mechanisms vary (re-entrant vs. triggered and automatic) and treatment varies accordingly.
- PSVT is a re-entrant arrhythmia usually seen more commonly in children. The re-entrant circuit usually involves an accessory conducting pathway and the AV node.
- AAT is more commonly seen in the pediatric population 2nd to the enhanced automaticity seen in children.

ICD-9-CM Code: 427.0 (Paroxysmal supraventricular tachycardia)

Etiology
- PSVT is due to re-entry, which generally involves the AV node and an accessory pathway. Accessory pathways are relatively common in children. Also assoc with WPW and Lown-Ganong-Levine Syndrome (LGL).

Risk
- SVT is assoc with advancing age, significant cardiac and pulm disease.
- PSVT is assoc with WPW, congenital heart disease, and mitral valve prolapse. It is more common in younger pts. Mechanism is re-entrant in nature.
- Atrial tachycardia (AAT) may be automatic, triggered, or re-entrant. It is seen more commonly in children and those with a HH of prior atrial surgery. In adults it is rare, though can be assoc with digitalis toxicity and hypokalemia.
- Multifocal atrial tachycardia (MAT) is seen in adult pts with critical illness or advanced pulm disease.

Worry About
- Electrolyte and acid-base balance (K, Mg, alkalosis)
- Digitalis toxicity

Perioperative Risk
- Myocardial ischemia assoc with tachycardia and resulting coronary insufficiency
- Circulatory compromise
- Increased risk of atrial thrombus
- Chronic sustained tachycardia can result in irreversible cardiomyopathy.

Perioperative Implications

Preoperative Preparation
- PSVT: Adenosine, esmolol, and amiodarone should be available.
- AAT: Check and optimize electrolyte (K, Mg) and acid-base status. Rule out digitalis toxicity.
- MAT: Optimize status of various organ systems incl cardiac, pulm, renal, and metabolic.

Monitoring
- ECG, ST segment analysis
- Consider arterial pressure or PAC monitoring depending on anticipated case and pt status.

Induction
- In the setting of LV dysfunction or cardiomyopathy aim for hemodynamically stable induction.

Management
- AAT and MAT: Use caution with medications or situations which increase pt’s heart rate (ketamine, pancuronium, desflurane, beta-agonists, light anesthesia)
- Maternal agents are not thought to increase incidence of PSVT, AAT, MAT with the possible exception of desflurane.
- Prophylactic beta-blockade may be useful intraop if the pt is able to tolerate them.

Exubation
- Avoid excess sympathetic stimulation around the time of extubation as this increases the incidence of tachyarrhythmias. Strategies aimed at mitigating airway stimulation and hyperdynamic circulation are helpful in this regard.

Adjuvants
- Avoid use of beta agonists and histamine-releasing drugs if at all possible.

Postoperative period
- Ensure adequate sedation and pain control.
- Use of beta-blockers as tolerated will reduce incidence of MAT, AAT postop.
- Optimize cardiopulmonary and metabolic status.

Anticipated Problems/Concerns
- PSVT: Be prepared to treat asl/aflutter with RVR or ventricular fibrillation with cardioversion and/or defibrillation, particularly in pts with WPW or LGL.
- Cardioversion of AAT or MAT may result in life-threatening arrhythmias.

Diseases

Shiroh Isono

Swallowing Disorders

Risk
- More than 10% of elderly individuals have an absent gag reflex
- Pts with bulbar paralysis of any etiology

Perioperative Risks
- Malnutrition and dehydration due to inadequate oral intake
- Presence of pneumonia due to chronic aspiration
- Increased risk of aspiration pneumonia postop
- Increased retained bronchial secretions

Worry About
- Aspiration pneumonia

Overview
- Condition usually assoc with impairment of any part of swallowing reflex arc, such as sensory receptors in pharynx and larynx, afferent nerves, CNS, efferent nerves, muscles
- High risk for aspiration pneumonia pre- and postop can be evaluated by video fluoroscopy
- Assoc with abn hygiene of upper and bronchial airways

ICD-9-CM Code: 787.2 (Dysphagia)

Etiology
- Depressed CNS by sedation, sleep, coma, or light anesthesia
- NM disorders such as polymyositis, progressive MD, MS, myasthenia gravis, Eaton-Lambert syndrome
- Regional anesthesia to upper airway
- Tracheotomy or prolonged ET intubation; surgery on the head and neck, cardiac surgery
- Precurarization
- Peripheral nerve disorders such as Guillain-Barré syndrome, acute porphyria, laryngeal nerve injury, parkinsonism; advanced age

Usual Treatment
- Control for underlying disorders if possible
- Cricopharyngeal myotomy sometimes indicated
- Nasogastric balloon tube reported useful


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<tr>
<td>HEENT</td>
<td>Aspiration</td>
<td>Cough</td>
<td>Check gag reflex</td>
<td>Videofluoroscopy</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Dehydration</td>
<td>Skin, orthostatic vital signs</td>
<td>UO</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumonia</td>
<td>Dyspnea, sputum production</td>
<td>Fever</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Dysphagia</td>
<td>Salivation</td>
<td>UA inspection</td>
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<tr>
<td></td>
<td>GE reflux</td>
<td>Repeated pneumonia</td>
<td>Laryngeal movement</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Cranial nerve IX or X or others dysfunctional</td>
<td>Eating/swallowing pattern</td>
<td>Cranial nerve examination</td>
<td></td>
</tr>
</tbody>
</table>


### Perioperative Implications

**Preoperative Preparation**
- Control underlying disorders and complications (pneumonia, dehydration).
- Correct malnutrition and dehydration by tube feeding, gastrostomy, or parenteral alimentation.
- Consider metoclopramide or domperidone as a part of preop medication to treat prolonged retention of stomach contents.
- H2 blocker or proton pump inhibitor to decrease effects of silent regurgitation due to use of anticholinergic drug
- Avoid deep sedation

**Monitoring**
- Routine + quantitative acceleromyographic monitoring

### Airway
- Tracheal intubation with cuffed ETT
- Suction of secretions above the tracheostomy tube
- Do not apply local anesthetics to upper airway

**Preinduction/Induction**
- Rapid induction/intubation of trachea after cricoid pressure
- Avoid precurarization, possibly leading to severe dysphagia or pharyngeal obstruction

**Maintenance**
- Minimize NMB

**Exubation**
- Aspirate stomach contents and clear the oronasal cavity before extubation
- Eliminate or reverse residual anesthetics and muscle relaxants before extubation
- Check recovery of swallowing reflex

### Possible Drug Interactions
- Light sedation may impair swallowing reflex
- Precurarization and residual muscle relaxants can severely impair swallowing
- Possible synergistic effect of low concentration of inhalational anesthetics and partial paralysis on impairment of upper airway muscles
- Regional anesthesia impairs other upper airway protective reflex (closure of the larynx, cough reflex)

### Postoperative Period
- Fowler position if possible
- Prophylaxis of and/or treat N/V
- Evaluate for presence of aspiration pneumonia

### Anticipated Problems/Concerns
- Aspiration pneumonia (chemical or infectious)
Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)

**Risk**
- Elderly pts
- Nursing home residents
- Planned major operations, esp. neurosurgical procedures
- Central nervous system disorders incl psychiatric diseases
- Cancer, esp small-cell lung cancer
- Lung disease

**Worry About**
- Other causes of hyponatremia such as heart failure, liver failure, renal failure, or pseudohyponatremia (e.g., hyperglycemia) (see Hyponatremia)
- Acuity and magnitude of hyponatremia influences the risk of CNS complications
- Central pontine and extrapontine myelinolysis caused by rapid correction of hyponatremia

**Overview**
- Hyponatremia is the most common electrolyte disorder in hospitalized pts (affects 15%) and SIADH is the most frequent cause of hyponatremia, but other causes of hyponatremia should be excluded before making a Dx of SIADH
- Normally, increased serum osmolality, hypovolemia, or hypotension triggers thirst and ADH release. ADH increases aquaporin-2 channels on the luminal surface of the distal tubules and collecting duct and acts to promote free water reabsorption. Thirst, free water intake, or hypotonic fluid administration combined with ADH-induced free water retention causes hyponatremia.

**Test**
- Avoid hypotonic IV fluids
- Hyponatremia reduces MAC
- Increase free water intake, but may occur at slower rates of correction.

**ICD-9-CM Codes:**
- 259.3 (Ectopic); 259.6 (Neurohypophyseal)

**Etiology**
- Malignant diseases causing ectopic ADH secretion: Lung cancer (esp. small-cell and mesothelioma); brain tumors, cancer of the duodenum, pancreas, head and neck, GU tract, lymphoma, and sarcomas
- Pulm disorders: Infections, asthma, cystic fibrosis
- CNS disorders: Infection, masses, head trauma, intracranial bleed, MS, Guillain-Barre syndrome, Shy-Drager syndrome, delirium tremens, acute intermittent porphyria
- Drugs: Incl, but not limited to, chlorpropamide, carbacholazepine, cyclophosphamide, SSRIs, TCAs, clofibrate, nicotine, NSAIDs, antipsychotics, narcotics, arginine vasopressin analogues: desmopressin (DDAVP), oxytocin, vasopressin
- Major surgery: Pain, stress, general anesthesia, PPV, neurosurgery
- Hereditary: Mutation of gene for renal vasopressin-2 receptor; mutation for gene affecting osmolarity sensing in hypothalamus

**Usual Treatment**
- Decision to treat depends on acuity and severity of hyponatremia or the presence of symptoms
- Treat underlying causes for SIADH when possible
- Water restriction to 500–1000 mL per day for asymptomatic or chronic SIADH
- Normal saline (0.9%, 154 meq/L) infusion and furosemide (20 mg) for hyponatremia of unknown duration or moderate CNS symptoms. Goal is to increase [Na+] by 8–10 mEq/L in first 24 hr. Measure [Na+] every 4 hr.
- Hypertonic saline (3%, 513 mEq/L) at 1–2 mL/kg/hr infusion and furosemide (20 mg) for acute hyponatremia assoc with coma or seizures. Goal is to increase [Na+] by 2 mEq/L/hr until symptoms improve. Measure [Na+] every 2 hr.
- Demeclocycline 300–600 mg po bid to diminish responsiveness of collecting tube to ADH for persistent hyponatremia unresponsive to other therapy
- Vasopressin-receptor antagonist such as conivaptan (20–40 mg IV qd) or tolvaptan (15–60 mg PO qd) as an adjunct to increase free water clearance and [Na+]
- Urea, 30 g PO qd, to enhance water excretion in chronic SIADH
- Infusion rate (mL/hr) = \[TBW \times ([Na+]_{\text{target}} - [Na+]_{\text{current}}) / ([Na+]_{\text{current}}) \times (1000 \text{ mL/L}) \times (1/t)\]
- Where: TBW = total body water (0.6 x body weight); [Na+]_{\text{target}} = target [Na+]; [Na+]_{\text{current}} = [Na+] of saline infusion; t = time to achieve target [Na+] in hr


**PERIOPERATIVE IMPLICATIONS**

**Preoperative Preparation**
- Medical evaluation for duration and other causes of hyponatremia
- Neurologic assessment for symptomatic hyponatremia

**Monitoring**
- Periop measurement of serum [Na+]
- CVP or pulm artery catheter if necessary to maintain euvoolemia

**Induction**
- Avoid drugs that may potentiate SIADH

**Maintenance**
- Hyponatremia reduces MAC
- Avoid hypotonic IV fluids

**Exubation**
- Symptomatic hyponatremia may contribute to delayed emergence from anesthesia
- Hyponatremia can cause obtundation and diminished ability to protect the airway

**Adjuvants**
- Normal saline (0.9%, 154 meq/L) and furosemide to maintain euvoolemia and normal [Na+]

**Postoperative Period**
- Free water restriction, avoid hypotonic fluids
- Monitor serum [Na+]
- Assess for CNS signs of hyponatremia: lethargy, confusion, seizures

**Anticipated Problems/Concerns**
- Major surgery causes increased ADH release
- Acute symptomatic postop hyponatremia is a medical emergency
- Practice of using hypotonic maintenance fluids in pediatrics is controversial
- Most reported cases of central pontine and extrapontine myelinolysis were assoc with rapid correction of hyponatremia at rates over 12 mEq/L per day, but may occur at slower rates of correction.
Syndrome X

Risk
- True incidence unknown, characterized by angina with or without ST changes, with or without reversible perfusion defects on stress test, with normal coronary angiograms.
- Postmenopausal or posthysterectomy women most often at risk
- Common cause of chest pain in women with angiographically normal coronary arteries
- No diagnostic test; mortality risk low, morbidity high due to recurring angina and readmission.

Perioperative Risks
- Acute withdrawal of sex hormone replacement can potentially lead to symptoms.
- Preop angina can delay procedures.

Worry About
- D/C of medications (HRT) could precipitate symptoms.

Overview
- Poorly understood multifactorial etiology makes specific treatment difficult.
- Multimodal approach to reducing oxidative stress and improving endothelial function may be beneficial.

ICD-9-CM Code: 413.9

Etiology
- Etiology unproved, but thought to be due to endothelial dysfunction, +/- vasospasm, with systemic inflammation (increased CRP) playing a role
- Bioavailability of NO plays a role
- Acute withdrawal of estrogen appears to be more significant factor than chronic withdrawal

Usual Treatment
- Beta blockade, Ca++ channel blockers, statins, folate found to be beneficial and should be continued periop.
- Estrogen patch has been found to significantly improve exercise tolerance and alleviating chest pain.


Perioperative Implications

Preoperative Preparation
- Estrogens are withdrawn due to threat of procoagulant activity. Pts with this syndrome may experience significant angina upon such withdrawal.
- Distinguish chest pain due to this syndrome from chest pain due to coronary insufficiency from other causes.
- Continue preop meds, with appropriate thromboembolic prophylaxis.

Monitoring
- ST-segment analysis, usual ASA monitors.
- Invasive as appropriate for procedure.

Preinduction/Induction
- Contingent upon type of surgery; may consider maintaining usual medications, with use of beta blockers as appropriate.
- No data as to preferred anesthetic technique, agents.

Anticipated Problems/Concerns
- Angina pre- or periop in a pt with known clear coronary arteries
- Continuation of HRT can increase coagulability.
- Continuation of beta blockers and calcium channel blockers can lead to expected use of vasopressors.

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<td>Angina (chest pain) Inflammation</td>
<td>Hx of exertional angina Hx of evaluations leading to catheterization Hx of hormone replacement therapy</td>
<td>Normal coronary angiogram in presence of chest pain Elevated CRP</td>
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Systemic Lupus Erythematosus

**Risk**
- Prevalence: 1/2500 in Northern Europeans; 1/500 in African-American population
- 90% of pts with lupus are female.
- 65% of SLE pts are diagnosed between ages 16 and 35; 20% of SLE patients present prior to age 16

**Perioperative Risks**
- Increased lupus activity is assoc with surgery and stress
- Infections can initiate lupus or cause a relapse

**Worry About**
- CV: Htn, CAD, Libman-Sacks endocarditis with mitral insufficiency, myo-carditis
- Pulm: Restrictive lung disease with decreased diffusion capacity
- Renal: Lupus nephritis and renal insufficiency
- Endo: Adrenal insufficiency and renal insufficiency

**Overview**
- Autoantibody-mediated tissue damage results in multisystem organ damage
- Biopsy demonstrates inflammation, deposition of autoantibodies and complement in skin and kidneys
- 15-20 survival with lupus is 80%
- Procedure, myalgias, myositis, Raynaud phenomenon
- Vasculitis and ulceration
- Peripheral neuropathy, stroke, psychosis, fatigue, seizures
- Glomerulonephritis, nephrotic syndrome, renal insufficiency, renal failure
- CNS Peripheral neuropathy, stroke, psychosis, fatigue, seizures
- MS/Derm Vasculitis and ulceration
- Arthritis, myalgias, myositis, Raynaud phenomenon

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Htn, ↑ CAD, pericarditis, endocarditis, myocarditis, CHF, conduction blocks, pulm hypertension</td>
<td>Chest pain, palpitations, dyspnea</td>
<td>Murmur</td>
<td>ECG, ECHO, CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive lung disease, alveolar hemorrhage, pleural effusion, pulm edema</td>
<td>Pleuritic chest pain, hemoptysis, cough, dyspnea</td>
<td>Pleural rub, cyanosis, ↓ lung volume, rales, crackles</td>
<td>CXR, PFTs, ABGs</td>
</tr>
<tr>
<td>GI</td>
<td>Gastritis/PUD 2° to medications, lupoid hepatitis, SLE vasculitis resulting in colitis, pancreatitis, and bowel ischemia</td>
<td>N/V, abd pain, ileus</td>
<td>Hepatomegaly, splenomegaly, jaundice</td>
<td>LFTs, PT/PTT/INR, CBC/platelet, PTT</td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenia, leukopenia, anemia, thromboembolism (lupus “anticoagulant” prolongs aPTT in vitro, but pts have prothrombotic tendency)</td>
<td>Bruising, thrombosis</td>
<td>Lymphadenopathy, splenomegaly</td>
<td>CBC/platelet</td>
</tr>
<tr>
<td>RENAL</td>
<td>Glomerulonephritis, nephrotic syndrome, renal insufficiency, renal failure</td>
<td>Fever, hematura, polyuria, oliguria</td>
<td>Costhrenic tenderness, edema</td>
<td>Uronalysis, BUN, Cr, TP, albumin, Renal U/S or scan</td>
</tr>
<tr>
<td>CNS</td>
<td>Peripheral neuropathy, stroke, psychosis, fatigue, seizures</td>
<td>Numbness, hemiparesis, paranoid states, hyperirritability</td>
<td>Psychosis, nystagmus, ptosis, diplopia, aphasia</td>
<td>EMG/NCS, MRI, CT scan, EEG</td>
</tr>
<tr>
<td>MS/DERM</td>
<td>Vasculitis and ulceration</td>
<td>Photosensitivity, ecchymosis, purpura, joint pain or immobility</td>
<td>Malar or butterfly rash, perioral ulcers</td>
<td>X-ray, ANA</td>
</tr>
</tbody>
</table>


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**Perioperative Implications**

**Preoperative Preparation**
- Consider hydrocortisone 100 mg IV prior to induction if on chronic steroid therapy
- Antibiotic prophylaxis if valvular disease present

**Monitoring**
- Caution with arterial line in pts with Raynaud phenomenon
- Consider PA catheter for pulm Htn or CHF
- Consider Foley catheter and CVP/PA catheter for fluid titration if renal involvement

**Airway**
- Occasionally reduced TMJ ROM and cricoarytenoid arthritis manifesting as hoarseness, stridor, or airway obstruction; consider fiberoptic intubation

**Preinduction/Induction**
- Consider stress dose corticosteroid therapy

**Maintenance**
- No specific agents indicated or contraindicated; consider myocardial function
- Regional acceptable if no coagulopathy
- Avoid renally excreted drugs and renal toxins if renal insufficiency is present.
- Cyclophosphamide inhibits plasma cholinesterase and may cause prolonged response to succinylcholine.

**Adjutants**
- Corticosteroids, supplemental O2, careful titration of fluids with renal involvement

**Extubation/Postoperative Period**
- Reassess resp, renal, CV status prior to extubation

**Anticipated Problems/Concerns**
- Adrenal insufficiency from chronic steroid suppression
- Postop infections and pulm compli-cations
- Postextubation laryngeal edema or stridor
- CAD, CHF and arrhythmias
- Renal insufficiency and volume status
- CNS dysfunction, seizures, neuropathy
- Thrombocytopenia, anemia, and thromboembolism
- Lupoid hepatitis
Tetanus

**Overview**
- Infection of penetrating wounds or devitalized tissue by spores of anaerobic, Gram-positive bacillus *Clostridium tetani*; enter the CNS via peripheral nerves and spread via retrograde neuronal pathways in the spinal cord and brain (glycine and GABA).
- **CNS** disinhibition characteristically begins with spasms of the masseter muscles (‘risus sardonicus’, lockjaw) and progresses to involve rest of the body, incl spasms of resp muscles (‘resp convulsions’) that cause glottic spasm, airway obstruction, hypoxia, and resp failure.
- Autonomic instability is a hallmark of the disease and may cause fatal cardiac arrest.
- The initial injury may be insignificant or unnoticed by the pt.
- Neonatal tetanus typically presents 6–8 d after birth with trismus and inability to feed.
- Tetanus may follow surgery (usually intra-abd or on contaminated tissues), burns, gangrene, dog bites, chronic infection, parenteral drug use, dental infection, abortion, and childbirth.

**ICD-9-CM Code: 037**

**Etiology**
- Infection of penetrating wound or devitalized tissue by spores of anaerobic, Gram-positive bacillus *Clostridium tetani*; they proliferate and produce a potent exotoxin, tetanospasmin.
- Tetanospasmin is taken up by motor nerve endings and spreads to other neurons in skeletal muscle, the spinal cord, and brain, where it principally inactivates inhibitory interneurons in glycergic and gamma-aminobutyric acid pathways.

**Usual Treatment**
- Neutralize circulating toxin with IV human antitetanus globulin.
- Eradication of the organism by wound care, surgical debridement, and antimicrobial therapy.
- High dose metronidazole or penicillin G (erythromycin if penicillin allergy) therapy IV for 10 d is effective at eradicating spores and bacilli.
- Control muscle spasms by sedation, other muscle relaxants, and NM paralysis.
- Supportive therapy incl ventilatory support, treatment of autonomic instability, nutritional support, prophylaxis of DVT, and prevention of nosocomial infection, particularly ventilator-assoc pneumonia (VAP).

**Risk**
- A major public health problem in the developing world, responsible for 200,000–300,000 deaths/y; of these, approx 180,000 were neonatal deaths (in 2002).
- Incidence in USA: 0.16 cases/million population (1998–2000).
- Highest incidence in USA is among the elderly (>60 y), persons of Hispanic ethnicity, older adults with diabetes, and parenteral drug users.

**Perioperative Risks**
- Difficult airway or intubation in the presence of masseter spasm, neck rigidity, or opisthotonus.
- Autonomic instability with sudden fluctuations in BP, arrhythmias, cardiac failure, and cardiac arrest

**Worry About**
- Spasms of the laryngeal and resp muscles can be life-threatening as a result of airway obstruction or chest wall rigidity respectively, and may mandate urgent ET intubation.
- Resp failure may require NM paralysis in addition to sedation for effective PPV in the presence of severe spasms.
- Autonomic instability: Tachycardia, bradycardia, HTn, hypotension, arrhythmias, cardiac failure, repeated cardiac arrest
- Pneumonia, sepsis, myoglobinuria, pulm embolism, bony fractures, hyperthermia

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIRWAY</td>
<td>Laryngospasm and glotic obstruction</td>
<td>Dyspnea, noisy breathing</td>
<td>Stridor, retractions of accessory muscles, limitation of mouth opening and ROM of neck</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Generalized or localized muscle rigidity and spasms</td>
<td>Dysphagia, drooling, spasms</td>
<td>Opisthotonus, trismus, ‘risus sardonicus’, onset of spasms with minimal stimuli, bony fractures</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cardiac failure, myocarditis, arrhythmias, HTn, hypotension, cardiac arrest</td>
<td>SOB, palpitations</td>
<td>Episodic fluctuations in BP, heart rate, arrhythmias, signs of cardiac failure</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoventilation, apnea, resp failure, pneumonia</td>
<td>Dyspnea</td>
<td>Hypoventilation, limited chest excursions, decreased breath sounds, rhonchi, cyanosis</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Rhabdomyolysis</td>
<td>Pink or red urine</td>
<td>Hematuria</td>
<td>Urinalysis, serum creatine kinase</td>
</tr>
</tbody>
</table>

Tetralogy of Fallot (TOF)

Veronica C. Swanson
Norah Janosy

Risk
• Occurs in 1 in 3000 live births
• Most common cyanotic CHD
• Slightly more common in males than females

Perioperative Risks
• If unrepaired: Tet spells >RVH>RV failure>death (50% in first year of life)
• Mortality after TOF repair: 5–8% in first 2 y post repair (if uncomplicated anatomy)
• Increased mortality if co-existing PA hypoplasia, atresia, or major AP collaterals

Worry About
• Decrease in SVR resulting in increased R → L shunt
• Increase in PVR resulting in increased R → L shunt
• Crying and agitation leading to tet spell, more hypoxemia, hypercarbia, acidosis
• Air bubbles in IV tubing
• Polycythemia and assoc thrombocytopenia
• RV failure after inadequate or late repair
• Arrhythmias following repair

Overview
• Anatomy: Tetralogy
  • RV outflow tract obstruction (RVOTO)
  • (Infundibular narrowing, pulmonary stenosis, PA hypoplasia, pulm atresia). May have bicuspid pulm valve.
  • VSD: Single or multiple; outlet.
  • Overriding aorta (<50%)
  • RV hypertrophy (5%) have anomalous origin of LAD from right coronary artery: Must confirm before OR.
  • 25% have right aortic arch.
• Degree of R → L shunting determined by fixed factors (degree of infundibular obstruction, size of pulm valve annulus, size of PA) and dynamic factors (infundibular muscle bundle spasm, PVR, SVR)
• Fixed factors determine amount of chronic cyanosis
• Dynamic factors determine tet spells
• Pink tets have minimal amount of PS
• Avoid hypoxa, acidosis, high airway pressures, excitement, agitation
• Dx by ECHO, cardiac catheterization, and/or MRI

ASSESSMENT POINTS

<table>
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<th>System</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENERAL</td>
<td></td>
<td>FTT, Clubbing</td>
<td>Growth charts</td>
</tr>
<tr>
<td>CHEST</td>
<td>RVH</td>
<td>Signs of right heart failure</td>
<td>CXR with boot-shaped heart</td>
</tr>
<tr>
<td>CARDIO</td>
<td>See Overview: Anatomy</td>
<td>Frequency and severity of tet spells</td>
<td>ECHO/cath/MRI</td>
</tr>
<tr>
<td>HEME</td>
<td>Polycythemia from chronic</td>
<td>Chronic cyanosis</td>
<td>HCT, plt count</td>
</tr>
<tr>
<td></td>
<td>hypoxemia</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Plt count may be low from</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>polycythemia</td>
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<td></td>
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</tbody>
</table>


Perioperative Implications
• Heavy premedication to avoid agitation, crying
• Phenylephrine appropriately drawn up and diluted
• Avoid increase in PVR and decrease in SVR
• Consider IM ketamine if no IV present. Caution if Hx of tet spells
• Narcotics may be used

Usual Treatment
• Primary repair: See TOF: Procedure chapter
• If not immediately operable (low birth weight, prematurity, other disease processes): Palliative shunts to increase pulm blood flow (Blalock-Taussig shunt, aortopulmonary shunts)
• β-blockers to decrease infundibular spasm and spelling
• Treatment of tet spell
  100% O₂ (pulm vasodilator)
  • Sedation (morphine/fentanyl)
  • Increased SVR (squatting, phenylephrine)
  • Propranolol (decreased contractility of infundibulum; decreased RVOTO)
  • Bicarbonate to correct metabolic acidosis

• Assoc with chromosome 22 deletions and DiGeorge syndrome, VACTERL, CHARcE, and velocardiofacial syndrome

ICD-9-CM Code: 745.2
**Thalassemia**

**Overview**
- Thalassemia is a heterogeneous group of inherited microcytic anemias that result from a genetic mutation causing a defect in the synthesis of one or more globin chain subunits of the adult hemoglobin tetramer (HbA), which is normally composed of two alpha and two beta chains (β2α2).
- Thalassemia is classified according to the genotype which correlates with clinical severity.

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Malar hypoplasia with relative mandibular hyperplasia</td>
<td>Prior difficulties with intubation</td>
<td>Airway evaluation</td>
<td>ECG, annual ECHO, CXR, Halter</td>
</tr>
<tr>
<td>CV</td>
<td>Cardiomyopathy, Arthritus, Pericarditis</td>
<td>Exercise tolerance</td>
<td>Dyspnea, Dysrhythmias</td>
<td>Murmurs</td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive lung disease</td>
<td>Exercise tolerance</td>
<td>Tachycardia, Splenomegaly</td>
<td>CBC, Type and screen</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, Splenomegaly, Alloimmunization</td>
<td>Exercise tolerance</td>
<td>Blood transfusion reactions</td>
<td>Coagulation studies</td>
</tr>
<tr>
<td>HEPATIC</td>
<td>Cirrhosis</td>
<td>Hepatomegaly</td>
<td>LFTs, coagulation studies, hepatitis serologies</td>
<td>Cortisol determination</td>
</tr>
<tr>
<td>ENDO</td>
<td>Diabetes mellitus</td>
<td>Cold intolerance, lethargy, depression, decreased metabolism</td>
<td>Fasting glucose, Glucose tolerance test, Thyroid function test</td>
<td></td>
</tr>
</tbody>
</table>

**Key Reference:** Rodgers GP. Hemoglobinopathies: The Thalassemias in Cecil medicine. 23rd ed. Chapter 166.

**Perioperative Implications**
- Thalassemia minor, in general, does not create anesthetic problems. In pts with thalassemia major, consideration has to be given to problems derived from the severity of the anemia itself, but also those related to transfusion therapy, and to bony malformations.

**Preinduction**
- Detailed airway evaluation
- Cardiac function evaluation
- Hemoglobin level should be determined and preop transfusion considered
- Cross-matched blood should be available (anti-body matched, leukocyte reduced for frequently transfused children); high degree of alloimmunization in this population exists.
- Evaluation for endocrine dysfunction (e.g., DM, hypopituitarism, hypothyroidism) and adequacy of treatment
- Hepatic function evaluation in light of risk of cirrhosis and iron or viral-induced damage
- Coagulation studies
- Presplenectomy antibiotics and immunizations (when appropriate)

**Monitoring**
- Consider the need for a Swan-Ganz catheter and measurements of CI, CO, mixed-venous oxygenation
• Consider arterial catheter and frequent hemoglobin, lactate, and blood gas analysis

**Induction/Maintenance**
• Preparation for possible difficult airway
• Close attention to the positioning in light of demineralization and scoliosis
• Careful monitoring of CV function; incl post-splenectomy Htn
• Beware of the effects of laparoscopy on circulatory and resp function
• Thromboembolism prophylaxis; SCD and/or pharmacotherapy when applicable
• Consider cell salvage

**General Anesthesia**
• Facial abn can present a difficult airway

**Regional Anesthesia**
• Osteoporosis, osteopenia, scoliosis are common
• Vertebral bodies maybe of reduced height as a result of osteoporosis; the segmental portion of conus medullaris may be lower than predicted
• Extramedullary hematopoiesis is uncommon in the intraspinal location, but if symptoms of spinal compression are suspected, MRI should be performed prior to regional anesthesia
• Consider epidural versus spinal in pts who need a regional anesthetic, but have CV pathology
• Evaluate closely coagulation studies prior to regional anesthesia
• Periop thromboembolism prophylaxis, esp. in post-splenectomy pts
• Spinal and epidural techniques have been performed safely in parturients

**Postoperative Period**
• Postop monitoring dependent on the preop status
• Prophylaxis for thromboembolism (post-splenectomy pts in particular)

**Anticipated Problems/Concerns**
• Intubation difficulties
• CV instability 2° to severe chronic anemia, cardiomyopathy, and endocrinopathies
• Coagulation abn: hyper- or hypo-coagulable
• Impaired drug metabolism 2° to cirrhosis
• Adrenal insufficiency complications
• Difficulty in obtaining cross-matched blood due to alloimmunization
**DISEASES**

**Thrombocytopenia**

### Risk
- Common in both adults and children
- Often assoc with systemic illness and pathologic conditions of pregnancy
- Heparin-induced thrombocytopenia (HIT) occurs in ~5% of pts exposed to heparin

### Perioperative Risks
- Bleeding assoc with invasive procedures (both anesthetic and surgical)

### Worry About
- Excessive periop bleeding
- Concurrent anemia, hypovolemia, and hemodynamic instability
- Potential need for blood and plt transfusions
- Underlying cause for thrombocytopenia

### Overview
- Definition: <150,000 plt/mm³
- Hemostasis is adequate with plt counts >100,000/mm³ unless concurrent plt dysfunction is present (e.g., antiplatelet agents, cardiopulmonary bypass).
- Plt transfusion may be necessary for plt counts between 50,000 and 100,000/mm³ to prevent/treat bleeding, depending on site and extent of invasive procedure

### Perioperative Implications

#### Preinduction/Induction/Maintenance
- Assess volume status and Hct
- Determine bleeding risk from physical exam, extent of thrombocytopenia, and type of surgical procedure
- Ensure that blood bank has adequate crossmatched blood and pltts available
- Plt transfusion immediately prior to surgical procedure if plt count <30,000/mm³

#### Monitoring
- Vigilance to volume status and blood/fluid replacement is essential
- Hesper administration can impede plt function

#### General Anesthesia
- Nasal intubation relatively contraindicated

#### Regional Anesthesia
- Epidural and spinal anesthetics can be safely administered with plt counts ≥100,000/mm³

#### Anticipated Problems/Concerns
- Bleeding from anesthetic or surgical procedures
- Potential for blood and plt transfusions

### Usual Treatment
- Treat underlying cause
  - D/C offending drug, treat infection, splenectomy
  - HIT treatment incl a direct thrombin inhibitor (e.g., bivalirudin, argatroban) to prevent/treat thrombosis in addition to D/C of heparin
  - ITP generally responds to corticosteroids (plus IgG therapy in severe cases); TTP requires exchange transfusion or plasmapheresis
- Decision to transfuse pltts depends on etiology of thrombocytopenia and relative risks of bleeding vs. transfusion; not useful for thrombotic syndromes (e.g., HIT, TTP)
- Each unit of transfused pltts should raise count ~10,000/mm³ but increases risk of future thrombocytopenia (alloimmunization occurs in 50% of pts transfused with pltts)

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<tbody>
<tr>
<td>HEENT</td>
<td>Mucosal hemorrhage</td>
<td>Petechiae, purpura, and ecchymoses of skin, oral mucosae, and conjunctivae</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Hypovolemia, anemia, pericardial effusion</td>
<td>Lightheadedness, syncope, palpitations</td>
<td>Tachycardia, hypotension, orthostasis, pericardial friction rub, pulsus paradoxus</td>
<td>ECG, CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm hemorrhage</td>
<td>Cough, hemoptysis</td>
<td></td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>GI bleeding</td>
<td>Hematemesis, hematochezia, melena</td>
<td></td>
<td>Stool guaiac</td>
</tr>
<tr>
<td>RENAL</td>
<td>Potential prerenal or renal azotemia, glomerulonephritis with specific disease entities</td>
<td>UO</td>
<td>BUN, Cr, urinalysis</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Intracranial hemorrhage</td>
<td>Change in mental status, Mental status, focal motor weakness</td>
<td>Head CT</td>
<td></td>
</tr>
</tbody>
</table>

### Key Reference
**Thyroid Neoplasms**

**Risk**
- Incidence in USA: 23,500 new thyroid cancer cases/y
- Approx 1% of new cancer diagnoses each year
- Hispanics, African-Americans, lower rate; Caucasians, moderate rate: Japanese, Chinese, Hawaiian, Filipinos, higher rate
- Overall incidence 3 × higher in women than in men, peaks in third and fourth decades of life

**Perioperative Risks**
- Large thyroid mass may produce airway compression, deviation, or vocal cord paralysis
- Decreased BP, decreased HR, asystole with manipulation of carotid sinus
- Postop complications: Phrenic nerve injury, pneumomediastinum, pneumothorax, tracheomalacia and tracheal collapse post extubation, hematoma or laryngeal edema leads to airway compromise; bilateral laryngeal nerve injury calls for tracheostomy; superior laryngeal nerve injury leads to aspiration
- Accidental removal and/or injury of parathyroid glands causes decrease in Ca²⁺

**Worry About**
- Occult pheochromocytoma: bilateral lobe medullary thyroid cancer is assoc with MEN IIa and IIb

**Overview**
- 4 types: Papillary (80%), follicular (10%), medullary (5–10%), 1° thyroid lymphoma (rare) and 1° thyroid sarcomas (rare)
- Prognosis of well-differentiated papillary cancer excellent, esp. for age <40 y with small tumors
- Prognosis worsens for large tumors with poorly differentiated, anaplastic histology
- Age at Dx, tumor burden, gender, extra-thyroidal invasion, and distant metastases: important prognostic factors
- Latest research: Define subcellular and molecular prognostic factors, genetic studies
- BRAF mutation most common mutation in papillary thyroid cancer and is assoc with disease aggressiveness

**ICD-9-CM Code:** 193

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Vocal cord dysfunction</td>
<td>Dysphonia</td>
<td>Neck mass</td>
<td>Indirect laryngoscopy</td>
</tr>
<tr>
<td></td>
<td>Tracheal obstruction</td>
<td>SOB, DOE, Wheeze/stridor</td>
<td>CT of neck</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Mediastinal mass</td>
<td>SOB, DOE, Wheeze, may be asymptomatic</td>
<td>Facial swelling</td>
<td>CT/MRI</td>
</tr>
<tr>
<td>RESP</td>
<td>Lung metastases</td>
<td>SOB, DOE, Wheeze, hemoptysis</td>
<td>CT/MRI</td>
<td></td>
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<tr>
<td></td>
<td>Lower airway obstruction</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>GI</td>
<td>Esophagel obstruction</td>
<td>Dysphagia</td>
<td>CT/MRI</td>
<td></td>
</tr>
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<td></td>
<td>Liver metastases</td>
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<td></td>
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<tr>
<td>ENDO</td>
<td>MEN IIa/IIb pheochromocytoma</td>
<td>HTN, esp. episodic Flushing Palpitations, episodic Sweating</td>
<td>CT/MRI</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>24 h urine epinephrine</td>
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<td></td>
<td></td>
<td></td>
<td>↑ Epinephrine/norepinephrine</td>
<td></td>
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<tr>
<td></td>
<td>Hyperparathyroidism</td>
<td>Colic Cramping Diarrhea Obstruction Mucosal neuromas in tongue, subconjunctival areas, or GI tract Thickened lips</td>
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<td></td>
<td>Ganglineuromatosis</td>
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<td>Hypercalciuria provocative test for calcitonin release</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>↑ Ca²⁺</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Bone metastases</td>
<td>Bone pain</td>
<td>Bone scan</td>
<td></td>
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<td></td>
<td>PTH-induced bone disease</td>
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</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Assess thyroid gland and/or tumor size
- Assess larynx and/or trachea compression
- May need smaller or armored ETI to prevent kinking (check CT scan)
- Record description of voice preop
- Correct abn Ca²⁺, TSHs prior to surgery
- Check serum calcitonin level if medullary cancer suspected; rule out pheochromocytoma

**Monitoring**
- Routine Airway
- Anticipate difficult airway

**Induction**
- Consider awake fiberoptic intubation for large thyroid masses

**Maintenance**
- No one agent or technique shown superior
- No CV instability may occur with manipulation of carotid sinus

**Exstution**
- May develop tracheomalacia
- May require reintubation owing to hematoma

**Postoperative Period**
- Metabolic: Decreased Ca²⁺, hypoparathyroidism
- Nonmetabolic: Unilateral or bilateral nerve injury, hemorrhage, airway obstruction

**Adjutants**
- May be performed under local anesthesia with IV sedation in selected cases
- Antiemetics, incl dexamethasone, effective in reducing postop N/V

**Anticipated Problems/Concerns**
- Pts with medullary thyroid cancer: Rule out occult pheochromocytoma
- Thyroid tumor can invade larynx, trachea, pharynx, or esophagus
- If esophageal wall invaded, reconstruction often by free microvascular jejunal transfer or gastric pull-up

**Etiology**
- Factors incl previous radiation, dietary iodine deficiency, goitrogens (chemical or dietary), pre-existing benign thyroid disease, and genetic factors (Gardner’s syndrome, Cowden’s disease)
- Association between primary thyroid cancer and ↑ incidence of subsequent breast cancer

**Usual Treatment**
- Surgery initial therapy of choice
- Lobectomy with or without isthmectomy, near-total or total thyroidectomy as indicated
- Radiiodine scanning and ablation commonly used after thyroidectomy in well differentiated tumors
- Radical debulking procedure (palliative) for large tumors invading airway and causing esophageal obstruction and bleeding
- Recurrences usually treated with surgery
- Combined chemo- and radiation therapy for poor prognosis cases
- Doxorubicin: Most active single agent; medullary thyroid cancer responds poorly
Transfusion-Related Acute Lung Injury (TRALI)

Risk

- All pts receiving blood or blood products incl pts, plasma, and cryoprecipitate
- Overall incidence probably <1%, increasing awareness of the syndrome has resulted in improved recognition.
- Use of leukodepleted blood has decreased the incidence of packed red cell–related lung injury.

Perioperative Risks

- Noncardiogenic pulm edema usually within 2–6 hr after transfusion
- Mortality reported, but rare

Worry About

- O₂ toxicity
- Barotrauma or volutrauma 2° to PPV

Overview

- Classic presentation is acute development of resp compromise indistinguishable from ARDS.
- Symptoms usually begin within 1–2 hr after transfusion and may be manifested by 2–6 hr.
- There are typically severe hypoxemia, hypotension, fever, and bilateral infiltrates on chest radiograph.
- Dx is one of exclusion (rule out fluid overload, CHF, sepsis)

ICD-9-CM Code: 999.8 (Transfusion, complication)

Etiology

- Classically, has been attributed to the presence of leukocyte antibodies in the plasma of multiparous donors directed against recipient WBCs.
- Alternatively, effect from biologically active lipids in stored cellular blood components
- Pulm edema arises from capillary injury rather than volume overload.

Usual Treatment

- Supportive care: Ventilation if required, supplemental O₂. No clear indications for steroids. Generally resolves within 1–4 d with appropriate care and no supervening complications.

Assessment Points

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Pulm edema</td>
<td>S₂, S₄</td>
<td>PA catheter, ECHO</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>Recent transfusion</td>
<td>Rales, hypoxemia</td>
<td>CXR—bilateral infiltrates, SpO₂</td>
</tr>
<tr>
<td>HEME</td>
<td>Leukoagglutination</td>
<td></td>
<td></td>
<td>Agglutination of recipient leukocytes by donor plasma: Contact blood collection agency</td>
</tr>
</tbody>
</table>


Perioperative Implications

Perioperative Concerns

- Acute resp compromise that may occur shortly after transfusion in healthy pt, but more typically 2–4 hr after transfusion

Monitoring

- PA catheter may aid in the exclusion of cardiac etiology

Postoperative Considerations

- Most require ventilatory support for several days
- Ventilator management appropriate for ARDS

Anticipated Problems/Concerns

O₂ toxicity and barotrauma
**Transverse Myelitis**

**Overview**
- Inflammatory disease of spinal cord causing demyelination/necrosis
- Ascending paralysis and sensory level; T8–12 assoc with pain and urinary retention
- Spinal cord swelling; increased CSF protein, WBC 10–200 (higher if culture positive)
- Onset over hours to days
- Antecedent febrile illness (33%)
- Variable recovery
- MS (subsequent demyelinating lesions) occurs in 5–10% of cases

**ICD-10-CM Code:** G 37.3 (Acute transverse myelitis)

**Etiology**
- Viral (polio, HSV, HIV)
- Bacterial, fungal, parasitic
- Noninfectious (postinfectious, postvaccine, lupus, immune compromise)

**Usual Treatment**
- High-dose steroids; immune globulin antibodies recently reported, stem transplant tried
- Acute antibiotics if infectious; long-term antibiotics if frequent UTI
- Chronic pain treated with spinal cord stimulator
- Avoid noxious stimuli below level of the lesion

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<td>↓ Visual acuity</td>
<td>Optic neuritis, ophthalmoscopy</td>
<td>Visual EPs</td>
</tr>
<tr>
<td>CARDIO (acute)</td>
<td>↓ BP, tachy- or bradycardia</td>
<td>Syncope</td>
<td>BP changes</td>
<td>Orthostasis Stop stimuli below lesion</td>
</tr>
<tr>
<td>CARDIO (chronic)</td>
<td>↑ BP, bradycardia</td>
<td>Headaches, flushing, sweating</td>
<td>Flushed skin above level of lesion, blanched skin below lesion</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm embolism</td>
<td>Dyspnea, tachycardia</td>
<td>DVT, cord signs</td>
<td>Doppler, V/Q scan</td>
</tr>
<tr>
<td>GI</td>
<td>Gastric atony</td>
<td>N/V, dyspepsia, early satiety</td>
<td>Tympany, CXR</td>
<td>Stomach bubble</td>
</tr>
<tr>
<td>CNS</td>
<td>Brain</td>
<td>Encephalitis</td>
<td>Mental status changes</td>
<td>MRI (brain) MRI (spine, LP)</td>
</tr>
<tr>
<td></td>
<td>Spine</td>
<td>Spinal level (sens/motor)</td>
<td>Paraplegia</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Bladder paralysis (acute)</td>
<td>Retention, oliguria</td>
<td>Palpation, catheterization</td>
<td>UA, bladder scan</td>
</tr>
<tr>
<td></td>
<td>Bladder spasticity (chronic)</td>
<td>Frequent urination</td>
<td>Residual urine volumes</td>
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**Perioperative Implications**

**Preoperative Preparation**
- Relieve gastric ileus, treat as full stomach

**Monitoring**
- Routine, unless dysautonomia

**Airway**
- Avoid succinylcholine, hyperkalemia

**Induction**
- Adequate hydration because of vasorelaxation acutely and dysautonomia chronically

**Maintenance**
- GA preferred or epidural; TM has been described after all anesthetics
- Spinal with caution, possible toxicity with usual doses; usually avoided
- Follow Train-of-4; variable response to NM blockers

**Extubation**
- Return of airway reflexes if demyelinating lesions in brain/brainstem

**Adjuvants**
- Resistance and sensitivity to nondepolarizing muscle relaxants reported
- Dysautonomia reported assoc with use of dexmedetomidine

**Anticipated Problems/Concerns**
- Unstable hemodynamics
- Possible hyperkalemia following succinylcholine
- Worsening of baseline symptoms and neurologic signs after anesthesia (transient)

**Perioperative Risks**
- Few data available (usually grouped with MS; demyelinating disease)
- Anesthetic effect (worsening) unknown, causative only question
- Sequelae of hypotension or Htn
- Urinary retention and UTI
- Delayed gastric emptying

**Worry About**
- Autonomic dysfunction (midthoracic lesion and above)
  - Autonomic dysautonomia from spinal shock
  - Chronic: Htn and bradycardia from mass reflex (autonomic dysreflexia)
  - Hyperkalemia from succinylcholine; prolonged NM blockade rarely

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Treacher Collins Syndrome

**Overview**
- Also known as mandibulofacial dysostosis and Franceschetti-Zwahlen-Klein syndrome
- Clinical features incl various degrees of mandibular hypoplasia, high arched or cleft palate, malar hypoplasia, ophthalmic abn (downward slant of palpebral fissures, lower lid coloboma, partial to total absence of lower eyelashes, visual loss), microtia and middle ear hypoplasia.
- Fishlike facies
- Choanal atresia may occur
- Conductive hearing loss due to ear abn is universal with varying degrees of severity.
- Airway compromise may occur due to maxillary hypoplasia (narrowed nasal passages resulting in choanal stenosis or atresia) and mandibular hypoplasia (tongue base is retrropositioned thereby obstructing oropharyngeal and hypopharyngeal spaces)
- Limited oropharyngeal and hypopharyngeal space may lead to obstructive sleep apnea, pulm Htn and in severe cases cor pulmonale
- Affected newborns and infants may have feeding difficulties

**ICD-9-CM Code:** 756.0

**Etiology**
- Abn bilateral first and second branchial arch development due to mutation in TCOFI gene on chromosome 5 (60% spontaneous/40% familial)
- When inherited shows autosomal dominance with variable penetrance and expression
- TCOFI mutation results in a deficiency of neural crest cells leading to failed development of cartilage, bone, and connective tissues particularly in the head and neck region.

**Usual Treatment**
- Prenatal detection of micrognathia and dysmorphic facial features on fetal US may prompt genetic testing and counseling if Treacher Collins is suspected
- Evaluation of airway and assessment of swallowing and feeding difficulties at birth. Some pts require ET intubation in delivery room
- Severe airway compromise and feeding issues may require tracheostomy and gastrostomy tube placement. Mandibular distraction procedures can be used to relieve airway obstruction and facilitate tracheal decanulation.
- Detailed assessment and imaging to determine the extent of craniofacial involvement during the first year of life. Repeated imaging may be needed prior to reconstructive procedures.
- Staged zygomatic, orbital, maxillomandibular and nasal reconstruction
- Surgical repair for cleft palate, choanal atresia
- Staged external ear reconstruction. Very few TCS pts are candidates for external ear canplasty to restore hearing.

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<td>HEENT</td>
<td>Limited airway</td>
<td>Hx of stridor, dyspnea, snoring, obstructive sleep apnea</td>
<td>Micrognathia, retrognathia, limited pharyngeal area</td>
<td>Hearing evaluation</td>
</tr>
<tr>
<td>--------------</td>
<td>---------------------------------------------</td>
<td>------------------------------------------------------</td>
<td>-----------------------------------------</td>
<td>-------------------------------------------</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cor pulmonale</td>
<td>Easy fatigability</td>
<td>S_{0}, hepatomegaly ⌊ Jugular venous pulsations</td>
<td>ECG: Right axis deviation</td>
</tr>
<tr>
<td></td>
<td>Pulm Htn</td>
<td></td>
<td>Heart murmur</td>
<td>P waves in II, IIIa, VF ECHO/ cardiac cath</td>
</tr>
<tr>
<td>GI</td>
<td>Difficulty feeding</td>
<td>Difficulty swallowing, chewing, poor Po intake</td>
<td>Poor wt gain</td>
<td>Upper endoscopy</td>
</tr>
<tr>
<td></td>
<td>GERD, esp. in pts with tracheostomy</td>
<td>Frequent regurgitation, discomfort after meals</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Obstructive sleep apnea</td>
<td>Loud snoring, intermittent complete obstruction, frequent arousal, daytime hypersomnolence or hyperactivity</td>
<td></td>
<td>Polysomnography</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Preparation**
-Thorugh airway assessment and review of previous anesthetics
-Review of pertinent labs, studies, and imaging
-Medical Hx inquiring about obstructive sleep apnea or cor pulmonale
-Antisialagogue for airway preparation

**Monitoring**
-Standard monitors
-Invasive monitoring for lengthy reconstructive procedures with anticipated blood loss

**Airway**
-Assume difficult intubation and prepare anesthetic plan in a case by case situation (ease of intubation with previous anesthetics may not guarantee ease of intubation with current anesthetic)
-Back up airway devices (fiberoptic bronchoscope, glidescope, bullard laryngoscope, LMA) and surgical airway preparation

**Preinduction/Induction**
- Avoidance of sedatives if Hx of severe OSA is present
- Inhaled sevoflurane induction with maintenance of spontaneous ventilation during laryngoscopy

**Maintenance**
- Avoidance of excessive opioids to minimize risk of postop resp depression

**Exubation**
-Strict extubation criteria
-Airway devices and staff support in case pt requires re-intubation

**Postoperative Period**
-Acute airway obstruction
-Consider steroids, racemic epinephrine to decrease airway swelling

**Anticipated Problems/Concerns**
-Obstructive sleep apnea, pulm Htn and cor pulmonale
-Difficult airway

Tricuspid Atresia

Risk

- Uncommon. Occurs in 0.056 per 1000 live births.

Perioperative Risks

- Hypoxia caused by limited pulm blood flow.
- Reliable systemic and pulm blood flow in these pts depends on existence of an unobstructed atrial level right to left shunt, an unobstructed left to right ventricular septal defect and intact pulm artery.
- There is obligatory mixing of systemic venous blood return to the heart from the vena cava (lower O2 sat) and blood return to the heart from the pulm veins (higher O2 sat).

Worry About

- Inadequate ability of systemic venous and pulm venous blood to mix caused by restrictive atrial septal defect (rare additional problem, but vital).
- Inadequate pulm blood flow caused by restrictive ventricular septal defect, pulm artery stenosis, pulm subvalvular obstruction or pulm atresia.
- Less common is the pt that presents with too much pulm blood flow and CHF (completely unobstructed pulm blood flow).

Overview

- Defined by the lack of a connection between the right atrium and hypoplastic (could be practically nonexistent) right ventricle.
- The tricuspid valve may be completely absent or there may be a rudimentary valve-like structure on the floor of the right atrium that is not patent.
- Basically, three major types
  - Tricuspid atresia with normally related pulm artery and aorta (70%–80%). There are three subtypes.
  - Tricuspid atresia with transposed great arteries, atresia of the pulm artery arising from the left ventricle, and a ventricular septal defect allowing systemic blood flow to occur through the aorta arising from the hypoplastic right ventricle (pulm blood flow completely dependent on the maintainence of a patent ductus arteriosis in the neonatal period).
  - Tricuspid atresia with transposed great arteries, hypoplasia of the pulm artery arising from the left ventricle and a ventricular septal defect.

ICD9-CM: 746.1 (Tricuspid atresia and stenosis, congenital)

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<tbody>
<tr>
<td>CV and RESP</td>
<td>More common, limited pulm blood flow causing hypoxia and possibly resp acidosis</td>
<td>Possibly requiring intubation or supplemental O2</td>
<td>Cyanosis, holosystolic murmurs, possible thrill</td>
<td>SpO2, ABG, ECHO</td>
</tr>
<tr>
<td>CNS</td>
<td>Stroke due to single ventricle physiology</td>
<td>Pt may have received a balloon septostomy</td>
<td>Varying levels of consciousness</td>
<td>CT, MRI</td>
</tr>
</tbody>
</table>


**Adjuvants**

- Heparin, usually 100 units/kg (more may be necessary), to maintain an activated clotting time of at least 200 sec during the creation of the surgical shunt. May require redosing, depending on the length of the procedure.
- Inotropes to maintain adequate contractility and systemic vascular resistance.

**Anticipated Problems/Concerns**

- A thorough understanding of how to balance and maintain as close to equal the pulm arterial and systemic arterial blood flow from a single ventricle (parallel physiology) is required.
Trigeminal Neuralgia
(TIC Doloureux)

**Risk**
- Trigeminal neuralgia (TN) has an incidence of 4/100,000
- More common in women, pts >50 y
- 1–5% of pts with MS have TN, and 2–4% of pts with TN have tumors in the posterior fossa.
  It is rarely seen in pts with Charcot-Marie-Tooth disease.

**Perioperative Risks**
- Evaluate co-morbidities with pts with MS
- Liver enzyme induction with use of anticonvulsant and/or antiepileptic drugs

**Worry About**
- Severe bradycardia with manipulation of the fifth nerve in the posterior fossa or with balloon ablation
- Oversedation and management of the airway in RFA procedures
- Postop exacerbation of MS

**Overview**
- TN is characterized by recurrent episodes of intense; lancinations pain over the distribution of the fifth cranial nerve more commonly the V2 and V3 divisions. Pain can be spontaneous or set off by stimuli in the trigger zones: light touch, cold air, talking or drinking. Pain is described as brief, like an electric shock, shooting or stabbing and terminates abruptly. Pts may have bouts over weeks or months with some spontaneous remissions up to 6 mo. Usually the bouts become more frequent and the pain more sustained. Pts with MS rarely have remissions.

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<td>CNS</td>
<td>Cranial nerve involvement, sensory loss</td>
<td>Pain Hx and distribution</td>
<td>Full neurologic exam</td>
<td>MRI, MRA</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preinduction/Induction/Maintenance**
- If the procedure calls for pt assistance in identifying the area for ablation then minimal sedation is used esp. longer-acting benzodiazepines
- NPO status must be observed.
- If the pt has MS then neurological status preceding the procedure should be documented.
- Pts on anticonvulant/antiepilepsy medications will be less responsive to induction agents and metabolize liver metabolized agents more quickly.
- Must take special care to avoid trigger zones when placing mask, nasal prongs

**Perioperative Implications**

**Monitoring**
- For MAC or GA for ablation procedures: Usual ASA monitors
- For posterior fossa craniotomy: ASA monitors, invasive arterial and venous lines, precordial Doppler for venous air embolism detection, nitrogen analysis, all depending on the position of the pt
- Consider means to pace the heart: Transesophageal pacemaker, transthoracic noninvasive pacer (Zoll pads)

**General Anesthesia**
- All usual considerations for posterior fossa craniotomy incl pt positioning, detection of air embolism, brainstem, and cranial nerve, manipulation resulting in bradycardia, asystole, tachyarhythmias and hyper- or hypotension
- Avoid succinylcholine in MS pts with extensive motor involvement.
- Expect rapid metabolism of opioids, nondepolarizing muscle relaxants in pts managed with anticonvulsants medications (cytochrome P-450).
- Surgeons may monitor BAERS for eighth nerve function.
- Arousal and extubation will require careful management of BP, HR.
- If manipulation of lower cranial nerves has occurred, the pt may not be able to protect the airway and delayed extubation may be planned.
- Surgeons will expect the pt to respond to commands before leaving the OR.

**Regional Anesthesia/Monitored Anesthesia Care**
- Judicious use of sedation required such that level can be increased during painful lesioning but the pt can be aroused for consultation. Agents used: Remifentanil, fentanyl, methohexital, propofol, dexmedetomidine

**Etymology**
- The pathophysiology responsible for the signs and symptoms of TN is unknown but several theories exist
- Idiopathic: More than 90% of cases are idiopathic with evidence of focal demyelination of trigeminal sensory fibers within the nerve root or within the brainstem. Usually due to compression by an aberrant artery or vein which results in close apposition of axons and absence of intervening glial processes. This favors ectopic impulses and ephaptic conduction at adjacent fibers. In the area of the trigeminal nerve entry zone demyelination would lead to ephaptic conduction between fibers for light touch and those for pain explaining the sensitivity of trigger zones.
- Symptomatic: These pts have less classical features of TN. Primary demyelinating disorders such as MS will lead to demyelination and plaque formation in the root entry zone but they may also have nerve compression by a vascular structure. Rarer presentations would incl compression by tumor, infiltration by tumor or amyloid, small infarcts or angioma in the brainstem.

**ICD-9-CM Code: 350.1**

**Usual Treatment**
- Pharmacologic: Despite few randomized trials, the mainstay of treatment is with anticonvulsant/antiepilepsy drugs. Initial response in over 70% of pt occurs with carbamazepine (Tegretol). Polypharmacy with other medications is common: baclofen, gabapentin, lamotrigine and oxcarbazepine. Pain or inability to tolerate these medications limits their use.
- Procedural:
  - Radiofrequency ablation (RFA): Under fluoroscopic control, a radiofrequency electrode is advanced into the foramen ovale and the pt awakened to describe the location of the parasethesis. The lesion made in cycles of 45–90 secs at temps 60–90° C. Glycerol, ethanol, and cryotherapy have been used to create a nerve lesion by this approach. Balloon compression of the ganglion for 1–6 min has been employed under general anesthesia.
  - Microvascular decompression (MVD): The posterior fossa is approached through a suboccipital craniotomy. The fifth nerve is identified and decompressed from the artery or vein and a surgical felt is interposed to protect the nerve. This procedure is performed under general anesthesia.
  - Stereotactic radiosurgery: the trigeminal nerve root at the point is targeted with a “Gamma knife” radiosurgery, a focused array of 201 intercepting beams of gamma radiation produced by separate cobalt sources. Few centers offer this treatment and long-term outcomes are yet unknown.


L. Jane Easdown

DISEASES

364
Truncus Arteriosus

**Risk**
- Uncommon congenital heart defect; <3% of all congenital heart defects
- No gender predilection

**Perioperative Risks**
- CHF
- Volatile agents may cause myocardial contractile depression and lower SVR.
- Inadvertent hyperventilation resulting in reduced PVR with excess PBF and worsening CHF
- Infective endocarditis
- Risks of CPB

**Worry About**
- Difficulty intubation due to assoc with velo-cardio facial syndrome (e.g., DiGeorge)
- Air embolus (VSD almost always present)
- Hyperoxia and hyperventilation result in pulm overcirculation.
- CV collapse at induction due to diastolic run off and associated trunval regurgitation with resulting coronary steal and myocardial ischemia
- Hypocalcemia due to parathyroid hormone dysfunction

**Overview**
- Single great artery arising from heart; supplies systemic, pulm and coronary circulations
- VSD almost always present; ASD in two-thirds
- Atria truncal valve; 50% are regurgitant
- Anomalies of coronary artery and aortic arch may be present.
- Pulmonary circulation arises directly from systemic circulation.
- Dominant physiology is a left to right shunt driven by relative lower resistance of the pulm vascular bed.
- Runoff from systemic circuit into PA during diastole may compromise myocardial perfusion.
- Primary goal is to balance PVR and SVR so that Qp:Qs is close to unity.
- Early development of pulm vascular obstructive disease due to excessive pulm blood flow.
- Preferred repair in early neonatal period before onset of pulm Htn.
- 30–40% of pts with TA have 22q11 deletion. Phenotypes incl DiGeorge syndrome and Sphrintzen syndrome.
- Uniformly fatal without surgical correction (50% die by 1 mo and 80% within 1 y)
- Approach 30% have 22q11 deletion resulting in phenotypic variants such as DiGeorge and Sphrintzen syndrome

**ICD-9-CM Code: 745.0**

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Difficult laryngoscopy and intubation</td>
<td>Difficulty feeding</td>
<td>Small mandible, small mouth</td>
<td>Pulse oximeter, ECG, ECHO, cardiac cath ±</td>
</tr>
<tr>
<td>CARDDIO</td>
<td>CHF-truncval valve regurgitation</td>
<td>Sweating during feeds</td>
<td>Cyanosis ± single S, Murmur—systolic or diastolic</td>
<td></td>
</tr>
<tr>
<td>RESPIRATORY</td>
<td>CHF—excessive pulm blood flow</td>
<td>Difficulty breathing</td>
<td>Tachypnea retraction</td>
<td>CXR (↑ pulm markings, cardiomegaly)</td>
</tr>
<tr>
<td>ENDO</td>
<td>Parathyroid hypoplasia</td>
<td>Seizures, tetany</td>
<td></td>
<td>Serum ionized Ca²⁺, parathyroid hormone level</td>
</tr>
<tr>
<td>IMMUNO</td>
<td>Cellular immunodeficiency</td>
<td>Recurrent infections</td>
<td>70% of 22q11 pts are immunosuppressed.</td>
<td>CBC, T-cell function</td>
</tr>
<tr>
<td>MS</td>
<td>Dysmorphic facies</td>
<td>Hypertelorism, low-set ears</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Perioperative Preparation**
- Treat CHF
- If intubated transport and ventilate with FIO₂ 21% aiming for SpO₂, 75–80% and appropriate hyperventilation to maintain PaCO₂, 45–50 mmHg and pH 7.25–7.35
- Avoid hyperventilation: Increase PBF and may compromise systemic and coronary perfusion.
- Check serum electrolytes, Ca²⁺, and Hct.

**Airway**
- High index of suspicion for difficult airway with appropriate precautions if velocardiofacial syndrome present.
- Maintain FIO₂ at 21% but may give a few breaths at 100% just prior to intubation.
- Once intubated return to FIO₂ 21% and avoid hyperventilation.

**Preinduction/Induction**
- Metabolic air bubble exclusion
- Preop antibiotics
- Consider inotropic support (e.g., dopamine at 3–5 mcg/kg/min) if MAP low prior to induction.
- Obtain baseline EKG prior to induction. Monitor for myocardial ischemia (best detected with EKG leads II and V) due to PA runoff.
- Volume infusion is unlikely to increase diastolic BP unless the pt is significantly volume depleted. Consider 1–2 mcg/kg bolus of phenylephrine for low MAP.
- Pts are usually ventricular volume overloaded and aggressive volume resuscitation will further elevate ventricular end-diastolic pressure compromising subendocardial perfusion.
- Balance PVR and SVR so Qp:Qs approaches unity. Key: Maintain SVR; keep SpO₂ below 90%.
- Surgeon must be in the OR prior to induction and prepared for sternotomy.
- For persistent hypertension rapid sternotomy followed by partial occlusion of PA to elevate MAP and reduce pulm overcirculation.

**Monitoring**
- Intra-arterial catheter and CVP. May have in situ umbilical arterial and/or venous lines.
- TEE valuable to assess truncal valve function, VSD patch leak, ventricular function and pulm artery pressure.
- Intracardiac line placement for postop management of preload.
- NIRs monitoring useful esp. during low flow CPB and DHCA

**Maintenance**
- Usually fentanyl infusion 2–4 mcg/kg/hr
- Deep hypothermia and circulatory arrest or low-flow CPB
- Inotropic drugs commonly used to facilitate weaning from CPB incl dopamine (1–5 mcg/kg/min), milrinone (0.5–0.75 mcg/kg/min) and low dose epinephrine (0.01–0.05 mcg/kg/min), calcium chloride (20–30 mg/kg/hr)
- Inhaled NO should be available in the OR for the post CPB period as high risk of pulm vasoactivity.

**Extracorporeal circulation**
- Postop ventilation usually required for at least 24 hr as pulm hypertensive crisis may occur. Not suitable for fast-tracking.

**Postoperative Period**
- Poor RV function (right ventriculotomy and Rastelli conduit placement): maintain appropriate inotropic support, afterload reduction and adequate preload for RV. Mechanical ventilation with minimal mean airway pressure.
- LV dysfunction (circulatory arrest, long bypass time, myocardial ischemia, truncal valve abn)
• Increased PVR and pulm Htn (low CO and low SaO₂) responds to hyperventilation, metabolic alkalosis, vasodilators (milrinone, PGE₁, NO), sedation (analgesia, paralysis).
• If PA pressures are high need to exclude residual VSD and RV outflow tract obstruction in addition to treating pulm Htn.

• Right to left shunting across PFO facilitates systemic cardiac output at the expense of SaO₂ in the face of RV dysfunction and elevated PA pressures.
• AV block requiring pacemaker.
• Bleeding
• Cardiac tamponade

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**Anticipated Problems/Concerns**

• CHF
• Truncal valve regurgitation and/or stenosis
• Pulmonary Htn
• Infective endocarditis
Tuberculosis (TB)

Risk
- Incidence in USA: 4.2 cases per 100,000 persons in 2008; worldwide over 9 million cases every year.
- Incidence of TB is decreasing in USA every year since 1992.
- Risk of TB is higher among homeless, elderly, Asian and Latin American immigrants, minorities and prisoners. Also immunosuppression (e.g., HIV infection, transplant recipients, CRF) increases the risk of TB infection.
- TB is the leading cause of death among HIV infected pts.

Perioperative Risks
- Risk to the pt and risk to medical personnel
- Pt risk depends on extent of pulm disease, other organ systems involvement, and overall health.

Worry About
- Overall health status of the pt, infectiousness of the pt, cross contamination through anesthesia machine and effects of anti-TB therapy on organ systems.

Overview
- Mycobacterium tuberculosis is the causative organism of TB.
- Pulm TB is the most common form of TB but other organ systems could be affected (e.g., intestine, spine and bones, kidneys, and brain).
- TB could be fatal if untreated.

ICD-9-CM Codes: 010–018

Etiology
- TB is transmitted by droplet nuclei produced by coughing, sneezing, or talking.
- TB does not spread by casual contact (e.g., shaking hands, sharing food or drink, or touching bed linens).
- Primary infection could be reason for up to one third of the cases.

Usual Treatment
- 2 mo therapy with isoniazid, rifampin, pyrazinamide, and ethambutol and then 4 mo therapy with only the first two drugs. All medicines taken orally.
- All four drugs are recommended in drug-resistant cases of TB for 6 mo.
- AIDS/HIV pts need a longer duration (9–12 mo).

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<tbody>
<tr>
<td>GENERAL</td>
<td>Night sweats, wt loss</td>
<td>Fever</td>
<td>Tuberculin skin test and in vitro T-cell release of IFN-gamma (IGRAS)</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Hilar or mediastinal lymphadenopathy, apical infiltrate or necrosis</td>
<td>Cough and hemoptysis</td>
<td>None or inspiratory rates in affected area</td>
<td>CXR, sputum culture</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Pericardial effusion, constrictive pericarditis</td>
<td>SOB</td>
<td>Signs of tamponade, muffled heart sounds</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>CNS</td>
<td>TB meningitis</td>
<td>Listlessness, headache, seizures, coma</td>
<td>Altered mental status, cranial nerve abs</td>
<td>LP, CSF analysis</td>
</tr>
<tr>
<td>GI</td>
<td>Peritonitis, enteritis</td>
<td>Abdominal pain, obstruction</td>
<td>Palpable mass, ascities</td>
<td>Endoscopy and biopsy, ascitic fluid analysis/culture</td>
</tr>
<tr>
<td>GU</td>
<td>Chronic cystitis, epididymitis, hydronephrosis, female genital tract disease</td>
<td>Late appearance of pyuria, hematuria</td>
<td>May have thickened epididymis</td>
<td>Urine analysis and culture, cystoscopy</td>
</tr>
<tr>
<td>Skeletal TB</td>
<td>Wt bearing joints (e.g., spine, hip, knee)</td>
<td>Pain, kyphosis</td>
<td>Spine tenderness</td>
<td>X-ray, CT, bone biopsy</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Properly fitted N95 mask use by the care team.
- Schedule TB and/or suspected TB pts at the end of the day to maximize time for cleaning and minimize spread.
- Use OR that has an anteroom; otherwise keep the doors closed, minimize traffic and use additional air cleaning (e.g., UVGI)

Postoperative Care
- Postop recovery in an AII room (AII room—an isolation room with single occupancy, negative pressure in the room, airflow rate at 6–12 ACH or equivalent).
- If all room not available, air cleaning technologies (e.g., HEPA filtration and UVGI) can be used.

Use disposable equipment, add bacterial filter (0.3 μm) to expiratory side or at Y-connection of anesthesia circuit.
- After use stop all gas flow through the anesthesia machine for at least 1 hr to avoid cross contamination.

Monitoring
- Standard ASA monitors
- Consider case by case according to co-morbidities and type of surgery.
Ulcerative Colitis, Chronic

Risk
- Incidence in USA and Northern countries of 35-100/100,000, incidence of 11/100,000/y with 2- to 4-fold increased frequency in Jewish populations
- Mortality highest in early years of disease, or with prolonged disease due to risk of colon cancer, 2 peaks for age of onset: 15–30 and 60–80
- M:F ratio: 1:1, smokers-nonsmokers 0.4:1, former smokers-nonsmokers 1.7:1. Up to 20% with positive family Hx.

Perioperative Risks
- Inflammatory mediators activate coagulation cascade in local blood vessels
- Chronic steroid use can cause adrenal insufficiency, delayed wound Hx.

Worry About
- Diarrhea causing metabolic acidosis, hypokalemia, electrolyte abn, intravascular volume depletion
- Defects in bleeding or clotting due to activation of coagulation cascade
- Bowel distension precluding use of NO and increasing risk of perforation
- Extracolonic manifestations: Primary sclerosing cholangitis and/or cirrhosis of liver; choose appropriate anesthetics, analgesics, and NMBs
- Spondylitis limited cervical range of motion, restrictive pulm mechanics

Overview
- Indications for surgery incl toxic megacolon, colonic perforation, massive hemorrhage, obstruction, and cancer prevention or resection. If pt is presenting for surgery, disease is in progressive stage and operation can be urgent/emergent in nature.
- Pts may have steroid dependence, hypovolemia, electrolyte imbalance, malnutrition, hypoalbuminemia, anemia, bleeding
- Sulfasalazine is mainstay of treatment for all stages of disease. Side effects incl blood dyscrasias, aplastic anemia, hemolytic anemia, hepatitis, pancreatitis, nephrotoxicity, hypersensitivity pneumonitis, impaired folate absorption

ICD-9-CM Code: 556

Etiology
- Etiology unknown
- Genetics, exogenous factors, host factors, and specific environmental factors are all hypothesized to play a role

Usual Treatment
- Mild: Sulfasalazine or other 5-aminosalicylates (5-ASA)
- Moderate: 5-ASA + glucocorticoid oral and enema, electrolyte repletion, parenteral nutrition
- Severe: 5-ASA, glucocorticoid enema, glucocorticoid PO or IV
- Fulminant: Glucocorticoid IV, cyclosporine IV, azathioprine PO, 6-mercaptopurine PO, infliximab IV

Perioperative Implications

Preinduction/Induction/Maintenance
- Fluid, electrolyte, volume repletion
- Stress dose steroids if needed
- Special attention to airway if ankylosing spondylitis
- Careful choice of anesthetics if hepatic or renal dysfunction
- Aggressive volume replacement

General Anesthesia
- Consider renal function for opioid dosing
- Consider renal, biliary function for NMB dosing
- Monitor ventilator settings carefully if restrictive pulm mechanics or toxic megacolon
- Beware of NO for risk of perforation

Regional Anesthesia
- Caution with local anesthetic esters: May decrease action of sulfasalazine

Postoperative Period
- Maintain normothermia for wound healing and coagulation
- Early parenteral nutrition

Anticipated Problems/Concerns
- Complicated operations with adhesions, obstructions, perforation risk
- Large intraop fluid requirement
- Need for stress dose steroids
- Correct electrolyte abn
- Risk of hemorrhage

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Tachycardia, hypotension,</td>
<td>BUN/Cr</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>orthostatic vital signs,</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>delayed capillary refill</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, thrombocytosis</td>
<td>Passing fresh blood</td>
<td>Pallor</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Metabolic acidosis, electrolyte abn</td>
<td>Tachypnea, oliguria</td>
<td>Electrolytes,</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>BUN/Cr, ABG</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive pulm mechanics</td>
<td>SOB, DOE</td>
<td>Cyanosis, SpO₂</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(if ankylosing spondylitis)</td>
<td></td>
<td>CXR, PFTs</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hyposensitivity pneumonitis from 5-ASA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Diarrhea, bowel obstruction/perforation</td>
<td>Diarrhea, no bowel</td>
<td>Electrolites</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hepatic steatosis, PSC/cirrhosis</td>
<td>movements</td>
<td>Abd X-ray</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Abd CT</td>
<td></td>
</tr>
</tbody>
</table>

Upper Respiratory Infections

Selina Read

Overview
• To cancel or not to cancel; that has been the dilemma of many anesthesiologists who are confronted with a pt that has recently, or currently has an URTI, who is scheduled for elective surgery.
• Several studies have linked URTIs to possible morbidity; however, none have linked it to increased mortality.
• Retrospective studies: Children with a recent URTI were at higher risk for laryngospasm, bronchospasm, and stridor. Children with URTI had a two to seven times greater incidence of resp complications. The complication risk increased to 11-fold if the trachea was intubated.
• Prospective studies: Children who developed laryngospasm were twice as likely to have a URTI.

ICD-9-CM Code: 465.9

Etiology
• Affect the airway by making them esp. susceptible to touch or chemical irritation, such as airway hyperreactivity from a viral etiology.
• Postulated that viruses release neuroamines which damage the M2 muscarinic receptors, increasing acetylcholine released at NM junctions setting off vagally mediated bronchoconstriction.
• Viruses also cause the release of chemical mediators, such as bradykinin, prostaglandins, and histamine that contribute to bronchospasm.
• URTIs increase airway secretions intensifying intraop atelectasis, decreasing diffusion capacities and increasing closing volumes.

Usual Treatment
• Postponing an elective case if the pt has a fever, purulent rhinitis, or productive cough.
• Laryngospasm is treated with PPV or small dose muscle relaxation.
• Bronchospasm treated by deepening the anesthetic, administering IV bronchodilators or inhaled beta-agonists.
• Hypoxemia treated with supplemental O2.
• Atelectasis can be decreased with incentive spirometry or sigh breaths intraop.
• Increased secretions can be managed by frequent suctioning.

Postoperative Period
• Almost all of the complications cited as a possible cause to cancel surgery can easily be treated by an experienced and diligent anesthesiologist along with proper monitoring and rapid response in the recovery room.
• Must monitor heart rate and pulse oximetry.

Anticipated Problems/Concerns
• Must have all available airway equipment such as ETTs and LMAs.
• Have rescue medications available and the ability to administer them in a variety of ways, esp. beta-agonists.

Assessment by Hx

Test

<table>
<thead>
<tr>
<th>System</th>
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<th>PE</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Tachycardia due to infection</td>
<td>Assess for possible CHD which can complicate the picture</td>
<td>Auscultation: BP, HR</td>
<td>None</td>
</tr>
<tr>
<td>RESP</td>
<td>Increased secretions, bronchospasm</td>
<td>Quantify cough, secretions</td>
<td>Auscultation: wheezes, ronchi</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Dehydration</td>
<td>Poor intake, UO</td>
<td>Skin turgor, sunken fontanelles</td>
<td>BMP</td>
</tr>
</tbody>
</table>


Upper Respiratory Tract Infections

Risk
• Most adults will suffer one upper resp tract infections (URTI) per year, this incidence jumps to approx six episodes per year in the pediatric population. Approx 95% of the infections have a viral etiology.
• URTIs are generally self-limiting, however, airway hyperreactivity may persist for several weeks.
• Adults are less likely to have URTI due to larger airways enabling them to compensate with edema and increased secretions.
• Those with underlying disease, esp. diseases affecting the airways, are more likely to have complications following anesthesia when confounded with URTI.

Perioperative Implications

Preinduction/Induction/Maintenance
• Evaluate whether symptoms are severe or likely due to infectious etiology. Examples are copious secretions and fever. If so, consider postponing.
• Minimize secretions, deep suctioning after pt deeply anesthetized.
• Avoid airway stimulation if possible, consider LMA or bag masking.
• IV necessary for adequate hydration and potential medications if bronchospasm encountered.
• Optimization of resp status is of utmost importance.

Monitoring
• Standard ASA monitors absolutely necessary: Heart rhythm, pulse oximeter and BP
• Have ABG monitoring available.

General Anesthesia
• Depending on the procedure, may be the best option to allow for deep anesthesia during stimuli, helping to prevent bronchospasm
• Agent used for induction can have effect on chance of bronchoconstriction: Propofol and sevoflurane best, thiopental worst.

Regional Anesthesia
• Useful as an adjunctive anesthetic.

Anticipated Problems/Concerns
• Viruses also cause the release of chemical mediators, such as bradykinin, prostaglandins, and histamine that contribute to bronchospasm.
• URTIs increase airway secretions intensifying intraop atelectasis, decreasing diffusion capacities and increasing closing volumes.

Perioperative Risks
• Complications incl bronchospasm, subglottic edema, stridor, desaturations, apneas, and atelectasis.
• A pt with a fever, purulent rhinitis or productive cough should have elective surgery cancelled.

Worry About
• Lung specific: Bronchospasm, desaturations, apnea, and atelectasis
• Cancellation of surgery and prolonged hospital stay.

Overview
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Urinary Lithiasis

Risk
- Annual incidence of stone disease: 16.4/10,000
- 12% of all individuals will experience calculous disease
- M:F ratio: 1.3–1.6:1
- Race with highest prevalence: Caucasians; rare in North American Indians and African-Americans
- Peak incidence: Third to fifth decade of life
- Recurrence rate after first stone: 15% at 1 y, 35–40% at 5 y, 50% at 10 y

Perioperative Risks
- Morbidity and mortality very low

Perioperative Implications
Preoperative Preparation
- If obese, require acid aspiration prophylaxis and airway evaluation

Monitoring
- Routine
- Temp monitoring during immersion lithotripsy essential because water temp may produce hyperthermia or hypothermia
- Shock waves synchronized to ECG to avoid dysrythmias

Preinduction
- Adequate padding to avoid nerve damage

Induction
- Sedation may be adequate for lithotripsy and minor ureteroscopy procedures, GA, spinal or continuous lumbar epidural with T8 level epidural are all acceptable depending on type of procedure, co-morbid diseases and pt preference.

Overview
- Urolithiasis refers to abn concretions occurring anywhere along collecting system of urinary tract
- Most stones seen in industrialized countries contain calcium oxalate (75%); remainder are composed of uric acid, struvite, or cystine
- If properly treated, urolithiasis does not adversely affect life expectancy
- Calculi <4 mm in diameter usually pass without intervention
- ~20% of stones cause enough symptoms to require surgical removal

ICD-9-CM Codes: 592.0 (Calculus of kidney); 592.1 (Calculus of ureter)

Etiology
- Intrinsic factors: Renal tubular acidosis, cystinuria, primary hyperparathyroidism
- Gout, Lesch-Nyhan syndrome, Dent's disease
- Extrinsic factors: Increased environmental temp resulting in increased perspiration and hyperconcentration of urine (southeast and southwest regions of USA); low-intake drinking habits resulting in low UO2; diet rich in calcium, animal fat (uric acid), or leafy vegetables (oxalate); immobility, incl sedentary occupations, obesity

Usual Treatment
- Observation, hydration, and symptomatic pain relief until spontaneous passage
- Medical expulsive therapy (MET) using nifedipine or tamsulosin can facilitate earlier stone passage
- If surgical intervention necessary (20%), choice based on stone size and location
- Shock wave lithotripsy
- Flexible ureteroscopy and Holmium laser lithotripsy
- Percutaneous nephrolithotomy
- Retroperitoneal laparoscopy

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<td>↑ Heart rate or BP 2° to pain</td>
<td>Tachycardia</td>
<td>Htn</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Grunting respiration during renal colic</td>
<td>Normal chest exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Abdo pain</td>
<td>N/V Moving irritation in abd</td>
<td>Tenderness to deep palpation of abd</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal colic characterized by unusually severe pain localizing to affected flank; pain may radiate to groin or abd</td>
<td>Sudden onset of flank pain</td>
<td>Flank tenderness to palpation over affected kidney</td>
<td>UA (hematuria), BUN/Cr Non-contrast CT scan (gold standard) plain film (KUB), intravenous pyelography (IVP)</td>
</tr>
</tbody>
</table>


Maintenance
- Central blood volume increases
- May become hypotensive 2° to warm water decreased SVR
- Vital capacity decreased and work of breathing increased
- Pleural effusion or hydropneumothorax may occur during percutaneous renal procedures

Adjuncts
- Visualization of stone may require iodine-containing contrast material
- Anticholinergic agents (glycopyrolate) occasionally given to shorten lithotripsy treatments; however, tachycardia can occur, resulting in myocardial ischemia in high-risk pts
- Most pts receive prophylactic antibiotics prior to urinary tract procedures

Anticipated Problems/Concerns
- Peroneal nerve compression from lithotomy position
- Allergic reactions in 5% receiving IV contrast media
- Steinstrasse, ureteral obstruction by fragmented calculi, may cause ureteral colic following lithotripsy
- Htn may occur following lithotripsy
- Septic complications occur in 1% after lithotripsy
- Ureteral injury occurs in 9% of ureteroscopy procedures, with 1.6% requiring further surgical intervention
- Bladder perforation may present as shoulder pain, unexplained Htn, tachycardia in PACU
Urticaria, Cold

**Risk**
- Prevalence is very low, < 1/100,000.
- Appears in all races and genders, reported between ages 3 mo and 74 y but seen typically at 18–25 y

**Perioperative Risks**
- Can develop urticaria or angioedema with skin cooling and rewarming
- Shock-like reactions can occur with whole body cold exposure
- Cooling with cardiopulmonary bypass can induce symptoms

**Worry About**
- Cold exposure of pt (e.g., cold room, cold fluids, cold instruments or devices to cool skin)

**Overview**
- Characterized by appearance of urticaria or angioedema after cold exposure
- Can be familial or acquired (most common). Acquired can be idiopathic or 2° to an underlying disease (e.g., malignancy, cryoglobulinemia, or infection).
  - In 2° syphilis, it is caused by the Donath-Landsteiner antibody.
  - Disease course varies widely. Symptoms may resolve after several months or can last 5–9 y or more. Symptoms in 2° cold urticaria usually regress with treatment of underlying disease.
- Dx by cold stimulation test (e.g., the ice cube test)

**Etiology**
- 1° cold urticaria appears related to skin mast cells sensitized to cold by a serum factor, very likely antibodies
- Sensitized skin mast cells (not blood basophils) release histamines on interaction with cold
- Similar activation by cryoglobulins appears in 2° cold urticaria

**Usual Treatment**
- Antihistamines, both H₁ and H₂, successful at reducing occurrences even for hypothermic cardiopulmonary bypass.

**ICD-9-CM Code:** 708.2

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
</tr>
</thead>
<tbody>
<tr>
<td>SKIN</td>
<td>Urticaria or angioedema</td>
<td>Hx of cold reactions</td>
</tr>
<tr>
<td>RESP</td>
<td>Angioedema</td>
<td>Hx of swelling on cold exposure</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Antihistamines H₁ and H₂ only if a cold challenge anticipated during surgery

**Monitoring**
- Temp, skin condition

**Maintenance**
- Warm IV fluids; keep room and pt warm

**Anticipated Problems/Concerns**
- Localized areas of urticaria and/or angioedema not of great concern, but serious widespread edema can compromise the airway or lead to fluid extravasation or shock
- Maintain temp
- Pretreatment with antihistamines if cold is unavoidable
Uterine Rupture

**Risk**
- Incidence varies: 1/1280–1/3000 for all vaginal deliveries
- Incidence of uterine rupture in women with unscarred uterus approx 0.01% in industrialized countries
- Incidence of rupture with prior C-section ranges from 0.2–0.8%.
- In women with previous upper uterine surgery (myomectomy), incidence of rupture can be as high 1.7%.
- Risk factors incl prior uterine scar; rapid, tumultuous labor; prolonged, augmented labor; trauma; and grand multiparity, polyhydramnios

**Perioperative Risks**
- Potentially catastrophic for mother and fetus. Maternal morbidity is ~0.1% and incl hemorrhage, shock, and hysterectomy. If fetus delivered within 30 min, fetal morbidity improved but still incl hypoxemia and/or acidosis, depressed apgar scores, and admission to neonatal ICU.
- Maternal mortality greatly increased in pts with traumatic or spontaneous rupture likely 2° to delayed suspicion and/or Dx and treatment.

### ASSESSMENT POINTS

<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Tachycardia, hypotension, shock</td>
<td></td>
<td>BP, HR, orthostasis</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Discomfort with breathing due to diaphragmatic irritation</td>
<td></td>
<td>Tachypnea, labored breathing</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Vaginal bleeding</td>
<td>Abd pain, shoulder pain, absence of contractions</td>
<td>Abdom tenderness, presenting fetal parts</td>
<td></td>
</tr>
<tr>
<td>FETUS</td>
<td>Category 2 or 3 fetal distress</td>
<td></td>
<td>HCT</td>
<td></td>
</tr>
</tbody>
</table>


**Induction/Airway**
- If mother grossly unstable or no epidural, proceed to GA
- Rapid sequence induction with cricoid pressure
- If severely hypovolemic, transfuse with induction. As well, use minimal vasodilating and/or negative inotropic induction agents to spare BP.

**Maintenance**
- If mother stable, may use continuous epidural for procedure
- If GA, 100% FIO₂ predelivery, volatile anesthetic at 0.5 MAC or less if maternal BP tolerates.
- Restore blood volume to keep Hgb >7 gm/dL and BP stable
- After delivery, if stable, consider titrating opioids.
- Fetus may require intensive resuscitation (neonatologists present).

**Worry About**
- Massive hemorrhage in mother
- Fetal hypoperfusion and hypoxemia

**Overview**
- Due to a breach in the myometrium, which is often 2° to a separation of a previous C-section scar, uterine rupture can occur antepartum, intrapartum, or postpartum. The lower uterine segment, at term, contains mostly connective tissue and little placental tissue. Therefore, most ruptures are asymptomatic and do not result in maternal and/or fetal compromise. However, if placental tissue is involved, massive bleeding with resultant need for emergent C-section and/or laparotomy can occur.
- Vaginal delivery is preferred over C-section as maternal blood loss and maternal morbidity are decreased.
- ACOG advocates trial of labor after delivery in pts with previous low transverse uterine scar. Mothers with classical C-sections and/or inductions with prostaglandins are both discouraged by ACOG as risk of rupture is greatly increased. As well, trial of labor after C-section is discouraged in hospitals where emergency C-sections cannot be performed within 30 min.

**Etiology**
- Separation of scar from previous C-section, often during trial of labor
- Rupture of myomectomy scar (highest incidence of rupture)
- Weak or stretched uterine muscles due to grand multiparity, polyhydramnios, or multiple gestations
- Precipitous labor or prolonged labor with oxytocin augmentation
- Traumatic rupture

**Usual Treatment**
- When occurring antepartum or during labor, urgent and/or emergent laparotomy with C-section and uterine repair or hysterectomy is the only treatment. Urgency is determined by speed of dx and maternal and fetal stability.
- If diagnosed incidentally postpartum, mother may undergo close observation without surgery.

**Anticipated Problems/Concerns**
- Other more common causes of antepartum hemorrhage incl placenta previa and placental abruption
- Pregnant mothers who hemorrhage can go into DIC quite quickly. Monitor coags, plt.
- Symptoms of rupture may be misleading. Must possess high index of suspicion to diagnose in a timely fashion.
- Rupture of classic scar or previous upper uterine surgery scar much more likely to result in severe hemorrhage

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**Matthew Fiegel**

**DISEASES**

**Overview**
- Diseases without central venous catheter
- ASA monitors, arterial catheter, and with or without central venous catheter

**Induction/Airway**
- If mother grossly unstable or no epidural, proceed to GA
- Rapid sequence induction with cricoid pressure
- If severely hypovolemic, transfuse with induction. As well, use minimal vasodilating and/or negative inotropic induction agents to spare BP.

**Maintenance**
- If mother stable, may use continuous epidural for procedure
- If GA, 100% FIO₂ predelivery, volatile anesthetic at 0.5 MAC or less if maternal BP tolerates.
- Restore blood volume to keep Hgb >7 gm/dL and BP stable
- After delivery, if stable, consider titrating opioids.
- Fetuses may require intensive resuscitation (neonatologists present).

**Exubation**
- Normal extubation criteria: Pt awake, strong, hemodynamically stable, no continuous bleeding, fairly normal acid/base, electrolyte status

**Postoperative Period**
- Consider ICU overnight
- EBL: 3000–6000 mL, check Hct, coags frequently
- Pain score: 6–8, consider IV PCA or postop epidural if coagulation status normal
Varicella-Zoster Virus

Overview
- Viral cause of varicella (chickenpox) and herpes zoster (shingles)
- Both nosocomial transmission and direct contact
- Development of herpes zoster common in immunocompromised pt and may be forerunner of AIDS
- Zoster is reactivated form of varicella from neural ganglion cells and can be assoc with severe pain
- May lead to congenital abn if contracted during 1st trimester of pregnancy.

ICD-9-CM Code: 053.9

Etiology
- Herpes group of viruses

Usual Treatment
- Varicella immune globulin
- Vaccine available but controversial
- Antiviral medications decrease the duration of symptoms and the likelihood of postherpetic neuralgia, esp. when initiated within 2 d of the onset of rash.
- Most common is acyclovir; valacyclovir, penciclovir, and famciclovir, are also available.
- Corticosteroid controversial for postherpetic neuralgia in pts with herpes zoster, controlled-release oxycodone was superior to placebo in the early period of pain.

Risk
- Prevalence: <10% of adults seronegative
- Usually contracted during childhood

Perioperative Risks
- Minimal additional risk to pt unless immunocompromised
- Risk of infection to caregivers

Worry About
- Encephalitis in immunocompromised pt
- Potential nosocomial transmission
- Acyclovir-induced nephrotoxicity
- Transmission to pregnant woman

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Pneumonia</td>
<td>Dyspnea</td>
<td>Rhonchi</td>
<td>CXR</td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenic purpura</td>
<td>Bleeding</td>
<td>Plts</td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Rash</td>
<td>Erythematous macules, papules, vesicles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Acyclovir nephrotoxicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalitis; transverse myelitis</td>
<td>MS changes</td>
<td>Vision changes</td>
<td>CT scan</td>
</tr>
<tr>
<td>PNS</td>
<td>Zoster shingles</td>
<td>Shingles in single dermatome</td>
<td>Multiple dermatomes in immunocompromised</td>
<td></td>
</tr>
<tr>
<td>IMMUNO</td>
<td>Assoc with AIDS</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Consider isolation precautions

Monitoring
- Routine

Airway
- Routine

Induction/Maintenance
- Routine
- May require modification of periop pain management regimen if treatment for post-herpetic neuralgia

Extubation
- Routine

Anticipated Problems/Concerns
- Multiple dermatomes may indicate immunocompromised individual
- Avoid exposing pregnant individuals to virus
Ventricular Fibrillation

Risk
- VFIB/VTach: Most frequent rhythm in sudden cardiac arrest
- At risk are the 1.5 million/y in the USA who have acute MIs: About 540,000 will die, 350,000 before they reach hospital (this incлеж death from dysrhythmia and myocardial failure)
- 1-y mortality in near-sudden death survivors: 20–30% if nonresponsive to antiarrhythmics (20–50% of near-sudden death survivors)

Perioperative Risks
- 1˚ VFIB, if assoc with acute infarction, when treated promptly with defibrillation, may not affect prognosis
- 2˚ VFIB (preceded by pump failure or hypotension) assoc with 75–80% mortality during hospitalization

Worry About
- Hypoxemia, hypercarbia, hyper- or hypokalemia, ischemia hypomagnesemia, digitalis toxicity, acid-base abn
- Antiarrhythmic drug levels
- Availability of defibrillator, myocardial ischemia and early revascularization

Overview
- Asynchronous, chaotic contraction of ventricles characterized by no organized ventricular depolarization and therefore no QRS; no cardiac output
- Coarse VFIB indicates recent onset, readily correctable with prompt defibrillation

- Fine VFIB (coarse asystole) indicates delay since collapse; successful resuscitation more difficult

ICD-9-CM Code: 427.41

Etiology
- Usually ischemic, often assoc with LV aneurysm
- Idiopathic cardiomyopathy
- Coronary spasm esp. in the immediate postop period
- Hypothermia
- Long QT syndrome is assoc with VTach, esp. torsades de pointes (one type of polymorphic VTach; other types not assoc with long QT)

Usual Treatment
- Definitive emergency Rx is always electrical defibrillation: External—either manual or automatic (AEDs)—or internal: May be implanted (ICDs)
- Time to defibrillation is a major determinant of survival, with chances of success reduced by 10% each minute
- Early bystander CPR and early defibrillation are the only factors that have increased, the rate of return of spontaneous circulation (ROSC) and decreased mortality.
- Vasopressors such as epinephrine and vasopressin are indicated after three successive countershocks fail to terminate VFIB. Vasopressors improve coronary and cerebral perfusion pressures; increased coronary perfusion pressure is assoc with increased likelihood of ROSC.
- Vasopressin may have fewer side effects than epinephrine, while being equally or more effective, particularly in acidotic pts. Vasopressin’s longer duration of action (10–20 min) has led to the recommendation of a single, one-time dose for VFIB.
- Amiodarone is the only antiarrhythmic assoc with improved resuscitation rates from VFIB; it is recommended after 3 successive shocks, an IV vasopressor (epinephrine or vasopressin), and a subsequent 4th shock are unsuccessful in restoring a perfusing rhythm
- Prospective trials of lidocaine and bretylium in VFIB pts have shown no benefit on outcome. However, based on historical use and the lack of side effects, lidocaine is considered an alternative to amiodarone in VFIB.
- Due to inconsistent availability, side effects, and lack of confirmed benefit, bretylium is no longer recommended for VFIB
- Evidence supporting procainamide use in VFIB is limited, while the need for slow infusion makes it less than ideal
- Magnesium may be beneficial in torsades de pointes (polymorphic VTach assoc with prolonged QT), but routine use does not improve outcome.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Right radical neck dissection assoc with increasing QT interval</td>
</tr>
<tr>
<td>CARDIO</td>
<td>No effective cardiac output</td>
</tr>
<tr>
<td>RESP</td>
<td>Apnea should be anticipated</td>
</tr>
<tr>
<td>CNS</td>
<td>Glucose administration may worsen CNS outcome</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Antiarrhythmic drug levels in optimal therapeutic range
- If for EPS, ablation, or ICD, antiarrhythmic drugs withdrawn on ECG monitoring
- Avoid anticholinergic premedication or sympathetic stimulation
- For pts with prolonged QT syndrome consider β-blockers or prophylactic left stellate ganglion block

Monitoring
- Consider ECG and pulse oximetry en route to OR.
- Consider arterial catheter for transport and in OR.

Airway
- Apnea expected with acute VFIB; ventilation should be supported with 100% O2.
- Airway secured with ETT if 3 successive countershocks fail to restore perfusing rhythm

Induction
- Avoid ketamine; intubate after adequate depth of anesthesia

Maintenance
- Suppress sympathetic responses to stimulation

Exubation
- Suppress sympathetic stimulation; extubate when spontaneous ventilation with oropharyngeal reflexes has been restored
- Reversal of NMBs acceptable

Regional: Serum levels of local anesthetics given epidurally may effect intraop defibrillation threshold testing during ICD placement
- Defibrillator should be available with sterile defibrillator paddles on surgical field; pharmacologic therapy for dysrhythmia conversion/main- tenance, for treating Htn and tachycardia, which frequently follow defibrillation; bradycardia may require pacing.

Postoperative Period
- Cardiac monitoring; resumption of preop antiarrhythmics, maintaining adequate oxygenation and ventilation
- Avoid and treat promptly electrolyte abn
- Post-defibrillation pain score: 1–3 from chest wall and psychic disturbances
- Psychiatric counseling if disturbed by shock or “out of body” experience

Anticipated Problems/Concerns
- PA catheter insertion may induce VTach or VFIB in dysrhythmia-prone pts; if PA catheter necessary consider central venous placement with advancement after ventricular dysrhythmia procedure completed
- For pts with prolonged QT syndrome avoid drugs that prolong the QT interval (class IA antiarrhythmic drugs such as quinidine and procainamide)
- Psychic disturbances from defibrillation in aware state
### Ventricular Preexcitation Syndrome

#### Risk
- A premature activation of ventricular myocardium via anomalous accessory pathways (AP) without physiologic delay in AV junction. Depending on the type of AP, several variants of preexcitation syndrome have been described.
- Wolff-Parkinson-White (WPW) syndrome is caused by an accessory pathway between atria and ventricles (bundle of Kent), which is found in 0.1–0.3% of the population, is more prevalent in males, and is characterized by a short P-R interval (<0.12 s), widened QRS (>0.10 s) and delta waves, and episodes of tachyarrhythmias.
- Anomalous pathways other than accessory AV pathways may cause preexcitation or participate in reentry tachycardia, but they are rarely diagnosed.
- Lown-Ganong-Levine (LGL) syndrome usually reflects abn antegrade and retrograde conduction through the ‘James bundle’ (atria to bundle of His)—intramural or parasural fast pathway. This syndrome is characterized by a short PR interval, normal QRS and no delta waves. It manifests itself through intermittent reentrant-type PSVT or paroxysmal atrial fibrillation or flutter. The frequency of LGL is up to 0.5% in adults.
- LGL may be more prevalent in women.
- Mohaim type, a less common form of preexcitation, is associated with the presence of Mahaim fiber (accessory connections running into the right ventricular muscle) and is characterized by normal PR, long QRS and delta wave, may trigger episodes of AFIB and SVT. Prevalence is unknown.
- Pregnancy, through an unknown mechanism, may predispose to exaggerate symptoms of WPW-related PSVT.

#### Perioperative Risks
- Asymptomatic forms of WPW (ECG pattern) may present no added risk. Symptomatic individuals with WPW are prone to PSVT (up to 80%), less commonly to AFIB (up to 30%) and AFLT (5%) with a rapid ventricular rate which may occasionally deteriorate to VTach, even VFIB.
- There is a danger that WPW patterns of ECG will be mistaken for a bundle branch block, due to the wide QRS, an MI, due to the negative delta waves simulating pathologic Q waves, other tachyarrhythmias (incl V-Tach). All prompt inappropriate treatment.
- General anesthesia may unmask asymptomatic WPW syndrome due to precipitating conduction in the pre-existing anomalous AP by volatile agents (particularly Halothane).
- Drugs used to suppress AV conduction (to slow the ventricular rate in treatment of AFIB/AFLT) may dangerously accelerate the rate in WPW.

#### Worry About
- Hyperadrenergic states, over stimulation and other interactions that may provoke or aggravate tachyarrhythmias
- High spinal anesthesia may be associated with an effective block of the sympathetic cardiac accelerator nerve and suppression of normal AV conduction. Along with relative parasympathetic predominance, it may further facilitate conduction by the AP, resulting in preexcitation and tachyarrhythmias.
- Effects of both inhalation and IV agents may suppress normal conduction pathways and increase conduction of AP, resulting in preexcitation.
- Some CV medications may have unfavorable effects, e.g., Ca+2-channel blockers (diltiazem, verapamil) and digitalis slow AV nodal conduction and may enhance conduction over AP, worsening WPW-related tachyarrhythmia.
- Potential of WPW-related PSVT to deteriorate into AFIB/AFLT, with danger of extremely rapid ventricular rates ensuing VTach/VFIB.

#### Overview
- SVT in pts with pre-excitation is generally owed to a reentrant mechanism, in which the AP plays an essential role. Re-entrant SVT involves the presence of dual conducting pathways between the atria and the ventricles: natural AV nodal His-Purkinje tract and AV accessory (or bypass) tract, such as Kent fibers, James bundle, or Mahaim fibers.
- Re-entrant orthodromic tachycardia during PSVT may produce normal looking narrow QRS complex in the absence of delta wave since normal pathway is used for ventricular depolarization. The AP, alternatively, is used for the retrograde conduction essential for the reentry mechanism. PVCs may also initiate orthodromic PSVT.
- Less common antidromic tachycardia may produce wide QRS complex with delta waves. In this case, a shorter refractory period in the AP may cause a block of ectopic atrial impulse in the normal pathway, with antidromic conduction down the accessory tract and then retrograde reentry of the normal pathway with ventricles activated via accessory pathways.
- Orthodromic and antidromic PSVT account for 70–80% of all paroxysmal tachycardia in pts with preexcitation syndrome, up to 30% of AFIB and up to 10% of AFLT. Primary VTach/VFIB is rare, but they may occur as a result of rapid SVT.
- The first episode of PSVT in many pts (regardless of ECG markers of pre-excitation during sinus rhythm) appears before the age of 20, rarely in middle age, and infrequently after the age of 50. The frequency of episodes of PSVT increases with age in pts with WPW.
- The rate of paroxysmal SVT may be faster in pts with LGL syndrome compared to pts with normal P-R intervals. In addition, the capacity for more rapid conduction may have an effect during AFIB/AFLT.

IDE-9-CM Codes: 426.7 (WPW); 426.81 (LGL)

#### Etiology
- Both WPW and LGL may be congenital or hereditary in nature.
- May be associated with congenital cardiac defects (e.g., Ebstein anomaly)
- May be associated with certain acquired cardiac defects, such as cardiomyopathy, idiopathic hypertrophic subaortic stenosis, or asymmetric septal hypertrophy.

#### Usual Treatment
- Caution should be exercised when treating WPW syndrome tachycardia. Agents with an AV node blocking effect, such as adenosine, Ca+2-channel blocker (diltiazem, verapamil) or digitalis, increase the chance of worsening existing tachycardia and precipitating AFIB/AFLT. Digitalis generally is not recommended in adult pts with WPW, particularly in treatment of AFIB/AFLT.
- Initial treatment of choice in WPW pts with hemodynamically unstable tachyarrhythmias (incl AFIB/AFLT) is direct-current synchronized electrical cardioversion. Minimum effective energy (100 joules) should be used initially; energy should be titrated to minimize potential injury to myocardium.
- Treatment of AFIB/AFLT and wide-complex tachycardia associated with WPW syndrome is necessarily different from the treatment of AFIB/AFLT in pts with a normal heart. In hemodynamically stable pts, AP suppressing agents would be the treatment of choice, e.g., procainamide (class Ia agent) and propranolol (class II beta-blocker). Amiodarone and sotalol, that influence both the AV node and the AP conduction, may also be used, particularly if VTach cannot be excluded. It is advisable to avoid lidocaine since it does not prolong refractoriness in the AP and may increase ventricular response.
- In termination of stable narrow-complex PSVT (reentrant type), vagal maneuver may be carefully attempted initially, followed by cautious introduction of adenosine or diltiazem. An external cardioverter-defibrillator should be immediately available in case AFIB occurs as a result of attempted treatment. In some cases amiodarone or a beta-blocker may be considered since neither facilitates AP conduction.
- In long-term pharmacologic maintenance, agents that prolong refractoriness in APs and encourage AV conduction are recommended, such as disopyramide, procainamide, and quinidine, and newer generation antiarrhythmics such as flecainide, propafenone, and moricizine. Amiodarone may be the treatment of choice in some pts since it also prevents recurrences of AFIB/AFLT and deterioration to VTach.
- For most pts with recurrent symptomatic tachyarrhythmias, the definitive treatment is catheter ablation of APs by delivering electrical or RF energy subsequent to EP studies and localization/mapping of APs.

#### ASSESSMENT POINTS

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<tr>
<th>System</th>
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</tr>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Arrhythmia, Palpitations, dizziness, syncope or near-syncope, angina, chest pain, cardiac arrest; sometimes asymptomatic LV function, Weakness, lassitude, exercise intolerance, CHF</td>
<td>Monitor BP; variable S2 pulse amplitude; fast regular, irregular, and/or weak pulse; S2; rales</td>
<td>12-lead ECG, Holter ECG, cardiac EP study, ECHO and possible further study</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Preparation**
- Cardiology consultation involving an electrophysiologist is necessary to determine pt’s status, specific type of arrhythmia, risk of symptomatic arrhythmia and management
- Regional anesthesia (provided hemodynamic stability) may have an advantage over GA owing to avoidance of multiple triggering drugs and noxious stimuli of airway instrumentation.
- A variety of antiarrhythmic drugs should be readily available, as well as cardioverter-defibrillator with synchronization capability.

**Monitoring**
- Standard ASA monitoring (incl ECG strip chart recorder), an arterial line if pt is at high risk for unstable tachyarrhythmias

**Induction**
- Smooth non-triggering induction is warranted to avoid unnecessary tachycardic response. Adequate depth of anesthesia, pain, and stress control with opioids and β-blockers prior to airway instrumentation may reduce the risk of tachyarrhythmias.
- In rare cases of ventricular dysfunction due to tachycardiomyopathy, the dose of induction agent should be appropriately titrated; etomidate and balanced anesthetic techniques may be considered.
- If neuraxial anesthesia is chosen, efforts should be made to avoid BP swings on induction since they may cause compensatory tachycardia. Vasopressors for BP correction should be used with caution to avoid tachycardic response.

**Maintenance**
- Some volatile agents (e.g., Halothane) may precipitate conduction via AP and should be avoided, whereas isoflurane and sevoflurane have no such effect or may even suppress AP conduction.
- Agents that can precipitate tachycardia should be used with caution or avoided. Among these are ketamine, pancuronium, atropine, and oxytocin. Fentanyl and droperidol, on the other hand, have been shown to be beneficial since they increase AP refractoriness.

**Extubation**
- Efforts should be made to avoid adrenergic hyperactivity on emergence and/or extubation. Adequate pain control with opioids or a regional block and β-blockers may be useful.

**Adjuncts**
- Agents that may suppress or enhance AV conduction should be avoided.
- Drugs that directly or indirectly increase adrenergic response should be used with caution.

**Postoperative Period**
- Adequate pain control is essential to reduce the likelihood of paroxysmal tachycardia.

**Anticipated Problems/Concerns**
- Possibility of unmasking WPW syndrome-related tachyarrhythmias under GA in previously asymptomatic pts.
- Risk of dangerous ventricular rates with AFIB/AFLT, with early deterioration into VFIB.
- Wide QRS complex of WPW patterns during antidromic PSVT or AFIB/AFLT mistaken for V-Tach/VFIB.
Ventricular Septal Defect (Congenital)

**Risk**
- Incidence is ~2–6/1000 live births

**Perioperative Risks**
- Mortality higher in older children, elevated PVR, and (>7 Wood’s units), and surgery complicated by complete heart block.

**Worry About**
- Worsening of L→R shunt with hyperventilation and increased FIO₂
- Paradoxical embolization
- Hypothermia
- Post-CPB pulm Htn and RV failure

<table>
<thead>
<tr>
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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Low forward cardiac output due to L→R shunt Pulmonary Htn due to excessive flow</td>
<td>CHF symptoms, FTT</td>
<td>Cyanosis, auscultation, ECHO, cardiac cath</td>
</tr>
<tr>
<td>RESP</td>
<td>Congestion/edema due to L→R shunt</td>
<td>Frequent URI</td>
<td>Rhonchi, CXR</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia in massive L→R shunt; polycythemia in R→L shunt</td>
<td>Pallor or cyanosis</td>
<td>Paleness or plethora, Hct</td>
</tr>
<tr>
<td>MS</td>
<td>Chronic hypoxemia due to late reversal of shunt flow (Eisenmenger’s syndrome)</td>
<td>Cyanosis</td>
<td>Clubbing of digits, Pulse oximetry</td>
</tr>
</tbody>
</table>

**Indications/Usual Treatment**
- 75% of small defects close spontaneously and require only antibiotic prophylaxis.
- Medical therapy for symptoms of CHF incl digoxin, ACE inhibitors, and furosemide.
- Surgery is indicated when CHF not amenable to medical treatment, or if failure to thrive
- Surgical repair contraindicated if PVR >10 Wood’s units, unless reactive to selective pulm vasodilators.

**Perioperative Implications**

**Preoperative Preparation**
- Digoxin and furosemide until day of surgery, ACE inhibitors controversial, but vasoplegic syndrome following CPB less common in pediatric pts.
- May not be possible to delay operation until free of upper resp symptoms

**Anesthesia**
- Limit FIO₂ to minimum necessary prior to CPB to restrict excessive pulm blood flow
- Maintain normal to slightly high Paco₂ to restrict excessive pulm blood flow
- Pts typically receive inhalational anesthetic for induction, if peripheral IV in place IV drugs can be administered alternatively.
- Avoid N₂O to prevent sequelae of paradoxical air embolization

**Monitoring**
- Indwelling arterial catheter for invasive monitoring in all pts.
- Central venous access and pressure monitoring in most pts undergoing surgery with CPB.
- Standard ASA monitoring, incl pulse oximetry, ECG, capnometry, multiple-site temp monitoring.
- Transesophageal echocardiography

**Induction/Maintenance**
- Mask induction with sevoflurane in most cases, IV drugs if peripheral IV in situ, IM induction possible for uncooperative pts.
- High-dose opioid anesthetic technique rarely used

**Surgical Stages**
- Pre-CPB
  - Low FIO₂, normal to high Paco₂
  - Avoid hemodilution with large amounts of crystalloid and/or colloid prior to CPB.
- CPB
  - After pt’s Hct has been obtained in the OR, dilutional Hct incl CPB prime is calculated. If calculated Hct is less than 25%, consider priming of CPB with whole blood or reconstituted whole blood (PRBC and FFP).
  - Inhalational anesthetic administration via CPB or continuous IV drug administration to allow for fast-tracking in most pts presenting for VSD repair.
- Post-CPB
  - Rule out residual shunting by transesophageal echocardiography.
  - Maintain Hct >25–30%

**Postoperative Considerations**
- Most pts presenting for VSD repair can be extubated at end of surgery
- Consider mechanical ventilation and sedation in the immediate postop period in pts prone to pulm hypertensive crises (e.g., Down’s syndrome)
- Infective endocarditis prophylaxis for 6 mo

**Anticipated Problems/Concerns**
- Imbalance in pulm to systemic blood flow ratio:
  - Excessive pulm blood flow results in high arterial saturation but with diminished tissue perfusion and metabolic acidosis
  - Diminished pulm blood flow results in good tissue perfusion but with cyanosis and potential injury due to hypoxia
- Postop ventricular dysfunction more likely with ventriculotomy
- Pulm Htn and/or right heart failure
- Coagulopathy, particularly in very small children.

Ventricular Septal Rupture (Defect), Post Myocardial Infarction

**Risk**
- Historically seen in 1–3% of MIs prior to era of acute revascularization
- Incidence 0.2% after lysis therapy or percutaneous intervention
- Majority occur within 1 wk; 20–30% in first 24 hr post MI
- Rarely occurs >2 wk post MI
- Medical management alone shows a mortality >90%

**Perioperative Risks**
- Accounts for 5% of MI-related deaths
- Without surgical therapy, survival is less than 10% at 1 month
- Surgical short-term survival 42–75%

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDO</td>
<td>Low forward cardiac output</td>
<td>Sudden onset of hypotension and shock</td>
<td>Loud holosystolic murmur and thrill</td>
<td>ECHO, cardiac catheterization</td>
</tr>
<tr>
<td>RESP</td>
<td>Congestion/edema</td>
<td>Resp distress</td>
<td>Rales</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL/HEPATIC</td>
<td>Dysfunction due to cardiogenic shock</td>
<td>Anuria</td>
<td>ABGs Foley catheter</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Consider elective tracheal intubation and PEEP
- Support cardiac output using inotropic agents
- Lower resistance to forward cardiac output using afterload reduction, incl intra-aortic balloon counterpulsation
- Obtain coronary angiogram. Concurrent revascularization can potentially improve outcome.

**Anesthetic Technique**
- High-dose opioid/muscle relaxant technique common
- Prior to CPB, use minimal FIO\textsubscript{2} and PEEP (maximizes PVR) to decrease L→R shunt across VSD

**Monitoring**
- Intra-arterial line
- Most use PA catheter due to pulm hypertension and for shunt quantitation. Step-up in saturation between right atrium and PA to measure degree of shunting.
- Thermodilution cardiac output falsely elevated.
- TEE to define anatomy, diagnose assoc papillary muscle rupture, monitor ventricular function (incl stroke volume), assess adequacy of surgical repair

**Airway**
- High airway pressures and frequent suctioning in the setting of pulm edema

**Induction**
- High-dose opioid technique to maintain hemodynamic stability. Avoid vasodilation assoc with volatile anesthetics

**Maintenance**
- If hypertensive, titrate low doses of volatile agent or benzodiazepines

**Surgical Stages**
- Pre-CBP
  - Median sternotomy with aortic and biatrial cannulation
  - May require vein or internal mammary artery harvest for concomitant myocardial revascularization
  - Lowest FIO\textsubscript{2} consistent with adequate oxygenation
- CPB
  - Maintain Hct using hemofiltration and transfusion
- Post-CBP
  - Inotropic support almost universally required for LV failure
  - RV failure common
  - Assess ventricular repair using TEE or right atrial-to-pulm O\textsubscript{2} sat ratio

**Airway**
- High airway pressures and frequent suctioning in the setting of urgent repair and posterior VSD
- Percutaneous device closure with GA and TEE have similar mortality

**Worry About**
- Assoc papillary muscle rupture
- Poor systemic perfusion and end-organ dysfunction
- Pulm congestion with massive L→R shunt

**Overview**
- Sudden onset of holosystolic murmur with thrill and hemodynamic deterioration (hypotension and pulm congestion)
- Despite advances in periop management, expect increased morbidity and mortality

**Expect complicated postop course with prolonged ICU stay**

**ICD-9-CM Codes:** 429.71 (Acquired cardiac septal defect); 410.00–410.92 (Associated MI)

**Usual Treatment**
- Repair of new VSD with hemodynamic deterioration using pericardial or prosthetic patch material
- Support preop with inotropic agents/intra-aortic balloon counterpulsation
- Percutaneous device closure as an alternative to surgery

**Postoperative Considerations**
- Postop renal/hepatic/neurologic dysfunction
- Postop LV, RV, or biventricular failure

**Anticipated Problems/Concerns**
- Cardiogenic shock with multi-organ dysfunction syndrome (MODS)
- Prolonged ventilatory dependency and ICU stay
- Course not dramatically improved with percutaneous device closure
Ventricular Tachyarrhythmias

**Overview**
- Ventricular tachyarrhythmias are characteristic by QRS $>$120 msec, rates $>$120 bpm; can be monomorphic (MVT) or polymorphic (PVT).
- Monomorphic ventricular tachycardias (MVT) have a single QRS morphology; can evolve into polymorphic ventricular tachycardia.
- Polymorphic ventricular tachycardias (PVT) are rapid rhythms with varying QRS morphology.

**Etiology**
- Structural heart disease, most commonly CAD and/or IHD and acute MI. Also, CHF, valvular disease, cardiomyopathy, myocarditis, RV dysplasia.
- Long QT ($>$450 msec) familial, idopathic or acquired $^2$ to hypokalemia, hypocalcemia, hypomagnesemia.

**Risk**
- Ventricular tachycardias (VT/VF) are uncommon but potentially fatal dysrhythmias.
- Risk increases with age due to higher incidence of structural heart disease.
- Primary cause of sudden death; incidence in USA: ~300,000/y, similar in other developed nations.
- Males have greater risk (46% vs. 34%) correlating with higher incidence of CAD, right ventricular dysplasia (2-fold male predominance) and Brugada syndrome (8-fold male predominance).
- Pts under 30 y with hypertrophic cardiomyopathy, RV dysplasia, myocarditis, long QT syndrome at higher risk of VT/VF.

**Perioperative Risks**
- Uncorrected electrolyte and/or acid-base imbalances.
- Uncontrolled sympathetic discharge.
- Cardiac function.
- Drugs: Sympathomimetants, antiarrhythmics (class 1 and 3), tricycles, antipsychotics, drugs that $>$QT; organophosphates
- Modulation of neuro-endocrine stress responses.
- More than three consecutive PVCs (considered VTach); duration $>$30 sec considered sustained VTach and should be treated.
- Six or more PVCs/min or multifocal PVCs

**Worry About**
- Electrolyte, acid-base, and metabolic disturbances esp. with ongoing myocardial ischemia.
- Cardiac function.
- Drugs: Sympathomimetants, antiarrhythmics (class 1 and 3), tricycles, antipsychotics, drugs that $>$QT; organophosphates
- Modulation of neuro-endocrine stress responses.
- More than three consecutive PVCs (considered VTach); duration $>$30 sec considered sustained VTach and should be treated.
- Six or more PVCs/min or multifocal PVCs

**ASSESSMENT POINTS**

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<th>Effect</th>
<th>Assessment by Hx</th>
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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Ischemia, MI, decreased cerebral perfusion, decreased cardiac output</td>
<td>Angina, palpitations, anxiety, lightheadedness, syncope</td>
<td>Pallor, diaphoresis, heart murmur, tachycardia, JVD, cannon a waves, displaced PML, S, gallop</td>
<td>12-lead EKG, TTE, TEE, Holter monitor, cardiac CT and cardiac MRI, cardiac catheterization (right or left heart), cardiac enzymes</td>
</tr>
<tr>
<td>PULM</td>
<td>Increased pulm venous pressure, pulm edema $^2$ to HF</td>
<td>Dyspnea, tachypnea, sleep apnea</td>
<td>Wheezing, course breath sounds, crackles</td>
<td>CXR, CT chest, PFTs, ABG</td>
</tr>
</tbody>
</table>

**Monitoring**
- Routine monitors incl ST segment trending and recorder.
- Arterial line for pts with cardiac disease, Hx of VT, or undergoing high-risk procedures.
- Consider TEE in pts with intraop hemodynamic turbulence, EKG changes, and/or MI.

**General Anesthesia**
- Avoid and treat electrolyte imbalances, esp. K and Mg.
- Check CVC if new PVCs occur after placement.
- Control/prevent sympathetic surges.

**Regional Anesthesia**
- Metliculous avoidance of intravascular injection
- Maintenance of adequate perfusion

**Postoperative Period**
- Adequate analgesia.
- Further workup and/or cardiology consult for episodes of periop VT

**Anticipated Problems/Concerns**
- SVT with aberrant conduction can be confused for MVT as it can be rapid and wide complex. Distinguishing the two rhythms may not be possible. Judicious use of adenosine helps with diagnosis. Pts with significant hemodynamic instability and a pulse with wide complex QRS tachycardia should be cardioverted.


**Preinduction/Induction/Maintenance**
- Pt Hx of VT/VF or CAD; functional status optimized, treatment of heart disease.
- Congenital and/or acquired long QT: Avoid and/or stop causative drug(s);
- TdP: Correct K$^+$ & Mg$^{2+}$. May require MgSO$_4$ or pacing to suppress TDP.
- Congenital TdP is best managed with beta blockade, RF ablation, and/or AICD.
- Minimize sympathetic response to stress; judicious choices of drugs to maintain adequate perfusion; avoid drugs that are sympathomimetic sensitizing.
- Nonsustained VT intraop without apparent cause needs postop evaluation. Sustained VT may need cardioversion (electrical/chemical), evaluation of acid-base status/electrolytes, and postop evaluation.

**Postoperative**
- Adequate analgesia.
- Further workup and/or cardiology consult for episodes of periop VT

**DISEASES**
Ventricular Tachycardia

**Risk**
- Structural heart disease (most commonly chronic phase of MI). Predictor of sudden cardiac death after MI.
- Most common cause of mortality with CHF
- Cardiomyopathies, both hypertrophic and dilated are assoc with VTach
- Seen in genetic syndromes such as long QT syndrome, Brugada syndrome and arrhythmogenic right ventricular dysplasia

**Perioperative Risks**
- Endogenous or exogenous catecholamines trigger VTach in susceptible pts
- Central venous, pulm artery catheters and intubation can trigger VTach
- Hyperventilation may decrease serum K+
- Precipitation of polymorphic VTach with agents that alter QT interval

**Worry About**
- Decreased vital organ perfusion related to low cardiac output
- Possible effect of antiarrhythmics on cardiac and pulm function
- Perioper ventricular dysfunction and/or ischemia
- Progression of VTach to VFIB
- Reduction of LV function due to IV antiarrhythmics

**Overview**
- Defined as 3 or more consecutive ventricular beats (usually at a rate >100 bpm)
- Sustained VTach persists for >30 sec or requires an intervention for termination

- Nonsustained VTach is ≤6 consecutive beats terminating spontaneously within 30 sec
- Possible signs of VTach incl a wide QRS (≥140 msec), presence of fusion beat, AV dissociation, LBBB morphology
- Must rule out SVT with aberrant conduction or pre-existing bundle branch block
- Torresade de pointes refers to VTach characterized by polymorphic QRS complexes that undulate in a regular fashion about baseline. Often assoc with prolonged QT interval

**ICD-9-CM Code**: 427.42

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Myocardial ischemia</td>
<td>Angina/anginal equivalent (syncpe, SOB, palpitations, and exercise intolerance)</td>
<td>Cardiomegaly, JVD</td>
<td>ECG, CXR</td>
</tr>
<tr>
<td></td>
<td>Hypotension</td>
<td>CHF</td>
<td>Cannon A waves; S1, S4</td>
<td>Electrophysiologic studies</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cardiac arrest</td>
<td></td>
<td></td>
<td>Ambulatory ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>SOB</td>
<td>Rales (wet or dry)</td>
<td>CXR, PFTs (A-a)O2 gradient</td>
</tr>
<tr>
<td>CNS</td>
<td>Syncope</td>
<td>Dizziness or loss of consciousness</td>
<td></td>
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</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Ascertain etiology of VTach and assoc problems
- Evaluate for Hx of palpitations, SOB, VTach, dizziness, syncope, chest pain
- Evaluate ECG for morphology of PVCs, QT interval, underlying BBB (important for Dx and therapy of wide complex tachycardia)
- Review electrophysiologic studies to determine optimal treatment of VTach
- Assess K+ and Mg2+ levels, digoxin level if indicated
- Pulm and thyroid function tests may be indicated for chronic amiodarone therapy
- Continue PO antiarrhythmic therapy
- Have defibrillator immediately available (near by) whenever inserting central venous catheters
- May need to have AICD deactivated for surgery to prevent firing with electocautery use

**Monitor**
- ECG for ischemia or QT prolongation
- Consider invasive hemodynamic monitor if suspect serious concomitant cardiac disease and major anesthetic/surgical intervention

**INDUCTION/MaintenACE**
- Avoid myocardial ischemia (maintain O2 supply and minimize O2 demand)
- Minimize surgical stimulus response and subsequent catecholamine release
- Avoid sympathomimetics, which may aggravate ventricular dysrhythmias
- Avoid hypokalemia, excessive hyperventilation

**Postoperative Period**
- Consider continuous arrhythmia monitoring.
- Continue parenteral antiarrhythmics until able to resume PO.
- ‘Treat Mg2+ and K+ deficits (common postop, esp. after major surgical procedures)
**Vitamin B_{12}/Folate Deficiency**

**Overview**
- Folate metabolism requires vitamin B_{12} dependent enzyme methionine synthase, which converts methylytetrahydrofolate and homocysteine to free tetrahydrofolate and methionine and is rapidly inactivated (T_{1/2} - 1 h) by N_{2}O
- Vitamin B_{12} required for two enzymes in humans: Methionine synthase and methymalonyl-CoA mutase (which converts L-methylmalonyl-CoA to succinyl-CoA)
- Tetrahydrofolate (in its free form and derivatives) needed for many metabolic processes, incl pyrimidine, purine, DNA synthesis; amino acid metabolism; formate elimination
- Vitamin B_{12}/folate required for synthesis and maturation of blood cells, integrity of CNS, GI function, growth of fetus and child
- Assoc with ↑ serum homocysteine levels and atherosclerosis

**ICD-9-CM Codes:** 281.0–281.2

**Etiology**
- Pernicious anemia (antibodies to gastric cells and lack of intrinsic factor) is most common cause
- Impaired nutritional intake, malabsorption (e.g., ileal resection), increased folate demand (e.g., pregnancy), treatment with antifolate drugs (e.g., methotrexate, prolonged N_{2}O exposure)

**Usual Treatment**
- Daily oral supplements of folate and/or weekly IM injections of vitamin B_{12}
- Folate treatment alone may produce partial hematologic remission due to vitamin B_{12} deficiency but mask vitamin B_{12} deficiency and result in irreversible neurologic abn
- Deficiencies assoc with N_{2}O exposure have been successfully treated with IM injections of vitamin B_{12}, IV administration of folic acid, oral methionine

**Risk**
- 5–20% of elderly
- Predisposed by prolonged exposure to N_{2}O, ICU pts, ileal resections, chemotherapy with anti-folates, ethanol abuse, AIDS, pregnancy

**Perioperative Risks**
- Worsening of pre-existing megaloblastic anemia and neuropathies after exposure to N_{2}O (infrequent)
- Anemia and limited O_{2}-carrying capacity
- Limb (positioning) injuries assoc with pre-existing neuropathy

**Worry About**
- Delayed onset of hematologic and neurologic abn after N_{2}O exposure—several weeks may pass before symptoms develop
- Untoward outcomes (e.g., death, infection) in critically ill pts with megaloblastic anemia undergoing anesthesia and surgery

**ASSESSMENT POINTS**

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<thead>
<tr>
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</tr>
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<tbody>
<tr>
<td>HEENT</td>
<td>Glossitis and painful tongue (infrequent)</td>
<td>Schilling test for malabsorption of vitamin B_{12}</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Anorexia, palpitations 2° to anemia</td>
<td>Serum levels of vitamin B_{12} and folate. RBC folate considered better indicator of tissue folate levels than serum folate.</td>
</tr>
<tr>
<td>HEME</td>
<td>Megaloblastic anemia</td>
<td>↑ Urinary levels of methylmalonic acid in vitamin B_{12} deficiency</td>
</tr>
<tr>
<td>GU</td>
<td>Impotence</td>
<td>Hematologic variables may be normal or abnormal; anemia, ↑ mean corpuscular volume</td>
</tr>
<tr>
<td>CNS</td>
<td>Subacute combined degeneration of spinal cord</td>
<td>Hypersegmented neutrophils may be present</td>
</tr>
<tr>
<td>PNS</td>
<td>Diminished vibratory sense, proprioception, and sensation; paresthesias, loss of deep tendon reflexes</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- If elective procedure, postpone to correct vitamin deficiencies and hematologic and/or neurologic abn

**Monitoring**
- Myocardial ischemia may occur with anemia and is assoc with increased homocysteine levels

**Airway**
- Large and painful tongue may be present

**Induction/Maintenance**
- Avoid NO if pt known to be vitamin B_{12}/folate-deficient and has hematologic and/or neurologic abn

**Adjuvants**
- Regional: Documentation of pre-existing neurologic deficits is required before proceeding with regional anesthesia

**Postoperative Period**
- Worsening of hematologic and neurologic abn may not occur until several weeks after N_{2}O exposure

**Anticipated Problems/Concerns**
- Anemia may result in impaired oxygenation of tissues and be assoc with myocardial ischemia
- CNS and PNS abn may exist.
- NO may exacerbate pre-existing hematologic/neurologic abn assoc with vitamin B_{12} and/or folate deficiency.
**Vitamin D Deficiency**

**Risk**
- High prevalence of deficiency, much more than previously recognized.
- At risk: Dietary insufficiency, elderly, nursing home residents, inadequate sun exposure, latitudes higher than 38 degrees, institutionalized, premature infants of VLBW; blacks, Hispanics, obese
- Genetically predisposed: Rickets, osteomalacia

**Worry About**
- Vitamin D–deficient pts are probably hypocalcemic. Without vitamin only 10–15% of dietary calcium and approx 60% of phosphorous is absorbed. Low total body magnesium is also likely.
- Calcitrol influences muscle function, CV homeostasis and immune response.
- Deficiency asso with Htn, myocardial infarction, CHF, and calcific aortic stenosis.
- Ample evidence to connect adequacy to risk and/or severity of certain cancers (colorectal, prostate, breast, leukemia) and autoimmune diseases (RA, MS, type 1 DM)
- Chronic vitamin D deficiency may lead to impaired mineralization of cervical spine (increased incidence of abn neck mobility; pedi atric pts with deformed chest wall may experience lowered FRC; increased incidence of resp infections)

**Overview/Pharmacology**
- There are two main forms of vitamin D. Vitamin D₃ (cholecalciferol) is synthesized in the skin by exposure to ultraviolet (UVB) radiation. Vitamin D₂ (ergocalciferol) is obtained through irradiation of ergosterol in plants and subsequent dietary intake.
- Amount of vitamin D obtained through food sources is minimal compared to that from the sun.
- Vitamins D₁ and D₂ are hydroxylated in the liver to 25 vitamin D (calcidiol), the major circulating form. Further hydroxylation in the kidney produces the active metabolite 1, 25 vitamin D (calcitriol). Calcitrol and not 25 vitamin D is the active form.
- Involved in functioning of hemopoietic cells, skin cells, cancer cells of various origins, islet cells of the pancreas, immune response, as well as CV function (via serum Ca⁺²)

**ICD-9-CM Code:** 268.9

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>Impaired mineralization</td>
<td>Bone pain, fracture</td>
<td>Dry, scaly skin</td>
<td>Bone density</td>
</tr>
<tr>
<td></td>
<td>Increased arthritis due to bone spur formation</td>
<td>Joint pain</td>
<td>Brittle nails</td>
<td>X-ray</td>
</tr>
<tr>
<td></td>
<td>Osteomalacia</td>
<td>Weak antigravity muscles</td>
<td>Coarse hair</td>
<td>Childhood</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td></td>
<td>Neck immobility</td>
<td>Osteoarthritis</td>
<td>Cardiac echo</td>
</tr>
<tr>
<td>CARDCO</td>
<td>CHD</td>
<td>Angina</td>
<td>Auscultation</td>
<td>ECG</td>
</tr>
<tr>
<td>CHF</td>
<td>Dyspnea</td>
<td>BP</td>
<td>Stress test</td>
<td></td>
</tr>
<tr>
<td>Irregular heart beat</td>
<td>Palpitations</td>
<td>Heart rate</td>
<td>Cardiac echo</td>
<td></td>
</tr>
<tr>
<td>Orthostatic hypotension</td>
<td>Fatigue</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Htn</td>
<td>Cardiac hypertrophy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vascular calcification</td>
<td>Stroke</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS/PNS</td>
<td>NM irritability</td>
<td>Muscle stiffness, rigidity</td>
<td>Seizure</td>
<td>Calcium levels</td>
</tr>
<tr>
<td></td>
<td>Numbness, paresthesias</td>
<td></td>
<td></td>
<td>PTH (if severe)</td>
</tr>
<tr>
<td></td>
<td>Muscle cramps</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Persistent, nonspecific</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>musculoskeletal pain</td>
<td></td>
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</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Both PTH and vit D (calcitriol) work to keep the level of ionized Ca²⁺ within tight range (4.01 mg/dl).
- Cimetidine: This hepatic P450-mediated metabolism inhibitor leads to decreased vitamin D plasma concentration
- Periop considerations are related to
  - Level of ionized Ca²⁺ (regulation of muscle contraction)
  - Neurotransmitter release
  - Blood coagulation

**Monitoring**
- ECG changes: Compare to previous tracing. Prolonged QT interval (adjusted to R-R interval; 2:1 intraventricular heart block)
- Easy availability of blood sample for immediate serum calcium assessment (art catheter vs. vein stick).

**Maintenance**
- ETCO₂: Avoid hyperventilation (alkalosis shifts ionized Ca²⁺ into the cells). Acute hypocalcemia increased chance of tetany.
- Monitor/replete Mg²⁺ as well

**Exubation**
- Laryngeal spasm on extubation in full awake pt is also likely. Predictor may be distal extremity paresthesia.
- Antibiotics: Rifampin, isoniazid

**Anticipated Problems/Concerns**
- Chronic anticonvulsant Rx (phenobarbital/phenytoin) may lead to hypocalcemia (↓ Ca²⁺ absorption from the intestine) and diminished vitamin D biosynthesis in the liver
- Vitamin D serum concentration is decreased when PTH is decreased (may occur with thiazide medications)
- Linked to Htn, CHD, vascular calcification, metabolic syndrome, autoimmune certain cancers
- Deficiency can be a result of deficient production of vitamin D in the skin, lack of dietary intake, impaired vitamin D activation or resistance to the biological effects of calcitriol
- Disorders of small bowel, hepatobiliary system, pancreas (bile salt deficiency, pancreas insufficiency, poor intestinal absorption of fat-soluble vitamins [A, D, K, E]) may cause malabsorption and/or malabsorption states
- Liver disease can impact CRF/ESRD (GFR < 25% of nml); moderate to severe impairment of renal phase synthesis of vitamin D with reduction of serum albumin

**Usual Treatment**
- Now recognized as essential supplement for most adults, esp. ages >50.
- Dose: Recommended doses higher than previously thought. Most adults require intakes of 800–1000 IU/day. Adults over 50 benefit from 1000 IU/day. Up to 2000 IU/day is safe in adults with renal insufficiency. Food sources incl fortified milk and/or dairy products, salmon, mackerel, cod liver, tuna, fortified cereals, and egg yolks.
- Toxicity: Margin of safety is large. Prolonged intake of doses >40,000 IU/d promotes bone demineralization, leads to hypercalcemia and enhances CV calcification.
- Prescribed for rickets, osteomalacia
- Vitamin D insufficiency: Vitamin D 800–2000 IU/d + elemental calcium 1200 mg/d
- Vitamin D deficiency: Elemental calcium 1200 mg/d plus ergocalciferol 50,000 IU/wk for 8–12 wk, then 2000 IU/d vitamin D.
- Uremia, CRF, and nephritic syndrome suppresses vitamin D action on gut
- Chronic renal failure acidosis leads to negative calcium balance; typical pts have increased phosphorous and/or decreased calcium serum levels
- Nephrotic syndrome causes vitamin D deficiency related to chronic proteinuria (loss of circulating 25 vitamin D$_3$–binding globulin). Symptoms present are 2$^\text{nd}$ hyperparathyroidism, low serum Ca$^{2+}$, osteomalacia.
- Vitamin D$_3$ (1,25 dihydroxycalciferol) directly facilitates Ca$^{2+}$, Mg$^{2+}$, and (PO$_4$)$^{3-}$ uptake by intestinal mucosa, their transport through intestinal cells and efflux.


### Vitamin K Deficiency

**Risks**
- Vitamin K deficient bleeding (VKDB) from abn factors II, VII, IX, and X
- Controversy exists regarding whether vitamin K deficiency leads to osteoporosis, abn cartilage calcification, and possible arterial calcification resulting in CV disease.

**Perioperative Risks**
- Minor or massive hemorrhage unrecognized as VKDB.
- Long-bone fractures during positioning the anesthetized pt (particularly in women).

**Worry About**
- Pts with underlying risk factors demonstrating unexplained coagulopathy
- Intracranial hemorrhage in infants (30-60% infants with VKDB) and other occult bleeding sites such as retroperitoneal hemorrhage (more commonly in infants)
- Avoid IM dosing of vitamin K if bleeding is present
- Anaphylaxis with IV vitamin K replacement (extremely rare)

**Overview**
- Vitamin K is cofactor for a carboxylase enzyme in liver, which is essential for normal function of factors II, VII, IV, X and proteins C, S and Z.
- Coagulopathy manifests as prolonged PT and INR (normal or prolonged aPTT) with normal fibrinogen and factor 5 (both lowered in liver disease and DIC).

**Fat-soluble vitamin K is absorbed in small bowel and colon and synthesized in gut by bacteria.**
- Poor oral intake alone not sufficient to cause vitamin K deficiency.
- Prevalence extremely rare in adults with adequate nutrition. Prevalence is as high as 30% in pts with chronic GI disorders. More frequent in infancy with classic VKDB occurring in 0–1.5% despite routine prophylaxis.

**ICD-9-CM Code: 269.0**

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEME</td>
<td>Insufficient Hemostasis/ Mucosal bleeding, acute or chronic anemia</td>
<td>Bleeding diathesis</td>
<td>Easy bruising, epistaxis, ICH (infants), retroperitoneal bleeding (infants)</td>
<td>Coagulation profile incl PT/INR, aPTT, fibrinogen, Hct, platelets</td>
</tr>
<tr>
<td>GI/RENAI</td>
<td>Mucosal bleeding/ inadequate production of clotting factors/ inadequate absorption of vitamins</td>
<td>Inadequate nutrition/parenchymal liver disease/coagulation disease/ malabsorption/bleeding diathesis</td>
<td>Hematuria/gastrointestinal bleeding/ wt loss/pundice/pale stools/dark urine/</td>
<td>Urinalysis, endoscopy</td>
</tr>
<tr>
<td>UTERUS</td>
<td>Mucosal bleeding</td>
<td>Bleeding diathesis</td>
<td>Vaginal bleeding</td>
<td></td>
</tr>
<tr>
<td>MET/OTHER</td>
<td></td>
<td></td>
<td>Antibiotic therapy/coumadin/other drugs</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preinduction/Induction/Maintenance**
- Suspect vitamin K deficiency in pts with underlying risk factors and unexplained anemia, bruising/bleeding, or prolonged PT/INR.
- Providers should have low threshold to correct unexplained prolonged PT/INR with vitamin K supplementation periop. With no signs of bleeding or easy bruising, a 1-mg IV dose of vitamin K is reasonable.
- If VKDB is present, PRBCs and FFP should be available and IV vitamin K should be given concomitantly to promote synthesis of clotting factors. Prothrombin concentrate, though less readily available, is more effective than FFP due to higher concentrations of factors II, VII, IX, and X.
- Large bore (16 gauge or larger in adults) IV access should be established prior to surgery to allow rapid volume resuscitation in the event of significant hemorrhage.

**Perioperative Implications**

**Monitoring**
- Anesthetic monitors recommended depend on etiology of coagulopathy and signs of bleeding. Consider Foley catheter, and CVP to monitor for volume status, and invasive arterial pressure monitoring to assess beat-to-beat BP during hemorrhage.

**General Anesthesia**
- Significant coagulopathy can result in easy bleeding with venipuncture, surgical incision, line placement, airway instrumentation.

**Regional Anesthesia**
- Prolonged PT and INR precludes neuraxial anesthetic 2” to risk of hematoma and subsequent neurologic injury.
- Prolonged PT and INR may result in hematoma formation during plexus anesthesia.

**Usual Treatment**
- Vitamin K can be administerted orally, intramuscularly, or intravenously with both route and dosage depending on urgency and degree of coagulopathy.
- Massive bleeding should promptly be treated with FFP, prothrombin complex concentrate, or factor VIIa along with IV administration of vitamin K (most sources recommend an adult dose of 10 mg IV and rarely more than 50 mg in first 6 hr)
- Labs for PT, INR, aPTT, fibrinogen, and plt count should be obtained in urgent situations to determine etiology of bleeding.
- When given IV, normalization of INR should be noted as soon as 30–120 min and no longer than 12 hr. If no correction noted or improvement in bleeding within 24 hr, an alternative etiology should be suspected, such as liver dysfunction or DIC.
- In non-urgent settings of prolonged INR w/o bleeding, other tests available incl serum vitamin K level or abn prothrombin level (most specific). If INR >9.0 recommend 5–10 mg oral vitamin K. If INR is between 5.0–9.0, recommended dosage is 2.5–5.0 oral vitamin K.
- Definitive diagnosis of vitamin K deficiency is made by correction of coagulopathy with vitamin K administration.
- For all routes of administration, sufficient serum vitamin K levels are present within 24 hr to reverse coagulopathy in most cases.
**Von Willebrand’s Disease**

**Risk**
- Incidence in USA: >1 million; 1% carry gene (severe disease 1/10,000–1 million)
- Race and gender with highest prevalence: None

**Perioperative Risks**
- Increased risk if hepatic dysfunction present
- Significant risk of bleeding if untreated

**Worry About**
- Excessive perioperative hemorrhage
- Concurrent antiplatelet agents or NSAIDs contributing to bleeding
- Adverse reactions to desmopressin therapy (seizures due to hyponatremia, hypotension, anaphylaxis)

**Overview**
- Coagulopathy characterized by quantitative/qualitative alterations in von Willebrand factor (vWF). vWF acts as a bridge between plt and vascular subendothelium, and stabilizes Factor VIII to prolong its circulating life.
- Presents as defect in primary hemostasis—mucocutaneous hemorrhage
- Highly variable severity; family Hx very helpful in predicting severity
- Diagnosed by prolonged aPTT, Factor VIII antigen and activity levels, vWF antigen, and ristocetin aggregation studies. Many disease subtypes further classified by band pattern of radiolabeled vWF after gel electrophoresis (multimeric analysis)
- Type I: Quantitative decrease in vWF of all sizes (most common, 70–80% of cases); Type II: Quantitative/qualitative alterations primarily in largest molecular weight vWF multimers, many subtypes exist (20–30% of cases), type IIIB may be accompanied by thrombocytopenia; Type III: Severe quantitative reductions or absence of vWF (rare, 2° to homozygous inheritance)

**ICD-9-CM Code: 286.4**

**ETIOLOGY**
- Autosomal dominant trait; variable penetrance and expression leads to unpredictable clinical severity; most severe disease in homozygotes
- Rarely, acquired disorder due to autoimmune disease or induced alterations in vWF function

**Usual Treatment**
- Must know disease subtype prior to therapy
- Desmopressin acetate (DDAVP), 0.3 μg/kg IV, stimulates release of endothelial vWF, variably effective in types I and II disease
- Desmopressin absolutely contraindicated in type IIIB
- Recombinant vWFs
- Pasteurized pooled factor VIII concentrates that preserve vWF (Humate-P) and solvent detergent heat-treated pooled concentrates (alphanate) are mainstays of therapy, if recombinant vWFs not available
- Cryoprecipitate best alternative if concentrates unavailable
- Antifibrinolytics often useful adjuncts

**ASSESSMENT POINTS**

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<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>GI bleeding</td>
<td>Melena, hematochezia</td>
<td>Stool guaiac</td>
</tr>
<tr>
<td>GI</td>
<td>Requirement for transfusion therapy</td>
<td>Random donor exposures</td>
<td>LFTs, hepatitis panel</td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulopathy, principal defect in primary hemostasis</td>
<td>Easy bruising, menorrhagia, epistaxis, pt or family experience during prior surgery or hemostatic challenge (e.g., dental extraction) vital to assessing periop risk, given variable severity of disease among individuals</td>
<td>PT, PTT, plt count often normal; platelet function assay; quantitative vWF antigen; ristocetin cofactor activity; multimeric analysis</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Collaboration with consultant hematologist and blood bank
- Desmopressin 1 hr preop in all but IIB subtype
- Antifibrinolytics for dental procedures

**Monitoring**
- Bleeding time/vWF activity periodically in prolonged procedures; $T_{1/2}$ of administered vWF about 8–12 hr

**Adjuvants**
- Consider regional anesthetics with caution
- Repeat desmopressin doses likely to be less effective than initial; reaccumulation of endothelial stores takes time

**Anticipated Problems/Concerns**
- Excessive intraop and postop blood loss
- Increased likelihood of infectious bloodborne disease

<table>
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<tr>
<th>System</th>
<th>Effect</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway</td>
<td>Laryngoscopy can lead to tissue trauma</td>
<td>Nasotracheal route best avoided</td>
<td></td>
</tr>
<tr>
<td>Induction</td>
<td>No specific recommendations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maintenance</td>
<td>Meticulous surgical hemostasis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extubation</td>
<td>Avoid coughing if possible; gentle orotracheal suction best performed under direct vision</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Waldenström’s Macroglobulinemia

**Risk**
- Rare hematologic neoplasm (accounts for 1–2% of hematologic malignancies)
- In USA, age-adjusted incidence of 3.4 per million among males and 1.7 per million among females. Median age 63–68 y
- Racial preponderance: Caucasians >> African-Americans
- Median survival ranges between 5 and 10 y; age >65 y, anemia, and organomegaly assoc with poor prognosis

**Perioperative Risks**
- Consequences of hyperviscosity
- Anemia and coagulopathy

**Worry About**
- Anemia
- Coagulopathy
- Hyperviscosity
- Hypervolemia
- Hepatosplenomegaly and lymphadenopathy in 15–25% of pts

**Overview**
- Uncommon B-cell lymphoproliferative disease characterized by bone marrow infiltration and production of monoclonal immunoglobulin M (IgM)
- Symptoms attributable to tumor infiltration and/or excessive IgM production
- Anemia most common finding, caused by combination of factors: decrease in red cell survival, impaired erythropoiesis, hemolysis, plasma volume expansion, blood loss from GI tract
- Potentially severe adverse neurologic, hematologic, CV problems peripop
- Anesthetic concerns similar to those in multiple myeloma except that hypercalcemia and bone lesions are rare; renal failure and proteinuria less common

**ICD-9-CM Code:** 273.3

**ASSESSMENT POINTS**

<table>
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<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hyperviscosity (high output cardiac failure, valvular dysfunction, MI)</td>
<td>Angina, Dyspnea, Fatigue</td>
<td>Venous thrombosis, Fluid overload</td>
<td>Serum viscosity &gt;4 centipoise (cP) (normal ≤1.8 cP)</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm involvement</td>
<td>Dyspnea</td>
<td>Hypoxia</td>
<td>CXR (pleural effusion, diffuse pulm infiltrates)</td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulopathy (multifactorial)</td>
<td>Episodic epistaxis, mucosal and gum bleeding, Fatigue</td>
<td>Pallor</td>
<td>Coagulation studies</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia (multifactorial)</td>
<td></td>
<td>CBC (normocytic, normochronic anemia)</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Cryoglobulinemia</td>
<td>Cold intolerance, Raynaud’s syndrome, Arthralgia</td>
<td>Purpura</td>
<td>Cryoglobulin assay</td>
</tr>
<tr>
<td>Lymph node involvement</td>
<td></td>
<td></td>
<td>Lymphadenopathy</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Glomerulonephritis</td>
<td>Dehydration, Uremic symptoms</td>
<td>BUN/Cr</td>
<td>Urinalysis (proteinuria)</td>
</tr>
<tr>
<td>CNS</td>
<td>Leukoencephalopathy</td>
<td>Headaches, Blurred vision</td>
<td>Mental status changes, Retinal hemorrhage, papilledema</td>
<td></td>
</tr>
<tr>
<td>PNS</td>
<td>Demyelinating peripheral neuropathy</td>
<td></td>
<td>Symmetric, distal sensorimotor neuropathy, ataxic gait</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Organomegaly secondary to IgM infiltration</td>
<td></td>
<td>Hepatomegaly, Splenomegaly</td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preinduction/Induction/Maintenance**
- Consider plasmapheresis and transfusion
- All drugs: Theoretical unpredictable pharmacokinetics due to alterations of relative proportions of globulins in blood and expanded plasma volume
- Judicious fluid management

**Monitoring**
- Normothermia to prevent cryoglobulin precipitation

**General Anesthesia**
- Macroglossia if amyloidosis (rare)

**Regional Anesthesia**
- Relative contraindication in presence of peripheral neuropathy

**Postoperative Period**
- Transient postop paresis due to disease rather than anesthetic management

**Anticipated Problems/Concerns**
- Hyperviscosity symptoms (20–30% incidence)
- Excessive amounts of circulating IgM impair transit of blood cells resulting in microvascular congestion and decreased tissue perfusion
- Capillary blood flow impaired, leading to decreased O2 delivery through microcirculation and tissue ischemia
- Classic triad of symptoms incl neurologic abn, visual changes, and bleeding
- CV manifestations 2° to expanded plasma volume: Angina, high output cardiac failure, valvular dysfunction, or MI
- Plasmapheresis is fastest, most effective method to reduce plasma viscosity
- Anemia
  - Hgb value may be artificially reduced by 2 g/dL.
  - Transfusion may precipitate CHF or hyperviscosity syndrome (by increasing serum viscosity) and potentially decrease O2 delivery
- Consider plasmapheresis before transfusion
- Coagulopathy
- Cryoglobulinemia (5% risk)
- Precipitation of cryoglobulins at cold blood temp triggers complement activation which results in immune complex vasculitis and ischemia
- Raynaud’s syndrome, arthralgia, purpura, peripheral neuropathy, hepatic dysfunction, and renal failure may develop

Wilms’ Tumor

Peter J. Davis

Diseases

Risk
- Most common malignant renal tumor in childhood
- 6% of all childhood malignancies
- 5–7.8 / million children <15 y
- M = F
- Peak age 1–3 y
- 5% bilateral
- Relapse-free survival rate at 2 y, 90%
- Pts with favorable staging 80–90% chance of cure. Pts with metastasis have 50% long-term survival.
- Overexpression of HER-2 oncoprotein good predictor of survival

Perioperative Risks
- Increased intra-abdominal pressure
- Immunocompromised
- Tumor extension into renal vein, IVC, and heart
- Some treated with chemo prior to surgery
- Assoc Htn

Worry About
- Anomalies
  - Aniridia 1%, hemihypertrophy 2%
  - Neurofibromatosis
  - Beckwith-Wiedemann syndrome
  - GU abn, horseshoe-shaped kidney, cryptorchidism, gonadal dysgenesis, hypospadias, duplication of collecting systems
  - Metastatic disease
  - Lymph nodes, lung, liver, brain

Overview
- Most common abd tumor of childhood; prognosis related to staging
- Because of location of tumor, blood loss can be significant
- Tumor is also assoc with other congenital abn, which may affect anesthetic and/or surgical management
- Tumor extension into IVC and heart carries increased morbidity and mortality
  - ICD-9-CM Code: 189.0

ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE | Test
---|---|---|---|---
HEENT | Beckwith-Wiedemann syndrome | Obstructive airway 2° to large tongue | Direct exam | Blood glucose levels
CARDIO | Htn | Asymptomatic | Htn | ECG
| Tumor extension into heart | | | CT abd
| | | US renal vein/IVC
RESP | Resp compromise | Abd distention | Hypoxemia | Pulse oximetry
| Metastatic disease | ECG
| Tumor embolization | | Cardiac ECHO
| | | Possible cardiac ECHO
GI | Gastric reflux | ↑ Intraop pressure | ↑ RR | Review CT scan
| Hx of reflux | | | 


Preoperative Indications
- Preoperative Preparation
  - Htn-controlled
  - R/O renal vein and/or IVC tumor involvement
  - Monitoring
    - Arterial catheter may be indicated
    - CVP catheter may be needed esp. if IVC and tumor extend mid-heart
    - Pre-existing hematuria. Foley catheter to aid in fluid balance.
    - IV catheters above diaphragm, large-bore catheters preferable
    - ETCO2 to rule out air and/or tumor embolus
  - Airway
    - May be a problem if Beckwith-Wiedemann syndrome present

Preinduction/Induction
- Age-appropriate use of sedation
- Rapid-sequence if increased intra-abdominal pressure
- Regional anesthesia; epidural for postop pain
- Pre-existing chem may have cardiac depressant effect
- IV access above diaphragm

Maintenance
- Prolonged procedure
- Avoid N2O
- Maintain temp
- Increased third space fluid requirements
- Procedure may be assoc with large blood loss
- Pulm function may be compromised, 2° to metastasis and/or tumor embolization, abd distention, and/or surgical traction

Extubation
- Expected if temp maintained and pt hemodynamically stable

Postoperative Period
- Pain control
- Third space fluid requirements
- Htn may still be present

Anticipated Problems/Concerns
- Risk of tumor and/or air embolus. If tumor extends into renal vein, IVC may have to be cross-clamped, the IVC opened, and the tumor removed.
- Intraop blood loss can be extensive
- Periop implications
Wolff-Parkinson-White (WPW) Syndrome

Risk

- WPW pattern (asymptomatic) prevalence: 0.15–0.25% in general population, increases to 0.55% in pts with a 1° relative with WPW.
- WPW syndrome (ECG pattern and arrhythmia): prevalence: 0.005–0.07% in general population, approx 2% out of pts with WPW

Perioperative Risks

- Atrioventricular reentry tachycardia (AVRT) (80% of pts WPW syndrome): rapid heart rate may cause hemodynamic instability and/or myocardial ischemia esp. if LV failure, LV hypertrophy, aortic stenosis, or mitral stenosis is present.
- Atrial fibrillation (AFib) (15–35%), increasing incidence with age. Major concern is rapid ventricular response due to antegrade conduction over accessory pathway (AP).
- Atrial flutter (5%) - Ventricle fibrillation (VFib)/sudden death (0–0.4%): Out of rapid ventricular response due to antegrade conduction over AP in Afib/AVRT.
- Myocardial ischemia in pts with CAD if SVT or AFib occurs

Overview

- Definition: WPW syndrome is a preexcitation syndrome. Ventricular depolarization occurs in part via an accessory pathway (AP) directly connecting the atrium and ventricle and thus capable of conducting electrical impulses into the ventricle bypassing the AV-His bundle conduction system. The AP in WPW syndrome is called the bundle of Kent and connects atrial and ventricular myocardium and is not connected to the conduction system.
- The accessory pathways electrical conduction may occur in an antegrade or retrograde fashion and is faster than the AV node and therefore the PR interval is shortened (<0.12 sec). Additionally, the mycardium around the AP insertion in the ventricle is activated earlier, i.e., pre-excitation. The impulse then spreads slowly from muscle fiber to muscle fiber until it joins the regular conduction system. This results in a slurred upstroke and widening of the QRS complex on the ECG (fusion complex from early but slow ventricular excitation via AP and rapid activation via the normal conduction system). These abn in the QRS complex may vary in magnitude and depend on relative contribution of normal AV nodal system and AP to ventricular depolarization.
- PSVT results from a re-entrant circuit involving the AV node and AP. The QRS complex during PSVT matches the usual QRS morphology when conduction is antegrade through the AV system and retrograde through the AP, i.e., orthodromic. 5–10% of the time, conduction through the AP is antegrade, i.e., antiodromic in the re-entrant circuit, producing a wide QRS complex. This rhythm may be confused with VTach.
- AFib and/or AFLT is more common in pts with WPW. Usually, but not always, AFib is precipitated by an episode of PSVT. Rapid (≥150 bpm) ventricular rates may occur in pts with APs with short refractory periods. These pts are at risk for developing VFib and hemodynamic collapse.

ICD-9-CM Code: 426.7 (Anomalous atrioventricular excitation)

Standard Therapy

- With severe hemodynamic compromise: Synchronized DC cardioversion (50–100 J)
- AVRT and/or narrow complex tachycardia: Apply vagal maneuvers or IV adenosine (6–12 mg IV). A small incidence of induction of AFib with adenosine therapy for PSVT in WPW has been described.
- AFib: Agents that reduce the accessory b bundle refractory period (digoxin, Ca2+-channel blockers, β-blockers, and adenosine) increase the risk of causing VFib and hemodynamic collapse in pts with WPW and AFib and should therefore be avoided.
- Broad complex tachycardia, i.e., antiodromic AVRT: It should be treated with IV procainamide or amiodarone.

Perioperative Implications

Preoperative Preparation

- If pre-excitation on ECG or Hx of WPW, consider cardiology evaluation
- If symptomatic, consider electrophysiologic study and catheter ablation.

Monitoring

- ECG for detection of periop PSVT or AFib
- Consider arterial line and CVP catheter if LV dysfunction or valve disease as these pts have a high dependence on preload and atrial kick.
- For emergency surgery, consider placement of defibrillator pads prior to induction.

Induction/Maintenance/Extubation

- Volatile anesthetics and IV induction agents such as propofol and benzodiazepines seem to have no influence on the conduction system and are safe to use. Enflurane and isoflurane as well as medications that enhance vagal tone (e.g., opioids, propofol infusion) actually decrease conduction via the AP and are safe to use as well.
- However increased sympathetic tone may decrease the refractory period and therefore accelerate the conduction in the AP and AV node. This may facilitate the precipitation of AVNRT, AFib, and/or VFib.
- Limit the use of vagolytic agents such as pancuronium and atropine, use β-1 agonists (phe
- nylephrine) instead of ephedrine to avoid positive chronotropy and arrhythmias.

Postoperative Period

- Pain management to avoid catecholamine excess
- If the delta wave appears in periop period, rule out myocardial infarction (decreased AV conduction second to ischemia facilitating increasing AP conduction).

Anticipated Problems/Concerns

- AV nodal blockers (digoxin, Ca2+-channel blockers, adenosine, and β-blockers) may shorten refractoriness in the AP and thereby provoke VFib in WPW pts with AFib.
- Hemodynamic collapse may occur when verapamil or β-blockers are used in the treatment of antiodromic (wide-complex) PSVT in pt with WPW that is mistaken for VTach.

ASSESSMENT POINTS

ECG Criteria:

<table>
<thead>
<tr>
<th>ECG</th>
<th>P wave &amp; PR interval</th>
<th>QRS</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classic (Type A)</td>
<td>Shortened PR interval, typically &lt;0.12 secs (left-sided bypass track)</td>
<td>Slurred upstroke (delta wave), widened QRS complex</td>
<td>The faster the AP conduction the more prominent the delta wave and the wider the QRS</td>
</tr>
<tr>
<td>Atypical (Type B)</td>
<td>Shortened PR interval (right-sided bypass track)</td>
<td>Q waves (inverted delta wave) in V1</td>
<td>May be confused with MI</td>
</tr>
<tr>
<td>Concertina effect</td>
<td>Periodically progressive shortening of the PR interval, with the P wave disappearing in QRS</td>
<td>The shorter the PR interval, the more pronounced is the delta wave (wider QRS)</td>
<td>This is the result from a periodically ↑ conduction via the AP</td>
</tr>
<tr>
<td>Intermittent WPW</td>
<td>May be mistaken for frequent ventricular premature beats, if it persists for several beats may be held for accelerated idioventricular rhythm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abdominal Aortic Aneurysm Repair

Risk
- AAA affects approx 5% of males and 1.7% of females over 65 y
- Risks: Smoking, males, age, white race, family Hx, Htn, atherosclerosis
- Risk of rupture: Female sex, diameter >5.5 cm, low FEV1, smoking, high MAP.
- Rupture: 80% mortality, approx 4500 deaths annually in the USA
- Incidence in USA: About 45,000 AAA repairs annually, resulting in 1400 deaths

Perioperative Risks
- Open repair (OR): Mortality is 4–8% for elective and 25–60% emergent/ruptured AAAs. Risks are higher in women, pts with more co-morbidities and emergencies.
- Morbidity: Renal 5% of infra- and 13% of suprarenal AAAs; MI 4–15%, resp 10%, ischemic bowel 3–4%, and paraplegia <1% for infrarenal and 1–5% for suprarectal AAAs.
- Endovascular aneurysm repair (EVAR): 1–2% 30-d mortality but significant periop complications: MI 7%, resp 10%, ARF 5.5% (dialysis < 0.4); conversion to open 1.6%, ischemic bowel 1.0%, bleeding requiring re-intervention 0.8%, thromboembolic 0.5 to 1.5%.

Worry About
- OR: Massive blood loss, hypovolemia, anemia, electrolyte and coagulation abn.
- OR: Ischemia-refrusion with acid-base derangement, hypothermia, and multi-organ injury.
- OR: Myocardial ischemia/infarction and renal insufficiency/failure.
- EVAR: Contrast nephropathy, myocardial ischemia/infarction.

Overview
- High incidence of co-morbidities: Htn, CAD, renal insufficiency, cerebrovascular disease.
- Emergent repair has much higher assoc mortality and morbidity.
- Need for invasive monitoring and significant IV access.

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CAD, MI, CHF, arrhythmias</td>
<td>Orthopnea, SOB, PND angina, palpitations</td>
<td>Pedal edema, JVD, S1</td>
<td>Noninvasive if &gt;3 of: CHF, CVA, DM, Cr&gt;2, MET&lt;8, Revasc.: STMI, unstable angina, 3-v dis</td>
</tr>
<tr>
<td>RESP</td>
<td>COPD</td>
<td>SOB, cough</td>
<td>Distal BS, wheeze</td>
<td>CXR, ABGs, PTTs, CT</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Reno-vascular Htn</td>
<td>Generalized edema</td>
<td>Serum Cr, creatinine clearance</td>
</tr>
<tr>
<td>CNS</td>
<td>CV disease</td>
<td>TIA, stroke, syncope</td>
<td>Focal deficits, bruits</td>
<td>Neuro assessment, carotid US</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
- ACE-inhibitors and ARBs held the day of surgery.
- β-blockers, ASA and statins continued.
- Thoracic epidural should be considered for OR postop analgesia.
- Large-bore IV access is essential.

Monitoring
- Invasive arterial pressure monitoring in both OR and EVAR.
- OR: CVP for all, PAC or TEE for poor LVEF and active CAD, and/or high clamp.

Anesthetic Technique/Induction
- OR: General anesthesia with epidural for postop pain.
- EVAR: Neuraxial or MAC anesthesia may be assoc with better outcomes than GA; anesthetic choice is based on team experience, complexity of repair and pts’ co-morbidities.

Surgical Stages: Open Repair
- Dissection
  - IV access and ability to transfuse blood products are essential.
  - Normotension is the goal of BP control to prevent premature rupture.
  - Mannitol (12.5–25 g) is a useful free radical scavenger but not a proven renoprotector.

  - Heparinization (75–100 U/kg) 5 min prior to aortic cross clamp; optimal ACT >250s.

  - Aortic Clamping
    - Hemodynamic goals: Avoid excessive Htn above the clamp but maintain adequate pressures below the clamp to ensure spinal, bowel perfusion. One strategy is MAP at 20% increase from baseline with good heart rate control.
    - For excessive Htn with infrarenal clamp the first choice is an afterload reducer (i.e., nicardipine), and for supraceliac a preload reducer (i.e., nitroglycerine).
    - Lactic acidosis below the clamp provides rationale for NaHCO administration: Higher base deficit and NaHCO requirements in supraceliac clamp.

  - Aortic Unclamping
    - Hemodynamic challenge: Reperfusion hypotension, arrhythmias, hyperkalemia, hypothermia.
    - Optimize preload, afterload; maintain NSR; CaCl, NaHCO3, and vasopressors ready to use.
    - Closing: Normalization of hemodynamics and coagulation, warming, dosing of epidural.

Postoperative Considerations
- ICU for continued hemodynamic and coagulation monitoring, possibly ventilatory support.
- Pain is significant; effective epidural promotes early extubation and pulm rehabilitation.

EVAR
- Bilateral groin dissection, insertion of an introducer sheath, and arteriogram.
- Stent is inserted over a guidewire, position confirmed radiologically, and deployed. Temporary balloon insufflation may be used to promote the seal.
- Conversion to OR is rare (<2%) but assoc with higher morbidity and mortality.
- Postop considerations: Lower extremity embolism, myocardial ischemia, renal insufficiency. Endoleaks are late complications. Routine EVAR requires an overnight stay.

Anticipated Problems/Concerns
- In the setting of potential blood loss early intraop epidural dosing is not advisable.
- Tenip management is difficult in OR as lower body warming cannot be used.
- Renal protection remains elusive. OR: Keys are short clamp time and maintenance of intravascular volume and CO. In EVAR, evidence additionally supports administration of NaHCO3 and acetylcysteine or use of CO2 contrast to attenuate contrast-induced nephropathy.
- High prevalence of CAD predisposes AAA pts to periop myocardial ischemia.
Abdominoperineal Resection

Risk
- About 40,000 cases of rectal cancer per year (abdominoperineal resection used in 18–27% of cases; remainder involve sphincter-preserving low-anterior resections)
- Average age at diagnosis is 67 (less than 4% of cases under 40 y)
- Slight male predominance (poorer prognosis)
- Slower progression in elderly
- African-Americans have highest incidence and mortality in USA

Perioperative Risks
- Periop mortality 2–3%
- Mortality related to pre-existing cardiac and pulmonary disease
- Significant impotence or urinary dysfunction 10–60%
- Urinary retention ~30%
- Perineal wound infection 16%; breakdown 7%

ASSESSMENT POINTS

System Effect Assessment by Hx PE Test
CARDIO Hypovolemia Bowel prep, N/V/D Hypotension, UO Orthostatics/ EKG
RESP Metastases Cough, SOB
GI Hepatic metastases Bowel obstruction Pain N/V, no flatus Tenderness Abd distension and tenderness CT scan Abd x-ray and/or CT scan
RENAL Metabolic alkalosis if NGT Obstruction High NG output Pain, oliguria Mental status BUN Cr, IVP or other imaging
ENDO Malnutrition Wt loss Muscle wasting Albumin, electrolytes
HEME Anemia GI bleeding, fatigue Pallor CBC


Preoperative Preparation
- Assess pre-existing conditions and volume status, electrolytes; aspiration premedication with H2 blockers, metoclopramide (contraindicated in obstruction), and sodium citrate
- Consider insertion of epidural catheter for periop pain control

Monitoring
- Routine monitors
- Arterial line in pts with significant pre-existing cardiopulmonary disease
- Insertion of CVP or PA cath for significant co-morbidities; not usually necessary
- Foley to monitor UO; assess hydration and urologic complications

Anesthetic Technique/Induction
- General endotracheal anesthesia; general/regional

Airway
- Pts at high risk for aspiration should undergo awake technique or rapid sequence induction; can still perform cricoid if pt has NG tube

Maintenance
- Prevention of hypothermia through the use of forced warm air, warm IV fluids, and warmed humidified gas to avoid postop wound infection and bleeding

Surgical Stages

Dissection
- Replacement of fluid deficit from bowel prep and NPO time; anticipate large 3rd-space losses; attempt to maintain euovolemia
- Lower midline incision on abd
- Abd portion of case involves mobilization of rectum, sigmoid colon, and creation of a descending colostomy with the pt in a modified lithotomy position
- During the abd phase steep Trendelenburg is usually needed for dissection. Watch for hemodynamic changes with pt position; ensure proper padding and securing of pt to OR table
- High peak airway pressures can occur in steep Trendelenburg

- Pelvic/abdominal abscess, ostomy problems, abd wound issues also occur
- Procedure involves removal of the distal colon, rectum, closure of anus, and creating a permanent colostomy

ICD-9-CM Code: 154.1 (Malignant neoplasm of rectum)

Indications and Usual Treatment
- Rectal adenocarcinoma involving levator ani muscle
- Inability to completely resect distal rectal cancer from abd approach
- Recurrent anal squamous cancer
- Tumors of anus too large to locally resect
- Decreasing in popularity in favor of low anterior resection and local excision of tumors
- Some pts with poor sphincter control benefit from APR instead of low anterior resection

Postoperative Considerations
- Extubation at end of case is typically routine
- Pain score: 7–8
- Postop analgesia: IV PCA or epidural
- Consider ICU admission

Anticipated Problems/Concerns
- Hypovolemic pts prone to hypotension when epidural used
- Male pts have narrower pelvis making dissection more difficult; watch for bleeding during pelvic dissection
- Watch for hypoventilation and hypoxemia postop
**Adrenalectomy for Pheochromocytoma**

**Risk**
- People within USA: 0.03%–0.04% (~100,000) by autopsy of unselected individuals; 0.1%–0.3% of individuals with sustained Htn
- Race with highest prevalence: Caucasian

**Perioperative Risks**
- Major goal to avoid pheo crisis; preop, intraop goals of management of extra-adrenal surgery no different from those of adrenal surgery; if α-blockade not present before surg, try to delay operation until degree of α-blockade judged appropriate by:
  - No BP > 165/90 mmHg for 48 hr
  - Presence of orthostatic hypotension, but BP on standing should not be < 80/45 mmHg
  - An ECG free of ST-T changes due to cardiomyopathy
- Absence of Sx of catecholine excess and signs of α-blockade (e.g., nasal stuffiness)
- If emergency use α-blockers, β-blockers, nitroprusside; keep in ICU till most painful time has passed or adrenergic control attained
- Increased risk of Htn crisis with bleeding into myocardium, brain, kidney or ischemia

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td></td>
<td>Nasal stuffiness (from α-adrenergic blockade)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Htn, dysrhythmias, AFIB, sinus tachycardia, mitral valve prolapse, CHF, myocardial fibrosis+/necrosis or myocarditis</td>
<td>SOB, exercise tolerance, palpitations, Htn (50% sustained, 40% paroxysmal)</td>
<td>Standard exam plus measurement of BP q1min in stressful environment plus orthostatic maneuvers with BP/HR measurement q1min</td>
<td>ECG, ECHO (if cardiomyopathy suspected)</td>
</tr>
<tr>
<td>GI</td>
<td>90% tumors adrenal or abd wt loss, diarrhea, dehydration</td>
<td>Be careful when palpating abdomen not to trigger pheo crisis</td>
<td>No different from normal</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td></td>
<td>Mild polycythemia, thrombocytopenia</td>
<td>Hgb (way to judge volume expansion)</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>↑ Catecholamine effects</td>
<td>Headache, tremor, anxiety, ↓ pain threshold, fatigue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Assoc with hyperparathyroidism</td>
<td>Glucose intolerance due to α-adrenergic glucagonemia, ↓ insulin secretion</td>
<td>Glucose often ↑ (insulin Rx prescribed before correct Dx of cancer made)</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Monitoring**
- Temp
- Art line placement before induction difficult
- Consider TEE if CV system severely affected; CVP rarely used

**Anesthetic Care/Technique**
- No technique assoc with better or worse outcome; use of droperidol controversial. Agents that block catecholamine reuptake (ketamine) or cause release might be avoided.

**Induction/Maintenance**
- Prehydrate liberally if tolerated
- Gentle induction with nitroprusside available
- Dopamine infusion in reserve
- Painful or stressful events often cause exaggerated stress response caused by release of catecholamines from nerve endings loaded by reuptake

**Adrenal Removal**
- Dissection to secure venous drainage, double ligature with transaction between; then arterial supply; then complete mobilization, liberation
- After predominant tumor removed, palpation of paraganglionic chain with observation to monitor for sudden increasing in BP or HR
- Goal in resection is securing venous supply from tumor(s). Pressure on tumor causing release can result in blood levels of 200,000–1 million pg/mL (ask for temporary stay of surgery while nitroprusside infusion increased)
- Good communication essential
- Relative hypotension often develops after venous drainage of tumor or its removal. If perfusion adequate can let BP stay at 80/40. Massive infusions of catecholamines occasionally required.

**Anticipated Problems/Concerns**
- Pheo crisis a life-threatening illness, manifest by increased temp, increased HR, alterations in consciousness

**Indications and Usual Treatment**
- 90% spont arise; 10% familial (autosomal dominant genetics involving chromosome 7 implicated)
- Assoc with MEA IIA (medullary thyroid cancer, 1° hyperparathyroidism), IIB (medullary thyroid cancer, mucosal neuromas; assoc with neurofibromatosis, von Hippel-Lindau syndrome, and retinal and cerebellar hemangioblastoma, ataxia-telangiectasia, Sturge-Weber syndrome)
- Prehydrate liberally over 6–60 d if CV status tolerates; expand with high salt and/or fluid diet while increasing α-blockade over 7–60 d period
Laparoscopic Adrenalectomy

Risk
- Pheochromocytoma when it is a well-defined tumor, maximum diameter of 7–8 cm, with good preop BP control.
- Aldosteronoma, nonfunctioning adenoma, cortisol producing adenoma. Cushing's disease, cysts, myelolipoma and metastatic adrenal tumor, when tumors are less than 10–12 cm in size.
- For adrenal cancer, open adrenalectomy is preferred esp. if the tumor is large.

Perioperative Risks
- Depend on tumor type and severity of symptoms.
- Main co-morbidities
  - Pheochromocytoma: Htn, cardiomyopathy, acute pulm edema, cardiac arrhythmias, myocardial ischemia or infarction
  - Adren adenoma and aldosteronoma: Htn, hypokalemia, hyperglycermia
  - Hemodynamic fluctuations during tumor manipulation may occur and when severe, can increase periop morbidity.
- During laparoscopic resection of a pheochromocytoma, pressure on the tumor caused by insufflation of gas can cause release of catecholamines from the tumor.

Perioperative Management
Preoperative Preparation
- Assess co-existing morbidities and optimize their medical management.
- Suplemental steroid therapy for those at risk for developing adrenal insufficiency.
- Continue alpha and beta blockade for pheochromocytoma

Monitoring
- Routine
- Arterial line and large-bore IV access.
- Central venous catheter to infuse fluids and vasopressors when needed. Position and pneumoperitoneum affect absolute valve of the CVP
- Pulm artery catheter in pts who have preexisting cardiac co-morbidities (cathoca-mine induced cardiomyopathy, LVH, myocardial ischemia, CAD) and severe pulm disease
- TEE for pts with significant left ventricular dysfunction
- Close monitoring of serum electrolyte and blood glucose concentration

Anesthetic Technique
- Adequate preop sedation and/or anxiolysis to prevent catecholamine release for anxiety.
- Midazolam is a good choice.
- Consider adding fentanyl for line placement before induction.
- Induction technique to prevent swings in heart rate and BP.
- Isoflurane, fentanyl, and vecuronium are good choices for maintenance. Isoflurane is a vasodilator and does not sensitize the myocardium to the effects of catecholamines. Desflurane is not the agent of choice because it may cause sympathetic stimulation. The use of sevoflurane is questionable if hypokalemic nephropathy and polyyuria are present (hyperaldosteronism).
- Increased intra-abdominal pressure opposes diaphragmatic descent and causes decreased FRC. This, coupled with reduced CO, results in V/Q mismatch.
- Hypercapnia from systemic absorption of CO₂ requires 1 minute ventilation and high peak inspiratory pressures, increasing the risk for barotrauma. If unacceptably high peak pressures are needed to maintain normocapnia, it may be necessary to allow a mild degree of hypercapnia and resp acidosis to prevent barotrauma.

Overview
- Safe and effective procedure for removal of small benign adrenal neoplasms
- For pts with pheochromocytoma, periop morbidity from adrenergic crisis can be greatly reduced by adequate preop medical management

ICD-9-CM Code: 194.0 and 227.0

Indications and Usual Treatment
- Aldosteronoma: Initial treatment with potassium supplementation, competitive aldosterone inhibitor (spironolactone) and antihypertensive drugs.
- Adrenal adenoma: Antihypertensives and drugs to control blood sugar, treatment of electrolyte imbalances.
- Pheochromocytoma
  - Alpha blocker (phenoxybenzamine) to lower BP, combined with beta blocker to treat cardiac arrhythmias.
  - A beta blocker should not be used as the initial or sole therapy as profound vasoconstriction from unopposed alpha receptor stimulation may lead to a hypertensive crisis or pulm edema.
- Calcium channel blocker (nicardipine) can be used instead of alpha blockers for BP control

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Htn</td>
<td>Palpitations</td>
<td>Standard BP measurement</td>
<td>EKG</td>
</tr>
<tr>
<td></td>
<td>Hypovolemia</td>
<td>SOB, orthopnea</td>
<td>Orthostatic hypotension</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>Dysthyrhythmias</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cardiomyopathy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electrolyte imbalance</td>
<td>Hypokalemia (aldosteronoma, adrenal adenoma)</td>
<td>Muscle weakness, cramps</td>
<td>EKG</td>
<td>Serum K level</td>
</tr>
<tr>
<td>METAB</td>
<td>Hyperglycermia (adrenal adenoma, cortisol) (pheochromocytoma, α mediated inhibition of insulin release)</td>
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</tbody>
</table>


Surgical Stages
- Positioning: Right or left lateral decubitus, the OR table is flexed to open up the area between the costal margin and iliac crest, the kidney bridge is elevated. For bilateral adrenalectomy, the pt may need repositioning or the surgeon may choose to use supine position.
- Pneumoperitoneum is established with CO₂ insufflation to 15 mm Hg via a Veres needle placed in the midclavicular line below the costal margin.
- Approaches: The transperitoneal approach is the most commonly used and provides the best access to the adrenal gland. A retroperitoneal approach may also be used, however because of the limited space in the retroperitoneum, this may be technically more difficult.
- The adrenal veins are ligated early to prevent spillage of catecholamines into the circulation (pheochromocytoma)
- Left laparoscopic adrenalectomy: Three trocars are placed along the costal margin. After mobilization of the splenic flexure and the spleen, the adrenal gland is exposed and the adrenal vein is clipped and divided. After further dissection and clipping or cauteterization of all blood vessels, the adrenal gland is freed and extracted in a sterile endocatch bag through the most anterior trocar.
- Right laparoscopic adrenalectomy: A fourth trocar is needed to retract the liver. After mobilization of the hepatic flexure, the adrenal gland is exposed. The right adrenal vein empties directly into the IVC and dissection to expose this may be difficult when the adrenal gland is enlarged. The vein is either clipped, or when short and broad, stapled and divided. After further dissection, the gland is freed and extracted.

Madhavi Naik
Postoperative Considerations

• Hemodynamic monitoring and treatment of any CV instability may require admission to the ICU, esp. in cases of pheochromocytoma.
• PONV and shoulder pain can be treated with antiemetics, NSAIDs and IV or PO narcotics.

Generally, the need for postop pain medications is significantly lower after laparoscopic adrenalec-tomy than open.

Anticipated Problems/Concerns

• Most serious periop complications are myocardial ischemia and infarction, acute pulm edema, acute right heart failure, cerebrovascular accident, rupture of intra-abdominal or cerebral aneurysm.
**Advanced Cardiac Life Support (ACLS)**

**Alan Jay Schwartz**

### Risk
- Incidence in USA: Based on data from the Resuscitation Outcomes Consortium, emergency medical services treat >294,000 out-of-hospital cardiac arrests annually
- On average, 31% of out-of-hospital cardiac arrests receive bystander CPR
- CHD caused about one of every five deaths in USA in 2005; the largest single killer of American males and females
- Nine risk factors: Cigarette smoking, abn blood lipid levels, Htn, diabetes, abd obesity, a lack of physical activity, low daily fruit and vegetable consumption, alcohol overconsumption and psychosocial index

### Perioperative Risks
- 1.7 cardiac arrests/10,000 anesthetics

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<tbody>
<tr>
<td>CNS</td>
<td>Arrhythmia may cause hypotension</td>
<td>Syncope</td>
<td>CNS exam</td>
<td>ECG, MRI</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Arrhythmia, Htn, valvar disease</td>
<td>CV status, Hx angina, SOB, palpitations</td>
<td>CV exam</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>SOB, orthopnea</td>
<td>Chest exam</td>
<td>SaO2, CXR</td>
</tr>
</tbody>
</table>

**Key Reference:** 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. http://circ.aha.org/journals/content/voll12/24_suppl/.

### Intraoperative Implications

**Monitoring**
- Routine; confirm ETT position using qualitative ETCO2 indicator, capnographic, capnometric and esophageal detector devices
- Invasive monitoring as indicated by the pts condition

**Management**
- Dependent upon type of rhythm and hemodynamic status
- Emergency drugs, incl epinephrine, lidocaine, and atropine, must be immediately available
- Cardiac defibrillator and means of cardiac pacing must be available.
- Initiate BLS with attention to maintenance of airway, ventilation, and circulation via chest compressions until definitive treatment established
- Supraventricular bradyarythmias
  - Treat if significant decrease in BP or cardiac output or in the presence of PAVC; atropine 0.5 mg IV, may repeat to total of 3 mg; transcutaneous or transvenous pacing; dopamine or epinephrine infusion; isoproterenol infusion used with extreme caution
  - Supraventricular tachycardia (PSVT) with severe hypotension and atrial fibrillation (AFib) or flutter with hemodynamic compromise
    - If stable in PSVT, vagal maneuvers followed by adenosine 6 mg IV; then adenosine 12 mg IV after 1–2 min if PSVT persists, treat recurrence with diltiazem or β-blockers.
    - If stable in AFib or atrial flutter, consider diltiazem or β-blockers.
- Unstable pt—immediate synchronized cardioversion (biphasic waveform recommended) (recommended starting dose)
  - AFib 100–200 joules (J)
  - Atrial flutter 30–100 J
  - VT and VFib 120–200 J (360 J with mono- or biphasic defibrillator)
- Stable pt
  - Narrow QRS (≤0.12 sec) (supraventricular tachycardia (SVT)) and regular rhythm-vagal maneuvers, adenosine 6 mg rapid IV, if no conversion 12 mg rapid IV, diltiazem or β-blockers
  - Narrow QRS and irregular rhythm (probable AFib, atrial flutter)—diltiazem or β-blockers
  - Wide QRS (≥0.12 sec) and regular rhythm (VT)—amiodarone 150 mg IV over 10 min to a maximum of 2.2 g/24 hr
  - Wide QRS (≥0.12 sec) and irregular rhythm (if AFib)—diltiazem or β-blockers
  - Torsades de pointes—magnesium 1–2 g over 5–60 min followed by a magnesium infusion
  - Premature ventricular beats
  - Look for treatable cause
  - Suppress with lidocaine 1.0–1.5 mg/kg IV; may repeat 0.5–0.75 mg/kg every 5–10 min to total of 3 mg/kg
  - VT
    - If stable, lidocaine 1.0–1.5 mg/kg IV; may repeat 0.5–0.75 mg/kg every 5–10 min to total of 3 mg/kg
  - If unstable, cardioversion with a biphasic defibrillator at 120–200 J (360 J with a monophasic defibrillator)
  - Atrial flutter often responds to lower energy
  - Ventricular bradyarythmias
    - Heart block with slow idioventricular escape rhythm and signs and/or symptoms of poor perfusion treated with transcutaneous or transvenous pacing
    - Atropine 0.5 mg IV or an epinephrine 2–10 mcg/kg or dopamine 2–10 mcg/min infusion until pacing is available; use of isoproterenol may precipitate VT or VFib
    - Unstable pt—immediate synchronized cardioversion (biphasic waveform recommended) (recommended starting dose)
      - AFib 100–200 joules (J)
      - Atrial flutter 30–100 J
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  - Premature ventricular beats
  - Look for treatable cause
  - Suppress with lidocaine 1.0–1.5 mg/kg IV; may repeat 0.5–0.75 mg/kg every 5–10 min to total of 3 mg/kg
- VT
  - If stable, lidocaine 1.0–1.5 mg/kg IV; may repeat 0.5–0.75 mg/kg every 5–10 min to total of 3 mg/kg
  - If unsuccessful or hemodynamically unstable, cardioversion
- VFIB
  - Rapid defibrillation 120–200 J biphasic defibrillator (360 J with a monophasic defibrillator)
  - If persistent, interspersed defibrillation with epinephrine 1.0 mg IV, vasopressin 40 U IV; then lidocaine 1.0–1.5 mg/kg IV followed by amiodarone, magnesium, and procainamide
  - Bradyasystole
    - High mortality, suspect hypoxia
  - Consider immediate transtunaneous pacing
  - Epinephrine is drug of choice along with atropine
  - Defibrillation may be tried as astystole may be unrecognized VFib
- Pulseless electrical activity (PAE)
  - Caused by acute derangements in preload (hypovolemia, cardiac tamponade, tension pneumothorax), afterload (pulm embolism), myocardial performance (acidosis, hypoxemia)
  - Treatment dependent on cause
  - Epinephrine and atropine may be given

### Indications and Treatment
- ACLS indicated for treatment of life-threatening/fatal arrhythmias or cardiac arrest, e.g., supraventricular bradycardias and tachycardias, ventricular fibrillation (VFib), bradyasystole and electrical mechanical dissociation
- Treatment depends on the arrhythmia or cause of cardiac arrest and the hemodynamic stability of pt.
- The quicker the restoration of normal rhythm, the more likely survival. Defibrillator early.

### Anticipated Problems/Concerns
- Following resuscitation, continued support of vital organ function required. CNS insult may cause increase in ICP or seizures; myocardial damage may result in persistent dysrhythmias or decreased contractility; renal damage may result in acute renal failure.

### PROCEDURES
Amputation, Above-Knee (AKA)

Risk
- 9 to 23 per 100,000 person y, depends on study. Ratio AKA/BKA = 3/2 in some centers
- Mean age 70 y, incidence increases with age between 45 and 85.
- MF ratio: 3:1 with 3:1 up to age 85; >85 male to female is 5:1
- Causes: DM, ESRD, vascular insufficiency, malignant neoplasm, trauma
- DM followed by ESRD, dominant underlying medical condition leading to limb ischemia

Perioperative Risks
- Operative mortality 3–5% (within 30 d), as high as 30% 1-y mortality
- Morbidity inc cardiovascular disease (myocardial ischemia, infarction, CHF, arrhythmias), renal failure/replacement, advanced age, emergency admission, nursing home admission
- Pulm emboli: 6–10%
- Nonhealing, infection common surgical problems

Worry About
- Underlying disease leads to AKA; 20% conversion from BKA to AKA
- Assoc infection, gangrene, tissue loss
- Cardiac disease
- Periop pulm emboli, thromboembolism, stroke
- Flexion contractures of involved limb, postop amputation
- Decubitis
- Phantom limb pain and/or stump pain

Overview
- Assoc with high periop mortality
- Vascular insufficiency to limb limits viability, increased risk of sepsis and complications of immobility; viability evaluated by Doppler, blood flow studies to determine level of amputation
- Preop epidural narcotics to eliminate rest pain
- Regional anesthesia, postop analgesia frequently used; contraindicated in pts receiving anticoagulants
- Rehabilitation more difficult with AKA than below knee amputation (BKA)

ICD-9-CM Codes: 747.64 (PVD); 897.2 (Trauma); 250.0 (DM); 585.6 (ESRD); 195.5 (Tumor)

Indications and Usual Treatment
- Amputations for trauma done early to decrease contamination
- Control infection, sepsis with antibiotics
- Control blood sugar with regular insulin in diabetic pts
- Smoking should be D/C 1 wk before operation or on admission
- Consider revascularization to improve changes for good results
- Below-knee amputation preferred for better rehabilitation
- Early management of systemic complications


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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CAD, Htn, hyperlipids</td>
<td>CV status, chest pain, previous MI, SOB</td>
<td>CV exam</td>
<td>ECG, stress test</td>
</tr>
<tr>
<td>RESP</td>
<td>Emphysema, COPD</td>
<td>SOB, smoking, exercise tolerance</td>
<td>Chest exam</td>
<td>O₂ sat, CXR, ABGs, PFTs</td>
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<tr>
<td>ENDO</td>
<td>DM</td>
<td>Polyuria, polydipsia, cardiomyopathy, neuropathy Autonomic neuropathy Delayed gastric emptying</td>
<td>Sensory exam</td>
<td>Glucose, orthostatic BP Hgb A1C</td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombophlebitis, bleeding</td>
<td>Pain, bruising, bleeding</td>
<td>Ulcerations, ecchymoses</td>
<td>PT, PTT, Hct, INR, WBC</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency/failure</td>
<td>Dialysis, transplant</td>
<td>BUN/Cr, K+</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>CVA, TIA</td>
<td>CNS deficits</td>
<td>CNS exam</td>
<td>Carotid studies</td>
</tr>
<tr>
<td>PNS</td>
<td>Poor circulation Posterior cerebral circulation</td>
<td>Claudication</td>
<td>Peripheral pulses Neck extension</td>
<td>Doppler, flow studies, angio</td>
</tr>
<tr>
<td>INFECTION</td>
<td>Malaise, swelling, gangrene</td>
<td>Fever, chills</td>
<td>Extremity ulcers, swelling, calor, rubor</td>
<td>Temp, cultures for organism</td>
</tr>
</tbody>
</table>

Intraoperative Management
- Volume status monitoring: Blood loss without tourniquet

Preoperative Preparation
- Antibiotics for trauma
- Ensure availability of blood products

Anesthetic Technique
- Either regional or GA appropriate in normal coagulation status

Monitoring
- Routine monitors incl ST-segment analysis
- Consider arterial line (FloTrac), CVP, or PA line if long surgery, depending on CV status

Airway
- In trauma, diabetes, dialysis, consider full stomach

Surgical Stages

Induction
- Spinal or epidural acceptable in pts with normal coagulation profile
- If GA, worry about myocardial ischemia and ventricular dysfunction

Skin Incision
- Circular incision at the level of amputation
- Observe blood loss; need for ligation of large vessels

Dissection
- Identify femoral artery, vein for clamping, division, ligation; sciatic nerve for ligation
- Assessment of viability of tissue by observing bleeding and/or blood loss

Definitive Surgery/Closure
- Femur is divided with a saw, filed
- Wound closed in 2 layers
- Sterile dressing applied
- Immediate postop prosthesis can be applied; more common BKA > AKA
- Approx duration: 1–2 hr
- EBL w/o tourniquet: 250–500 mL

Postoperative Considerations
- Blood loss may continue; replace as indicated by Hct/Hgb
- Venous thrombosis assoc with prolonged hospitalization, immobilization, venous stasis
- Aggressive pulm toilet to prevent atelectasis, pneumonia
- Phantom limb sensation in 100% of pts; usually resolves in 1 y
- Pain score: 5–10
- Pain relief by PCA if epidural is contraindicated

Anticipated Problems/Concerns
- CV morbidity, mortality common 5–30%
- Amputation for acute ischemia has higher co-morbidities
Amputation, Lower Extremity (LEA)

**Risk**
- Over 100,000 pts undergo LEA annually
- Approx 50% are the result of vascular disease
- Estimated that two-thirds of these pts are also diabetic
- Other etiologies incl trauma, malignancy, and congenital anomalies
- Incidence increases with age, more predominant in males, esp. in trauma pts

**Perioperative Risks**
- Highest morbidity: Cardiac (10%) followed by resp (pneumonia)
- Increased risk of arrhythmias, MI, CHF
- Most common cause of death: Cardiac, followed by resp failure
- Overall 30-d mortality around 9%; worse for AKA (17%) than BKA (6%)
- Survival in elderly diabetic amputees less than 50% at 3 y

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Risk Factors/Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CAD, MI, CHF Autonomic neuropathy</td>
<td>SOB, angina, stents, Sx Syncope</td>
<td>JVD, rales, S1 Orthostasis</td>
<td>ECG, enzymes, ECHO Holter, tilt-table</td>
</tr>
<tr>
<td>RESP</td>
<td>Asthma Obesity, sleep apnea COPD</td>
<td>Smoking Difficult intubation SOB/bronchospasm</td>
<td>Wheezing Airway exam Auscultation</td>
<td>PFTs</td>
</tr>
<tr>
<td>GI</td>
<td>Gastroparesis</td>
<td>GERD, early satiety</td>
<td>N/V, NG tube</td>
<td>CXR</td>
</tr>
<tr>
<td>ENDO</td>
<td>Diabetes</td>
<td>DKA or hypoglycemia</td>
<td>AMS may be 2/2 ↓ glu</td>
<td>Accuchek, UA</td>
</tr>
<tr>
<td>HEME</td>
<td>Pts on anticoagulants Anemia</td>
<td>Bleeding Transfusion</td>
<td>Bleeding, bruises Pallor</td>
<td>PT, INR, PTT Type and cross</td>
</tr>
<tr>
<td>GU</td>
<td>ESRD</td>
<td>Dialysis</td>
<td>Fistula, graft, or vas cath</td>
<td>Electrolytes: K, Cr</td>
</tr>
<tr>
<td>MS</td>
<td>Infections/sepsis Rhabdomyolysis</td>
<td>Antibiotics, cultures</td>
<td>VS, wound</td>
<td>WBC with diff, Cx</td>
</tr>
</tbody>
</table>


**Indication/Maintenance**
- Vigilant control of hemodynamic variables and fluids shifts in pts with heart disease

**Operation**
- Procedure length, blood loss and morbidity depend on type of amputation
- Hip disarticulation: 3–4 hr, 1,000–2,000 mL blood loss, ICU postop care
- More distal procedures are generally faster, with less bleeding, and PACU postop care
- Use of a tourniquet also significantly affects the amount of blood loss
- Access for pain during dissection of musculature and bone
- Monitor for major bleeding during dissection of vasculature
- Clean wounds will be covered with a flap then closed
- Contaminated wounds often left open (Guillotine) and a vacuum dressing is placed

**Postoperative Considerations**
- If significant heart disease consider need for postop monitored care for 24–48 hr
- Blood glucose control in diabetic pts may be difficult to achieve
- 26% of vascular pts undergo subsequent amputation within 1 y
- Early as well as late postop pain issues common

**Indications and Treatment**
- Open (guillotine) amputations common in severe infection and/or gangrene; require extensive revision
- Below-knee-above-knee amputation ratio in USA is around 3:1
- Revisions can infrequently convert BKAs into AKAs
- Preservation of knee joint important for more efficient ambulation; less energy expenditure
- Adequate stump flap essential for weight bearing on prosthesis
- Over two-thirds of BKA pts are rehabilitated with prosthesis versus less than one-third of AKAs

**Perioperative Management**

**Preoperative Preparation**
- CV assessment
- Diabetic pts: Control glucose, preserve renal function, provide prophylaxis against aspiration
- Know hemodynamic status for trauma pts (Hb/Hct)

**Anesthetic Technique**
- Can be performed with regional, general, or combined anesthetic techniques
- Regional anesthesia may be contraindicated if pts septic and/or on antithrombotic Rx
- Pts with contaminated wounds needing serial I & Ds may benefit from a continuous epidural catheter
- Postop phantom limb pain may decrease with use of continuous regional anesthesia and/or analgesia

**Monitoring**
- Rarely need a-line, CVP, or PA catheter, except in pts with severe heart disease

**Airway**
- Consider possibility of difficult airway in diabetics
- Preoxygenation, sniffing-position, shoulder ramp to facilitate intubation in obese pts

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### Aneurysm Coiling

#### Risks
- Prevalence of intracranial aneurysms is approx 2.3% in the general population. At least 5.4% of these have multiple aneurysms. Aneurysms may develop from a congenital abn of the arterial wall, are more common in those with connective tissue disorders and those with polycystic kidney disease and may be seen in association with other vascular defects such as arteriovenous malformations. Cerebral injury, infections, and tumors have also been assoc with aneurysm formation. Smoking, Htn, atherosclerosis, and drug abuse (esp. cocaine) are also risk factors. Oral contraceptives have been postulated as a risk factor to explain the slightly increased prevalence in women.
- The annual risk of subarachnoid hemorrhage (SAH) from aneurysmal rupture is 1.3% per year. The risk of rupture increases with the size of the aneurysm, is greater for those located in the posterior cerebral circulation, and is more likely for those who have already experienced the rupture of an intracranial aneurysm at another location. Other risk factors for aneurysmal rupture incl Htn, smoking, alcohol abuse, drug (cocaine) abuse, a family Hx of cerebrovascular disease and features related to aneurysmal shape.
- The 30-d mortality rate for SAH from aneurysmal rupture is 30–40%, and only one third of those who survive remain functionally independent. Additional aneurysmal events are assoc with even higher mortality.

#### Perioperative Risks
- Rupture of the aneurysm from procedural manipulation or Htn during the intervention
- Cerebral infarction due to thrombosis, misplaced embolic material, interruption of flow from vessels originating from the aneurysm neck or dome, or vasospasm
- Complications related to anticoagulation which generally incl heparin and some type of antiplatelet therapy. These can result from hemorrhage in the brain or other sites, reactions to heparin (heparin-induced thrombocytopenia) and reactions to protamine when it is used to reverse heparin.

#### Worry About
- Contrast reactions which can range from direct cardiac depression to anaphylactic reactions. Although the use of non-ionic contrast agents has not decreased the incidence of fatal contrast reactions, it has decreased that of mild to moderate ones.
- Contrast nephropathy, Risk factors incl DM, the large dye loads that are common during prolonged endovascular procedures, volume depletion, pre-existing renal dysfunction, and concurrent administration of other nephrotoxic medications.
- Hypothermia
- Radiation exposure of pt and caregivers
- Distance from pt’s airway due to interposed imaging equipment and radiation shields.

#### Overview
- Endovascular approaches are used to secure both intact aneurysms and those assoc with prior SAH in order to prevent additional events and their resulting morbidity. Endovascular approaches may be advantageous for aneurysms whose location is not anatomically favorable for surgical interventions, such as the posterior cerebral circulation. Endovascular approaches generally favor aneurysmal geometry where the neck is narrow relative to the dome. Overall, as endovascular technique advances, the available literature appears to demonstrate the noninferiority of endovascular approaches for equivalent lesions.
- The 1° technique involves the use of multiple platinum coils advanced through a microcatheter placed at the opening of the aneurysm and detached from the pusher wire. Coil dimensions refer to the length and diameter of the coil. Detachment of the coil once it is in place occurs by passing an electrical current through the insulated pusher wire to initiate electrolysis of the junction between it and the coil. Coils are packed into the aneurysm until contrast dye no longer opacifies the aneurysmal lumen. The placement a fenestrated stent over the aneurysmal orifice makes endovascular treatment possible for aneurysms with wider necks where the coils would be otherwise expelled. To date, there is no consensus as to whether various bioactive coatings on the coils facilitate or contribute to the effectiveness of the intervention.
- To facilitate manipulation of the microcatheter and coil placement a “roadmap” is generated by subtracting the background image obtained from a scout film, and then superimposing the image of the vascular anatomy obtained by an injection of contrast dye on the fluoroscopic image. Clearly, such a technique requires immobility, often to the point where respiration must be suspended for short periods of time.
- Access to the arterial circulation is generally obtained through a sheath in one of the femoral arteries. However, access can also be obtained through the brachial and carotid arteries.
- Anticoagulation therapy is an important component of endovascular therapy and is generally comprised of longer-term antiplatelet therapy initiated prior to the procedure and heparin administered during the procedure which is later reversed with protamine. An exception to this may be when treating an aneurysm assoc with prior SAH. Then, anticoagulation is often held until successful placement of the initial coil(s), an indication that the aneurysm will eventually be secured. At that time antiplatelet medications can be administered by gastric tube or per rectum, and heparin can be administered IV.

### ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Ability to maintain airway while supine and during sedation</td>
<td>Obstructive sleep apnea</td>
<td>Airway adequacy</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>SAH is assoc with ECG changes and left ventricular dysfunction Fluid shifts assoc with contrast dye administration may lead to congestive failure</td>
<td>Exercise tolerance, angina</td>
<td>Signs of congestive failure</td>
<td>ECG, cardiac echocardiogram, cardiac enzymes</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema is common following SAH Exercise tolerance, prior smoking Hx</td>
<td>Rales</td>
<td></td>
<td>CXR, ABGs</td>
</tr>
<tr>
<td>RENAL</td>
<td>Assessment of risk for contrast nephropathy Diabetes mellitus Evidence of decreased intravascular volume and electrolyte abn that may accompany SAH</td>
<td></td>
<td></td>
<td>Cr, serum glucose, Na⁺, K⁺, Ca²⁺, Mg²⁺</td>
</tr>
<tr>
<td>CNS</td>
<td>Determination of prior SAH. Baseline exam for post-procedure comparison, and, if prior SAH, prognostication via SAH grading scale (see chapter on Cerebral Aneurysm Clipping) Headache, or established history of prior aneurysmal rupture</td>
<td>Nuchal rigidity, level of consciousness, cranial nerves, focal deficits</td>
<td></td>
<td>Prior cerebral imaging</td>
</tr>
<tr>
<td>HEMAT</td>
<td>Pts frequently will have anticoagulants administered during the procedure, thrombocytopenia following SAH does occur</td>
<td>Brusing, bleeding from other sites</td>
<td></td>
<td>Plt count, PT, PTT</td>
</tr>
<tr>
<td>IMMUNE</td>
<td>Contract reaction, protamine allergy, heparin allergy Prior Hx of contrast reaction, allergies to protamine or heparin</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Management**

**Preoperative Preparation**
- Antiplatelet therapy (e.g., aspirin and clopidogrel bisulfate) for those with intact aneurysms.
- Prophylaxis for those with contrast dye allergy (e.g., for scheduled procedures: Prednisone 50 mg, orally 13, 7 and 1 hr prior to procedure with diphenhydramine 50 mg po and ranitidine 150 mg po 1 hr prior to procedure; for emergent procedures: hydrocortisone 200 mg IV with diphenhydramine 50 mg IV and ranitidine 50 mg IV).
- Prophylaxis for those at risk for contrast nephropathy (e.g., sodium bicarbonate 154 mEq/L, 3 mL/kg for 1 hr prior to procedure then 1 mL/kg for 6 hr and acetylcysteine 600 mg bid po prior to procedure)

**Monitoring**
- Standard ASA monitors
- Arterial catheter for those with SAH and those at high risk of rupture. Many would place an arterial catheter even in those presenting for elective procedures to facilitate management in the event of a rupture and to permit ongoing assessment of the extent of heparinization. The catecholamine surge that accompanies rupture can sometimes produce vasospasm so marked that radial artery catheters are unreliable, and arterial pressure monitoring from the femoral artery should then be provided by the interventionalist.
- A urinary drainage catheter is necessary given the large fluid shifts and so that accompany the large dose of contrast dye.
- NMB monitoring is essential for techniques using NMB in order to render the pt immobile without excessively prolonging the time until emergence.

**Anesthetic Technique/Induction**
- Anesthetic techniques can range from local, to sedation, to general anesthesia. The level of noxious input is primarily from catheter insertion and is comparable to that for diagnostic angiography for which general anesthesia is rarely necessary. If general anesthesia is not used, the pt must be capable of lying flat, and sedation should not be such that the pt becomes uncooperative, or so deep that resp artifacts from airway obstruction become significant. The use of nasal airways to relieve airway obstruction can be problematic in an anticoagulated pt. Although the ability to monitor the neurologic exam is lost with general anesthesia, it provides a level of immobility which facilitates the procedure. Sedatives that might later interfere with the neurologic exam are best avoided with any technique.
- For general anesthesia, almost any technique with IV or inhalational anesthetics is appropriate as long as the induction is smooth, the response to laryngoscopy blunted, the emergence is crisp and immobility is assured. In pts with unruptured aneurysms, a decreased transmural pressure (proportional to cerebral perfusion pressure) is desirable. However, for those with vasospasm following SAH, it may be necessary to maintain higher Bps to provide adequate cerebral perfusion. Despite its benefit in facilitating rapid emergence, concern exists about the capacity of NO to increase the size of microbubbles introduced into the cerebral circulation.

**Surgical Stages**

**Procedure**
- Access of arterial system
- Detailed angiography to plan the embolization
- Stent placement if dictated by anatomy
- Coil placement to occlude the aneurysm
- Exit from arterial system. The sheath is often left in place or a vascular closure device is used, obviating a prolonged period of pressure to the groin at the conclusion of the procedure.
- Although the treated aneurysm is considered secure, if other lesions are present the impact of their rupture would be compounded by ongoing anticoagulation. In these instances, it is essential to avoid Htn during emergence.

**Postoperative Considerations**
- The pt should be in a situation permitting frequent neurologic assessments.
- Pts are encouraged to remain supine without flexion of the joint adjacent to the vascular access site.

**Anticipated Problems/Concerns**
- Aneurysmal rupture during the procedure can be catastrophic with mortality similar to unwwnessed SAH. The incidence is reported to be 3–5% and is more common with lesions of the posterior circulation, but may be less in experienced hands. Rupture is generally accompanied by a massive Cushing’s response. If technically feasible, the ruptured aneurysm is packed with coils. Further treatment consists of controlling intracranial pressure and systemic BP. Placement of an intraventricular catheter may be beneficial. It will be necessary to reverse any heparin with protamine, and administer working plt’s if the pt received antiplatelet therapy.
- Thrombus formation may be revealed during the procedure, or during angiography administered in response to a neurologic deficit upon emergence from anesthesia. Thrombolytic therapy administered directly to the thrombus may necessitate resumption of general anesthesia. Regardless, an increase in cerebral perfusion pressure is desirable.
Anterior Cervical Discectomy and Fusion (ACDF)

Risk
• ACDF is one of the most commonly performed spine procedures with an 8-fold increase in prevalence from 1990–2004
• M:F ratio: 3:2

Perioperative Risks
• <1% in hospital postop mortality assoc with cervical spine surgery
• Postop airway complications in 6% of cases of anterior cervical spine surgery
• Incidence of recurrent laryngeal nerve (RLN) injury: 5%

Worry About
• Airway management in pts with limited cervical spine mobility resulting in increased risk of difficult intubation.
• Pts at risk for postop airway complications incl those with prolonged procedures; exposure of more than three vertebral levels; involvement of C2, C3, or C4; and more than 300- mL EBL. These risks (and thus complications) more commonly assoc with complex corpectomy and fusion procedures rather than 1–2 level ACDF.

• Surgical complications incl RLN paralysis, esophageal perforation, cerebrospinal fluid leak, injury to vertebral artery, carotid artery, jugular vein, postop dysphagia, and pharyngeal edema or hematoma.
• Pts having previously undergone ACDF may have unrecognized pre-existing RLN injury. If contralateral approach planned, preop fiberoptic exam of cord function should be considered.

Overview
• Indications incl cervical radiculopathy and/or myelopathy 2° to degenerative disc disease and/or spondylosthesis
• The clinical outcome of this procedure is good or excellent in the majority of cases.
• Anterior approach provides good access to vertebral bodies and transverse processes of C2–C7
• Right anterior approach assoc with increased risk of damage to RLN but decreased risk of damage to thoracic duct.
• Anterior plate almost always used, thus securing intervertebral graft placement
• Although fusion rate is greater with autograft (iliac bone) vs. allograft (cadaveric fibula or iliac bone), morbidity assoc with taking bone graft plus improved fusion rates with anterior instrumentation have made the use of allografts for ACDF the standard at most centers. Titanium cages also utilized for interbody fusion and corpectomy/fixation.

Indications and Usual Treatment
• Cervical herniated disk or spur
• Degenerative or traumatic hypermobility or subluxation
• Radiculopathy with foraminal stenosis
• Decreased AP diameter of spinal canal
• Compressive myelopathy
• Degenerative kyphoscoliosis
• Alternative Rx incl posterior cervical approach and conservative management with NSAID, rest.

ASSESSMENT POINTS

System Effect Assessment by Hx PE Test

HEENT Access limited 2° to pain, anatomy, prior fusion, neurologic Sx ↑ Neurologic Sx or pain with movement Oral opening, cervical ROM, thyromental distance, neuro exam Review of cervical imaging studies

PNS Radicular Sx, myelopathy Onset of pain, numbness, weakness, bowel or bladder Sx Motor, sensory assessment, single root vs. cord compression Review of cervical imaging studies (X-ray, MRI, CT; myelogram)


Perioperative Management

Preoperative Preparation
• Careful exam of the airway incl evaluation of cervical mobility
• Consider awake fiberoptic intubation (FOI) or having immediate access to glidescope or Bullard laryngoscope in those pts with potentially difficult airways.
• Surgical positioning generally requires greater neck extension than required for routine direct laryngoscopy.
• In pt with traumatic spine injury or evidence of unstable spine consider awake FOI and positioning.

Monitoring
• Generally routine
• Consider invasive monitoring if multiple levels involved and/or significant co-morbidities
• Arms and neck inaccessible during procedure so invasive monitoring is placed prospectively. Consider placing a second IV catheter for this reason.

• Neurophysiologic monitoring frequently utilized, particularly for more invasive corpectomy and fusion. May incl somatosensory monitoring and/or motor-evoked potential monitoring (MEP).

Anesthetic Technique
• General endotracheal due to poor access to airway and surgical traction on trachea, esophagus
• Pt is placed supine with the neck extended
• Consider narcotic-based anesthesia to assist with neurophysiologic monitoring
• Muscle relaxation assists in distraction of the cervical spine but may interfere with MEP monitoring

Surgical Stages

Dissection
• Trachea and esophagus retracted medially
• Carotid artery retracted laterally
• RLN retracted inferiorly
• Superior laryngeal and hypoglossal nerves retracted superiorly
• Vertebral artery ascends via foramina of transverse processes.

Other Intraoperative Considerations
• Cervical traction occasionally requested of the anesthesiologist to facilitate graft placement
• Coughing or bucking on the ETT at conclusion of case may rarely lead to anterolateral expulsion of the graft in the absence of anterior plating.
• EBL and third space losses are generally small.

Postoperative Considerations
• Postop dysphagia is the most common complaint 2° to traction on esophagus.
• If iliac bone graft is harvested this can be more painful than surgical site (pain score: 6–8). Discomfort can be attenuated with local anesthetic injection pre-emergence.

Anticipated Problems/Concerns
• Airway management in pts with limited cervical spine motion
• Post-operative airway compromise 2° hema
toma formation, soft tissue swelling, or neurologic deficit (RLN injury)
• Failure of fusion now rare given instrumentation, rates range from 2–10%.
**Aortic Valve Replacement**

**Risk**
- Incidence in USA: 27,000 operations in 2005
- M:F ratio: 3:2

**Perioperative Risks**
- For isolated AS, 3–8% periop mortality for pts <70 y, 3–16% for pts >70 y
- Mortality for elective AVR in pts with chronic AR is 4–10%
- Increased mortality with emergency surgery, advanced age, AI, decreased LV function, CAD, HTN, preop pacing, dialysis-dependent renal failure, infective endocarditis
- Morbidity incl heart block, CVA

**Worry About**
- Maintaining optimal hemodynamic variables for valvular lesion
- Decreasing LV function preop, esp. with AR
- Arrhythmias

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<tbody>
<tr>
<td>HEENT</td>
<td>Co-existing dental infection—↑ risk for postop endocarditis</td>
<td>Oral hygiene</td>
<td>CV exam</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>– Rhythm</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>– Murmur</td>
<td>Cardiac cath</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>– Gallop</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Aortic valve dysfunction</td>
<td>Angina, syncope, CHF</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Arrhythmia</td>
<td>Palpitation, syncope</td>
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<tr>
<td></td>
<td>Ischemia</td>
<td>Angina</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>LV dysfunction</td>
<td>DOE, fatigue</td>
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</tr>
<tr>
<td>RESP</td>
<td>Pulm vascular congestion from ↑ LVEDP</td>
<td>Dyspnea</td>
<td>Rales</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Orthopnea</td>
<td>S3</td>
<td>So2</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td></td>
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</tr>
<tr>
<td>CNS</td>
<td>TIA, RIND, CVA</td>
<td>Chronic AFIB</td>
<td>Carotid bruit</td>
<td>Carotid Doppler</td>
</tr>
<tr>
<td></td>
<td>Syncope</td>
<td>Endocarditis</td>
<td>Neuro deficit</td>
<td>TEE (atrial thrombus, valvular vegetation)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carotid disease</td>
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</tbody>
</table>


**Intraoperative Management**

**Preoperative Preparation**
- Cautious premed; prevent anxiety but avoid acute changes in HR, preload, and afterload
- Adequate vascular access, blood products available

**Anesthetic Technique—AS**
- Periop hemodynamic goals: Maintain preload, afterload, avoid lowering BP (coronary perfusion pressure essential with LVH), maintain sinus rhythm, normal rate, aggressive Rx for dysrhythmias (loss of atrial kick/rapid rate poorly tolerated) may require synchronized cardioversion

**Anesthetic Technique—AR**
- Periop hemodynamic goals: Maintain preload, maintain arterial dilation (nitroprusside, nicardipine, clevidine), avoid significant myocardial depression, maintain high-normal HR (90–100/ min); bradycardia results in increasing regurgitation leading to increased LVEDP; inotropic support frequently required post bypass if decreased LVEF
- IABP contraindicated in presence of AR

**Monitoring**
- A-line, ECG with ST-segment analysis
- CVP vs. PAC (CVP may grossly underestimate LVEDP, but placement of PAC has potential for dysrhythmias, PA rupture)

**Cardiopulmonary Bypass**
- Cardioplegia: Antegrade ± retrograde to allow infusion without interruption
- In AR, prevent LV distention at initiation of CPB by maintaining sinus rhythm:
  - IV esmolol 1–2 mg/kg
  - IV lidocaine 1–1.5 mg/kg
  - Defibrillation or urgent aortic cross-clamping/LV venting poss necessary
- Evacuate air and assess valve function with TEE before D/C CPB
- Inotropic support frequently required post bypass if decreasing LVEF (AR)

**Postoperative Considerations**
- Compliance of hypertrophic ventricles after AVR unchanged, need adequate preload + NSR
- Control Htn to avoid bleeding and dissection
- Anticoagulation for mechanical valves; coumadin usually begun 2–3 d postop

**Anticipated Problems/Concerns**
- Decreased preop LV function may require support
- AFIB/AFLT poorly tolerated with LVH; consider low-dose β-blocker to prevent arrhythmias
- Heart block can develop on postop day 1. Atrial, ventricular pacing wires recommended. Bundle of His prone to injury, permanent pacemaker possibly required.
Aortopulmonary Window

Scott Watkins

Risk
- 0.2–0.6% of congenital HD
- Assoc with secundum ASD, PDA, VSD, aortic origin of right PA, interrupted aortic arch/coarctation (23%), tetralogy of Fallot, anomalous origin of coronary arteries
- Assoc with 22q11 chromosome deletion
- No gender predilection

Perioperative Implications
- Periop mortality rate <3%
- Classic defect just above the sinus of Valsalva, near left main coronary artery orifice, which can be damaged during repair

Anesthetic Technique
- General anesthesia with narcotic-based technique usual
- Avoid increases in PVR in the neonatal pt or those with reactive pulm vascular bed
- Excessive diastolic runoff may exist during periods of hypotension, resulting in reduced coronary perfusion, ischemia, and arrhythmias

Monitoring
- Arterial line
- Two peripheral IVs or CVL
- Right and left atrial lines placed by surgeon at conclusion of CPB for monitoring

Airway
- No assoc airway anomalies

Induction
- If IV access available can use opioids, benzodiazepine, ketamine, or etomidate
- If pt is without IV access, sevoflurane mask induction is acceptable
- Use principles applied to VSDs, PDAs, other L → R shunting lesions

Surgical Stages
Dissection
- Under CPB through midline sternotomy

Definitive Surgery
- Involves placement of pericardial or Dacron patch
- Performed under moderately hypothermic conditions

Overview
- Manifests similarly to VSD or PDA with over circulation of pulm vascular bed
- Rx of CHF incl FTT, diaphoresis, dyspnea, recurrent resp infections

ICD-9-CM Code: 745.0

Indications and Usual Treatment
- Surgical correction indicated in most cases using CPB with patch closure (Dacron or glutaraldehyde-treated pericardium) via transaortic approach
- Children presenting beyond 3 mo of age may require cardiac cath to assess reversibility of pulm vascular resistance
- Assoc congenital heart lesions may result in postop problems unrelated to the aortopulmonary window repair
- Eisenmenger’s syndrome with physiologic changes from long-standing left to right shunting only contraindication to surgical closure

Key Reference:

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<tbody>
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<td>CARDIO</td>
<td>CHF, pulm Htn, Eisenmenger’s syndrome</td>
<td>Exercise intolerance, dyspnea, cyanosis</td>
<td>CV exam, bounding pulses, harsh systolic murmur, cardiomegaly, PA sat &gt; RV sat</td>
<td>ECG, ECHO, CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Reactive pulm vascular bed</td>
<td>Tachypnea, cyanosis</td>
<td>Auscultation</td>
<td>O₂ sat, CXR</td>
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ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE | Test |
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</table>

Key Reference:
Effect
Age-related considerations; dehydration 2° to fever, UA
CNS exam
Mental status changes assoc with dehydration;
Assessment by Hx
ECG if indicated,
Leukocytosis, with left shift hemconcentration;
CBC with differential

• Routine,
or determined by pt pathophysiologic indicators.

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Age-related considerations; dehydration 2° to fever, emesis; ↓ PO intake</td>
<td>CV status, Hx of angina, SOB, exercise tolerance</td>
<td>CV exam</td>
<td>ECG if indicated, orthostatics to assess vol</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp impaired 2° to abd pain/splinting in elderly; tachypnea, hyperpnea may suggest perforation/sepsis; full-stomach considerations</td>
<td>Chest exam</td>
<td></td>
<td>CXR if indicated</td>
</tr>
<tr>
<td>HEME</td>
<td>Leukocytosis, with left shift hemconcentration; 4% of pts have normal WBC, differential</td>
<td>CBC with differential</td>
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<tr>
<td>RENAL/CNS</td>
<td>Mental status changes assoc with dehydration; electrolyte abn, early sepsis; ↓ UO 2° to ↓ IV vol</td>
<td>UO, mental status</td>
<td>CNS exam</td>
<td>UA</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
• Restore IV vol temp management

Anesthetic Technique
• GA generally indicated for laparoscopic appendectomy
• General endotracheal anesthesia: Rapid-sequence induction and intubation with Sellick’s maneuver or awake if difficult airway
• Regional: Spinal vs. epidural if no absolute contraindications, pt adequately hydrated, and cooperative; high abd exploration unlikely – most often used with open appendectomy
• Small surgery, but can be fraught with unexpected findings and complications. Be prepared.

Monitoring
• Routine, or determined by pt pathophysiologic indicators.

Airway
• Routine

Induction
• Consider IV volume status when choosing induction agents
• Consider possibility of myopathy in children esp. males, in choosing muscle relaxant

Maintenance
• Usually volatile agent ± N₂O, narcotic, relaxant, use N₂O cautiously in laparoscopy

Surgical Stages

Skin incision
• McBurney’s incision (RLQ)

Dissection
• Extent depends on appendix location, degree of inflammation; third space volume a consideration
• Retrocecal position, 65%
• 30° tip in pelvis

Closure
• Perforated skin closure if abscess present, surgeon may place drain; minimal if laparoscopic
• EBL: <75 mL
• Vol requirements
• Replace deficit and 5–8 mL/kg/hr with normal saline or lactated Ringer’s solution
• Transgastric — laparoscopic assisted appendectomy: Undergoing clinical trials

Postoperative Considerations
• Pain score: 5–7; PCA for postop pain

Anticipated Problems/Concerns
• Similar for open versus laparoscopic appendectomy
• Sepsis, paralytic ileus, atelectasis
• Aspiration risk, esp. during emergence
• Prolongation of NMB drugs 2° to interaction with antibiotics, esp. aminoglycosides

Indications and Usual Treatment
• Suspected appendicitis
• Differential Dx in young children: Acute gastroenteritis, mesenteric lymphadenitis, pyelitis, Meckel’s diverticulitis, intussusception, pneumonia
• Dx In teenagers and adults depends on gender: in females, ruptured ectopic pregnancy, mittelschmerz, endometriosis, salpingitis, regional enteritis; in males, regional enteritis, renal calculi, testicular torsion, acute epididymitis
• Differential Dx in older adults: Diverticulitis, perforated ulcer, acute cholecystitis, pancreatitis, intestinal obstruction, perforating cecal cancer, torsion of ovarian cyst, mesenteric vascular occlusion, rupturing abd aortic aneurysm
• Differential Dx made easier with US, spiral CT, or MRI
• Urine 5-hydroxyindolacetic acid (U-5-HIAA) increases significantly in the early stages of acute appendicitis
• LRG (leucine-rich α-2 glycoprotein): Elevated in the urine of children with acute appendicitis – [LRG] increased with severity of appendicitis
Atrial Fibrillation Ablation

Risk
- Incidence in USA: More than 2.5 million Americans have atrial fibrillation. It is the most common cardiac cause of stroke.
- Predominantly a disorder of the elderly pts and is more frequent in males.
- Assoc co-morbidities: Htn, sleep apnea, valvular heart disease, congestive failure, chronic obstructive and/or restrictive lung disease
- Antiarrhythmic drugs (AADs) are usually only 60% effective in maintaining long-term sinus rhythm (SR) and so radiofrequency ablation has become a popular alternative in the management of this arrhythmia.

Perioperative Management

Anesthetic Technique
- Options for anesthesia incl: MAC or GA or a combination of both.
- The mapping is usually done under MAC with minimal sedation to minimize interference with the stimulation protocol.
- The choice of agent for sedation depends on the discretion and judgment of the anesthesia provider. Midazolam and fentanyl are commonly used.
- If the case was done under general anesthesia (GA):
  - Fluctuations in the hemodynamics during induction and maintenance of general anesthesia
  - Motion of the ablation catheter through the respiration cycle with higher tidal volumes during PPV.
- Few centers have recently adopted the use of high frequency jet ventilation (HFJV) as a mode of ventilation to minimize resp motion and hence have a relatively stable position of the ablation catheter during ablation.
- Use of paralytic agents can compromise ability to assess proximity of ablation lesion to phrenic nerve – done by pacing at ablation site and detecting movement of the ipsilateral diaphragmatic muscle.
- Pts can be fully anticoagulated (on Coumadin and IV heparin) at the time of the procedure.
- Air embolism, development of intracardiac thrombus, thromboembolic complications (incl stroke and/or peripheral arterial compromise), development of cardiac tamponade, pulm vein stenosis, esophageal thermal injury

Overview
- Since its original description in 1998, AF ablation procedure has become a popular treatment strategy. It involves creation of serial circumferential radiofrequency ablation (RFA) lesions around PV ostium (os) to electrically isolate it from rest of the left atrium (LA). This is accomplished by demarcating the PV os either with the help of a circular multipolar mapping catheter (Lasso) and/or utilizing 3-dimensional mapping to define the PV/LA junction.
- To accomplish this, multipolar catheters are inserted percutaneously via the right and left femoral veins into the right atrium. Via transseptal puncture, the Lasso and the mapping/ablation catheter are advanced into the LA.
- Using a combination of orthogonal fluoroscopy, intracardiac US and 3-D mapping (aided by CT or MRI segmented anatomy), the pulm vein/ left atrial junction is identified. RFA involves the delivery of energy via the generator to the tip of the ablation catheter which is moved sequentially around individual or ipsilateral PV os (typical RF duration 20–40 sec; power 20–40 watts). After completion of PV isolation, standard stimulation protocol (decremental overdrive pacing with and without isoproterenol infusion) is performed to assess AF inducibility and unmask any remaining non PV triggers of AF.
- After completion of the ablation protocol, catheters and sheaths are withdrawn into the RA, intracardiac echocardiography (ICE) is utilized during the course of the procedure to monitor for development of any pericardial effusion and periodically assess flow velocities across the PV os.
- At the end of the procedure all catheters are removed, heparin is stopped and if there are no contraindications then a small dose of IV protamine is administered. The sheaths are left in place and removed subsequently once the activated clotting time is ≤180 sec.
- Pts are subsequently monitored on telemetry and stay on bed rest for 6 hr after sheath removal following which heparin is restarted (without bolus) and all previous medications (incl Coumadin and AADs) are resumed.
- A follow-up transthoracic ECHO is performed within 24 hr of the procedure and pts are typically discharged home within 48 hr.

ICD-9-CM Codes: 427.31 (A FIB); 427.2 (Paroxysmal atrial tachycardia, unspecified)

Indications and Usual Treatment
- As per the AF consensus statement, AF catheter ablation is indicated in pts with symptomatic paroxysmal and/or persistent AF that have failed and/or are intolerant to ≥1 membrane stabilizing antarrhythmic drug. In rare clinical situations, it may be appropriate to perform AF ablation as first line therapy. Selected symptomatic pts with heart failure and/or reduced ejection fraction. The presence of a LA thrombus is a contraindication to catheter ablation of AF.

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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Myocardial ischemia</td>
<td>Angina symptoms, Exercise tolerance, DOE</td>
<td>S3, rales, Irregular irregularity</td>
<td>ECG, pharmacologic or exercise stress testing</td>
</tr>
<tr>
<td>RESP</td>
<td>Pt co-morbidities may incl COPD, obstructive sleep apnea (OSA), and amiodarone toxicity</td>
<td>Exercise tolerance, DOE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Edema</td>
<td>BUN, Cr</td>
<td></td>
</tr>
<tr>
<td>NEURO</td>
<td>CV disease</td>
<td>Stroke, TIAs, Bruits</td>
<td>Carotid duplex</td>
<td></td>
</tr>
<tr>
<td>LYTES</td>
<td>Reversible causes of arrhythmias</td>
<td>Diuretic Rx</td>
<td>Serum K+ and Mg2+</td>
<td></td>
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</tbody>
</table>

Monitoring
• ASA standard monitors
• Invasive BP monitoring
• Intracardiac ECHO
• Frequent measurements if ABGs
• Activated clotting time (ACT)

Airway
• Endotracheal intubation for the majority of the procedure; airway instrumentation equipments should be readily available during sedation and before induction of general anesthesia at the time of percutaneous access.
• If HIJFV is used, the ETT cuff may be left deflated to minimize barotrauma, thereby creating an open system. This approach, however, results in greater entrainment of room air. Lung atelectasis may develop and frequent recruitment maneuvers are necessary.

Surgical Stages
• Surgical preparation and draping of the operative site (both groins and infrequently the right neck for internal jugular vein access).
• Infiltration of the skin with local anesthetic
• Obtaining venous access through both femoral veins. Confirm venous access cannulation using fluoroscopy.
• Placement of multiple sheaths: Typically two 8F sheaths in the right femoral vein, two 7F sheaths and one 9F sheath in the left femoral vein; intrarterial access for invasive BP monitoring is achieved either via a radial or 5F left or right femoral arterial sheath.
• After sheaths are in place, 2 decapolar catheters are advanced via the 7F sheaths in left groin and positioned in the posterior RA and CS. The ICE catheter is advanced via the 9F sheath and positioned in the mid RA. The 8F sheaths are replaced serially by a deflectable 12F and long 8F sheaths and in each instance using a Brockenbrough needle, transseptal puncture is performed. The mapping/ablation catheter and the Lasso catheter are then deployed in the LA via the 12F and 8F sheaths respectively.
• A detailed shell of the LA (incl the PVs) is created on the 3-D mapping system and merged with the segmented anatomy of the chamber obtained from the CT or MRI scan.
• With the ablation and Lasso catheter positioned in the left and right sided veins, stimulation protocol is performed to identify PV arrhythmogeneity.
• With the Lasso catheter positioned at PV ostia, RF lesions are targeted at the PV-LA junction encircling the veins (either individually or as ipsilateral common) to achieve electrical isolation (loss of PV entry and exit), which is reassessed 30–60 min after initial ablation. Next, the stimulation protocol is repeated to identify and target any non-PV triggers.
• After achieving successful PV isolation and if necessary targeting non-PV triggers, catheters are withdrawn, heparin is D/C, individual PV flow velocities are assessed, and protamine is administered.
• Sheaths are eventually removed (usually 1–2 hr later when ACT is ≤180 sec) and the pt is kept on bed rest for 6 hr. Heparin is then started (without bolus) and all prior medications incl Coumadin and antiarrhythmic drugs are resumed.

Postoperative Considerations
• Common problems in PACU incl postop pain, N/V, and hypothermia.
• Pts with Hx of obstructive sleep apnea should be subject to extended periods of monitoring for desaturation and apneic episodes.
• Typically postprocedure pts are kept in the hospital for 24–48 hr during which they are monitored on the telemetry and are maintained on IV unfractionated heparin.
• A transthoracic echocardiogram is obtained the following day to assess chamber dimensions, valve function, and pericardial effusion.

Anticipated Problems/Concerns
• Complications of percutaneous vascular access incl bleeding, hematoma, development of arteriovenous fistula, and development of pseudoaneurysm.
• Procedural complications incl pericardial effusion/tamponade, development of PV stenosis, thrombo-embolic complications incl cerebrovascular accidents, formation of intracardiac thrombus, injury to the phrenic nerve, and damage to the esophagus, which can potentially result in the development of atrio-esophageal fistula.
Atrial Septal Defect, Repair of

Joshua D. Stearns
Charles W. Hogue, Jr.

Risk
• ASD is the most common congenital heart defect.
• Ostium secundum represents 7% and 40% of all congenital heart disease in children and adults, respectively
• MF ratio: 1:2, except for ostium primum ASD, for which ratio is 1:1

Perioperative Risks
• Risk dependent on age, degree of reversibility of increased PVR; mortality usually <1%
• Supraventricular arrhythmias incl atrial flutter, AFib
• AV conduction block possible if ASD close to AV node

Worry About
• Paradoxical venous embolism
• Right heart volume overload with RV dysfunction
• MVP and mitral regurgitation assoc with ostium secundum

• Partial anomalous pulm venous drainage assoc with sinus venous defect
• Cleft anterior mitral valve leaflet associated with ostium primum defect
• Endocarditis prophylaxis
• Left to right shunt leading to pulm Htn and eventual Eisenmenger’s syndrome (right to left shunt); concern greater with large defects (>1 cm diameter)

Overview
• Classified by location: Ostium secundum (70% of ASDs) involves midseptal fossa ovalis; sinus venosus, may occur near RA/SVC or RA/IVC junction; ostium primum, in inferior septum, represents an endocardial cushion defect
• Unless heart murmur heard, usually not diagnosed until symptomatic in third or fourth decade of life
• Degree of left to right shunting dependent on size of ASD, relative compliance of ventricles, relative SVR, PVR

Indications and Usual Treatment
• Surgical closure for uncomplicated ASD with pulm-to-systemic flow ratio >1.5
• Optimal age of repair may be <5 y
• PVR at rest ≥ 8 U/m² that fails to lower to <7 U/m² with pulm vasodilators usually contraindication to surgery
• Lung Tx considered with correction of ASD or heart and lung Tx for irreversibly raised PVR
• Transcatheter closure possible in some centers

ICD-9-CM Code: 745.5

ASSESSMENT POINTS

System Effect Assessment by Hx PE Test
CARDIO Pulm Htn, RHF SOB, DOE, fatigue ↑ RV impulse, JVD ECG (R axis deviation in ostium secundum vs. L axis deviation in ostium primum)
Fixed split S₂, hepatomegaly, ascites, edema
ECHO (RAE, RVE; paradoxical septal motion; Qt:Qs calculation from R- and L-sided stroke volumes; color-flow Doppler; check for anomalous pulm veins and MVP/mitral valve regurgitation), cardiac catheterization

RESP Infection Cough, spuitum Rhonchi, wheezing, consolidation CXR, CBC, cultures

HEPAT Passive edema Hepatomegaly, jaundice, ascites Liver enzymes, albumin, PT, PTT


Perioperative Implications

Anesthetic Technique
• General
Monitoring
• CVP
• Arterial line
• Consider TEE with color-flow Doppler, LAP
Induction/Maintenance
• Anesthetic technique guided by preferences, age, condition; extubation early after surgery in most pts
• Maintain HR, preload, contractility
• Avoid large increases in PVR or large decreases in SVR to minimize R → L shunt

Surgical Stages
• Median sternotomy most common approach, but anterior lateral thoracotomy, and cosmetic submammary incision becoming more common
• Cardioplegic arrest
• Direct suture (primary) closure if ASD small, pericardial patch closure for larger defects. Dacron graft, Gore-Tex CV patch suitable substitutes for pericardial patch in some cases.
• Anomalous pulm venous drainage repaired with pericardial patch “baffle,” redirecting pulm venous flow into LA
• Mitral valve repair for some pts with ostium primum and subsequent mitral cleft

Postoperative Considerations
• LAP/PCWP may be high, 2° to MR, LV diastolic dysfunction from co-existing disease or shift of I-IV septum
• RV dimension, hemodynamics improve shortly after surgery
• Supraventricular arrhythmias incl AFib/flutter
• Reoperation uncommon, but occasional dehiscence of atrial baffle
• Pulm and peripheral thromboembolism with AFib (warfarin commonly started second day after surgery for 8–12 wk)
• Risk of heart block, esp. in ostium primum repairs and in primary closer of atrial defect
AV Graft for Hemodialysis

Randall F. Coombs

Risk
- Incidence in the USA: 26 million Americans have chronic kidney disease
- End stage renal disease (ESRD) is defined as either a glomerular filtration rate less than 15 mL/min/1.73 m² or a need for dialysis or renal transplantation.
- >355,000 on hemodialysis in the USA

Perioperative Risks
- Perioperative mortality is low compared to complications of ESRD (electrolyte-induced arrhythmias, cardiac decompensation)
- Fistula failure rate is 10–20% due to thrombosis or inadequate flow through graft.
- Other surgical complications: Infection, venous aneurysm, venous Htn, vascular steal

Worry About
- Adequate preop Rx of Htn, CAD, and DM. More than 70% of diabetic ESRD pts have CHF, and almost 70% have CAD. In nondiabetic ESRD pts, CHF and CAD occur just more than 40% for both diagnoses. CV disease is the number one cause of mortality in pts with kidney failure.
- Hypovolemia and hypokalemia (esp. if recently dialyzed)
- Hypertension, hyperkalemia, and acidosis (esp. if not recently dialyzed)
- Possible marked Htn in response to surgical stimuli under general anesthesia
- Adequate intravascular volume and BP to preserve graft patency

Overview
- Causes of ESRD: Glomerulonephritis, diabetes, Htn, pyelonephritis, polycystic disease, collagen diseases, reflux or obstructive nephropathy, renal vascular disease, drug toxicity
- Manifestations of chronic renal failure (Stenling’s anesthesia and co-existing disease 5th ed., 2008)
  - Electrolyte imbalance: Hyperkalemia, hypermagnesemia, hypocalcemia, increasing phosphate
  - Metabolic acidosis
  - Unpredictable intravascular fluid volume status
  - Anemia: Increased cardiac output, oxyhemoglobin dissociation curve shifted to the right
  - Uremic coagulopathies: Ptt dysfunction
  - Neurologic changes: Encephalopathy, peripheral neuropathies
  - Cardiovascular changes: Htn, CAD, CHF, angina

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<tr>
<td>CARDIO</td>
<td>CAD, CHF, LVH, arrhythmias</td>
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<tr>
<td>RESP</td>
<td>Pulm edema</td>
</tr>
<tr>
<td>GU</td>
<td>↓ Concentrating ability, uremia, electrolyte/acid-base abn, oliguria/anuria</td>
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<tr>
<td>ENDO</td>
<td>↑ Glucose, ↓ glucose after treatment</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, Platelet dysfunction</td>
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<tr>
<td>CNS</td>
<td>Encephalopathy</td>
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<td>PNS</td>
<td>Peripheral neuropathy</td>
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<td>Chest pain, exercise tolerance, Htn, palpitations</td>
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<tr>
<td>SOB, orthopnea, PND</td>
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<td>Anorexia, N/V, diarrhea, malaise</td>
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<tr>
<td>Hx DM?, daily glucose values</td>
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<tr>
<td>Fatigue, SOB, bruising</td>
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<tr>
<td>↓ Mental acuity, somnolence</td>
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<td>Weakness, numbness, paresthesias</td>
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<tr>
<td>Rales, respiratory rate and pattern</td>
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<td>Wt</td>
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<tr>
<td>Evidence of PVD and/or poor healing</td>
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<tr>
<td>Pallor, bruising</td>
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<td>Mental status exam, sedation assessment</td>
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<tr>
<td>Motor and sensory neurologic exam</td>
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<td>ECG, CXR</td>
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<tr>
<td>CXR</td>
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<tr>
<td>K, Cr, BUN, HCO₃⁻</td>
</tr>
<tr>
<td>Hemoglobin AIC, blood glucose</td>
</tr>
<tr>
<td>Hgb/Hct</td>
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<tr>
<td>Pt count</td>
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Perioperative Management

Anesthetic Techniques
- Monitored anesthesia care (MAC)
  - Local anesthesia by surgeon with IV sedation by anesthesiologist
  - Best when surgical dissection is minimal (i.e. radial-cephalic anastomosis at the wrist)
  - Regional (brachial plexus block)
  - Vasoconstriction helps surgeon identify best vein to anastomose to
  - Increased blood flow decreases incidence of periop thrombosis
  - Better hemodynamic stability and less N/V than with GA
  - Contraindications: pt objection, anticipated technical difficulty
  - General anesthesia (GA)
  - Better for uncooperative pt
  - Potential for prolonged drug effects and drug interactions
  - Potential for complications related to electrolyte/acid-base abn
  - Potential for airway problems and resp depression

Monitoring
- Place IV, BP cuff, pulse oximeter on nonop arm
- Five-lead electrocardiogram
- Temp (skin or esophageal)
- ETOC, for MAC and plexus block as well as GA if sedative drugs given

Induction/Maintenance
- Non-K⁺-containing, careful fluid balance monitoring
- Large volume of NS leads to ↑ metabolic aci-dosis and thus higher K⁺ than when equal volume of LR is given

Induction/Maintenance
- Decreasing plasma proteins lead to prolonged and increased effect of highly protein-bound drugs
  - Acidosis increases non-ionized, unbound drug (active portion of drug is non-ionized)
  - Uremia inhibits the sympathetic nervous system, thus decreasing compensatory vasoconstriction
  - Increased CNS effects of anesthetic induction drugs due to uremic disruption of the blood brain barrier

- Maintenance of general anesthesia
  - Inhalation agents (sevoflurane, isoflurane, desflurane) all acceptable
  - Total IV anesthesia (remifentanil, propofol, and cisatracurium) also acceptable
  - Muscle relaxants
  - Avoid succinylcholine if K⁺ ≥ 5.5 (K⁺ may increase by 0.5–1.0 mEq/L after succinylcholine)
  - Delayed excretion of pancuronium, thus increasing the duration of action

- Duration of action of vecuronium, and rocuronium are not significantly prolonged
- Clearance of mivacurium, atracurium, and cisatracurium is independent of renal function
- Opioids
  - Increase magnitude and prolonged duration (decrease protein binding and delayed clearance)
  - Accumulation of morphine metabolite, morphine glucuronide leads to resp depression
  - Fentanyl may be given judiciously (large doses accumulate and prolonged effect)
  - Local anesthetics
  - Increase susceptibility to toxicity due to reduced protein binding, metabolic acidosis, and delayed elimination

- Sympathetic nervous system activity due to treatment with antihypertensive drugs
  - Renal osteodystrophy
  - Pruritus
  - Autonomic dysfunction (e.g., Stokes-Adams attacks)
  - Poor nutritional state: Fatigue, general malaise, and anorexia

Indications and Usual Treatment
- ESRD: Cr clearance ≤ 5 mL/min or serum Cr > 1200 μmol/L
- Methods of dialysis: Peritoneal dialysis (PD) versus hemodialysis (choice depends on the pt’s general condition and preference)
- PD preferred in young children, elderly, difficult vascular access, pt’s preference for independent self-care
- Contraindications to hemodialysis
  - Absolute: CHF or hemodynamic instability,
  - Relative: Psychosis and/or severe mental retardation, carcinomatosis, multiple nonrenal complications, e.g., blindness, diabetic neuropathy
  - AV fistula created 1–2 mo prior to expected start of hemodialysis to allow time for maturation of shunt

PROCEDURES
Surgical Stages

Location
- Should be easily accessible for dialysis
- First fistula: As far distal as possible (adequate size of vein is the determining factor)
- Non-dominant arm if possible
- Wrist: Radial artery to cephalic vein
- Forearm (most common first site): Radial, ulnar, or brachial artery to brachial vein
- Upper arm: Brachial artery to basilic or axillary vein

Blood Flow
- 200–300 mL/min immediately after anastomosis formed
- 200 mL/min required to complete dialysis in 3–4 hr
- Doppler often used during closure to check shunt for patency

Types of Access
- Fistulas: Side of artery to side of vein, or side of artery to end of transected vein
- Prosthetic graft: Usually Teflon (polytetrafluoroethylene)

Procedure Duration
- Variable, ~ 1–3 hr

Blood Loss
- Usually minimal (depends on access and ease)

Postoperative Considerations
- Medical management
  - Monitor and manage co-existing diseases (i.e., DM, CAD, CHF, and Htn)
  - Assess fluid volume status and serum electrolytes (esp. serum K+)
  - Consider early postop dialysis (but not via new fistula).
- Operated arm
  - Elevated for several hours and avoid constrictive clothing since arm may swell
  - Avoid venipunctures and BP measurements.
  - Monitor fistula blood flow (palpate thrill, auscultate, Doppler).
- Early surgical revision for inadequate fistula blood flow
- Pain management
  - Medication initially unnecessary if brachial plexus block was performed
  - PO analgesia usually adequate

Anticipated Problems/Concerns
- IV access can be difficult in these pts. Central venous line may be necessary.
- Avoid fistula use for initial 3 wk to prevent aneurysm formation
- Thrombosis is the most common complication: up to 20% in first few weeks after surgery.
- Infection: Higher incidence with graft fistulas

Prognosis
- Fistulas have a finite life span. Revisions and repeat procedures are common.
  - Less than 50% patent at 1 y, less than 40% patent at 2 y
- Renal transplantation is the optimal treatment for ESRD in suitable pts

Maturation of fistula
- Blood flow increases with time (up to 1200 mL/min)
- Venous wall thickens (prevents venous tears)
- Maturation period is variable but generally takes longer for smaller vessels.
Blalock-Taussig Shunt (BTS)

**Risk**
- Indicated for infants with congenital heart lesions resulting in either severely reduced pulm blood flow (PBF) (e.g., tetralogy of Fallot, pulm and tricuspid atresia) or as the first stage of single ventricle palliation (e.g., hypoplastic left heart syndrome [HLHS]).

**Perioperative Risks**
- Fewer BT shunts are performed now compared to previous decades and operative mortality has fallen despite a higher percentage of pts with single ventricle physiology.
- Periop complications incl Horner’s syndrome, chylothorax, phrenic nerve damage and acute arm ischemia with classic BTS.

**Worry About**
- BTS for decreased PBF keep PVR as low as possible (high FIO₂, avoid hypercarbia and acidosis)
- BTS for single ventricle staged palliation essential to balance SVR and PVR (often requires FIO₂ 0.17 - 0.21 and normocarbia)

**Overview**
- One of multiple types of systemic to pulm shunts to increased PBF
- Familiarity with underlying anatomy and physiology is essential to management.
- Subclavian artery (opposite side of arch to limit kinking) to PA anastomosis performed directly (classic BTS), or most common is using a Gore-Tex tube graft (modified BTS)

**Indications and Usual Treatment**
- Infants with decreased PBF who are unable to have a 1˚ surgical correction of their congenital heart lesion may have a BTS to improve PBF and allow PAs to grow.
- Infants with single ventricle physiology have a BTS as part of a three-stage surgical palliation (Stage 1 Norwood procedure).

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>↑ Incidence</td>
<td>Other congenital abl</td>
<td>Airway</td>
<td>FISH for DiGeorge syndrome Chromosomes</td>
</tr>
<tr>
<td></td>
<td>of craniofacial defects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>CHD</td>
<td>Single ventricle physiology Qp-Qs</td>
<td></td>
<td>ECHO Cardiac cath</td>
</tr>
<tr>
<td>RESP</td>
<td>↓ Pulm blood flow</td>
<td>‘Tet’ spells (hypcyanotic episodes from dynamic RVOT narrowing in tetralogy of Fallot)</td>
<td>Cyanosis</td>
<td>O₂ sat, Hct ABGs CXR</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Pts often already intubated, consider placing a nasal ETT for infants under 12 mo (tube more stable and makes placement of TEE probe easier)
- Arterial line: For cardiac lesions with ↓ PBF place A-line in contralateral radial artery from BTS or in the femoral artery. For single ventricle lesions place an A-line in right radial artery if low flow cerebral perfusion technique to be used for Stage I Norwood and/or in the umbilical or femoral artery.
- Central venous line: All cases for inotropes. Consider co-oximetry central venous catheter.

**Intraoperative Period**
- May use inhalation induction. Single ventricle pts often have IV access.
- Infants with tetralogy of Fallot may have tet spells due to worsening RVOT obstruction.
- Lateral thoracotomy classic approach: Anticipate worsened hypoxia with PA clamping, blood product transfusion usually not required.
- Sternotomy and CPB for complex and single ventricle cases

**Postoperative Period**
- Goal is adequate but not excessive pulm blood flow; shunt should be slightly restrictive to avoid CHF. Calculate Qp:Qs.
- Advantages of modified BTS incl: preservation of subclavian artery, technically easier anastomosis and later take down, greater PA growth with less distortion of PAs and low shunt failure rate
- Goal: Adequate but not excessive PBF
- Nonconfluence of PAs, distal PA stenosis and single ventricle lesions require cardiopulmonary bypass (CPB) and more extensive surgery

**ICD-9-CM Code:** 745.2 (Tetralogy of Fallot)

**ICD-10-PCS Code:** 12.0 (Subclavian-Artery-to-Pulmonary-Artery-Bypass)
Blood Components

Overview
- The U.S. blood supply is dependent on volunteer donations
- Whole blood donations are processed into the various components (RBC, FFP, cryoprecipitate, and Plts). Components can also be collected individually via apheresis.
- Citrate is the anticoagulant in all components.
- Preservative solutions are added to RBC units to extend the shelf-life up to 42 d

Indications and Usual Treatment
- RBC: Given to increase O₂ carrying capacity due to acute blood loss or chronic anemia. Dose: 1 unit, expect increase in Hgb by 1 g/dL or Hct by 3% in adults.
- Plt: Given to prevent or treat bleeding due to thrombocytopenia or plt dysfunction. Dose: Whole blood-derived plt 1 unit/10kg, expect each unit to increase plt count 5000/μL; Apheresis plt 1 unit, expect 1 unit to increase plt count 30,000–60,000/μL.
- FFP: Given to prevent or treat bleeding due to depletion and/or deficiency of multiple coagulation factors, incl. urgent warfarin reversal, DIC, massive transfusion. Dose: 10–20 mL/kg, expect factor levels to increase by 20%
- Cryo: Given to treat bleeding in hypofibrinogenemia or dysfibrinogenemia. Second line therapy to treat Hemophilia A only when Factor VIII concentrate is not available, and von Willebrand’s disease only when pt is not DDAVP-responsive, and vWF concentrate is not available. Dose: 1 unit/7–10 kg, expect fibrinogen levels to increase by ~50 mg/dL.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Component</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td>O₂ delivery</td>
<td>Anemia; blood loss; chemotherapy; cardiopulmonary reserve</td>
<td>Pallor; acute blood loss; hypotension; tachycardia</td>
<td>Hgb, Hct, O₂ extraction ratio</td>
</tr>
<tr>
<td>PLT</td>
<td>Coagulation</td>
<td>Mucosal bleeding; diseases or drugs affecting plt function; blood loss ≥1 blood volume</td>
<td>Microvascular bleeding, petechiae, purpura</td>
<td>Plt count; plt function testing</td>
</tr>
<tr>
<td>FFP</td>
<td>Coagulation</td>
<td>Personal/family Hx of bleeding; ecchymoses; liver disease; warfarin therapy; blood loss ≥1 blood volume</td>
<td>Microvascular bleeding</td>
<td>PT, PTT</td>
</tr>
<tr>
<td>CRYO</td>
<td>Coagulation</td>
<td>Personal/family Hx of bleeding; vWF disease; congenital dysfibrinogenemia; blood loss ≥1 blood volume</td>
<td>Microvascular bleeding</td>
<td>Fibrinogen</td>
</tr>
</tbody>
</table>

Worry About
- Pt willingness to accept transfusion
- Presence of RBC antibodies detected on antibody screen
- Availability of components from blood bank

Risk
- One out of every 10 hospitalized pts receives a transfusion
- Approx 29 million components transfused annually

Perioperative Risks
- Transfusion-transmitted infection (HIV, hepatitis, West Nile, etc.)
- Hemolytic transfusion reactions (acute and delayed)
- Nonhemolytic reactions (fever, allergy, anaphylaxis, transfusion-related acute lung injury)
- Immunosuppression (increased infection rate, cancer recurrence)

Perioperative Management

Preoperative Preparation
- Type and screen: ABO/Rh type, and detection of clinically significant RBC antibodies
- Type and crossmatch: Same as above, plus testing to demonstrate compatibility between donor and recipient
- ABO compatibility is important for all components; Rh compatibility is important for RBC and plt components
- Coagulation testing is not advocated in pts with no Hx or exam suggestive of abn

Induction/Maintenance
- Hypotension can further compromise O₂ delivery in anemic pts
- Periop cell salvage and acute normovolemic hemodilution can reduce allogeneic RBC transfusions
- Massive blood loss (>1 blood volume) will likely require transfusion of RBC, FFP, plt, and cryo; ideally, component therapy is driven by laboratory and clinical assessment

Postoperative Considerations
- Continued blood loss may necessitate additional transfusion

Anticipated Problems/Concerns
- Risk of transfusion and/or transmitted infection is lower now than at any other time
- Some pts refuse transfusion based on religious beliefs. An in-depth discussion with the pt can elucidate what he/she will allow/refuse. This should be thoroughly documented.
- The blood bank will need additional time to find compatible RBC units for pts with antibodies. A discussion with the blood bank can provide insight regarding the relative ease or difficulty in finding compatible units based on the antibody(ies) present.

Blowout Orbital Fracture

Kathryn E. McGoldrick

**Risk**
- Rare
- Pts subjected to blunt trauma assoc with battery, motor vehicle accidents, and sports injuries with a nonpenetrating object (e.g., fist)
- Racial predominance: None

**Perioperative Risks**
- In absence of serious assoc injuries, rare periop mortality (<0.1%)
- Postop risk of visual disturbances, incl blindness, infection, and cosmetic deformity

**Worry About**
- Intraocular damage (ruptured globe rare in isolated orbital blowout fracture owing to release of compressive forces into the maxillary sinus)
- Assoc nonophthalmic injuries (intracranial injury, cervical spine fracture or subluxation, Le Fort fractures with basilar skull fracture)
- Prolapse and incarceration of orbital soft tissues
- Preop systemic steroids (to distinguish NM edema or related motility disturbance from true entrapment, unmask enophthalmos, and reduce discomfort) predispose to sinus-orbital infections
- Hemostasis for delicate surgery

**Overview**
- Fractures of orbital floor are repaired by various surgical approaches
- Intraop infiltration with lidocaine with 1:100,000 epinephrine for hemostatic effect
- Entrapped tissues freed with care taken to avoid infraorbital neurovascular tissue trauma
- Autologous or alloplastic implant material placed over fracture site; reconstruction typically done with mesh, implants, or solvent-preserved bone graft

**ICD-9-CM Code:** 802.6

**Surgical Indications/Usual Treatment**
- The three standard indications for surgical intervention are enophthalmos, motility disturbance ≥2° to entrapment, hypo-ophthalmos
- Timing of surgical intervention depends on assoc injuries and pt’s age, general health, and preference; often preferable to delay in order to permit some resolution of edema and bleeding; technical ease and functional result typically enhanced by surgery in 5–14 d or longer; rarely considered urgent unless muscle entrapped and possibly ischemic

**ASSESSMENT POINTS**

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Trauma may produce orbital subcutaneous emphysema, restriction of globe motility, globe ptosis, enophthalmos, retinal or choroidal injury</td>
<td>Inspection</td>
<td>Palpation</td>
<td>CT scan</td>
</tr>
<tr>
<td>CNS</td>
<td>Trauma may produce head injuries</td>
<td>CNS Hx</td>
<td>CNS exam</td>
<td>CT scan</td>
</tr>
<tr>
<td>MS</td>
<td>Trauma may produce assorted MS and organ injuries</td>
<td>Pain</td>
<td>Palpation</td>
<td>Plain films as indicated</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Children with a trap door injury may require treatment for vagal reactions, incl nausea and syncope, caused by trapped parasympathetic nerve fibers that travel with the injured muscle.

**Anesthetic Technique**
- GA usual; also local

**Monitoring**
- Routine

**Airway**
- Other facial injuries may complicate airway management

**Induction/Maintenance**
- Without assoc injuries, few hemodynamic perturbations
- Avoid Htn (to minimize bleeding)
- Adequate depth of anesthesia to prevent pt movement during delicate surgery
- Consider anti-emetic prophylaxis

**Surgical Stages**

**Dissection**
- Minimal blood loss

**Definitive Surgery**
- Minimal blood loss (<100 mL) and fluid shifts
- Approximate duration: 2 hr

**Postoperative Considerations**
- Mild postop pain
- IV PCA usually not necessary
- Without other injuries, discharge on day of surgery

**Anticipated Problems/Concerns**
- Blindness
- Intraorbital paresthesia
- Implant extrusion
- Diplopia and delayed extraocular muscle restriction
- Obstructive sinus disease
- Infection
- Return to potentially violent environment
Bone Marrow Transplantation
(Harvest Procedure)

Risk
- Autologous: Pts with certain malignancies that respond to chemotherapy with need for marrow reconstitution and for cell-based therapies (experimental treatment for acute MI, refractory CHF)
- Allogeneic: HLA-matched healthy donor for recipient with malignancy or marrow failure as alternative to peripheral blood–stem cell transplant

Perioperative Risks
- Postop morbidity is high 2° to underlying disease of autologous donor.
- Life-threatening complications extremely rare (0.27%) for allogeneic (healthy) donor

Overview
- Multiple (often 100–200) needle punctures of posterior iliac crest (occasionally other sites) made to obtain stem cells for reinfusion following marrow ablative chemotherapy for malignancy or as protocol directed for cell-based therapy.
- Operating physician may be a hematologist (not surgeon)

Indications and Usual Treatment
- Marrow failure, hematologic malignancy, selected chemotherapy-responsive solid tumors
- Recipient anticipates prolonged hospitalization in major medical center; aggressive support for anemia, thrombocytopenia, neutropenia, GVHD
- Autologous transplantation may follow first or subsequent remission for hematologic malignancy or be performed prior to chemotherapy for solid tumor of sites remote from bone marrow stores
- Cell-based therapy (protocol-driven) uses bone marrow stem cells for experimental treatment of acute MI and refractory CHF. (Smaller quantities of marrow may be harvested under local anesthesia.)

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<tbody>
<tr>
<td>CARDIO</td>
<td>CHF, dysrhythmias</td>
<td>Doxorubicin, pericardial effusions</td>
<td>CV exam</td>
<td>ECG, consider ECHO/MUGA</td>
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<tr>
<td>GI</td>
<td>Electrolyte imbalance</td>
<td>Vomiting, melena</td>
<td>Edema, orothostasis</td>
<td>Na+, K+, Ca2+</td>
</tr>
<tr>
<td>HEME</td>
<td>Blood loss, infection</td>
<td>Recent chemotherapy</td>
<td>Petechiae, ecchymosis</td>
<td>CBC, platelets</td>
</tr>
</tbody>
</table>

*Applies primarily to autologous donors with malignancy.


Intraoperative Management

Monitoring
- Large-bore IV (x2); meticulous sterile technique
- Consider CVP, if access difficult
- Check availability of irradiated blood
- Urinary cath may be necessary (large fluid shifts)
- Arterial access may result in hematoma (thrombocytopenia); useful if BP is labile
- Pulse oximetry may guide O2 requirements: Acutely reduced ETCO2, may indicate embolism from marrow space

Induction
- Decrease the dose of induction agent if there is Hx of cardiotoxic chemotherapy

Surgical Stages
- Establishment of adequate venous access
- Induction of general anesthesia (spinal or epidural possible for allogeneic donors)
- Establish prone position
- Supine to prone position change requires minimun of 4 persons
- Wt supported on chest/pelvis with arms extended on armboards
- Avoid pressure on eyes, throat, genitals
- Abd position for free excursion
- Aspiration of marrow
- Cell count of marrow determines volume needed
- Return to supine position
- Duration 2–4 hr
- Postop pain score 1–4; usually PO drugs (acetaminophen with codeine) adequate

Anticipated Problems/Concerns
- Familiarity with pt's specific chemotherapy and protocols
- Volume replacement with crystalloid, albumin
- Avoid starch (interferes with processing of marrow)
- Avoid non-irradiated RBCs (potential engraftment of random donor nucleated cells)
- Prior steroid Rx may result in reduced adrenal reserve
- Avoid unnecessary O2 enrichment if prior chemotherapy incl bleomycin
- Air and/or O2 may be used (N2O inhibits methionine synthetase resulting in marrow toxicity)
**Bowel Resection**

**Risk**
- Incidence depends on disease process
- Person living in North America has an average lifetime risk of 6% of developing colorectal cancer
- Crohn’s disease has an annual incidence of 4 cases per 100,000

**Perioperative Risks**
- Periop mortality 0.5-5% mainly due to underlying disorder and other co-morbidities
- Periop morbidity: Prolonged ileus 5–10%; anastomotic leak 2–4%; wound dehiscence 1–2%, bleeding 1%

**Worry About**
- Decreased intravascular volume and electrolyte abn, esp. hypokalemia due to bowel prep
- Bowel obstruction, esp. in the small bowel can lead to bowel necrosis, perforation, and development of septic shock
- Risk for aspiration with bowel obstruction
- Contamination of peritoneum with bowel perforation
- Development of PTE

**Overview**
- Bowel resection of the large and small bowel is performed for a variety of reasons
- Crohn’s disease most common surgical disease of the small intestine
- Small bowel resection involves varying amounts of mesentery
- Large bowel resection requires mobilization prior to resection; 1° anastomosis vs. colostomy depends on several factors—infammation, prepped vs. unprepped bowel, local ischemia

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<tbody>
<tr>
<td>HEENT</td>
<td>Hypovolemia</td>
<td>Dizziness</td>
<td>Orthostatic BP</td>
<td>Electrolytes, Hct</td>
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<tr>
<td>CARDIO</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Aspiration risk</td>
<td>N/V</td>
<td>Bowel distention</td>
<td>ABG, O2 sat, CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoventilation and hypoxemia</td>
<td>Splinting</td>
<td>Auscultation</td>
<td></td>
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<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Signs of hypovolemia, dry mucus membranes</td>
<td>Electrolytes, BUN, Cr</td>
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<tr>
<td>GI</td>
<td>Diarrhea, N/V</td>
<td>Nausea</td>
<td>Orthostatic vital signs</td>
<td>Electrolytes</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Volume replacement and correction of electrolytes prior to induction
- Aspiration prophylaxis with sodium citrate and H2 antagonist
- Avoid metoclopramide in pts with bowel obstruction or perforation
- Appropriate antibiotic prophylaxis

**Monitoring**
- Routine monitors
- Consider CVL if significant co-morbidities or pt has difficult venous access
- Consider arterial line for continuous BP measurement and blood sampling
- Foley catheter to monitor urine output

**Anesthetic Technique**
- General anesthesia
- Consider combined GETA/epidural for postop analgesia
- Avoid LMA due to risk of aspiration
- Large bore IV access
- Monitor hemodynamics and airway pressures closely during abd insufflation if laparoscopic approach

**Airway**
- Increased incidence of gastric aspiration due to bowel obstruction or emergency surgery
- Consider placement of NG tube prior to induction to suction gastric contents

**Induction**
- Often RSI due to aspiration risk
- Consider awake intubation
- Induction agents depend on volume status and assoc co-morbidities

**Maintenance**
- Avoid NO in cases of bowel obstruction
- Relaxation with non-depolarizing agent is often needed in order for surgeon to have optimal working conditions

**Surgical Stages**

**Dissection**
- May have extensive adhesions if previous surgeries
- Hypotension with manipulation of perforated or strangulated small bowel
- May have hypotension after peritoneum opened, esp. if intra-abdominal bleeding has been tamponaded

**Definitive Surgery**
- Large third-space loss, depends on degree and duration of bowel exposure, may require extensive crystalloid and colloid
- Risk of hypothermia when large amounts of bowel exposed
- Risk of ureteral injury during mobilization of pelvic colon
- Suction enterostomy of obstructed bowel will lead to additional fluid requirements

**Postoperative Considerations**
- Consider epidural analgesia or IV PCA for pain
- Hypoventilation and hypoxemia due to splinting, consider supplemental O2 during postop period
- Ileus from bowel manipulation and narcotics

**Anticipated Problems/Concerns**
- Aspiration on induction due to bowel obstruction
- Hemodynamic instability due to hypovolemia from bowel prep
- Septic shock if bowel perforation preop

**ICD-9-CM Codes: 153.9 (Obstruction, volvulus, intussusception, tumors, trauma, Crohn’s disease) 555.9 (Crohn’s disease)**
## Brain Cortex Resection (for Epilepsy)

### Risk
- Incidence in USA: 300,000
- 3000 ablative operations/y
- Racial predilection: None

### Perioperative Risks
- Depends on procedure—potential unintentional interruption of neural pathways
- Combined morbidity, mortality rates <5% for epileptogenic focus resection, <11% for corpus callosotomy, <17% for functional hemispherectomy
- Blood loss diathesis (functional hemispherectomy)
- Risk of craniotomy incl hypercoagulable state (PE, thromboembolism), intracerebral hemorrhage, air emboli, infection

### Worry About
- Intraop seizures
- Aspiration; airway obstruction

### Overview
- Used for intractable seizures
- Requires discussion with surgeons and neurologists; concerns about intraop electrocorticography (ECoG), and functional testing when elegant brain cortex is at risk
- Concerns about seizures, possible status epilepticus
- Concerns about craniotomy, incl blood loss, adequate operating conditions, postop responsiveness
- Pt may have co-morbid conditions—e.g., psychiatric disorders, tuberous sclerosis, neurofibromatosis, obesity

### Usual Treatment
- Pts recommended for cortical resection for epilepsy have met these criteria:
  - Have focal seizure not responding to adequate trial of antiepileptic agents
  - Seizures significantly interfere with pt's overall function
  - Surgery appears to offer reasonable opportunity for improvement of overall function
- Preop testing likely incl Wada's test to determine hemispheric dominance, functional MRI testing, and both invasive and non-invasive EEG localizations

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<tbody>
<tr>
<td>CARDIAC</td>
<td>Arrhythmias</td>
<td>Rx with carbamazepine and phenytoin Valproic acid</td>
<td>Clinical cardiac exam</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>Wt gain</td>
<td></td>
<td>Calculate BMI</td>
<td></td>
</tr>
<tr>
<td>HEENT</td>
<td>Gum hypertrophy</td>
<td>Phenytoin Topiramate, eye pain, vision less</td>
<td>Airway exam</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acute myopia and 2° narrow angle glucoma</td>
<td>Carbamazepine Vigabatrin</td>
<td>May require specialized ophthalmologic exam</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diplopia</td>
<td></td>
<td>Cranial nerve exam</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Peripheral field vision loss</td>
<td></td>
<td>Visual field exam</td>
<td></td>
</tr>
<tr>
<td>GI/HEPATIC</td>
<td>Hepatitis, pancreatitis</td>
<td>Most commonly with valproic acid, trimethadione, mephenytoin, lamotrigine, felbamate (more common in females), abd pain</td>
<td>Hepatomegaly, jaundice</td>
<td>Routine biochemical monitoring (ALT, AST, GGT, amylase) is NOT indicative of extent of liver damage. Blood lipase levels if pancreatitis suspected.</td>
</tr>
<tr>
<td>HEME</td>
<td>Blood dyscrasias related to antiepileptic drugs</td>
<td>Weakness, fatigue, Rx with carbamazepine, ethosuximide, mephenytoin, phenytoin, trimethadione, lamotrigine, phenobarbital, valproic acid, felbamate</td>
<td>Petechiae, rash</td>
<td>CBC</td>
</tr>
<tr>
<td>RENAL</td>
<td>Nephritis</td>
<td>Trimethadione therapy Carbamazepine, oxcarbazepine Zonisamide, topiramate</td>
<td>BUN, Cr, Na, Bicarb, ABG</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hyponatremia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEURO</td>
<td>Lethargy, depression, insomnia, dizziness, etc., related to anticonvulsants</td>
<td>Rx lamotrigine, topiramate (decreased attention, memory impairment), zonisamide, valproic acid Carbamazepine</td>
<td>MS exam</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tremor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ORTHIO</td>
<td>Osteoporosis</td>
<td>Phenytoin, carbamazepine, barbiturates, valproic acid</td>
<td>Neurologic exam</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Factsures</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SKIN</td>
<td>Rash</td>
<td>Highest risk: Phenytoin, lamotrigine, carbamazepine; may occur with all anti-epileptic drugs</td>
<td>Skin exam</td>
<td></td>
</tr>
</tbody>
</table>

### Key Reference:
Perioperative Management
• Cortical resection under general anesthesia is most frequently performed for temporal lobe operations where structural lesions have been identified and intraop testing of speech, motor and other functions is unnecessary.

Intraoperative Electrocorticography/Conscious Sedation
• Discussion by surgeon, anesthesiologist about specific pt’s suitability for an awake craniotomy
• Work to establish rapport with the pt; preop orientation to intraop testing
• Pts position themselves as comfortably as possible; O2 delivered by nasal prongs
• Avoid benzodiazepines if intraop ECoG or serial neurocognitive testing is planned

Monitoring
• Anesthesiologist’s continuous awareness of pt’s well being
• Capnography by nasal prongs
• Urinary cath
• 1 large-bore IV line
• Arterial line placement based on co-morbidities

Airway
• Careful inspection mandatory: Difficult intubation anticipated in lateral position with pre-existing airway abn
• Rigid pinion fixation may be used; fiberoptic equipment should be available.
• Some centers use LMA in an “asleep/awake/asleep” technique.

Induction
• If intraop ECoG, general anesthesia chosen, induction can be with propofol, maintenance with N2O, propofol, narcotic technique supplemented with low-dose isoflurane 0.25%. Avoid N2O if craniotomy for electrode placement within previous 2 wk.
  • LMA with spontaneous breathing
  • Anesthesia maintenance with remifentanil (0.01 to 0.05 μg/kg/min) infusion often satisfactory for intraop ECoG recording
  • D/C propofol and/or inhalation agents 20 min prior to ECoG
  • Narcotic requirements may be increased
  • During closure of craniotomy, care must be taken to avoid Htn, movement by pt
  • For craniotomy, conscious sedation under local anesthesia, two generally accepted techniques: Remifentanil 0.03 to 0.09 μg/kg/min and propofol 30 to 180 μg/kg/min or dexmedetomidine 0.3 μg/kg bolus and infusion at 0.2 μg to 0.7 μg/kg/hr with occasional fentanyl 50 μg bolus
  • Infusions D/C 20 min before pt needs to be responsive to ECoG recordings
  • Consider glycopyrrolate prior to dexmedetomidine bolus
  • Local infiltration of scalp by surgeons (careful attention to local anesthesia overdose)
  • Consider addition of morphine 5–10 mg for postop analgesia

Surgical Stages
• Scalp incision: Should be comfortable with adequate local anesthesia
• Removal of bone flap: Pt may be bothered by drilling (should be warned)
• Stripping of dura: May cause N/V
• Meningeal vessel manipulation: May cause pain
• Blood loss: While usually not great, replaced to avoid hypovolemia
• N/V: Can be controlled with metoclopramide 5–10 mg or ondansetron 4 mg
• Pt at risk for seizures
  • Often eliminated by cold saline applied to cortical surface
  • May require therapy with propofol, IV methohexital 0.5–1.0 mg/kg if ECoG anticipated; following ECoG, benzodiazepines acceptable.

Postoperative Considerations
• Fluctuating blood levels of anticonvulsants
• Possible postop agitation
• Control of hemodynamics, possibility of recurrent seizures
• Pain score ~5–10; careful narcotic titration

Anticipated Problems/Concerns
• Difficulties with seizure control
• Possible brain swelling or intracranial hematoma related to resection
• Pt anxiety related to lengthy operation time
• Intraop N/V
Breast Biopsy

Overview

- Palpable mass, nipple discharge, or abn finding on mammogram indication for surgery
- Excisional biopsy removes benign lesion in entirety
- Lumpectomy removes cancerous tissues with sufficient tumor-free margins
- Sentinel lymph node biopsy performed by injecting marker (blue vital dye or low radioactive tracer) for lymphatic mapping. Node closest to location of breast cancer has 97% accuracy in predicting metastasis of tumor to nonsentinel nodes.
- Nondiagnostic sentinel node biopsy mandates auxiliary dissection. Positive node greater than 0.2 mm requires complete auxiliary dissection.

ICD9-CM: 174 (Malignant neoplasm of female breast)

Indications and Usual Treatment

- Non OR diagnostic procedures may incl US guided, mammographically guided, MRI-guided biopsy or stereotactic core biopsy.
- Open breast biopsy, breast biopsy with sentinel lymph node biopsy are OR procedures
- Excisional biopsy for benign lesions
  - Palpable lesions (e.g., fibroadenomas)
  - Non-palpable lesions (e.g., nipple discharge from intraductal papillomas)
- Needle localized biopsy for potentially malignant, nonpalpable abn seen on screening mammograms
- Sentinel lymph node biopsy for small invasive breast cancers without clinical evidence of lymph node involvement

ASSESSMENT POINTS

System | Effect | Assessment by Hx | Test
--- | --- | --- | ---
HEENT | Obesity may make airway management difficult for sedation | OSA | Prior difficult intubation
GI | GERD | Reflux | 
CNS | Ability to tolerate with sedation | Anxiety level |


Airway

- Surgical field may limit access to airway
- Supplemental O₂ via nasal cannula or face mask
- May need airway adjuncts for pts with OSA done under sedation. Consider GA with LMA

Induction and Maintenance

- IV sedation: Propofol infusion plus anxiolytic (e.g., midazolam) and/or short acting narcotic
- GA standard induction. Usually performed as outpatient procedure: any technique that allows rapid emergence. Mask or LMA in appropriate pts

Surgical Stages

- Dye injected to identify lymph nodes frequently causes transient drop in pulse oximeter reading
- Use of electrocautery may cause increased discomfort requiring deep sedation or GA
- Postop considerations
  - Pain score 2–5
  - Pain management PO analgesics
  - PONV may require rescue antiemetics

Anticipated Problems/Concerns

- Depth and size of mass may necessitate converting from local with sedation to GA
- Use of injectable dye may cause blue urine, emesis or stool for 24–48 hr. Reaction to isosulfan blue may incl itching, blue hives, decreased BP
Bronchoscopy, Fiberoptic

Andranik Ovassapian

Overview
- Fiberoptic bronchoscopy enables endoscopist to go deeper into bronchial tree for evaluation, biopsy of lesions not commonly accessible to rigid bronchoscopy
- Fiberoptic bronchoscopy assoc with repeated coughing, Htn, tachycardia often due to inadequate topical or general anesthesia
- Hypoxemia common when performed without supplemental O₂ Rx
- Blood loss from biopsy site of lower airway lesions can be troublesome.
- Tracheal and sometimes bronchial intubation, separation of lungs possibly necessary for major hemoptysis

ICD-9-CM Code: 162.9 (Lung cancer)

Indications and Usual Treatment
- Evaluation of upper, lower airway problems, Dx of pulm disease
- Most useful for diagnosis and staging of lung cancer
- Has diagnostic, therapeutic, and problems-solving indications
- Rx of acute atelectasis performed by saline lavage, aspiration of thick secretions
- Transbronchoscopic bronchial biopsy, brushing cytology, transbronchial needle aspiration biopsy, bronchialveolar lavage performed
- Absolute contraindications incl acutely unstable CV system, current life-threatening cardiac arrhythmia, severe hypoxemia

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Upper airway obstruction due to tumor, edema Limitation of movement</td>
<td>Degree of compromise Airflow pattern Snoring Stridor</td>
<td>Mandibular subluxation Size of tongue Head and neck anatomy Mouth opening Neck ROM</td>
<td>Lateral x-ray CT scan Barium swallow Flow-volume loop</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Ischemic CAD potential</td>
<td>CAD Hx: Chest pain CHP Sx</td>
<td>Heart rate and rhythm, S₃, rales</td>
<td>ECG with stress</td>
</tr>
<tr>
<td>GI</td>
<td>Aspiration potential</td>
<td>GE junction integrity by Hx of regurgitation Nighttime cough Sour nighttime taste</td>
<td>Evaluate nutritional status</td>
<td>Esophagoscopy Examination of larynx</td>
</tr>
<tr>
<td>CNS</td>
<td>Sleep dysfunction due to obstruction</td>
<td>Sleep Hx</td>
<td>CNS exam</td>
<td>MRI</td>
</tr>
<tr>
<td>RESP</td>
<td>Upper airway obstruction due to tumor</td>
<td>Tumor Hx</td>
<td>Wheezing Cyanosis Clubbing</td>
<td>Flow-volume loop, ABGs</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
- Depends on indication
  - If laryngeal tumors, evaluate for degree of airway compromise
  - If stridor present, closely observe, preferably in ICU: Humidified O₂, and steroids may be given to avoid severe and/or complete airway obstruction.

Monitoring
- Routine

Anesthetic Technique
- Topical anesthesia and light sedation for most adults and some children
- General anesthesia with laryngeal mask airway in children.
- Laryngeal mask airway provides open airway and easy access for bronchoscope
- Short-acting IV drugs (e.g., alfentanil, midazolam, esmolol) may provide analgesia and attenuate CV response to rigid laryngoscopy
- Blood loss negligible

Postoperative Concerns
- CV hyperactivity
- Status of upper airway

Anticipated Problems/Concerns
- Hypoxemia in immediate postop period
- Acute ventilatory failure
Bronchoscopy, Rigid

Andranik Ovassapian

**Risk**
- First performed by Killian in 1895. Its use has declined continuously after introduction of fiberoptic bronchoscope.
- Performed for removal of foreign body, massive hemoptysis, to dilate tracheobronchial structures, laser resection of airway tumors, and stent placement.

**Perioperative Risks**
- Depends on nature of disease
- Cardiac arrhythmias, hypoxemia, increased BP and HR, myocardial infarction
- Incidence of reintubation reported at 0.39% when panendoscopy performed for upper airway path

**Worry About**
- Ventilation, oxygenation in pts with chronic lung disease, airway pathology
- Endoscopist, anesthesiologist share airway, complicating ventilatory management
- Massive hemorrhage
- Postbronchoscopy ventilatory failure

**Overview**
- Assoc with severe CV response manifested with tachycardia, Htn
- Bleeding from biopsy site could be troublesome
- Level of the lesion critical
- Ventilation performed through side arm
- Requires communication with surgeon throughout procedure

**Indications and Usual Treatment**
- Removal of foreign bodies
- Management of major hemoptysis
- Establishing emergency airway
- Dilatation of tracheobronchial stricture
- Laser resection of airway tumors
- Bronchoscopy in infants, small children
- Bronchoscopy, esophagoscopy, laryngoscopy for staging of oropharyngolaryngeal malignant lesions
- Biopsy of endobronchial lesion
- Evaluation of the lower airway
- Placement of stent

**ICD-9-CM Code:** 146.9 (Oropharyngeal cancer)

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Airway compromise</td>
<td>Wheezing, stridor</td>
<td>Lung, airway exam</td>
<td>CXR</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Exercise intolerance, angina, Hx of CV disease</td>
<td>S3, rales</td>
<td>ECG</td>
<td>Stress test</td>
</tr>
<tr>
<td>RESP</td>
<td>Wheezing, exercise tolerance impairment, Hx of smoking</td>
<td>Clubbing, cyanosis, wheezing</td>
<td>PFTs</td>
<td>ABGs</td>
</tr>
<tr>
<td>AIRWAY</td>
<td>Tumors</td>
<td>SOB</td>
<td>Wheezing</td>
<td>X-ray studies</td>
</tr>
<tr>
<td></td>
<td>Infections</td>
<td>Hemoptyis</td>
<td>Use of accessory muscles</td>
<td>PFTs</td>
</tr>
<tr>
<td></td>
<td>Compressions</td>
<td></td>
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</tbody>
</table>

**Perioperative Management**
- Usually done as outpatient procedure with pt in sitting or supine position
- Antisialagogue (glycopyrrolate 0.2 mg IV or 0.4 mg IM) to minimize secretions, enhance topical anesthesia of airway
- Appropriate size bronchoscope, ancillary equipment should be available

**Anesthetic Technique**
- Routine
- Usually done under sedation, topical anesthesia
- Lidocaine 4% spray of oropharynx followed by translaryngeal injection of 3 mL 4% lidocaine provides excellent topical anesthesia
- Spray-as-you-go technique used to anesthetize rest of bronchial tree
- General anesthesia for children
- Short-acting IV drugs used during fiberoptic bronchoscopy
- Jet ventilation can be applied through fibroscope but not commonly practiced

**Postoperative Concerns**
- Hypoxemia treated with supplemental O2
- Irritable airway, coughing, tachycardia, Htn common during early recovery
- Bleeding from biopsy site may necessitate repeating the procedure

**Anticipated Problems/Concerns**
- Inadequate ventilation, hypoxemia with heavy sedation
- Airway obstruction, hypoxemia, barotrauma of lungs possible
- Consider precautions to minimize fire hazards during laser surgery

Burr Hole

**Risk**
- Often indicated for cranial decompression, esp. traumatic brain injury
- Annually, 470,000 people sustain traumatic brain injury.

**Perioperative Risks**
- Depending on pathology and severity of injury, mortality ranges 24–50%.

**Worry About**
- Proper maintenance of CPP (MAP-ICP)
- Compromised CBF because of altered cerebral autoregulation

**Indications and Usual Treatment**
- Hematoma evacuation
- Pneumocephalus and hydrocephalus evacuation
- Drainage suprasellar cysts
- Placement of intraventricular drain
- Evacuation subdural empyemas
- EEG electrode placement

**ASSESSMENT POINTS**

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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>C-spine injury creating difficult</td>
<td>Mechanism of injury (blunt vs. penetrating)</td>
<td>Airway exam but assume unstable neck</td>
<td>CT, C-spine series</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Autonomic dysfunction, blunt chest trauma concerns (PTX, hemothorax), pre-existing cardiac arrhythmias</td>
<td>Hx atrial fibrillation requiring anticoagulation</td>
<td>Auscultation</td>
<td>CXR, EKG</td>
</tr>
<tr>
<td>RESP</td>
<td>Brainstem injury, Cushing's reflex, blunt chest trauma concerns (PTX, hemothorax)</td>
<td>Abn respiration</td>
<td>Bilateral breath sounds, wheezing</td>
<td>ABGs, pulse oximetry, CXR</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia may compromise cerebral O2 delivery</td>
<td>Hx anticoagulation (warfarin)</td>
<td>CBC, PT/INR, plt</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>ARF due to hypotension. Electrolyte abn—hyperkalemia, hyponatremia, hyperglycemia</td>
<td>Recent osmotic diuretic, hypertonic saline, steroid administration</td>
<td>Na+, K+, BUN, Cr, serum, urine osmolality</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Impaired LOC, increased ICP</td>
<td>Glasgow coma scale assessment</td>
<td>Gross neuro exam, CN exam, pupil exam</td>
<td>CT, MRI</td>
</tr>
</tbody>
</table>

**Perioperative Management**

**Preoperative Preparation**
- CT/MRI scans to confirm side of lesion. Assess midline shift and ventricular compression.
- Routine medical management: Avoid hypoxemia, hypotension, hypercapnia. Elevation of head 15–30 degrees, mannitol (0.25–1 g/kg), neutral head position, antibiotics.
- Prophylactic hyperventilation (PaCO₂ of 25 mm Hg or less) is not recommended.
- Correct underlying coagulopathy, esp. if Hx of warfarin.

**Anesthetic Technique**
- Local anesthesia, MAC, or GA

**Monitoring**
- Consider large-bore IV access. Central line may be necessary if mannitol, hypertonic saline indicated.
- Consider arterial line, esp. if hyperventilation needed.
- Consider ICP monitoring, Foley catheter (esp. if osmotic diuretics administered).
- Nasal ETCO₂ monitoring with local/MAC technique.

**Airway**
- If trauma pt, RSI and inline stabilization for presumed C-spine injury.
- Since airway is not easily accessible, ensure ETT secured well.

**Induction/Maintenance**
- Avoid increased ICP. Avoid coughing due to light anesthesia. Turning head may obstruct venous drainage. Consider elevation HOB 30 degrees. If severe increased ICP, consider total IV anesthetic.
- Prepare for increased HR and BP during intubation and possible pin placement. Consider propofol, increased inhalational agent, and beta blockade.
- Hyperventilation should be limited in first 24 hr from injury because of decreased CBF, esp. if hypoxemia and hypertension also present.
- Avoid long-acting opioids to facilitate prompt neuro exam.
- Avoid secondary insult—hypoxia, hypercarbia, hyperglycemia.

**Surgical Stages**

**Dissection**
- Risk of accidentally tearing dura

**Definitive Surgery**
- Beware sudden bleeding if sinus or middle meningeal artery encountered.
- Air embolism risk increased if venous sinus encountered and elevated HOB

**Conclusion**
- Prompt neuro exam to assist in management in ICU as well as determine need for additional surgery.
- Low threshold to remain intubated if unable to assess CNS status.
- EBL 50–200 mL

**Anticipated Problems/Concerns**
- Still concern for intracranial Htn, despite Burr hole drainage.
- Appropriate sedation to prevent postop Htn (>160 mm Hg) leading to increased CBF, ICP, bleeding, and cerebral edema.
- Strongly consider specialized neuro ICU to detect neurologic changes.
# Bypass, Femoral-Femoral

## Risk
- Incidence in USA: ~10,000/y, number is decreasing; number of aortic and iliac stents is increasing.

## Perioperative Risks
- Related to diffuse arteriosclerosis, CV disease
- Periop MI 3–10%
- Mostly geriatric pts with age-related risk factors and accompanying diseases: DM, COPD, Htn, renovascular
- Potential for improvement and/or more distal occlusion with risk of need for amputation

## Worry About
- Thrombosis and embolism of leg or graft
- Blood loss

## ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>High incidence of lung disease that could have caused this procedure to be selected</td>
<td>Smoking, chronic cough, dyspnea</td>
<td>Chest exam, wheezing, clubbing cyanosis</td>
<td>CXR, ABGs, spirometry</td>
</tr>
<tr>
<td>CARDIO</td>
<td>High incidence of CAD, myocardial ischemia, and/or infarction</td>
<td>Angina, MI, CHF, dysrhythmia, PCI (metallic/DE stents), CABG, exercise tolerance, activity level</td>
<td>Chest auscultation, VS</td>
<td>ECG, stress ECHO, dipyridamole, thallium or dobutamine test</td>
</tr>
<tr>
<td>Chronic HTN</td>
<td></td>
<td>Antiplatelet Tx?</td>
<td>ECG, CXR, retinal funduscropy</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency 2nd to age, arteriosclerosis, and multiple dye studies</td>
<td>Hx edema and intolerance to salt load</td>
<td>BP Rx, drug interaction, LVH</td>
<td>BP</td>
</tr>
<tr>
<td>CNS</td>
<td>Possible carotid disease</td>
<td>Syncope, stroke, or TIAs</td>
<td>Neuro exam</td>
<td>Carotid angiogram, CT, MRI</td>
</tr>
<tr>
<td>ENDO</td>
<td>DM</td>
<td>Infections, stress/gestational</td>
<td>Skin infections</td>
<td>CNS exam, UO; glucose</td>
</tr>
<tr>
<td>HEME</td>
<td>Periop heparin, t-PA, or aspirin</td>
<td>Petechiae, nasal bleed</td>
<td>Carotid bruist</td>
<td>PT, PTT, ACT</td>
</tr>
</tbody>
</table>

## Indications and Usual Treatment
- An extra-anatomic procedure
- Performed in pts with lower extremity ischemia, gangrene, or severe short-distance claudication; in one limb more than the other
- Replace the standard anatomic bypass of ABI or ABF in pts with the following conditions:
  - S/P abd procedure
  - S/P extensive abd radiation
  - Intestinal stoma
  - Abd infection
  - Poor medical condition
  - S/P iliac: Aortic graft stent
  - Ischemic limb from thoracic dissection

## Overview
- Femoral-femoral bypass is suggested for pts too risky to undergo abd procedure (ABI, ABF)
- Done with prosthetic graft. This is a nonanatomic graft that might be fed by a nonoptimal vessel.
- Regional anesthesia can be beneficial in its effect on coagulation and graft patency
- Procedure sometimes can be performed also under monitored anesthesia care

ICD-9-CM Code: 440.2x (Atherosclerosis, Peripheral)

## Monitoring
- ECG, arterial BP, UO
- Consider CVP or PA line in pts with severe CAD or severely ↓ EF.
- Monitoring for myocardial ischemia; ECG with continuous 3-lead ST-segment analysis, PA catheter, or TEE
- Monitoring of vol replacement: CO, CVP, PAOP, or with TEE

## Postoperative Period
- Mild postop pain: 5–7
- Ischemic leg pain should improve
- Epidural extended for postop pain control

## Anticipated Problems/Concerns
- High incidence of periop cardiac and resp complications
- Inappropriate blood flow from donor side tends to make the bypass not functional
- Infection of prosthetic graft at the groin is relatively frequent, compared to other prosthetic graft locations
- If the femoral-femoral bypass is not functional, a bypass such as axillary femoral-femoral or amputation may be needed

## Key Reference
Bypass Graft Procedure, Infrainguinal

R. Yan McRae
Grace L. Chien

**Risk**
- 0.4–14% prevalence of intermittent claudication in adults; occurs most frequently in individuals age 50–75 y old; peripheral vascular disease asymptomatic in >50% of pts, identified only by noninvasive testing (ankle/brachial index; ABI)
- Co-existing conditions commonly incl: tobacco use, Htn, diabetes, CAD, COPD, renal insufficiency

**Perioperative Risks**
- Mortality: 5.8% at 30 d, 16% at 1 y; approx half of periop deaths due to cardiac complications
- Common morbidity: 2–6.5% incidence of periop myocardial infarction; respiratory complications; poor wound healing

**Worry About**
- Reperfusion syndrome after prolonged ischemia: Hyperkalemia, metabolic acidosis, hypotension, transient increase of O2 consumption, hypothermia
- Intra- and periop cardiac complications: Myocardial ischemia, myocardial infarction, dysrhythmias, CHF
- Graft thrombosis/failure

**Overview**
- Wound healing incl need for periop glyceric control
- Determine urgency of surgery (see Indications below)
- Consider the need for myocardial revascularization prior to infrainguinal revascularization (see Coronary Artery Revascularization Prophylaxis (CARP) Trial) if pt has an indication for myocardial revascularization
- Reduce risk of periop thrombosis in pre-existing coronary or vascular stents, or new graft, through coordinated anesthetic and surgical plan for periop antiplatelet or anticoagulant therapies
- Reduce periop risk of cardiac complications through initiation and/or continued periop administration of beta-blocker, clonidine, and/or statin therapy
- Manage intraop hemodynamics and hematocrit to maintain adequate myocardial O2 supply/demand matching
- Provide tight glyceric control

**No definitive benefit of regional anesthetic over general anesthetic in reducing cardiac complication or increasing graft patency rates**

**Indications and Usual Treatment**
- Two primary indications:
  - Claudication: Exercise-induced leg pain relieved with rest. Initially treated with trial of lifestyle modification, smoking cessation, exercise and medical management. Surgery primarily to improve quality of life, done when anatomy suggests possible durable, long lasting repair. Elective nature of procedure allows careful preop evaluation and treatment to reduce periop morbidity and mortality.
  - Critical limb ischemia defined as persistent, recurring ischemic rest pain. Ankle systolic pressures usually lower than 50 mmHg +/- visible tissue damage. Generally requires intervention despite operative risks and graft failure risks. Surgery may briefly be delayed to optimize pts with CHF; hemodialysis for volume or hyperkalemic issues, anticoagulation management, etc.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by HX</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CAD, previous MI, CHF, arrhythmias, upper extremity PVD</td>
<td>Angina, orthopnea, prior stents and stent types</td>
<td>Pedal edema, S3, murmurs, discordant BP in arms</td>
<td>ECG, CXR, noninvasive cardiac testing, cardiac cath</td>
</tr>
<tr>
<td>RESP</td>
<td>COPD, pulm edema</td>
<td>Dyspnea, S3 gallop</td>
<td>Cracks or wheezes, barrel chest</td>
<td>CXR/CT, PFTs, ABG</td>
</tr>
<tr>
<td>Renal</td>
<td>CRI, dialysis</td>
<td>Htn, timing of most recent dialysis, recent/anticipated IV contrast load</td>
<td>Fistula patency, edema, dyspnea</td>
<td>K+, BUN, creatinine</td>
</tr>
<tr>
<td>CNS</td>
<td>Carotid and cerebral atherosclerosis</td>
<td>Amaurosis fugax, TIA, stroke</td>
<td>Neurologic deficits, carotid bruits</td>
<td>Neuro exam, carotid US, CT/MRI</td>
</tr>
</tbody>
</table>


**Anesthetic Technique/Induction**
A general or regional anesthetic may be appropriate and needs to be tailored to each individual pt and procedure. Spinal anesthetic is effective if expected operative times permit. Epidural allows easier control of duration of block and postop analgesia but postop anti-coagulation plans must be considered.

If a general anesthetic is chosen then hemodynamic stability at induction is important 2° to co-existing CAD, Htn, and CHF.

**Surgical Stages**

- **Dissection**
  - Often a very stimulating procedure at beginning due to long incisions and dissection but maintenance period may have much lower anesthetic requirements.
  - Heparin usually given just prior to arterial clamping

- **Arterial Clamping**
  - Clamping of artery, unlike more proximal arterial clamping, usually has little effect on overall BP or cardiac afterload.
  - Blood loss usually well controlled, however, can be difficult to detect or quantify 2° to distant surgical sites and loss into absorbent drapes.
  - Important to ensure adequate normovolemia, Hct and pt temp before a cold, ischemic leg is unclamped.

- **Arterial Unclamping**
  - Hypotension may occur when reperfusion of the ischemic limb allows venous return of acidic, hyperkalemic blood and various metabolic waste products.
  - Possible hyperkalemia with reperfusion is esp. a concern in the settings of renal failure or extremely long ischemic time.
  - Risk of increased blood loss at Anastomosis sites at time of reperfusion.
  - Heparin reversal with protamine creates chance of significant allergic reaction
  - Preparation for unclamping generally involves brief increase in minute ventilation, increase percentage O2 delivered and ready availability of inotropic/pressor support.

**Postoperative Considerations**
- Important to continue tight BP and HR control in postop period as this is time most cardiac complications occur; risk peaking in postop d 2–3.
- Shivering after anesthesia 2° to hypothermia or as side effect of anesthesia should be prevented or treated to avoid increased metabolic demands on the cardiac system.
- Post-extubation pulm status may be compromised 2° to COPD and/or atelectasis resulting from prolonged GA.
• Disposition to ICU versus ward is highly dependent on need for graft/limb monitoring, and on preop cardiac and pulm status. Pt may require ICU or step-down unit care to monitor for cardiac ischemia or arrhythmias.

**Anticipated Problems/Concerns**

- Induction of general anesthetic or neuroaxial blockade may be assoc with profound hypotension 2° to a relative volume contraction due to chronic Htn or as an interaction of antihypertensive medications (e.g., ACE inhibitors, beta blockers) with the anesthetized state.

- Rebound tachycardia 2° to beta-blocker withdrawal must be avoided through peri- and intraop use of beta-blockers. However caution is advised as the POISE trial has recently demonstrated that acute high doses of beta-blockers may increase risk of stroke and death.
Carcinoid, Excision of

Risk
- Incidence: 1.5 cases/100,000; 1/300 appendectomies; 1/2500 proctoscopic exams
- Race and gender predominance: None
- Age with highest incidence: Fourth and fifth decades

Perioperative Risks
- The presence of carcinoid heart disease and high urinary 5-HIAA levels are significant risk factors for periop complications incl death
- 1.5–10% periop mortality reported before use of somatostatin analogue

Worry About
- CV: Carcinoid crisis (CV collapse 2/2 release of vasoactive mediators)
- Respiratory: Bronchospasms. Bronchial tumor leads to compression and/or obstruction of bronchi.
- Cardiac: Right heart involvement leads to tricuspid insufficiency or pulmonic valve stenosis.
- Autonomic: Stimulation (pain, hypercarbia, hyperthermia) or sympathoinnervation leading to tumor mediator release and carcinoid crisis
- GI: Increased motility and secretion of H₂O, NaCl, and K⁺ leading to fluid and electrolyte abn. Midgut (ileal, jejunal) carcinoids leading to fibrosis of mesentery. GI tumor leading to compression and/or obstruction of small bowel.

Overview
Pathophysiology
- Slow-growing malignancies capable of metastases. Derived from APUD cells of embryonic neuroectoderm capable of synthesizing and secreting peptide hormones and monoamines, most commonly serotonin (5-HT). Other neurohumoral agents involved in carcinoid syndrome incl dopamine, histamine, bradykinin, kallikrein, prostaglandins, gastrin, corticotropin, neuron-specific enolase, substance P, neurotensin, vasoactive intestinal peptide, and somatostatin.
- 75% originate in GI tract (from esophagus to rectum). Most common site is the appendix (rarely metastasize or produce carcinoid syndrome). Tumors in ileocecal region have highest incidence of metastases. Carcinoid tumors have also been reported to arise from lung, head and neck, gonads, breasts, and urinary tract. The liver is the most common site for metastases.
- Carcinoid syndrome is usually assoc with ileal carcinoid tumors that have metastasized to the liver (hypothesized that metastasis impair hepatic metabolism of mediators released by the tumor) or from a 1° tumor draining into the systemic circulation.
- 20–40% of pts with carcinoid syndrome have carcinoid heart disease. Vasoactive substances released by the tumor cause fibrosis of the heart valves leading to pulmonic stenosis, tricuspid insufficiency, and/or myocardial plaque forma. (Cardiac involvement is usually right-sided because mediators are cleared or inactivated in the lungs before reaching the left side of heart.)

Presentation
- Pts with nonfunctional tumors (or whose tumor mediators are inactivated by the liver) present with pain, GI bleeding, or intestinal obstruction
- Pts with carcinoid syndrome present with flushing 74%, intestinal hypermotility 68%, cardiac symptoms 41%, and wheezing 18%. Only 7% of pts with a carcinoid tumor have carcinoid syndrome.

ASSESSMENT POINTS

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</tr>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Carcinoid crisis, right heart involvement, hemodynamic instability (usually hypotension), 5-HT has indirect positive chronotropic and inotropic effects (mediated by norepinephrine)</td>
<td>Hx carcinoid syndrome, fatigue, ascites, edema</td>
<td>Hemodynamic collapse; JVD, murmur, pulse</td>
<td>ABGs</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchospasms, bronchial obstruction</td>
<td>SOB</td>
<td>Wheeling</td>
<td>O₂ sat</td>
</tr>
<tr>
<td>GI</td>
<td>Diabetes +/- electrolyte disturbances</td>
<td>Abd pain, wt loss</td>
<td>Flushing</td>
<td>Ca²⁺, PO₄³⁻, prolactin, gastrin, blood glucose</td>
</tr>
<tr>
<td>ENDO</td>
<td>10% MEN (hyperplasia of parathyroid, pancreas, pituitary) hyperglycemia</td>
<td>Ulcers, renal calculi</td>
<td>Lipomas</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Postop sedation (2/2 5-HT)</td>
<td>Diarrhea</td>
<td>Dermatitis, dementia</td>
<td>Serum protein</td>
</tr>
</tbody>
</table>


Operative Management

Preoperative Preparation
- Evaluate for carcinoid syndrome and the extent of assoc multisystem disease: Urinary 5-HIAA (5-HT metabolite), imagining of 1° tumor and metastases (CT, MRI, abd US, barium GI x-ray films, radionuclide scans) echo to evaluate for carcinoid heart disease, CXR or broncoscopy for suspected bronchial carcinoids
- Correct CV instability, volume depletion, bronchospsam, hyperglycemia, and hypoproteinemia
- Premedication (aimed at minimizing tumor mediator release or activity): First line octreotide 100 µg SC (peak level in 30 min and half-life of 100 min) followed by octreotide infusion 50 µg/hr or 25–50 µg bolus prn and before tumor manipulation. Second-line drugs incl H1 & H2 receptor blockers, aprotinin, ketanserin, cyproheptadine. Consider endocarditis prophylaxis for pts with significant carcinoid heart disease.
- Avoid triggers of mediator release (anxiety, pain, hypoxia, hypercarbia, tumor compres) and drugs that cause histamine or catecholamine release.

Monitoring
- Arterial catheter
- Consider CVP cath for vol monitoring ( substitue PA cath or TEE if valvular lesions)

Anesthesia Technique
- Induction/maintenance: Etomidate or propofol appropriate for induction, maintenance with volatile agent (isoflurane)
- Paralysis: Caution with succinylcholine (may cause fasciculation of abd wall leading to mechanical compression of tumor and release of vasoactive peptides). Succinylcholine, atracurium, cisatracurium, and mivacurium stimulate the release of histamine.
- Analgesia: First line is fentanyl. Caution with morphine and meperidine, which stimulate the release of histamine, and light sedation, which increases the sympathetic response.
- Regional: With a neuraxial regional anesthetic, hypotension and reflex sympathetic stimulation can be minimized using titratable techniques such as epidural rather than spinal anesthesia.
• Carcinoid crisis:
  * Stop manipulation of tumor, 100% O₂,
  reduce concentration of volatile anesthetics if
  hypotensive, restore intravascular volume, admin-
  ister drugs to decrease mediator release and effect
  * Rx: First line is octreotide, which is effective
    in controlling flushing, hypotension, diarrhea,
    and bronchospasm in 75% of pts (80% of tumors
    have somatostatin receptors.) Second-line thera-
    pos incl drugs with rapid onset and brief clinical
effect for hemodynamic instability (phenolamine,
esmolol, and phenylephrine) vasopressin and
angiotensin for refractory hypotension, ketanserin
(a 5-HT antagonist for H₃m) H₁ & H₂ blockers,
and aprotinin (inhibitor of kallikrein)
• Avoid direct sympathomimetics (epineph-
    rine, norepinephrine), drugs that cause reflex
sympathetic stimulation 2/2 peripheral vasodilata-
tion (nitroprusside) and histamine-releasing drugs
(thiopental, succinylcholine, atracurium, cis-
tracurium, mivacurium, morphine, meperidine)
• Bronchospasm
  * Usually responds to octreotide. Use cor-
ticosteroids, inhaled ipratropium bromide, and
antihistamines as adjuvants.
  * Avoid beta-2 agonists, theophylline, and epi-
nephrine, which can exacerbate bronchospasm.

Surgical Stages
• Skin incision-tumor resection
  * Sympathetic and/or mechanical stimulation
    incl skin preparation, incision, and manipula-
tion of tumor may release vasoactive substances lead-
ing to carcinoid crisis.
  * Dissection may be extensive if small-bowel
    fibrosis has occurred.
  * En bloc resection of ileal carcinoids justified
due to frequent presence of multiple tumors

Closure and Postoperative Considerations
• About one third of pts with advanced (hepatic)
disease require blood transfusions intraop
• Postop PCA usual
• SQ octreotide resumed with IV supplement
  for hypotensive episodes
• Pain score depends on location: abd 4–7

Anticipated Problems/Concerns
• Carcinoid crisis can result in abrupt CV col-
lapse or severe bronchospasm
• When correcting for low cardiac output 2/2
  tricuspid insufficiency, avoid drugs that increase
  pulm vascular resistance such as vasopressin and
  angiotensin. Acidosis, hypercapnia, and hypo-
thermia can also increase pulm vascular resistance
and should be avoided.
Cardiopulmonary Bypass (CPB)

Veronica A. Matei

Risk
• Over 2,000,000 CPB procedures are performed annually worldwide.

Perioperative Risks
• Dependent on pt preop status, emergent institution of CPB, duration of CPB
• Cardiac, renal, resp, and CNS complications contribute to CPB morbidity.
• Variable degrees of renal and neurologic deficits are common post-CPB. Incidence of stroke is 5%. Incidence of acute renal failure requiring dialysis is 4%.

Worry About
• Potential catastrophic events during CPB: Inadequate coagulation with clotting of CPB machine components, circuit disconnection with massive air embolism and/or exsanguination

ASSESSMENT POINTS

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<tbody>
<tr>
<td>CARDS</td>
<td>Severity of cardiac disease dictates pre/post CPB management</td>
<td>Angina, CHE, arrhythmias, exercise tolerance</td>
<td>Cardiac</td>
<td>ECG, ECHO, cardiac cath</td>
</tr>
<tr>
<td>RESP</td>
<td>Prior resp dysfunction ↑ risk of “pump lungs”</td>
<td>Smoking Hx, dyspnea</td>
<td>Resp</td>
<td>CXR, PFTs, ABG</td>
</tr>
<tr>
<td>ENDO</td>
<td>CPB-induced hormonal stress response</td>
<td>Diabetes</td>
<td></td>
<td>Blood glucose</td>
</tr>
<tr>
<td>HEME</td>
<td>Prior anemia, plt dysfunction or coagulopathy ↑ chance for blood products transfusion pre/post CPB</td>
<td>Hx of bleeding disorders, antplatelet or anticoagulant medication use</td>
<td>Ht, PT/INR, PTT, plt count</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Prior renal dysfunction ↑ risk of renal complications post CPB</td>
<td>Hx of renal dysfunction, diuretic use</td>
<td>BUN, Cr, UA, FeNa</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Prior CVA/TIA ↑ risk of neurologic complications post CPB</td>
<td>Hx of CVA, TIA</td>
<td>Neuro exam</td>
<td>Carotid US</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
• Assess co-existing morbidities and define anesthetic goals for individual cases.

Monitoring
• Standard ASA monitors (temp is monitored at multiple simultaneous sites)
• Invasive arterial pressure monitoring essential
• Large-bore vascular access mandatory. Central vascular access preferred for administration of vasoactive drugs.
• Laboratory parameters: Blood gases, Hct, serum potassium, ionized calcium, glucose measurement
• Adequacy of anticoagulation (ACT goal >400 sec)
• CVP, PAC if ventricular function compromised (LVEF <35%) or pulm Htn
• Cardiac output (via PAC or TEE), O2 (goal >1 mL/kg/hr)
• Intraoperative TEE
• Additional monitoring during CPB: Pump flow rate, venous reservoir level, arterial inflow line pressure, blood and myocardial temp, in-line (arterial and venous) O2, sat
• Surgical field monitoring essential

Anesthetic Technique/Induction
• All variations of carefully conducted anesthesia techniques can be employed.

Surgical Stages
• Skin incision, sternotomy, and pericardectomy are stages of intense stimulation. Titration of anesthetic agents is necessary to avoid tachycardia and Htn

• Communication among surgeons, perfusionist, and anesthesiologist is of vital importance.

Overview
• CPB technology temporarily replaces cardiac and pulmonary functions during surgery. CPB is a form of extracorporeal circulation.
• Full CPB requires an oxygenator and a blood pump. Systemic venous blood is drained (via venous cannula) to a venous reservoir. From the reservoir, the blood is pumped through an oxygenator and then returned (via arterial cannula) to the arterial system.
• Partial CPB requires a blood pump that supports only a portion of the body (usually the infra-diaphragmatic portion). The oxygenation can be accomplished by a mechanical oxygenator (femoral vein to femoral artery partial CPB) or by pt’s lungs (left atrium to femoral artery partial CPB)

ICD-9-CM Code: 39.61

Indications
• Heart and great vessels surgery: CABG, valve surgery, heart transplant, congenital heart defects surgery, repair of aortic or cerebral aneurysms, removal of intracardiac and great vessel masses, pulm thromboendarterectomy
• Treatment of severe hypothermia
• Local anesthetic toxicity (bupivicaine)

PROCEDURES

Anticipated Problems/Concerns
• Re-do surgery can result in massive bleeding during sternotomy. In some cases, initiation of CPB (via femoral-femoral cannulation) is achieved before sternotomy.
• Heparin is an essential drug for CPB. For patient with Hx of HIT, alternative drugs are used for systemic anticoagulation.
• Controversies incl the use of pulsatile vs. non-pulsatile flow during CPB, acceptable arterial pressure during CPB, blood gas management (alpha-stat vs. pH-stat), need for routine use of PAVs, BIS monitoring, optimal site for temp monitoring.
Cardioversion

**Overview**

Brief procedure where either electrically or chemically an attempt is made to convert an abn heart rhythm to a normal rhythm. Electrical cardioversion is usually done for atrial fibrillation; however it can also be done for a flutter, supraventricular tachycardia, and ventricular tachycardia.

Cardioversion from atrial flutter and v. tach usually requires a small amount of energy. Atrial fibrillation and ventricular fibrillation usually require a larger number of muscle fibers to be depolarized and thus a larger amount of energy.

For supraventricular arrhythmias, cardioversion should be synchronized and occur during the R wave of the QRS cycle to prevent depolarization during the vulnerable period of the ventricle which could lead to a more unstable arrhythmia. Defibrillators currently used typically deliver energy in a variety of waveforms generally classified as monophasic or biphasic. Biphasic energy delivery requires lower energies and is assoc with greater first shock efficacy as compared with monophasic defibrillation. Monophasic defibrillation however is still in common use and quite effective and currently there are no data to suggest a clear clinical outcomes benefit of one over the other.

**ICD-9-CM Code:** 427.31 (AFib)

**Indications and Usual Treatment**

- AFib
- Cardioversion from AFib to NSR does not reduce the pt’s risk for stroke but can improve the pt’s symptoms. May also be necessary for acute rate control when rate or rhythm control medications have not succeeded. Particularly true if assoc with hemodynamic instability.
- In general, for AFib of duration less than 48 hrs, the risk of embolism with cardioversion is low (<1%) and cardioversion may be performed without anticoagulation. For AFib that has lasted longer than 48 hrs; 3 wk of anticoagulation with or without a consideration of TEE is recommended prior to cardioversion. TEE may also be used to assess for the presence of left atrial thrombus as an alternative to 3 wk of anticoagulation.

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hemodynamic instability</td>
<td>Cardiac symptoms functional status wt fluctuations</td>
<td>Heart and lung exam murmur, S3/4, edema venous stasis</td>
<td>ECG Consider ECHO</td>
</tr>
<tr>
<td>CHF Acute ischemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>↓ Pulm function OSA</td>
<td>Exercise tolerance Recent infection Tobacco use</td>
<td>Wheezes Crackles Rales</td>
<td>Room air SpO₂ Blood gas</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Potential ischemic/embolic stroke</td>
<td>Functional status TIA/CVA</td>
<td>Neuro exam document deficits</td>
<td>Possible head CT/MRI</td>
</tr>
</tbody>
</table>


**Anesthetic Considerations**

- Often a small bolus of IV sedative or a brief general anesthetic (GA) is employed to provide pt comfort and tolerance of the procedure. Each treatment and medicine choice carries its own relative risk/benefits profile and should be selected accordingly. The duration necessary for sedation/esthesia will vary based on whether TEE is employed and number of defibrillation attempts required.
- An important initial treatment consideration is whether a GA with or without intubation is necessary. The procedure is typically brief and induction medications are titrated to loss of consciousness or deep sedation. Intubation may be necessary due to aspiration risk. Pts with a Hx of or risk factors for a difficult intubation or BMV should be approached with caution and appropriate rescue equipment should be available.

**Induction Agents**

- Propofol: Short duration, anti-emetic
- Etomidate: Hemodynamic stable
- Ketamine: Some pain control, potential for continued spontaneous respirations
- Benzodiazepines: Amnesia

**Some Advantages of Selected Induction Agents**

- Ketamine: Myocardial ischemia, tachycardia, increased ICP
- Benzodiazepines, prolonged sedation

**Some Disadvantages of Selected Induction Agents**

- Propofol: Hypotension
- Etomidate: Myoclonus (making ECG reading difficult), adrenal suppression

**Postoperative Management**

- Typically minimal, possible recall and/or pain etc. (see Periop Risk).
- Observation until awake and stable (institutional variation on postop management)

**Anticipated Problems/Concerns**

- CNS event: Immediate CNS evaluation necessary, recall possible, pain
- Cardiac: Possible myocardial ischemia, Htn, tachycardia, arrhythmia, hypotension
- Pulm: Acute pulm edema, airway difficulties, aspiration
- Dermatologic: Thermal injury
## Carotid Endarterectomy

### Risk
- Incidence in USA: About 100,000 CEAs performed annually
- Risk factors for carotid disease: Tobacco, DM, HTm, age, male sex, hypercholesterolemia, hyperlipidemia, obesity, family HS
- Prevalence of significant carotid stenosis (60-99%) is 1%. TIA the most common presentation
- Annual stroke rate is about 2% for asymptomatic and 13% for symptomatic carotid stenosis
- Ulcerated plaque presents an increased risk of stroke independent of the degree of stenosis

### Perioperative Risks
- Main periop risks are death, stroke and MI. All are higher in symptomatic pts.
- Mortality from 0.3–1%
- Stroke from 0.8–2.7%
- MI from 1.7 to 4.3%
- Independent risks for postop mortality: TIA/stroke, age, CHF, obesity, CRF

### Worry About
- CNS status: Asymptomatic versus symptomatic
- Degree of carotid stenosis and status of collateral/contralateral circulation
- Co-morbidities: CAD, HTm, COPD, renal disease
- Pt cooperativity for an awake procedure

### Postop: Monitored care, CNS assessment, BP control, hematoma/airway, nerve injury

### Overview
- Carotid endarterectomy is a stroke-preventative surgery.
- Risk of cerebral ischemia during surgery due to embolism or hyperperfusion
- Risk of postop hyperperfusion cerebral injury
- Possibility of persistent postop cognitive decline
- Intermediate cardiac risk surgery, as per ACC/AHA 2007 Guidelines
- Periop mortality highest from cardiac event, followed by cerebrovascular event
- Anesthetic techniques incl general (GA) and regional anesthesia (RA)
- RA does not seem to improve cardiac and CNS outcomes or hospital stay, but may be assoc with more hemodynamic stability and lower vasopresor requirements
- Periop ASA and statins are CNS protective; beta-blockers per ACC/AHA Guidelines.
- ICD-9-CM Code: 443.1 (Arteriosclerosis of carotid artery)
- ICD-9-CM Code: 38.12 (Carotid endarterectomy)

### Indications
- Indications are based on the degree of stenosis and symptoms.
- Symptomatic pts (RIND, TIA, or stroke) benefit from CEA when carotid stenosis is >70%.
- In stenosis of >50% risk reduction is lower. Maximum benefit is achieved when surgery is performed within 2 wk from the onset of symptoms.
- Asymptomatic pts: Carotid stenosis >60% in men. No benefit has been shown in asymptomatic women because of the high periop risk.
- Surgery is beneficial if stroke rates of the perip team are lower than 3% for asymptomatic pts, 5% for TIA and 7% for stroke pts.

### Usual Treatments
- Surgical plaque removal by longitudinal or evasion endarterectomy, the former with or without a patch. A temporary shunt can be placed between the common and internal carotid artery to prevent and/or treat cerebral hyperperfusion during the carotid artery clamp.
- An alternative to surgery is endovascular angioplasty/stenting. Approx 7000 endovascular procedures are performed annually in the USA.

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<tbody>
<tr>
<td>CARDO</td>
<td>CAD, Htn, CHF, PVD Renovascular disease</td>
<td>Chest pain, MI, CHF, SOB, dyspnea, exercise tolerance</td>
<td>HR, BP both arms; S or S, murmurs, dysrhythmia, bruits</td>
<td>ECG, ECHO stress test</td>
</tr>
<tr>
<td>RESP</td>
<td>Tobacco use COPD, OSA</td>
<td>SOB, cough, exercise tolerance, Orthopnea, use of CPAP, O2</td>
<td>Decreased breath sounds, wheezing</td>
<td>CXR, PFTs</td>
</tr>
<tr>
<td>ENDO</td>
<td>DM, obesity</td>
<td>Diet, PO meds or insulin control Duration, effectiveness of treatment</td>
<td>Wt, BMI Peripheral neuropathy</td>
<td>Serum glucose HbA1c</td>
</tr>
<tr>
<td>CNS</td>
<td>TIA, RIND, stroke</td>
<td>Tinnitus, dizziness, vision changes, weakness, slurred speech, paralysis</td>
<td>Motor, visual, or speech</td>
<td>US, CT, EEG Angio/MRI</td>
</tr>
</tbody>
</table>

### Key Reference

### Intraoperative Management

#### Monitoring
- Standard ASA monitors. ECG incl leads II and V5.
- Arterial line for continuous BP and blood sampling (ABGs, glucose, etc.)
- CNS function
  - Regional anesthesia: Mentation, speech, motor function
  - General anesthesia: EEG (raw or processed), SSEP, transcranial cerebral oximetry (rSO2), transcranial Doppler (TCD), jugular venous oximetry
  - Monitoring modalities have similar sensitivities and specificities. rSO2 is gaining favor because of the ease of use and low failure rate. TCD differentiates hyperperfusion from embolic neurologic events, but is technically not feasible in 15–20%.
  - There is no consensus on multimodal monitoring strategies.
  - Stump pressure helps with the decision whether to shunt; cannot be monitored continuously.

#### CNS Protection
- Glycemic control
- Maintain normocarbia. Consider FI02 of 100% if neurologic deterioration.
- BP management: Preop ABP control, optimally <180/110 mmHg; intraop at baseline; during clamp most clinicians increase ABP by 20% or higher, guided by CNS monitoring; postop at baseline and/or normotensive, to avoid hyperperfusion injury.

#### Anticipated Problems/Concerns
- Periop TIA or stroke
- Unstable BP and myocardial ischemia
- Hyperperfusion syndrome with Htn, esp. after repair of high-grade stenosis
- CNS deficit (ischemia, emboli, intimal flap, thrombosis, etc.)
- Hematoma with possible airway compromise
- Cranial n. injury (XII, recurrent, etc.) or persistent phrenic n. block from regional anesthesia

### Surgical Stages
- Intraop considerations
- Dissection: Concern is embolization from surgical manipulation. Blood loss usually not a concern.

### Anticipated Problems/Concerns
- Cross-clamping: Follows heparinization (ACT>250s). Assess adequacy of cerebral perfusion if awake or by CNS monitors if asleep. Shunt, FIO2, and BP control accordingly.
- Unclamping and reperfusion: Avoid Htn. Continue neurologic assessment.
- Closure: Ideally heparin should not be reversed Postoperative Considerations
- Altered BP control and occasional bradycardia
- Hyperperfusion syndrome with Htn, esp. after repair of high-grade stenosis
- CNS deficit (ischemia, emboli, intimal flap, thrombosis, etc.)
- Hematoma with possible airway compromise
- Cranial n. injury (XII, recurrent, etc.) or persistent phrenic n. block from regional anesthesia

### Anticipated Problems/Concerns
- Periop TIA or stroke
- Unstable BP and myocardial ischemia
- Hyperperfusion from upper airway obstruction or phrenic nerve paralysis
- Difficult neurologic assessment in oversedated awake pts or after general anesthesia
- Rare but potentially challenging urgent conversion from regional to general anesthesia
Carpal Tunnel Syndrome

**Overview**
- Accounts for approx 90% of all entrapment neuropathies
- Due to entrapment of the median nerve in the carpal tunnel at the wrist
- Most common symptom is burning pain associated with tingling and numbness in the distribution of the median nerve distal to the wrist
- Symptoms often first appear during the night (nocturnal paresthesia), since many people sleep with flexed wrists
- Dx should be based on Hx, physical exam, and results of electrophysiological studies
- Gold standard test is nerve conduction studies
- Mild symptoms can be managed with conservative treatment, incl steroid injections
- Moderate to severe symptoms are treated via surgical intervention.

**ICD-9-CM Code**: 354.0

**Etiology**
- Two distinct types: Acute and chronic
- Acute type is uncommon and is due to rapid and sustained pressure in the carpal tunnel
- Acute type is assoc with burns, coagulopathy, fractures, infection, and injections
- Chronic type is common and slow in progression

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effects</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEURO</td>
<td>Pain with activity, sensory deficits, and motor deficits</td>
<td>Paresthesias, weakness, Hx of dropping objects, nocturnal sensory abn</td>
<td>Diminished pinprick, decreased hand grip, Thenar atrophy, Tinel's sign and Phalen's test</td>
<td>Nerve conduction study, US</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Evaluate for and/or optimize underlying causes such as diabetes, hypothyroidism, etc.
- Optimize volume status in order to decrease edema (diuretics, dialysis, etc.).
- Consider Hx of corticosteroid usage and NSAIDs prior to surgical intervention.
- Document musculoskeletal and neurologic exam prior to performing regional anesthetic.

**Monitoring**
- Standard ASA monitoring

**Anesthetic Technique/Induction**
- Goal: To maintain a bloodless operative field without anatomical distortion
- Options incl:
  - Local anesthetic infiltration
  - Advantages: Simple, economical, rapid onset, no motor block, good pt satisfaction
- Disadvantages: Tourniquet pain, anatomical distortion
  - IV regional infiltration
  - Advantages: Simple, rapid onset
  - Disadvantages: Short post-block analgesia, local anesthetic toxicity, motor block
  - Peripheral nerve block

- Advantages: Tourniquet tolerance, no anatomical distortion, good postop analgesia
- Disadvantages: Time consuming, technically difficult
- General anesthesia
- Advantages: Rapid anesthesia, no anatomical distortion, no tourniquet intolerance
- Disadvantages: Postsurgical pain, dizziness, nausea, and vomiting

**Surgical Stages**
- Open carpal tunnel release surgery is easy to perform and leads to symptomatic relief in most pts. It is performed by making a curved longitudinal inter-osseous incision approx 4–5 cm in length. The subcutaneous tissue, superficial fascia, transverse carpal ligament, and 2–3 cm of distal forearm fascia is opened under direct vision. The canal is then inspected for mass lesions or anatomical abn.
- Endoscopic carpal tunnel release can be broadly divided into single portal and dual portal techniques depending on the number of ports used to access the carpal tunnel. A ½-inch incision is made in the wrist and palm through which a camera is inserted through a tube. The camera enables the surgeon to indirectly visualize and release the transverse carpal ligament.

**Anticipated Problems/Concerns**
- Early post-surgical complications: Incomplete release of the transverse carpal ligament, neuropraxia or injury to median or ulnar nerve, inadvertent entry to Guyon's canal (tunnel between pisiform and hamate bone and the ligament connecting both bones), injury to the palmar cutaneous or recurrent motor branch of the median nerve, and injury to the superficial palmar arch or ulnar artery
- Late post-surgical complications: Scar tenderness, loss of grip strength, pillar pain (tenderness in the thenar or hypothenar eminence), complex regional pain syndrome, and bow stringing of flexor tendons

**Risk**
- M:F ratio: 1:3
- Incidence: 0.125%–1%
- Peak incidence around 55 to 60 y old
- Classically involves the thumb, index and middle fingers, and radial half of the ring finger
- Often bilateral, but dominant hand is involved initially
- Common causes incl occupational trauma, congenital predisposition, pregnancy, diabetes, and obesity

**Perioperative Risks**
- Burning, tingling, and/or numbness in the distribution of the median nerve distal to the wrist
- Loss of coordination and weakness in the affected hand
- Exacerbated with activity or work

**Worry About**
- Assoc with conditions with higher risk anesthetic implications incl gout, rheumatoid arthritis, diabetes, obesity, pregnancy, lupus, renal failure, hemodialysis, alcoholism, and acromegaly
- More susceptible to compression with edema (fluid retention) and sustained wrist flexion
- Sensory and/or motor deficits

- Chronic type is assoc with local (infection, trauma, tumors), regional (arthritis, gout), or systemic causes (pregnancy, diabetes, obesity, renal failure, etc).

**Usual Treatment**
- Nonsurgical methods effective in mild to moderate carpal tunnel syndrome, i.e., pts with no muscle weakness or atrophy, absent denervation with EMG, and only mild abn on nerve conduction studies
- Nonsurgical treatments incl: Splinting of the wrist, hand brace, rest, ice, NSAIDs, local injections with corticosteroids, vitamin B6 (pyridoxine), US therapy, yoga, and workplace modifications
- Surgery is indicated in almost all pts with moderate to severe carpal tunnel syndrome
- An absolute indication for surgery is muscular atrophy
- Surgery consists of division of the transverse carpal ligament, which releases pressure on the median nerve by increasing the volume of the carpal tunnel
- Two different surgical approaches: Open and endoscopic release

- The Cochrane database group reviewed all available evidence comparing various alternative surgical techniques and found no strong evidence to favor endoscopic repair against the open technique. There was no conclusive evidence favoring either technique when assessing a pt’s symptom relief and return to work.
Cataract ± lol

**Risk**
- Incidence in USA: >1.5 million cataract operations/y
- Gender predominance: None
- Advanced age
- Direct trauma
- Response to other intraocular conditions, incl chronic uveitis, glaucoma, retinal detachment
- Systemic diseases (diabetes mellitus, myotonic dystrophy, galactosemia)
- Chronic use of topical or systemic corticosteroids
- Congenital (idiopathic, familial, assoc with pre-natal infection)

**Perioperative Risks**
- Periop mortality exceedingly rare
- Surgical morbidity: Bleeding into anterior chamber; capsule rupture; posterior dislocation of lens into degenerative vitreous; loss of vitreous, producing retinal detachment and macular edema; expulsive hemorrhage

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>OCULAR</td>
<td>Determination of lens power of intraocular implant</td>
<td></td>
<td></td>
<td>Ultrasonic measurement of axial length of eye; optical measurement of corneal curvature</td>
</tr>
<tr>
<td>CARDIOPULM</td>
<td>Impaired ability to lie flat; chronic coughing; SOB, orthopnea</td>
<td>Inspection, auscultation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Impaired ability to follow instructions and remain motionless because of age, anxiety, claustrophobia, deafness, tremors</td>
<td>CNS Hx</td>
<td>CNS exam</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- None

**Anesthetic Technique**
- Can be performed under regional (peribulbar or retrobulbar), general, sub-tenon’s, or topical anesthesia

**Monitoring**
- Routine
- Invasive monitoring seldom indicated

**Regional Techniques**
- Eye in neutral gaze to minimize risk of optic nerve injury
- Small-gauge needle, no longer than 31 mm (1¼ inch) to ↓ risk of globe perforation
- Consider general or topical anesthesia if high risk (e.g., extreme myopia; severe enophthalmos; staphyloma; previous ocular complications of regional anesthesia; severe vascular disease; bleeding diathesis; one-eyed pt) for complications assoc with retro- or peribulbar block
- Avoid deep orbital penetration
- Avoid heavy sedation

**Worry About**
- Anesthetic morbidity following retrobulbar block
  - Retrobulbar hemorrhage (1–3%)
  - Perforation of globe (0.1% or less)
  - Central spread of local anesthesia that may affect brainstem (0.1%)
  - Intra-arterial injection with immediate seizures (<0.1%)
  - Optic nerve injury (<0.1%)

**Overview**
- Removal of cloudy lens with small incision(s), with aspiration or ultrasonic fragmentation
- Assoc with extremely low mortality, although complications of retrobulbar block (e.g., brainstem anesthesia) can be life threatening
- Morbidity can incl blindness in operated eye

**General Anesthesia**
- Meticulously secure endotracheal tube or laryngeal mask airway to prevent intraop extubation
- NM paralysis with appropriate monitoring to avoid coughing or bucking that can cause loss of intraocular contents
- Consider prophylactic antiemetic

**Surgical Stages**
- The vast majority of cataract procedures are extracapsular because an intact posterior capsule may reduce posterior segment complications: retinal tear, retinal detachment, macular edema
- Incision for extracapsular procedure: With the pupil fully dilated, an anterior capsulotomy is performed
- Definitive Surgery: Central anterior capsule removed, with irrigation and aspiration of lens nucleus through wound. Alternatively, nucleus may be fragmented ultrasonically (phacoemulsification) behind iris plane to avoid corneal epithelial damage. Phacoemulsification is generally considered the preferred technique.
- Incision partially closed and every precaution taken to maintain normal anterior chamber depth (by air infusion, fluid infusion, or injection of viscoelastic solution) to prevent corneal endothelial damage during intraocular lens insertion
- Supporting loops of posterior chamber lens inserted into capsular bag or ciliary sulcus
- Minimal hemodynamic disturbance
- Approx duration: 1 hr or less

**Postoperative Considerations**
- EBL: Negligible
- Minimal postop pain
- Pts instructed to avoid bending, lifting, straining

**Anticipated Problems/Concerns**
- Prosis
- Postop wound dehiscence
- Iris prolapse
- Infectious endophthalmitis
- Retinal tear or detachment
- Cystoid macular edema
- Delayed posterior capsule opacification

**Indications and Usual Treatment**
- Based on degree of visual impairment in relation to visual needs of the individual, as well as anticipated visual improvement and risk of serious complications
- With congenital cataracts, risk of amblyopia dictates that surgery be performed within the first few months of life

**PROCEDURES**

**ICD-9-CM Code**: 366.9
Cerebral AVM Repair

Risk
- <2000–3000/y
- More commonly diagnosed in pts age 20–40 y
- Risk of intracranial hemorrhage is 2–4%/y; 7–33% incidence of rebleeding
- Mortality 1%/y; 10–15% after first bleed
- Racial predominance: None
- Risk of hemorrhage is increased with male gender, size and location of lesion

Perioperative Risks
- Preop embolization decreases bleeding, facilitates resection, and decreases hyperemic complications
- 30-d operative morbidity 20–40%; worse with higher clinical grade
- 30-d mortality 6–9%
- Unique complication: Normal perfusion pressure breakthrough (NPPB) syndrome; overall risk 1–18%; at highest risk are high-flow lesions, border-zone AVM location, large AVMs (19–37% risk); lesions with severe hypoperfusion or steal around AVM; severe CNS deficit (may significantly improve over time)
- Postop CNS deficits may predispose to airway obstruction, aspiration

Worry About
- Blood availability
- Effect of preop embolization: New neuro deficit; bleeding; palm embolus
- Emergence: To allow early CNS assessment
- Massive brain swelling; cerebral hemorrhage
- High-dose barbiturate Rx to prevent edema, intracranial Htn
- Tight BP control on emergence, early postop

Overview
- Congenitally abn connections between arterial and venous cerebral circulation, without intervening capillaries; 80% are supratentorial
- Mass of thin-walled vessels with abn vasomotor response, deficient muscularis layer, dilated veins, and chronically ischemic surrounding brain tissue


ASSESSMENT POINTS

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hyperdynamic CHF in small children, FTT</td>
<td>Recurrent resp failure, prolonged ventilatory support</td>
<td>Auscultation, hepatomegaly</td>
<td>JVD, diaphoresis</td>
</tr>
<tr>
<td>RESP</td>
<td>May aspirate during seizure, hemorrhage</td>
<td>Review with family; records from ER, ICU</td>
<td>Auscultation</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Dehydration and/or mild renal insufficiency from multiple CNS imaging</td>
<td>Chart review</td>
<td></td>
<td>BUN, Cr</td>
</tr>
<tr>
<td>CNS</td>
<td>Seizures, chronic ischemia of brain surrounding AVM; hydrocephalus, intracranial Htn</td>
<td>LOC, headache, diplopia, nausea; family to describe Sx</td>
<td>MS</td>
<td>MRI, angio, clinical AVM grade: Size, location, drainage (deep vs. superficial)</td>
</tr>
</tbody>
</table>

Indications and Usual Treatment
- Stereotactic craniotomy for smaller lesions (<3 cm); scalp flap with craniotomy and surgical excision for larger lesions (>3 cm) if resectable with low risk of deficit; progressive Sx; refractory seizes; <40 y
- Alternate Rx: Stereotactic radiosurgery or embolization for older population, location in eloquent area, symptom control. Embolization is palliative, not curative.

Intraoperative Management

Preoperative Preparation
- Avoid premedication if mental status impaired or ICP high
- Determine risk of NPPB

Anesthetic Technique
- Requirements include brain relaxation, mildly decreased CPP; early emergence (or in pts at high risk for NPPB, high-dose barbiturates)
- Hyperventilation to Paco2 of 25–30 mmHg to improve regional distribution of CBF
- Avoid glucose-containing fluid.

Monitoring
- Routine
- Direct arterial pressure prior to induction, placing transducer at level of head to reflect cerebral BPs; CVP for larger, deeper AVMs; PA catheter, ECHO for CV compromise
- Consider EPs
- Consider EEG for barbiturate effect
- ICP for emergence and postop

Anticipated Problems/Concerns
- Rx for NPPB: Hyperventilation, diuretics, propofol, mild hypothermia, BP control

Surgical Stages

Induction
- Avoid succinylcholine with hemiplegia, increased ICP
- Maintain BP control; avoid coughing

Skeletal Fixation
- Avoid BP spike during fixation

Skin Incision
- Avoid Htn; may see effects of local anesthesia, epinephrine
- Consider beginning mannitol 0.5–2.0 g/kg

Dissection
- Arterial supply resected first; severe episodic bleeding, esp. if dural sinuses involved
- After resection, test for bleeding by normalizing BP
- Consider barbiturate infusion to prevent NPPB

Definitive Surgery
- May develop edema (NPPB) and hyperemia of surrounding tissue with occlusion of AVM
- Rx for NPPB: Hyperventilation, diuretics, propofol, mild hypothermia, BP control

Closure/Postoperative Period
- EBL: 300–2000 mL; depends on AVM size, location; no need for third-space allowance
- Careful BP control during emergence/postop period, consider fast-acting agents
- Ensure normal coag status
- Controlled emergence to minimize coughing/bucking, sympathetic response to pain
- Rx small doses of IV opioid, if CNS status OK

Anticipated Problems/Concerns
- Intraop concerns: Bleeding, brain volume control; CNS assessment at end of procedure
- Postop concerns: Brain swelling (NPPB); bleeding into surgical site; hydrocephalus, seizure

Logan S. Emory
Armin Schubert
Cervical Spine Fusion

Douglas Hester

Perioperative Management

Preoperative Preparation
- Sedation for either awake airway management or placement of neck traction prior to induction
- Usually routine monitors are sufficient
- Upper extremities inaccessible; consider needs for arterial catheter placement
- Large peripheral access sufficient; consider CVP and Doppler if pt will be in sitting position (VAE risk)
- Neurologic monitoring (SSEP, MEP)

Anesthetic Induction/Maintenance
- General endotracheal
- Monitoring may impact (e.g., SSEP might be optimized with narcotic-based technique)
- Stimulating parts of procedure incl: incision and initial dissection, retraction at neck, and bone graft harvesting (if done)

Airway Management
- Consider awake intubation
- Consider difficult airway equipment
- Airway will be difficult to access during case; consider wire reinforced ETT to avoid kinking/obstruction

Emergence
- Rapid emergence facilitates timely neurologic examination

Surgical Stages

Transoral Approach
- Pins, head brace, or Gardner-Wells tongs used to provide cervical traction (5–20 lbs)
- Dingman retractor used to facilitate access; soft palate also retracted via sutures; hard palate sometimes transected

Anterolateral Approach
- Neck is hyperextended; straps, brace or Gardner-Wells’ tongs used to provide cervical traction (5–20 lbs)
- Trachea, esophagus, carotid, and neural structures retracted to allow bone graft or plating

Posterior Approach
- Pt either prone or sitting (usually done in Mayfield pin system)

Postoperative Considerations
- Cervical collar often placed before emergence
- Airway edema
- Dysphagia
- Pain at autologous bone graft donor site

Anticipated Problems/Concerns
- Airway management (resp insufficiency, hematoma, recurrent laryngeal nerve injury)
- Dysphagia
- Pain at autologous bone graft donor site

Blood loss: Usually less than 500 mL, unless corpectomy planned (can be up to 1000 mL)

Indications And Usual Treatment
- Upper cervical spine: Atlantoaxial or occipitouatantal instability, odontoid fractures, congenital or neoplastic lesions; cervical instability, prior failed surgery, spinal tumor
- Mid and lower cervical spine: Herniated disks, spinal cord compression (myelopathy), cervical spondylosis, cervical radiculopathy (nerve-root compression), cervical instability; prior failed surgery, kyphotic deformities, spinal tumor

Cesarean Section, Emergent

**Risk**
- Incidence in USA: 30.3% of all deliveries
- 24.5% of all C-sections are performed for an emergent indication
- Racial predominance: Slightly non-Hispanic black predominance
- Obesity is a risk factor for both elective (odds ratio 1.87) and emergency (odds ratio 2.23) cesarean delivery

**Perioperative Risks**
- In 2003, anesthesia was the 7th leading cause of maternal mortality in the USA.
- Most maternal anesthesia related death occurs during emergent C-section.
- Most frequent cause: Failed airway management during or just after emergence from general anesthesia or related to intubation for high neuro-axial block.
- Nonanesthetic risks: Embolism, hypertensive disorders, bleeding

**Worry About**
- Difficult airway
- Rapid desaturation
- Full stomach if in labor or exposed to any opioids (even neuroaxial)

**ASSESSMENT POINTS**

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<tbody>
<tr>
<td>HEENT</td>
<td>Upper airway edema</td>
<td>Severe nasal congestion</td>
<td>MP – may increase by 1 class with labor</td>
<td></td>
</tr>
</tbody>
</table>
| CARDIO | Increased CO and total plasma volume, total blood volume=95ml/kg Biventricular hypertrophy, MR, TR, PI, Arrhythmias Aorto-caval compression | SOB, chest pain, peripheral edema, palpitation, irreguar heartbeats | Wide split S$_2$, possible S$_3$, 3/6 syst M of MR | ECG: Left axis deviation, S1Q3, inverted T in II and AVF, arrhythmias (SVT, nonsustain VT)
ECHO: MR, TR, PI, aortic dilation RVH = 20% LVH = 6% |
| RESP   | Hyperventilation, resp alkalosis, 20% increase in O$_2$ consumption (14) Decreased vertical size of the chest, decreased diameter | Barrel shape chest High position of the diaphragm | MV increased 50%, FRC decreased 20% Normal ABG: pH = 7.44, PaCO$_2$ = 32mm Hg, HCO$_3$ = 20 mmol/L, PaO$_2$=105mm Hg |
| OB     | Preeclampsia, eclampsia, HELLP syndrome | Headache, RUQ pain, blurry vision | Elevated BP, severe per edema, petechia, jaundice, papilledema, seizures | Elevated creatinine and uric acid, proteinuria, elevated LFTs, thrombocytopenia, prolonged PT, PTT |
| GI     | Decreased LES tone, poss. Increased acidity | Heartburn | Increased risk of aspiration of gastric content | |


**Perioperative Management**

**Preoperative Preparation**
- Administer oral antacid (sodium citrate 30 mL), if not contraindicated otherwise, IV H2-blockers (cimetidine), metoclopramide 10–20 mg
- Position the pt with left uterine displacement
- If pt is bleeding or has high risk of placenta accreta, use 2 large-bore IV

**Monitoring**
- Standard ASA monitoring, fetal HR monitoring, Foley catheter
- A-line if pt is bleeding or has high risk of placenta accreta
- CVP, PAP, BIS monitoring are rarely indicated. CVP or PA catheters—insertion of catheters in pregnant pts carries higher risk of complications.

**Anesthetic Technique/Induction**
- Modern U.S. data is unavailable.
- In United Kingdom, anesthetic technique for Grade I cesarean section
  - Primary GA: 35.5%
  - Converted to GA: 9%
  - Regional: 55.5%
  - In Singapore: 90% of all Grade I cesarean section is performed under GA

**Induction**
- Don’t induce until obstetrician is ready to make skin incision. Surgery can be started only after airway is secured. Instruct obstetrician to begin surgery after you say “cut”, do not use words like “begin”, “start”, or “lets go”; can be confused with command to start the induction.

**Medications**
- Ketamine: 1 mg/kg. Hallucinations and emergence phenomena are dose-related and less frequent in obstetric pts. Sympathomimetic effect limits its use in preeclamptic pts.
- Thiopental: 3–7 mg/kg. Doses <4 mg/kg unlikely to cause fetal depression, doses >7 mg/kg are liable to do so.
- Propofol: 2.5 mg/kg propofol may cause severe bradycardia in combination with succinylcholine at 1–1.5 mg/kg.
- Although pseudocholinesterase activity is decreased about 24% at term, it does not cause clinically significant prolongation of paralysis after one dose of succinylcholine. Fasciculation may be absent, esp. in pts on magnesium therapy.
- Vecuronium/rocuronium: Avoid, if necessary titrate to effect, start with 20% of standard dose), esp. for pre-ecclamptic pts on magnesium therapy or/and infected pts (chorioamnionitis) on gentamicin or clindamycin. Even defasciculating dose can cause total muscle paralysis.

**Extension of Preexisting Epidural Block**
- Lidocaine:2% + Epinephrine 1:200,000-18mL+ Bicarbonate 2mL or/2-Chloroprocaine 3%–18mL+ Bicarbonate-2 mL.
- Spinal
  - More appropriate for Grade II-III C-section
  - Hyperbaric bupivacaine 0.75%–1.8 mL, may be mixed with fentanyl 10 mcg and preservative-free morphine–200 mcg for better pain control during and up to 15–18 hr after surgery.
  - Spinal after failed epidural

**Indications and Usual Treatment**
- Maternal: bleeding (abruption placenta, pla-centa previa, aneurysm rupture), high risk of uterine rupture (laboring patient with history of classical incision for cesarean section or multiple low transverse cesarean section), uterine rupture, quick deterioration of maternal condition due to decapsulation of comorbidities (CHF secondary to congenital heart disease, acute renal failure in a patient with chronic renal disease etc.) or progression of preeclampsia (HELLP, acute fatty liver).
- Fetal: prolapsed umbilical cord, shoulder dystocia, vasa previa, severe distress (category III tracing), laboring malpresentation

**ICD-9-CM Code**: 656.31 (Fetal distress resulting in delivery)
• To minimize risk for high block perform intrathecal injection in sitting position, reduce dose of spinal bupivacaine by 20%, let pt sit for 60-90 sec after injection.

**Surgical Stages**

**Dissection Till Delivery**
- Inhalation agent should be started after intubation. MAC is reduced in pregnancy by 25–40% (32). All volatile agents, except NO, are potent uterine relaxants.
- Hyperventilate the pt to maintain normal for term pregnancy resp alkalosis with pH = 7.4, PaCO₂=32, HCO₃ = 20. Normoventilation will cause acute resp acidosis.
- Avoid opioids.

**Delivery of placenta, closing.**
- Start oxytocin infusion 20–40 units in 1000 mL of LR.
- Uterus will contract in response to oxytocin if MAC is <0.8 (33). Reduce concentration or stop halogenated agent, give 50–70% NO in O₂ and midazolam/opioids combination.
- Uterine atony-
  - Methergine 0.2 mg IM (contraindicated during preeclampsia), Hemabate 250 mcg IM (contraindicated for pts with asthma).
  - If massive blood loss: Transfuse RBC/FFP/platelets-1:1:1. Cryoprecipitate to keep fibrinogen higher than 100 mg/dL.

**Postoperative Consideration**
- None of maternal death happened during induction or maintenance of anesthesia, but all of them during the emergence or soon thereafter.

**Anticipated Problems/Concerns**

**General Anesthesia**
- Problems during induction
  - Rapid desaturation
  - Difficult airway
  - High risk of aspiration
  - Higher risk of intraop recall

**Regional Anesthesia**
- Local anesthetic toxicity
- Use amiodarone to treat arrhythmia and vasopressin to treat CV collapse
- High block
- Hypotension
- Failed block
- Rate of failed block converted to GA: 14% (35); spinal to GA: 1.4–2.1 % (7, 48).
Cesarean Section, Planned

Andrew P. Harris

Risk
- Incidence in the USA increasing: 1995, 785,000/y; 1998, 900,000/y; 2007, 1.37M/y
- Racial predominance: African-American higher, Hispanic lower rate

Perioperative Risks
- Periop pulm morbidity varies by type of anesthesia. GA is assoc with increased pulm morbidity
- Low mortality, but anesthesia (5–24/100,000) significant contributor to mortality

Worry About
- Inability to intubate
- Unintentional high regional block
- Unanticipated blood loss
- Spinal headache
- Embolism: Thromboembolism, air embolism, amniotic fluid embolism
- Postop endometritis

Overview
- Significant other frequently accompanies pt to the OR
- Hysterotomy usually through the lower uterine segment
- Occasional uterine atony after delivery Rx with oxytocic agents, occasionally progressing to cesarean hysterectomy
- Exteriorization of the uterus during closure assoc with greater intraop discomfort if regional anesthesia

Indications and Usual Treatment
- Maternal preference or conditions that would result in increased perinatal morbidity for mother or fetus if vaginal delivery attempted; most common examples incl Hx of classic (or maybe any) cesarean section, macrosomia, Hx of CPD, twin gestation
- May also be emergent (see Cesarean Section, Emergent, in Procedures section)
- Vaginal birth after C-section may be attempted in lieu of elective repeat cesarean, but now risks seem to be greater than benefits

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>AIRWAY</td>
<td>Engorgement of vessels and enlargement of breasts</td>
<td>Airway exam</td>
<td>Mallampati class</td>
<td></td>
</tr>
<tr>
<td>PULM</td>
<td>↑ Minute ventilation</td>
<td>SOB</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ O₂ consumption</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>↑ Cardiac output</td>
<td>Cardiac failure</td>
<td>Hgb</td>
<td>Antibody screen</td>
</tr>
<tr>
<td></td>
<td>↑ Dilutional anemia</td>
<td>Supine hypotensive syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Delayed gastric emptying</td>
<td>Regurgitation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>↑ Back pain</td>
<td>Back pain, sciatica</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Preoperative Management

Preoperative Preparation
- Antacids or H₂ blocker and/or metoclopramide
- Left uterine displacement

Anesthetic Technique
- Any: General, local, spinal, epidural, combined spinal/epidural; regional preferred due to potential airway problems

Monitoring
- Fetal HR monitoring during induction if possible or indicated

Airway
- Engorgement leads to easy bleeding, difficult intubation
- −1/300 unanticipated difficult intubation
- Airway cart and/or equipment available

Induction/Maintenance
- 33% less local anesthetic required for regional anesthesia
- T4 level during regional anesthesia desirable
- If GA, D/C halogenated agents (if possible) after delivery to reduce blood loss
- Prophylactic antibiotic usually prior to incision
- Oxytocin, methylergonovine, carboprost available for uterine atony after delivery

Surgical Stages
- Skin incision to delivery: Amniotic fluid embolism possible
- Closure: Venous air embolism, uterine atony, or hemorrhage possible
- EBL: 750-1000 mL normal; can be much greater with uterine atony

Postoperative Considerations
- Uterine atony, hemorrhage possible
- Pain score: typically 4–8
- IV PCA or epidural PCA for 1–2 d

Anticipated Problems/Concerns
- Inability to intubate
- High block with hypotension, sudden bradycardia
- Hemorrhage
- Postop headache

ICD-9-CM Code: V22.2 (Pregnancy)
Cholecystectomy, Laparoscopic

Stephen Aniskevich  
Sorin J. Brull

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<th>PE</th>
<th>Test</th>
</tr>
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<tbody>
<tr>
<td>CV</td>
<td>Likely co-morbidities: PVD, CAD</td>
<td>CV exam</td>
<td>ECG</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Co-morbidity likely: COPD (elderly) Sleep apnea</td>
<td>Chest exam</td>
<td>Neck circumference</td>
<td>CXR</td>
</tr>
<tr>
<td>HEME</td>
<td>Intraop blood loss (cystic artery laceration) Preop dehydration (N/V, elderly) Orthostasis</td>
<td>Hct, electrolytes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Impairment 2$^{nd}$ to age, co-morbidity</td>
<td>CNS Hx</td>
<td>CNS</td>
<td>BUN/Cr</td>
</tr>
</tbody>
</table>


Monitoring
• Routine, UO (Foley catheter)
• ETCO$_2$, not good substitute for arterial Pco$_2$

Airway
• Change in position from head-up to head-down may displace ETT into endobronchial position

Anesthetic Technique
• GA, controlled ventilation with cuffed ETT (prevent aspiration during pneumoperitoneum); regional (axial) anesthesia not advocated

Surgical Stages

Induction
• CV instability if co-existing disease, elderly
• Trocar insertion: Injury to viscera
• Trendelenburg position
  • CV effects: Improves venous return, CO, BP; pulm effects: reduced VC, atelectasis, shunting
  • Pneumoperitoneum creation
  • SQ emphysema from poorly placed insufflating needle
  • CV effects: Intra-abd pressure <15 mmHg assoc with minimal CV changes (slight increase in MAP, no change in CO)
• Pulm effects: Hypoventilation, resp acidosis, hypoxemia, tension pneumothorax (via patient pneumoperitoneum canal), atelectasis, shunting; exogenous CO$_2$, insufflation—rapid absorption, necessitating controlled ventilation; arrhythmias, catecholamine release; possible CO$_2$, pulm embolism, esp. at release of pneumoperitoneum

Definitive Surgery
• Postop N/V high (42%); prophylaxis recommended: Metoclopramide, droperidol, ondansetron
• Avoiding neostigmine, narcotic requirements by using NSAIDs (ketorolac) also effective
• Narcotic-induced spasm of Oddi spasm reversed with narcotic antagonists, local anesthetic infiltration, glucagon
• Use of N,O not recommended because of bowel distention, postop N/V
• Pre-emptive/adjuvant anesthesia with local anesthetic infiltration of skin, gallbladder bed may reduce postop pain
• Blood loss: Minimal
• Surgery Duration: ~1–3 hr
• Fluid shifts: Minimal
• Pain score: 2–5; same day or next day hospital discharge
• Long-term benefits of laparoscopic technique: lower incidence of bowel obstruction

Anticipated Problems/Concerns
• Intraop: Tension pneumothorax, CO$_2$ absorption, arrhythmias, hemodynamic compromise from pneumoperitoneum, visceral damage from surgical trocar, CO$_2$ embolism
• Conversion to open procedure (1–7% incidence) due to technical factors

Worry About
• Intraop hemorrhage
• Visceral damage. Bile duct injury is an iatrogenic catastrophe with significant morbidity, mortality, and reduced quality of life.
• Bacterbilia, sepsis
• PE, arrhythmias (CO$_2$ absorption)
• SQ emphysema from improperly placed CO$_2$ insuffling needle
• Hemodynamic consequences of pneumoperitoneum
• CO$_2$ absorption, position changes; CO$_2$ embolism

Overview
• Laparoscopic procedure increasing in frequency
• Lower incidence of complications than with open but mortality is the same

Risk
• Incidence in the United States: 20 million with gallstones
• 600,000 cholecystectomies/y
• Prevalence increases with age; higher incidence in women, 17%; men, 8%
• Among Pima Indian women, 75% affected
• Incidence in African-Americans lower than in Caucasians

Perioperative Risks
• Periop mortality ~0.1%, morbidity 4–6x lower than in open procedure (2–9%)
• Most benefits derived from avoidance of large ab incision
• Shorter hospital stay (~3d), more rapid convalescence (~22d)

Indications and Usual Treatment
• Indications: Chronic cholecystitis, symptomatic cholelithiasis
• Early contraindications: Large stones in common bile duct, acute inflammation, pregnancy, obesity, but now used in acute cholecystitis and pregnancy
• Considered technique of choice in octogenarians
• Alternative Rx: Open procedure, stone/contact dissolution, biliary lithotripsy, cholecystolithotomy

Overview
• Gasless (traction) laparoscopic techniques are less expensive and may help increase (or maintain) cardiac index.
• De Vinci surgical surgery is an emerging laparoscopic technology. Its benefits are yet to be established over traditional laparoscopic techniques.
• Miniport and single-incision (2-cm) laparoscopic techniques use smaller ports than the traditional 5- and 10-mm diameter ports, but their benefits have not been established.
• Ultrasonic energy dissection (Harmonic scalpel) may result in shorter operative time, lower gallbladder perforation risk, and shorter hospital stay than traditional electrocautery dissection techniques.
• Early laparoscopic cholecystectomy for acute cholecystitis appears safe and shortens total hospital stay.
• Low-pressure pneumoperitoneum procedures (intra-abdominal pressures < 12 mmHg) decrease postoperative pain and analgesic consumption.
• Natural-orifice trans-luminal endoscopic surgery (NOTES) technique provides an incisionless operation by insertion of operative instruments into peritoneal cavity through the GI tract (stomach) or urogenital tract (vagina). Advantages include less pain, no hernias, no surgical wound infection, and better cosmetic results.
### Cholecystectomy, Open

#### Risk
- Incidence in the USA: 20 million
- 600,000 cholecystectomies/y
- Prevalence increases with age; in women incidence is 17%, in men 8%
- Pima Indian women, incidence is 75%
- Incidence in African-Americans lower than in Caucasians

#### Perioperative Risks
- Periop mortality: 0–0.5% (0.1% in pts <50 y
- In elderly: Up to 10%
- Morbidity: 5–25%, esp. 2° to impairment of pulm mechanics (abd incision)

#### Worry About
- Intraop hemorrhage
- Hepatic failure

#### ASSESSMENT POINTS

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<thead>
<tr>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Rule out angina vs. cholecystitis ECG changes and arrhythmias</td>
<td>Rule out CAD relief by nitroglycerin (relieves both angina and biliary colic)</td>
<td>Pulm reserve Exercise tolerance</td>
<td>Auscultation CXR</td>
</tr>
<tr>
<td>RESP</td>
<td>Co-morbidity likely: CORD (elderly)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>N/V</td>
<td>N/V</td>
<td></td>
<td>RUQ pain</td>
</tr>
</tbody>
</table>

#### Key References:

#### Perioperative Management

<table>
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<th>Perioperative Evaluations</th>
</tr>
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<tr>
<td>Assess CV system, CAD: pulm system, predict postop resp issues</td>
</tr>
<tr>
<td><strong>Anesthetic Technique</strong></td>
</tr>
<tr>
<td>GA</td>
</tr>
<tr>
<td>Regional (axial) anesthesia may not be appropriate due to high level of sensory denervation required (at least T4)</td>
</tr>
<tr>
<td>Local anesthetic for cholecystectomy</td>
</tr>
<tr>
<td>Adjunct techniques: Paravertebral block, intercostal nerve block, interpleural cath</td>
</tr>
<tr>
<td>Prophylaxis for postop N/V</td>
</tr>
<tr>
<td><strong>Monitoring</strong></td>
</tr>
<tr>
<td>Routine</td>
</tr>
<tr>
<td><strong>Airway</strong></td>
</tr>
<tr>
<td>May warrant rapid-sequence induction 2° to pain, ↓ gastric emptying, preop N/V</td>
</tr>
<tr>
<td><strong>Induction</strong></td>
</tr>
<tr>
<td>Narcotic-induced sphincter of Oddi spasm reversed with narcotic antagonists, injection of local anesthetic, or glucagon</td>
</tr>
</tbody>
</table>

#### Indications and Usual Treatment
- Chronic cholecystitis and symptomatic cholelithiasis
- Biliary colic treated with parenteral narcotics; antibiotic therapy for pts over age 60 with chronic cholecystitis, and for pts with acute cholecystitis or with concomitant common duct stones
- NG suction and low-fat diet not proven beneficial
- Other Rx: Gallstone dissolution; percutaneous shockwave lithotripsy; contact dissolution; percutaneous cholecystolithotomy; laparoscopic cholecystectomy

#### Postoperative Considerations
- N/V: Consider prophylaxis
- Pain score: 6–10; narcotic requirements ↓ by "pre-emptive analgesia," adjunct techniques (paravertebral block, intercostal nerve block, interpleural cath), local anesthetic infiltration of gallbladder bed, postop NSAIDs for visceral pain

#### Anticipated problems/concerns
- Severe postop pain (incisional) may lead to ↓ ambulation; splinting ↓ cough, mobilization; atelectasis, pulm infection
- Open procedures assoc with longer hospital stay and convalescence.
- No difference in mortality between laparoscopic and open cholecystectomy techniques.
- The routine use of postop drains to prevent subhepatic abscesses or bile peritonitis results in higher abd wound and chest infection and is not recommended.

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**ICD-9-CM Code:** 574.0 (Cholelithiasis)
Risk
- ∼2 million/y procedures are performed
- Generally performed in neonatal period

Perioperative Risks
- Considered minimal 0.2–0.6% (aspiration, bleeding, hematoma, malignant hyperthermia reported 1 series [2/476], postop fever); complications from local anesthesia rare
- Local skin necrosis after dorsal penile nerve block (<0.5%)

Worry About
- Complicated preop neonatal course: sepsis, hypospadias, immaturity

Overview
- Most common surgical procedure in USA. Uncommon in most other parts of the world. Religious followers of Jewish and Islamic faiths practice circumcision for religious and cultural reasons. Circumcision rates also vary among racial and ethnic groups, with whites being much more likely to be circumcised.
- Rate increased from 1985 to 1992; largest increment following 1989 American Academy of Pediatrics statement of “potential benefits and advantages” of procedure
- According to the Agency for Healthcare Research and Quality 36% of males were circumcised in 2005; however, some estimate the percentage in the mid- to high-60s. Risk from UTI in uncircumcised males is 4–10 times greater than in circumcised, with the greatest risk in infants <1 y. Absolute risk of developing UTI in an uncircumcised male infant is low (∼1%).

Intraoperative Management
- Considerable evidence that newborns circumcised without analgesia experience pain and physiologic stress, manifested by changes in heart rate, BP, O₂ saturation, and cortisol levels
- In the past, the procedure was performed without analgesia or anesthesia. Recommendations now emphasize procedural analgesia.
- Subcutaneous ring block: A SQ circumferential ring of 0.8 mL of 1% lidocaine without epinephrine injected at the midshaft of the penis was found to be more effective than either EMLA cream or dorsal penile nerve block (DPNB)
- 1–2 g of EMLA cream is applied to the distal half of the penis and wrapped in an occlusive dressing 60–90 min before the procedure. There is a risk of methemoglobinemia from a metabolite of prilocaine, which can oxidize hemoglobin to methemoglobin.
- DPNB: A 27-g needle is used to inject 0.4 mL of 1% lidocaine, administered at the 10- and 2-o’clock positions at the base of the penis. The needle is directed posteriorly 3–5 mm on each side until Buck’s fascia is entered. After aspiration, the local anesthetic is injected. Bruising may be seen from the injection. A GA may be preferred in children or adults.
- Monitoring
  - Routine
  - Airway
  - Routine: Mask, laryngeal mask airway, intubation

Surgical Stages

Skin Incision/Definitive Surgery
- Two methods, sleeve or freehand, in which ring incision made around prepuce, or using a clamp (Plastibell, Gomco, or Mogen). In either, maximal surgical stimuli at this point; no dissection. Bleeding controlled with compression or electrocautery; suture placement rare.
- Commonly, petrolatum-based gauze used to dress wound edges, which are brought together
- Adult circumcisions are frequently performed without clamps and may require 4–6 wk to heal.

Postoperative Considerations
- Pain score: 2–4
- Pain relief by rectal acetaminophen in neonates
- Older individuals may require opiates

Anticipated Problems/Concerns
- Newborn infants experience pain manifested by physiologic changes (increase in BP, HR, sweating, decrease in oxygenation), behavioral changes, which persist for at least 22 h; physiologic, behavioral effects attenuated by local or regional anesthesia
- Pain often undertreated
- Infection remains a possibility in neonates, because hygiene may be compromised.
Cleft Lip Repair

Risk
- 1/700 live births
- Racial predominance: Asian and Native American 1/500; Caucasian 1/4,000; African descent 1/2,500
- Gender predominance: Male 60–80%; female 20–40%
- Assoc with cleft palate in 70–85% of cases
- Cause of orofacial clefting is multifactorial: Genetic and maternal influences incl smoking, alcohol use, phenytoin, folate deficiency

Perioperative Risks
- Extremely low morbidity and/or mortality
- When assoc with cleft palate repair, the most significant risk is postop airway obstruction

Overview
- Congenital condition by 7th wk intrauterine life; unilateral left–sided cleft is most common
- Strong genetic influence; one-quarter of cases bilateral cleft lip
- Timing of repair follows “rules of ten”: age >10 wk, wt >10 lbs, hemoglobin >10, WBC <10

ICD-9-CM Code: 749.10

Indications and Usual Treatment
- Single stage cleft lip repair, cheiloplasty, is performed if infant is appropriate surgical candidate
- Functional repair of lip and orbicularis oris muscle for feeding, facial expressions, normal facial growth, and speech development
- Cosmetic repair to facilitate parental bonding and social integration

ASSESSMENT POINTS*

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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Difficult airway</td>
<td>Snoring, grunting, difficulty with oral feeds</td>
<td>Airway exam: particular attention to head and neck mobility, palate, presence of mandibular hypoplasia</td>
<td>Otoscopic exam</td>
</tr>
<tr>
<td></td>
<td>Otitis media</td>
<td>Ear pain, fever</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Assoc CHD</td>
<td>SOB, cyanosis, poor growth</td>
<td>CV exam</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>URI</td>
<td>Cough, fever, rhinorrhea</td>
<td>Auscultation, chest exam</td>
<td>CXR, ABGs</td>
</tr>
<tr>
<td></td>
<td>Aspiration</td>
<td>Coughing with oral feeds, cyanosis</td>
<td></td>
<td></td>
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<tr>
<td>GI</td>
<td>Impaired deglutition</td>
<td>Nasal regurgitation</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Malnutrition</td>
<td>Poor growth</td>
<td>Observe feeding</td>
<td>Albumin</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td>Malnutrition</td>
<td>Pallor</td>
<td>Hgb/Hct</td>
</tr>
<tr>
<td>GU</td>
<td>Assoc congenital defects</td>
<td>UTI</td>
<td>UA, BUN/Cr</td>
<td></td>
</tr>
</tbody>
</table>

*Inclusive for associated cleft palate.


Preoperative Preparation
- Identify and evaluate assoc birth defects
- Prepare for potentially difficult airway/intubation with a variety of face masks, oropharyngeal airways, nasopharyngeal airways, laryngoscope blades, LMA’s, and possibly a video laryngoscope or fiberoptic scope

Anesthetic Technique
- Mask induction with sevoflurane, oral intubation using appropriate–sized uncuffed RAE tube well secured to mandible, reinforce tape since very close to surgical field
- Maintenance with inhalational agent; NMB not needed
- Pain control with short–acting opioid titrated to effect, consider bilateral infraorbital nerve block

Monitoring
- Standard ASA monitors; Precordial stethoscope esp. important since intraop access to airway severely limited
- Temp monitoring, forced air warmers, and warming blankets critical in infants

Surgical Stages
- Placement of pharyngeal pack, potential for ETT displacement if pack is placed
- Surgical field block with local anesthetic with 1:200,000 epinephrine
- Tissue flap elevation followed by closure of mucosa, orbicularis oris muscle, and skin
- Procedure time: 45–90 min
- EBL: Minimal

Postoperative Considerations
- Confirm that pharyngeal pack has been removed before extubation.
- Avoid disrupting sutures and/or repair during suctioning, extubation, and postop airway interventions.
- If pt less than 55 wk postconceptual age consider oximetry and apnea monitoring for 24 hr
- Oral or rectal acetaminophen usually sufficient for pain control
- Oral feeding can start with clear liquids as soon as pt is awake

Anticipated Problems/Concerns
- Undiagnosed cardiac anomalies in neonate
- Postop airway obstruction due to congenital airway abn, forgotten pharyngeal pack or airway edema
Cleft Palate Repair

Elizabeth A. Hein
C. Dean Kurth

**Risk**
- Incidence of cleft palate is about 1/1000 live births
- Repaired before speech develops, usually at age 6–12 mo

**Perioperative Risks**
- Periop mortality rare in pediatric centers

**Worry About**
- Assoc deformities, their risks: Congenital heart disease (SBE prophylaxis, cyanosis, CHF), micrognathia (difficult intubation), retroglottis (difficult mask airway), upper airway congestion (laryngospasm)
- Airway: Difficult mask ventilation, difficulty placing oral airway, difficult intubation; tube occlusion, inadvertent extubation, endobronchial tube advancement during surgery

**ASSESSMENT POINTS**

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Palate defect, other deformities, rhinorrhea</td>
<td>Apnea, known syndrome</td>
<td>Defect size, airway exam, nasal secretions</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cardiac defect</td>
<td>Slow feeding, diaphoresis</td>
<td>Murmur, liver size, cyanosis, HR, RR</td>
<td>ECG/CXR ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchitis, chronic aspiration</td>
<td>Cough, fever, feeding problem</td>
<td>Rhonchi, wheeze</td>
<td>O, sat CXR</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td>Age 3–9 mo</td>
<td>Pallor</td>
<td>Hct</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Premedication: If no obstructive apnea, then midazolam PO, atropine if young infant
- Consider cross-match, depending on surgeon, pt's Hct

**Anesthetic Technique**
- No special techniques

**Monitoring**
- Routine incl forced air warmer

**Airway**
- Oral RAE tube secured tracheal tube at midline, flat against chin, bend of tube at lip; use water-resistant tape, maintain small leak or use low-pressure ETT
- Flex, extend head to check for bronchial intubation or inadvertent extubation

**Surgical Stages**
- Palate infiltrated with epinephrine before incision; keep dose <10 μg/kg
- Tissue on both sides of defect mobilized to create flap
- During dissection, observe wound for bleeding, but transfusion rarely required
- After defect, check palate for edema, gauge airway caliber
- At end, heavy ligature may be placed through tongue, or NP airway may be inserted
- Wound may be injected with bupivacaine for postop analgesia; keep dose <2 mg/kg
- Surgical duration: 2–4 hr
- Blood loss variable

**Indications and Usual Treatment**
- Defects are surgically repaired if life expectancy reasonable
- Bottle-fed with special nipple before defect closed; caloric intake, growth monitored
- Otitis media often occurs; antibiotic prophylaxis common preop
- Myringotomy tubes frequently placed concomitantly with repairs

**Overview**
- Usually isolated deformity; it can also be part of syndrome (e.g., Pierre Robin)
- Repaired to separate oral, nasal cavities; improve feeding, speech; prevent middle ear disease, hearing loss; aspiration
- Surgical position: Supine, head extended, mouth open (Dingman gag), pharyngeal packs in
- Before incision, palate is infiltrated with epinephrine for hemostasis
- Surgery involves undermining tissues around defect to create flap to cover it; soft palate edema; opioid administration may contribute to postop obstructive apnea

**ICD-9-CM Codes**: 749.0; 749.2

**Postoperative Considerations**
- Analgesia with acetaminophen, or opioid (usually fentanyl); be careful with opioid dose to avoid obstructive apnea
- Pulse oximetry, cardiorespiratory monitoring recommended for 24–48 hr on floor unless other medical problems warrant ICU stay

**Emergence**
- Before extubation, ensure that the pharyngeal pack is gone and that the oral cavity is dry.
- Extubation best done when pt is awake
- Restrain arms to prevent child from pulling at oral suture line

**Risk**
- Incidence of cleft palate is about 1/1000 live births
- Repaired before speech develops, usually at age 6–12 mo
Colostomy

**Risk**
- All colostomies 42–65,000/y; 10–15/100,000 for Ulcerative Colitis (UC); 7/100,000 for Crohn’s
- M:F risk equal, increased incidence in non-Jewish and Jewish Caucasian pts; Crohn’s – M:F
- Colorectal Ca incidence per 100,000, all races male 57.3%, female 42.8%
- White male 56.9%, female 42.1%; black male 69.3%, female 53.5%
- Emergency surgery for obstruction and/or trauma, 10–15% have perforated, some resulting in ostomy

**Perioperative Risks**
- Risks are related to underlying disease process and surgical procedure.
- Mortality when a colostomy is an option is rare.
- Morbidity dependent on prior treatments, surgical procedure, and ongoing diseases: Wound healing problems and infections from irradiation or steroid use, perirectal hernia from APR, stoma retraction, bleeding, common postop fecal incontinence, constipation, and pain

**Perioperative Management**

**Preoperative Preparation**
- Restoration of fluid deficits, correction of electrolyte and acid-base imbalances if feasible
- Mechanical bowel preparation less de facto practice
- Corticosteroid supplementation as needed
- H/PPI and promotility agents as needed
- SQ LMWH and/or fondaparinux for DVT prophylaxis
- Antibiotics IV prior to skin incision
- Discuss FastTrack anorectal surgery with surgeon if appropriate

**Monitoring**
- ASA monitors
- Foley catheter
- Consider CVC based on need for pressure infusion, postop access

**Worry About**
- GI: Assessment of nutritional status, acid-base status, electrolytes, intravascular volume
- Renal: Assess electrolytes, acid-base, intravascular volume
- Intravascular volume: Bleeding, anemia, emesis, diarrhea, bowel prep, NPO status, anorexia
- Aspiration risk
- Steroid use: Htn, osteoporosis, infections, electrolytes, adrenal insufficiency
- TPN use: Liver problems, metabolic imbalances, bacteremia
- Metastatic disease to liver, lungs, brain from colon cancers.
- Trauma: Concurrent injuries and bleeding
- Smoking and/or heavy alcohol consumption

**Overview**
- Colostomy is a stoma that connects the colon to surface of the abd, can be permanent or temporary
- Transverse colostomy: 2 types, ascending and descending

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Ankylosing arthritis</td>
<td>C-spine mobility and neurologic complaints</td>
<td>C-spine ROM, airway and neuro exam</td>
<td>CT scan and/or MRI if positive neurologic findings</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Volume depletion, tachycardia, hypotension</td>
<td>Blood loss, emesis, diarrhea, anorexia, bowel prep, MET score, orthostasis</td>
<td>Vital signs, mental status</td>
<td>EKG; further testing as required in relation to urgency of surgery (stress test, troponin)</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm disease from metastases, smoking</td>
<td>Cough, hemoptysis, dyspnea, MET score</td>
<td>Auscultation, percussion</td>
<td>CXR, PFT, CT scan and/or MRI as time permits</td>
</tr>
<tr>
<td>GI</td>
<td>Aspiration risk</td>
<td>Obstruction, NPO status, GERD, pregnancy, trauma, N/V</td>
<td>Abd. exam, percussion, auscultation</td>
<td>KUB, FAST US, CT scan/MRI as time permits</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td>Orthostasis, pallor, exercise tolerance</td>
<td>Vital signs, cardiac exam</td>
<td>Hct</td>
</tr>
</tbody>
</table>


**Indications and Usual Treatment**
- Used to divert fecal content proximally from distal pathology
- Used as blow-hole to decompress distal blockage
- Colon Ca: Resection with postop radiation or chemotherapy
- IBD: Sulfasalazine, steroids, bowel rest preop
- Trauma: Exploratory laparotomy with colostomy or 1st anastomosis
- Intestinal ischemia: Vascular surgery, bowel resection, colostomy, open abd
- Distal obstructions, fistulas

**Postoperative Considerations**
- SICU for pts with hemodynamic instability, extensive surgery requiring ongoing resuscitation, and severe co-existing disease
- Continue corticosteroid use if adrenal suppression is anticipated
- Establish pain control regimen with epidural catheter and/or PCA
- High FIO2 supplementation may decrease wound infections and anastomotic breakdown
- Continue chemical DVT prophylaxis

**Anticipated Problems/Concerns**
- Postop resp problems with large fluid shifts or concurrent trauma injuries
- Postop pain control from surgery or concurrent trauma injuries

**ICD-9-CM Code:** 46.1 (Colostomy)
Coronary Artery Bypass Graft (CABG)

Risk
- 500,000 CABG annually, although number has been declining
- Risk factors for CAD: Htn, DM, hyperlipidemia, lipoprotein a genotype, smoking, age, male sex, family Hx

Perioperative Risks
- Overall mortality 0.5–4 % (average 2%)
- Main cause of death during and after bypass is MI (2–10%)
- Mortality and/or complications increase with age >70, decreased EF, DM, COPD, chronic renal failure
- Stroke 1–2%, primarily elderly; 30–55% pts have less than perfect cognitive function postop; mental status changes decrease as time post surgery lengthens

3% require exploration 2° to bleeding lead to ↑ risk of chest infection and lung complications
- Mortality of female > male

Worry About
- Periop ventricular function
- Myocardial protection, periop ischemia, early graft closure
- Completeness of surgical revascularization
- Bleeding with reperopations

Overview
- Occluded or severely diseased coronary arteries bypassed with venous or arterial grafts
- Anesthesia technique, monitoring, postop ventila
tory care affected by pt’s physical condition

ICD-9-CM Code: 36.11 (aortocoronary bypass of one coronary artery) to 36.14 (aortocoronary bypass of four coronary arteries)

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td></td>
<td>Risk factor search; Smoking stain; hypercholesterolemic lesions</td>
<td>McArdle’s earlobe</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>↓ LV or RV compliance</td>
<td>SOB, DOE, Angina</td>
<td>IHR/BP prior to and after 2-stair climb if stable enough to do so; S; rales; JVD; use character and rhythm</td>
<td>ECG, CXR, stress ECHO or dipyridamole thallium or ambulatory Holter</td>
</tr>
<tr>
<td>RESP</td>
<td></td>
<td>Nocturnal cough, orthopnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td></td>
<td>Perfusion insufficiency</td>
<td>Nocturia</td>
<td>BUN/Cre</td>
</tr>
<tr>
<td>CNS</td>
<td>Autonomic pain syndromes</td>
<td>Pain in neck or left arm Stroke/TIA Hx</td>
<td>CNS and cranial nerve exam; mental status exam</td>
<td>Carotid Doppler</td>
</tr>
<tr>
<td></td>
<td>Other atherosclerotic syndromes</td>
<td></td>
<td></td>
<td>ANS testing</td>
</tr>
</tbody>
</table>


Intraoperative Management

Preoperative Preparation
- Continue all preop medications, esp. aspirin, beta-blockers and statins
- Statins reduce early death, atrial fibr, and stroke

Monitoring
- 5-lead for ST segment analysis
- Arterial (radial, ulnar, brachial, femoral) line
- Central venous access for assessing volume status, cardiac function, fluid, and vasoactive drugs administration
- Core temp and peripheral temp monitoring
- UO monitor, if ↑ risk of hemodynamic complications during periop period, Category II, monitors left ventricle systolic function, hemodynamic/volume status, MR 4% assoc with CAD, assessment of viability, assess adequacy of cardiopulmonary distribution
- PAC (controversial) to measure pulm vascular resistance, filling pressures, and serial CO/CI

Anesthetic Technique
- Narcotics, volatile agents, benzos, muscle relaxants, Amicar
- Any technique is appropriate with concurrent monitoring of hemodynamics, setting of goals and keeping variables within desired parameters
- Technique can vary depending on whether beating heart or open circulatory arrest, and variants

Induction
- Avoid increased myocardial O2 consumption
- Avoid decreased myocardial O2 supply

Surgical Stages (Sternotomy Surgical Technique)

Skin Incision
- ↑ anesthesia to prevent ischemia during peri
dua or stimulation (incision, sternotomy, sternal spreading)
- Worry a lot about errant vessels adhering to sterna
num in redo ops or ops after a prior sternotomy
- Harvesting of internal mammary and vein grafts
- Temp suspend ventilation during sternotomy to prevent injury to lung and right side of heart
- Heparin 300 units/kg given centrally, and monitor effectiveness prior to institution of bypass
- Venous cannula placed in RA or vena cava

CPB
- Act >400
- ↑ potential for recall anesthesia narcotics, benzo, propofol infusion, precedge infusion
- Pt cooled to approx 28°C
- Stop ventilation when CPB full flow
- Aortic cross clamp
- Cardiopulgia blood or crystalloid given in aortic root proximal to cross clamp (antegrade ) or directly into coronary ostia and through coronary sinus ( retrograde)
- Repeat dose of cardiopulgia depending on time and reappearance of electrical activity

- MAP 60–80 mm Hg cerebral autoregulation fail below cerebral perfusion pressure of 50–55 mm Hg
- Maintain MAP by ↑ arterial flow or give phenylephrine

Termination of CPB
- Normothermia
- Resume mechanical ventilation with 100% O2
- SR with slightly ↑ rate, epicardial or trans
evus pacing can be used
- Return blood to heart
- Calcium to treat hypocalcemia, hyperkalemia, ↑ SVR, enhance contractility
- CPB terminated with acceptable pressure IABP
- Cl 32 for adequate organ perfusion
- TEE to evaluate RWMA, ventricular function, filling defects
- Stop Amicar
- Reverse heparin with protamine 1.3 mg/100 units slowly to prevent protamine reactions
- Suction to bypass machine stopped and vents removed with 50% protamine in ACT, ABG, electrolites

Anticipated Problems/Concerns
- Myocardial infarction due to embolism, hypoperfusion or graft failure
- Postperfusion syndrome, or pump head
- Acute renal failure due to embolism or hypoper-
fusion
- Stroke 2° to embolism or hypoperfusion
- Extrabub as soon as possible

Paul L. Samm

PROCEDURES

441
Craniotomy

Risk
- Intracranial tumors
- Vascular abn (aneurysms, AVMs)
- Traumatic Brain Injury (TBI)
- Stroke
- Intractable seizure disorder
- Intracranial infections (abscess, CJD)

Perioperative Risks
- Tumors: Size and grade can effect neurologic outcome
- Vascular: Stroke, vasospasm, bleeding
- TBI: Cardiomyopathy, cervical spine trauma
- Infection: Meningitis, sepsis
- Overall: Risk of stroke, hemiplegia, coma, and seizure

Worry About
- CPP, MAP-ICP (or CVP). Increases in ICP need to be matched with increases in MAP. In TBI and lesions with edema, perfusion can be more of a challenge.

In vascular lesions, maintaining normal pressure to not rupture lesion until clipped or ablated. Once clipped, increasing MAP to aid collateral perfusion and avoid ischemia.
- Once dura has been opened, optimizing surgical field to avoid herniation

Overview
- Craniotomies are performed to correct intracranial lesions such as tumors, abscesses, aneurysms, AVMs. Can also be decompressive in cases of subarachnoid hemorrhage, TBI, or stroke.
- Autoregulation of ICP and MAP lead to good compensation in chronic neurologic disease. Acute changes such as subarachnoid hemorrhage, TBI, or stroke lead to rapid decompen-sation of neurologic status.
- Vital signs changes that can lead to decomposition incl increased ICP, hypercapnia, hypoxemia, and malignant Htn or prolonged hypotension.

ASSESSMENT POINTS

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<tbody>
<tr>
<td>HEENT</td>
<td>TBI: Concurrent cervical/facial trauma</td>
<td>Hx of acute trauma</td>
<td>Facial trauma</td>
<td>CT scan of head and neck</td>
</tr>
<tr>
<td></td>
<td>Concussion/bleed can cause uneven pupils</td>
<td></td>
<td>Cervical trauma/c-collar</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pupillary exam</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>TBI: Tachycardia/cardiomyopathy from catecholamines</td>
<td>Often have concurrent Htn/CAD/PVD</td>
<td>BP</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>Aneurysms: Htn/bradycardia</td>
<td></td>
<td>HR</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Brainstem pressure causes apnea/resp depression</td>
<td>Agonal/Cheyne-stokes</td>
<td>Breath sounds</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Arterial bleed 2° to steroids</td>
<td>Hx of steroid use</td>
<td>Melena, coffee ground emesis</td>
<td>EGD</td>
</tr>
<tr>
<td>CNS</td>
<td>Change in consciousness, seizure, weakness/dysrhythm</td>
<td>Acute vs. chronic changes in neurologic exam</td>
<td>Focal motor/sensory dysfunction; awake/lethargic/comatose</td>
<td>MRI or CT of brain EEE</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Emergent: Secure airway, hyperventilation, avoid hypoxia. Maintain MAP.
- Elective: Minimize preop sedation. Evaluate symptoms and radiologic imaging to determine anesthetic approach. Give steroids, diuretics (mannitol, lasix), and anticonvulsants.

Monitoring
- Intra-arterial BP
- Consider CVP in SAH
- UO
- EEG, SSEP, MMEP, cranial nerves (acoustic)

Airway
- TBI: May have cervical spine trauma
- Aneurysm: Treat Htn assoc with laryngoscopy
- Avoid hypoxia and hypercapnia

Preinduction/Induction
- IV induction with propofol, barbituate, or etomidate
- NMBA to prevent coughing, facilitate intubation. Use rocuronium if RSI is indicated.
- Avoid succinylcholine and ketamine due to increase in ICP.
- Treat hypotension with phenylephrine, Htn with nicardipine or labetalol

Maintenance
- Low dose volatile agent
- Consider propofol infusion with severe cerebral edema.
- NMBA unless MMEP, EMG, or CN monitoring
- Fentanyl bolus vs. sufentanil or remifentanil infusion
- Ventilate to a PaCO2 of 26-30
- Propofol/barbituates decrease ICP, inhaled decreases CBF, opioid no effect

Extubation
- Neurologic exam before extubation
- Smooth extubation with no coughing or bucking
- If unable to assess neurologic status, postop CT scan
- Low preop GCS should remain intubated

Anticipated Problems/Concerns
- Intraop: Bleeding, stroke, seizure, venous air embolism
- Postop: Hypo/Htn, stroke, seizure, cerebral edema, vasospasm, neurologic changes
- Postop neurologic changes necessitate physical exam and possible CT imaging.
Craniotomy, Awake

**Indications**
- Resection of tumor or vascular malformation
- Resection of epileptic focus
- Deep brain stimulation for Parkinson’s disease, essential tremors or dystonia

**Perioperative Risks**
- Seizures
- Excessive sedation, resp depression, airway obstruction
- N/V, aspiration
- Disinhibition and lack of pt cooperation, agitation, postural discomfort
- Intracranial Htn and bleeding
- Hypercarbia and brain edema
- Venous air embolism
- Poor neurologic outcome

**Overview**
- Awake craniotomy performed for resection of space occupying lesions (tumors or vascular malformations) or epileptic foci or to place deep brain stimulation electrodes
- It is used when the pt's conscious response and cooperation is necessary for intraop functional testing such as speech mapping and motor mapping.
- Awake intraop mapping allows wider tumor excision and lower periop morbidity

**Preoperative Preparation**
- Appropriate pt selection and very thorough explanation of procedures and expectation of pt. Pts with morbid obesity, OSA, severe mental retardation, extreme anxiety, claustrophobia, and pts with documented difficult airway may not be good candidates for awake craniotomy.
- Minimize preop sedation. Avoid and/or minimize benzodiazepines preop because of the risk of disinhibition. Antiepileptic drugs should be given on the day of surgery.
- Antacids and nausea prophylaxis may be indicated.
- Neurologic exam and documentation of baseline status.

**Monitoring**
- Standard ASA monitors plus invasive BP monitoring
- Electrocorticography and/or direct cortical stimulation (Ojemann stimulator)
- UO

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<tbody>
<tr>
<td>HEENT</td>
<td>Airway access, risk of aspiration</td>
<td>BMI, difficult airway, sleep apnea</td>
<td>Mallampati exam, neck mobility, neck circumference</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Htn and bradycardia if severe increase in ICP</td>
<td>PMH: Htn, CAD</td>
<td>BP, HR, rhythm</td>
<td>EKG, ECHO and functional studies as indicated</td>
</tr>
<tr>
<td>RESP</td>
<td>Sleep apnea, smoker, COPD</td>
<td>Significant use of antacids, H2 blocker or proton pump inhibitor use, steroid usage, Hx of postop N/V</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Level of consciousness, seizures, headache, focal deficits, speech</td>
<td>Seizures, focal deficits</td>
<td>Neurologic evaluation</td>
<td>Head CT/MRI</td>
</tr>
</tbody>
</table>


**Anesthetic Technique/Induction**
- Sedation during with scalp infiltration or specific scalp blocks of branches of trigeminal nerve and cervical plexus with 0.5% bupivacaine or ropivacaine.
- Most common sedation drug combinations that have been used successfully incl a short-acting narcotic (fentanyl, sufentanyl or remifentanil) combined with propofol or dexmedetomidine.
- Dexmedetomidine alone administered as a bolus at 1 mcg/kg over 10min followed by infusion at 0.2–0.7 mcg/kg/min provides sedation without resp depression.
- Awake-Awake-Asleep technique: GA for craniotomy via LMA or ETI with inhalational agents and/or propofol for maintenance. The pt is awakened for functional testing and then asleep for closure of craniotomy.

- Local infiltration only with no sedation
- Hemodynamic management
- Normal BP or mild hypotension during brain exposure to minimize bleeding and brain swelling

**Worry About**
- Resp depression and need for emergent airway control
- Hypercarbia leading to brain edema
- Pt movement, disorientation, and lack of cooperation

**Etiology**
- Brain tumor
- Epileptic foci
- Parkinson’s disease refractory to medical management

**Indications and Usual Treatment**
- Space occupying lesions in eloquent areas of the brain
- Craniotomy under general anesthesia with electrocorticography
- Deep brain stimulation improves function in 15% of pts with Parkinson’s disease.

**Emergence**
- Prompt emergence and extubation in asleep cases for neurologic exam
- Prevent or treat Htn at emergence with antihypertensive of choice.

**Postoperative Considerations**
- Frequent neurologic exam

**Anticipated Problems/Concerns**
- Postop brain edema
- Hypovolemia from intraop osmotic diuretics and fluid restriction
- Seizures

**ICD-9-CM Code:** 239.6 (Brain tumor)

**Letha Mathews**

**PROCEDURES**
**Craniotomy, Sitting Position**

**Risk**
- Pts with infratentorial tumors (pineal, floor of fourth ventricle, pontomedullary junction, vermis, cerebellopontine angle)
- Trend decreasing, but still used in some institutions in USA, esp. for pineal surgery

**Perioperative Risks**
- Venous air embolism (as high as 80%)
- Paradoxical air embolism (probe patent foramen ovale in 25% of adult population)
- Hypotension
- Brainstem and/or cervical spinal cord ischemia
- Airway obstruction
- Tension pneumocephalus
- Macroglossia

**Worry About**
- Subdural hematoma due to major brain shift (excessive CSF drainage)
- Venous and/or paradoxical air embolism
- Brainstem, lower cranial nerve injury
- Poor cerebral venous drainage with acute flexion of head on neck

**Overview**
- Acoustic neuroma most common infratentorial tumor in adults
- 2 mmHg reduction in cerebral BP with every inch of elevation above heart
- Head, neck markedly flexed for better exposure
- Operation is performed around brainstem centers vital to respiration and circulation
- N₂O avoided during closure of dura

**Indications and Usual Treatment**
- Surgical indications
  - Supracerebellar infratentorial approaches to pineal region, midline, 4th ventricular lesions, CP angle tumor
- Contraindications
  - Ventriculoatrial shunt in place and open
  - Cardiac diseases
  - Hydrocephalus
  - Autonomic dysfunction
  - Extremes of age
  - Cerebral ischemic disease (stroke)

**ICD-9-CM Code:** 239.6 (Brain tumor)

**Key Reference:** Wong Ay, Irwin MG. Large venous air embolism in the sitting position despite monitoring with transoesophageal echocardiography. *Anaesthesia.* 2005;60(8):811–813.

**Perioperative Implications**
**Preoperative Preparation**
- Antishock trouser (MAST suit)
- Precordial Doppler
- Multiorificed RA catheter
- Adequate hydration

**Anesthetic Technique**
- General with controlled ventilation

**Monitoring**
- PA catheter or CVP
- ETCO₂
- Precordial Doppler or TEE
- Direct intra-arterial BP: Transducer zeroed at head level
- SSEP and BAER
- EMG-facial muscle, tongue, shoulders

**Airway**
- ETT may be kinked by acute flexion of neck
- Allow at least 2 fingerbreadths between chin, sternum

**Induction/Maintenance**
- Isoflurane, N₂O, low-dose fentanyl most common technique
- Use of short-acting NMB (for quick reversal for facial nerve monitoring)

**Surgical Stages**
**Dissection**
- Sudden onset of tachycardia/bradycardia, PVCs, hypotension

**Definitive Surgery**
- Except for extra-axial lesions in cerebellopontine angle, surgery for pathologic Dx, and/or to reduce mass effect

**Anticipated Problems/Concerns**
- CV complications resulting from venous air embolism
- Tension pneumocephalus
- Cranial nerve paresis, aspiration
- Macroglossia
- Failure to awaken from anesthesia, possible brainstem or subdural hematoma

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<tbody>
<tr>
<td>HEENT</td>
<td>Dysphagia, facial paralysis</td>
<td>Choking, hoarseness</td>
<td>ENT exam</td>
<td>Cine x-ray</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Patent foramen ovale predisposes to paradoxical air embolism</td>
<td>Easy fatiguability</td>
<td>Auscultation</td>
<td>CXR cardiac cath</td>
</tr>
<tr>
<td>RESP</td>
<td>Aspiration</td>
<td>Coughing</td>
<td>Auscultation</td>
<td>CXR</td>
</tr>
<tr>
<td>CNS</td>
<td>Ventriculoatrial shunt predisposes to venous air embolism, hydrocephalus predisposes to tension pneumocephalus</td>
<td>Shunt surgery</td>
<td>CNS exam</td>
<td>Head CT scan</td>
</tr>
</tbody>
</table>

**Blood loss usually not significant**

**Postoperative Considerations**
- Pain score: 0–3
- Minimize coughing, straining on ETT
- Cranial nerve dysfunction, esp. cranial nerve 7, 9
- Extubation determined by extent of surgery
- Postop Htn possibly caused by brainstem compression due to hematoma

**Anticipated Problems/Concerns**
- CV complications resulting from venous air embolism
- Tension pneumocephalus
- Cranial nerve paresis, aspiration
- Macroglossia
- Failure to awaken from anesthesia, possible brainstem or subdural hematoma

- Muscle relaxant avoided by some because of facial nerve monitoring; when used, 2–3 twitches of train-of-4 usually maintained
**Electroconvulsive Therapy (ECT)**

**Risk**
- Lifetime prevalence of major depressive disorder (MDD): 16.2%

**Perioperative Risks**
- Periop mortality rare
- Dysrhythmias and Htn common
- Cognitive dysfunction common post-treatment

**Worry About**
- Sympathetic stimulation producing myocardial ischemia and infarction
- Assoc between MDD and CHD
- Risk of dysrhythmia as a result of parasympathetic and sympathetic stimulation
- Cognitive dysfunction and short-term memory loss from Rx
- Adequate medical Hx frequently difficult to obtain from depressed pt

**Overview**
- ECT remains the most effective acute antidepressant intervention
- Induced seizures produce multiple neuroendocrine changes (increased ACTH, cortisol, epinephrine, norepinephrine, growth hormone, decreased GABA) and changes in serotonergic and dopaminergic receptor function in the brain; these and other effects of ECT are well-documented but which of these produce antidepressant effects is unclear and the mechanism(s) by which ECT affect mood remain unclear.

**Indications and Usual Treatment**
- Clinical indications
  - Failure to respond to conventional pharmacologic Rx
  - Medical contraindication to pharmacologic Rx (e.g., cardiac conduction defect)
  - Profound depression if delay in Rx places pt at unacceptable risk for suicide
  - Maintenance ECT (weekly to monthly) being used in pts at risk for relapse
  - More controversial indications incl schizophrenia, mania, eating disorders, and catatonia.
- Relative contraindications incl intracranial space-occupying lesion, recent MI, recent CVA, pheochromocytoma, long-bone fractures and pregnancy.
- Alternative therapies incl the use of antidepressants and psychotherapy. Recent advances in brain stimulation techniques such as transcranial magnetic stimulation, direct-current stimulation, and deep brain stimulation have not been shown to be superior to ECT in terms of response rates or speed of response.

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<tbody>
<tr>
<td>CARDIO</td>
<td>CAD ↓ LV function</td>
<td>Angina, prior MI ↓ Exercise tolerance</td>
<td>Enlarged heart, JVD, orthopnea</td>
<td>ECG or stress test as indicated by Hx</td>
</tr>
<tr>
<td>GI</td>
<td>GE reflux</td>
<td>Syncope or near syncope, Medications</td>
<td>Rhythm</td>
<td>ECG or Holter as indicated</td>
</tr>
<tr>
<td>MS</td>
<td>Fracture or vertebral collapse</td>
<td>Pain or trauma</td>
<td>Palpation</td>
<td>X-ray, CT as indicated</td>
</tr>
<tr>
<td>NS</td>
<td>Confusion/delirium</td>
<td>Prior Hx</td>
<td>CNS exam</td>
<td>Routine workup for change in MS incl</td>
</tr>
<tr>
<td>PSYCH</td>
<td>Competent for consent</td>
<td></td>
<td></td>
<td>Determined by psychiatric team</td>
</tr>
</tbody>
</table>


**Perioperative Implications**
- Pt comfort and amnesia
- Minimize risk of physical injury from generalized seizure
- Management of hemodynamic changes assoc with treatment
- Rapid return to conscious state
- Recent interest in evaluating anesthetic techniques which may ameliorate cognitive deficits inherent to treatment

**Monitoring**
- Routine incl ECG, pulse oximetry, capnography and NIBP
- EEG to determine adequacy, duration of seizure
- In high-risk pts consider invasive monitoring for initial treatments.

**Induction/Maintenance**
- Ga, usually by mask
- IV induction
  - Thiopental (0.5–1.0 mg/kg): Decreased seizure threshold, increased seizure duration, rapid awakening
  - Propofol (0.5–1.0 mg/kg): Decreased seizure duration, rapid awakening
- Ketamine (1–3 mg/kg): Increased seizure duration
- Methohexital (0.5–1.0 mg/kg): Increased seizure duration
- Succinylcholine (0.5–1.0 mg/kg): Increased seizure duration
- Paralysis reduces potential for injury from generalized seizure; mivacurium when succinylcholine contraindicated
- Hyperventilation with 100% O₂ by mask before stimulus produces reduced seizure threshold and may prolong seizure duration.
- Propofol is considered an initial parasympathetic stimulation assoc with bradycardia and hypotension; transient asystole possible.
- Subsequent sympathetic stimulation assoc with increased HR, increased BP, increased CO₂, increased myocardial O₂ consumption; rate

**System**

- **CARDIO**: CAD (ventricular function)
  - ↓ Angina, MI
  - ↓ Exercise tolerance
  - ↓ Orthopnea
- **GI**: GE reflux
  - Syncope or near syncope
- **MS**: MS fracture or vertebral collapse
  - Pain or trauma
  - Palpation
- **NS**: Confusion/delirium
  - Prior Hx
- **PSYCH**: Competent for consent

**Assessments by Hx**

- **PE**: Enlarged heart, JVD, orthopnea
- **Test**: ECG or stress test as indicated by Hx

**PROCEDURES**

- **Induction/Maintenance**: Ga, usually by mask
- **IV induction**
  - Thiopental (0.5–1.0 mg/kg): Decreased seizure threshold, increased seizure duration, rapid awakening
  - Propofol (0.5–1.0 mg/kg): Decreased seizure duration, rapid awakening
- **Ketamine (1–3 mg/kg)**: Increased seizure duration
- **Methohexital (0.5–1.0 mg/kg)**: Increased seizure duration
- **Succinylcholine (0.5–1.0 mg/kg)**: Increased seizure duration
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- **Subsequent sympathetic stimulation** assoc with increased HR, increased BP, increased CO₂, increased myocardial O₂ consumption; rate

**ICD9CM Codes**
- 296.2 (Major depression, single episode)
- 296.3 (Major depression, recurrent episode)
- 311 (Depressive disorder, not elsewhere classified)
PROCEDURES

pressure product (HR × SBP) increases 50–400%; regional wall motion abn common by TTE
• Other physiologic effects incl increased CMRO₂, increased CBF, increased ICP, increased intragastric pressure, increased IOP.
• Vasodilators (sodium nitroprusside, nitroglycerin) and β-blockers (esmolol, labetolol) effective for ameliorating hemodynamic effects of seizure
• Bradycardias rarely require Rx.

Management
• Waveform, frequency, and duration of stimulus adjusted to produce desired seizure
• Some psychiatrists utilize the isolated limb technique: Inflate BP cuff on extremity prior to succinylcholine administration to monitor motor response to seizure
• Seizures ideally 25–120 sec but controversial whether clinical efficacy actually related to seizure duration; prolonged seizures assoc with increased cognitive deficit

Anticipated Problems/Concerns
• Pts frequently have multiple co-morbidities and thorough preop interview may be difficult or impossible given psychiatric illness.
• Repetitive short anesthetics require efficient system to record medical Hx, allergies, drug dosages, complications, etc.
• Rx produces sympathetic activation in pt population deemed medically unfit to tolerate conventional pharmacologic Rx
• Pts commonly complain of headache or myalgias post ECT; this can be treated with acetaminophen or ketorolac.
Endoscopic Sinus Surgery (ESS)

Risk
- Incidence in USA: Chronic rhinosinusitis affects nearly 15% annually
- Males and females equally affected
- Most frequently pts undergo bilateral surgery

Perioperative Risks
- Major complications (0.3% to 1%) incl CSF leak, meningitis, massive bleeding, visual impairment (to diplopia and blindness), stroke, death.
- Minor complications (<1% to 20%) incl periorbital emphysema and ecchymosis, middle turbinate adhesions, epistaxis, facial pain.

Worry About
- Periop control of asthma and airway reactivity
- Quantification of intraop blood losses and/or postop bleeding

Overview
- ESS is performed to restore normal sinus ventilation and mucosal function in the context of chronic sinusitis of the paranasal sinuses. Also utilized for tear duct surgery, orbital decompression, and drainage of mucoceles.
- Major risks are related to the possibility of perforation of the ethmoid sinus roof, orbital penetration through a dehiscence or fracture of the lamina papyracea, injury to the carotid artery and the optic nerve.
- Surgeon’s knowledge of normal anatomy of the nasal cavity and its many variations is of paramount importance in avoiding major complications. Preop CT scans of the nasal cavity and paranasal sinuses are extremely useful to the surgeon as a diagnostic tool and to provide important landmarks for surgery.

Indications and Usual Treatment
- Failure of medical management of recurrent or chronic rhinosinusitis (steroids, antihistamine, antibiotics, decongestants)
- Chronic sinusitis assoc with obstructive nasal polyps
- Treatment of mucoceles
- Dx of neoplasms of the nasal cavity and paranasal sinuses and orbital cellulitis

ASSESSMENT POINTS

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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Postnasal drip</td>
<td>Persistent cough/aspiration</td>
<td>Airway</td>
<td>Laryngoscopy</td>
</tr>
<tr>
<td>CARDIO</td>
<td>CAD, toxicity from medications</td>
<td>SOB, exercise tolerance, Hx of palpitations and/or chest pain</td>
<td>CV exam</td>
<td>ECG/other testing as indicated per guidelines for non-cardiac surgery; theophylline level</td>
</tr>
<tr>
<td>RESP</td>
<td>Frequent assoc with asthma, allergies, bronchitis</td>
<td>Frequency of asthma exacerbations, hospitalizations with intubation, chronic steroids, known allergens</td>
<td>Chest, larynx (vocalization)</td>
<td>Spirometry</td>
</tr>
<tr>
<td>GI</td>
<td>N/V</td>
<td>N/V</td>
<td>Neuro</td>
<td>Theophylline level</td>
</tr>
<tr>
<td>CNS</td>
<td>Rule out meningitis</td>
<td>Headache, fever, N/V, visual impairment, mental status, seizures</td>
<td>Neuro</td>
<td>Tests as indicated by Hx/PE</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
- Bronchodilator puffs prior to surgery
- Consider stress dose of steroids
- Oxymetazoline spray for topical decongestion

Monitoring
- Standard

Anesthetic Technique/Airway
- General anesthesia preferred technique
- MAC possible only for minor procedures
- Oral intubation +/- oropharyngeal packing or LMA

Induction/Maintenance
- TIVA with propofol and/or remifentanil proposed as better choice (N/V, hemodynamic stability)
- Eye protection with clear tape to allow for orbit inspection by surgeon
- Avoid histamine-releasing medications.
- Intraop anti-emetic drug indicated to prevent N/V
- Controlled hypotension to maintain clear surgical field and facilitate hemostasis not always necessary: always wt benefits against risks of cerebral and/or coronary hypoperfusion (age, co-morbidities).

Surgical Stages
- Lidocaine plus epinephrine injection may cause arrhythmia and/or tachycardia.
- When mucosa is removed from posterior ethmoid cell, risk of perforation of the roof of the ethmoid sinus. During exposure, risk of penetration of the lamina papyracea.
- Injuries to brain, optic nerve, and carotid artery may occur during sphenoidectomy.

Extubation/Postoperative Considerations
- Nasal packing placed after surgery
- Removal of oropharyngeal packing if present and suction of oropharynx/esophagus/stomach are of paramount importance before emergence and/or extubation.
- EBL is usually modest (even if difficult to quantify) unless arterial injury occurs
- Postop pain control is generally easily achieved with opiates and/or acetaminophen.
**Endovascular Aortic Stent Repairs**

**Alexandru Gottlieb**

### Risk
- Incidence in the USA: About 15,000 aortic stents/y. The number is increasing dramatically (originally was reserved only for high-risk pt, is now indicated for all pts)

### Perioperative Risks
- Mostly related to the diffuse arteriosclerotic CV disease, unknown long-term mortality and/or morbidity
- Periop MI: 3–10%
- Geriatric pts with age and other related risk factor: Htn, DM, and COPD
- Potential for thrombosis, embolization, occlusion, or bleeding

### Worry About
- Failure of deployment and need to convert to open procedure
- Leak to aortic sack—repeat endovascular intervention
- Device cost may be limiting
- Dissection of the aortic wall.
- Organ ischemia, esp. with the new fenestrated and high thoracic repairs

**Overview**
- Open repair is still the gold standard. Aortic stent repair was initially suggested for high-risk pts, but successful enough that it is recommended for all reconstructive aortic pts, provided that they do not have contraindication for the operation (see Indications)

### Indications and Usual Treatment
- Indication: AAA, occlusive disease, or aortic dissection
- Contraindications: Young pt (long-term prognosis—unknown); aneurysm neck: 4 cm long, >3.5 cm wide irregular, calcified, mural circumferential thrombus; iliac artery disease: occlusive, calcified; dominant inferior mesenteric artery

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<tr>
<td>RESP</td>
<td>High incidence of lung disease that could have caused this procedure to be selected</td>
<td>Smoking, chronic cough, dyspnea</td>
<td>Chest exam, wheezing, clubbing, cyanosis, dyspnea</td>
<td>CXR, ABGs, spirometry</td>
</tr>
<tr>
<td>CARDIO</td>
<td>High incidence of CAD, myocardial ischemia, and/or infarction</td>
<td>Angina, MI, CHF, dysrhythmia, PCI, stents, CABG, exercise tolerance, activity level</td>
<td>Chest auscultation, Vital signs</td>
<td>ECG stress test: ECHO, dipyridamole, thallium, dobutamine</td>
</tr>
<tr>
<td>Chronic Htn</td>
<td>BP Rx, drug interaction</td>
<td>BP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>High incidence of renal insufficiency 2° to age, arteriosclerosis, and multiple dye studies</td>
<td>Hx of edema and intolerance to salt load</td>
<td>Presence of edema, Anorexia</td>
<td>Urea, Cr, electrolytes</td>
</tr>
<tr>
<td>CNS</td>
<td>Possible carotid disease</td>
<td>Syncope, stroke, or TIAs</td>
<td>Neuro exam Carotid bruit?</td>
<td>Carotid angiogram CT, MRI</td>
</tr>
<tr>
<td>ENDO</td>
<td>High incidence of DM</td>
<td>Hx of infections Stress-related/gestational DM Polydipsia, polyuria, coma/stupor Hx of abnormal glucose</td>
<td>Skin infections</td>
<td>CNS exam Urine output Serum glucose</td>
</tr>
<tr>
<td>HEME</td>
<td>Some pts on periop heparin, t-PA, or aspirin</td>
<td>Hx of petechiae, nasal bleed</td>
<td>Presence of petechiae or clinical bleeding</td>
<td>PT, PTT, ACT</td>
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**Postoperative Consideration**
- Mild postop pain (3–4)
- Groin hematoma, seroma, or bleeding

**Anticipated Problems/Concerns**
- Periop cardiac, pulm complications
- Thrombosis and/or embolization of lower limb
- Leak around aortic stent
- Decreased UO
- Spinal ischemia

**Pathology Findings**
- Continuous persistent leak
- Potential for decreased renal function
- Long-term prognosis after stent repair unavailable

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Esophagectomy

Risk
• Esophageal carcinoma: Worldwide sixth leading cause of cancer.
• Adenocarcinoma more common in western countries, among males; assoc with gastro-esophageal reflux and resulting Barrett's esophagus
• Squamous cell more common among African-Americans, males (3:1), tobacco abusers (4:1); alcohol abusers (6:1), Hx of achalasia, caustic burns to esophagus, Paterson-Kelly syndrome (iron-deficiency anemia, esophageal webs, glossitis)

Perioperative Risks
• Operative mortality less than 5% reported by some centers but may be as high as 10–14%
• Multiple risk factors reported for morbidity and mortality incl age, tumor stage, pulmonary dysfunction, smoking Hx, diabetes, cardiac dysfunction, hepatic dysfunction, and impaired functional status
• Periop complication rate of 10–27% incl anastomotic leak (risk for sepsis due to mediastinitis with intrathoracic anastomosis), pulmonary insufficiency, delayed emptying of intrathoracic stomach, diaphragmatic herniation of abdominal viscera, chylothorax, massive aspiration, pancreatitis, delayed splenic rupture, and dysrhythmia

Worry About
• Aspiration risk
• Hemodynamic effects of blunt dissection
• Consequences of N2O technique if colonic interposition performed
• Recurrent laryngeal nerve injury if cervical anastomosis performed
• Consequences of periop TPN (hypoglycemia, increased CO2 production)

Overview
• Midline laparotomy to explore for metastases
• Mobilization of stomach (Kocher’s maneuver), pyloromyotomy
• Mobilization of esophagus: Depends on site of lesion, surgeon’s preference; may occur via transhiatal approach, right (Ivor-Lewis) thoracotomy or three-incision approach with the addition of a left cervical incision.
• Reconstruction: Stomach is preferred conduit, but colon or jejunum may be used

Postoperative Considerations
• Possible esophagorespiratory fistula (may need to maintain spontaneous ventilation)
• Use of gel lubricants on ETT cuff may decrease risk of aspiration intraop

Induction
• Potential for significant hypotension if dehydrated
• If esophagorespiratory fistula: Avoid PPV, vent stomach if necessary, isolate fistula with appropriate double-lumen ETT

Surgical Stages
Dissection
• Initial laparotomy
• Depending on approach may have right thoracotomy
• Cervical incision if cervical anastomosis to be performed (necessary with transhiatal approach, in addition to thoracotomy with three-incision approach)

Definitive Surgery
• Blunt dissection during transhiatal approach may result in compression of vena cava or heart with resultant hypotension or dysrhythmias
• Impaired gas exchange may occur during l-lung ventilation if required
• EBL: 500–1500 mL

Anticipated Problems/Concerns
• Possibility for unrecognized pneumothorax with transhiatal approach


Perioperative Management

Anesthetic Management
• General or combined technique (thoracic epidural frequently employed, increasing experience with paravertebral techniques)
• Evidence suggests improved outcome with addition of thoracic epidural. However, concern for decreased perfusion of distal end of gastric tube reconstruction—may have implications for BP management
• If transhiatal or left thoracotomy approach, single-lumen ETT is adequate. Right thoracotomy approach requires placement of a double-lumen tube or bronchial blocker.
• Consider lung protective ventilation strategy – may have beneficial effect on postop SIRS

Monitoring
• Large-bore IV access
• Consider arterial catheter
• Consider central venous catheter

Airway
• Full-stomach precautions
• Possibility of tracheal compression if significant mediastinal lymphadenopathy

• Minimally invasive endoscopic resection incl thoracoscopy, laparoscopy, and robot-assisted techniques are increasingly employed. Outcome benefit has not been convincingly demonstrated. Some surgeons employ prone positioning for mobilization of the esophagus.

ICD-9-CM Code: 150.9 (Esophageal carcinoma)

Indications and Usual Treatment
• Surgery only curative treatment (5-y survival as high as 70% if limited to stage I disease)
• At presentation disease is usually advanced with overall 5-y survival of only 25%
• Palliative Rx incl chemotherapy, combined chemo/radiation, or radiation alone
• Postop chemotherapy has not resulted in improved survival
• Preop (neoadjuvant) chemotherapy with irradiation may downstage tumors before resection; role in possible improved survival is controversial and not clearly established
• If albumin low, consider preop enteral nutrition

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<td>HEENT</td>
<td>Aspiration risk, esophagorespiratory fistula</td>
<td>Dysphagia, heartburn, N/V</td>
<td>Barium swallow</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tracheal compression</td>
<td></td>
<td>CT, flow loops</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Chemotherapy-induced cardiomyopathy</td>
<td>DOE, PND, orthopnea,</td>
<td>Auscultation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>exercise tolerance</td>
<td>JVD, edema, heptojugular reflex</td>
<td>CXR, ECHO, MUGA</td>
</tr>
<tr>
<td>RESP</td>
<td>Chronic aspiration</td>
<td>Wheezing, dyspnea</td>
<td>Auscultation</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Malnutrition</td>
<td>Wt loss, fatigue</td>
<td>Cachexia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>GERD</td>
<td>Heartburn</td>
<td>Serum protein</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Albumin, pre-albumin</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Dehydration</td>
<td>Orthostatic VS</td>
<td>BUN, Cr</td>
<td></td>
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<tr>
<td></td>
<td>Electrolyte wasting</td>
<td></td>
<td>Serum electrolytes</td>
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<tr>
<td>HEME</td>
<td>Anemia</td>
<td></td>
<td>CBC</td>
<td></td>
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<tr>
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<td>Impaired immune function</td>
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Extracorporeal Membrane Oxygenation (ECMO)

**Risk**
- Proven utility in newborn resp failure
- Common causes of newborn resp failure are meconium aspiration, persistent fetal circulation, persistent pulm Htn (PPH), congenital diaphragmatic hernia, sepsis
- May be indicated in children and adults with potentially reversible resp failure unresponsive to conventional therapy
- Used for cardiac and pulm support after cardiac surgery in infants and children

**Perioperative Risks**
- Survival depends on the underlying condition
- Neonatal resp failure has an 80% survival for those thought to have a 20% survival without ECMO

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<td>Myocardial dysfunction</td>
<td>Hypoxemia, acidosis</td>
<td>CV exam</td>
<td>ECHO Hemodynamic variables ABGs</td>
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<td>PPH</td>
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<td>Hypoxemia R → L shunt through PDA or PFO</td>
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<td>NEURO</td>
<td>Hemorrhage</td>
<td>Intracranial hemorrhage</td>
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<td>Cranial US</td>
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**Monitoring**
- ECG, temp, UO
- Arterial and central venous pressures
- Arterial and mixed venous blood gases
- Pulse oximetry if cardiac ejections present

**Surgical Stages**

**Anesthetic Choice**
- Cannulation often performed at the bedside in ICU with local anesthetic infiltration. Narcotics and muscle relaxants may be used and continued while cannulas in place.
- ECMO may be initiated in OR after failure to wean from CPB

**Initiating ECMO**
- Venoarterial ECMO can be done with extrathoracic cannulation (carotid artery and internal jugular vein or femoral artery and vein) or by transthoracic cannulation through a median sternotomy (aorta and right atrium)
- Original cannulation sites are used after failure to wean from CPB in OR
- Right internal jugular vein is commonly used for venovenous bypass
- Heparin (100–150 units/kg) is given and the vessels are cannulated
- Bypass is initiated slowly by increasing extracorporeal flow rate
- After reaching full flow rates, ventilate at non-traumatic settings

**Worry About**
- Mechanical complications (cannula dislodgment, clot in circuit, air in circuit)
- Bleeding complications (particularly intracranial hemorrhage)
- Multisystem organ failure

**Overview**
- Provides total resp support with venovenous or venoarterial bypass
- Venoarterial bypass also provides total hemodynamic support

**Intraoperative Management**
- Survival after repair of CHD in infants is largely dependent on the severity of the underlying lesion (43–54% in TOF, 14% in HLHS)

**Risk**
- Pt is anticoagulated to maintain an activated clotting time (ACT) of ~200 sec

**Indications and Usual Treatment**
- Criteria vary among institutions
- Contraindications: Congenital abs not compatible with meaningful life, profound neurologic impairment, irreversible lung disease, prolonged ventilatory support (>7–10 d), estimated gestational age <35 wk, evidence of intracranial hemorrhage

**Ecmo Management**
- Heparin to maintain ACTs at approx 200 sec.
- If bleeding occurs, the ACT can be maintained at lower levels (140–180 sec).
- Plts transfused to maintain 100,000/mm³
- As cardiopulmonary function improves, the pump flow is decreased. Prior to decannulation, the pt is given a trial without ECMO support.
- After decannulation, the vessels are usually ligated.

**Anticipated Problems/Concerns**
- Hypoxia and hemodynamic instability prior to bypass
- Bleeding complications
- Multorgan failure
- Mechanical problems with ECMO circuitry
Lithotripsy (ESWL)

**Overview**
- Shock waves propagated through body to pulverize urinary stones. First-generation lithotriptors require immersion in water bath, which complicates monitoring, airway management, and induce changes in cardiopulmonary physiology; regional (usually epidural) anesthesia preferred for procedures in these machines.
- Second-generation “dry” lithotriptors eliminate these problems, generate less powerful shock waves, so lighter planes of anesthesia or sedation combined with topical anesthesia may be effective.
- Third-generation lithotriptors use piezoelectric crystals with smaller focal zone, are essentially painless; may also be used to pulverize gallstones.

ICD-9-CM Codes: N20.0 (Kidney stones); N20.1 (Ureteral stones); N20.9 (Urinary stones)

**Indications and Usual Treatment**
- Preferred choice for upper urinary tract stones <2 cm; lower stone-free rate compared to invasive methods, esp. with stones >1 cm. Absolute contraindications: Urosepsis, pregnancy, coagulopathy, uncorrected obstruction; relative contraindications: morbid obesity, renal malformations, intra-abd calcific processes (e.g., AAAs).
- Urolithiasis may also be Rx by alkalization of the urine using potassium citrate.
- Urinary stones can be extremely painful; pts may be taking variety of analgesic drugs incl NSAIDs, opioids.

**Risk**
- Annual incidence of urolithiasis: 1.5/1000

**Perioperative Risks**
- Shock waves can trigger cardiac dysrhythmias if not delivered during ventricular refractory period.
- Shock waves can damage kidney resulting in perinephric hematoma, parenchymal injury with loss of renal function, hematuria, new-onsset Htn.
- Shock waves can cause pancreatic, hepatic injury resulting in elevations in amylase, lipase, bilirubin, lactic dehydrogenase, transaminases, CK; changes are usually mild, transient.

**Worry About**
- Cardiac dysrhythmias
- Cardiopulmonary derangements resulting from immersion
- Electrical safety in water bath
- Renal insufficiency
- Plt dysfunction

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<td>Delayed gastric emptying, gastritis, PUD</td>
<td>Reflux Sx, dyspepsia, abd pain</td>
<td>Hgb, endoscopy, upper GI x-ray, stool heme</td>
<td></td>
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<tr>
<td>HEME</td>
<td>Anemia (due to renal failure, GI losses), Plt dysfunction (due to analgesia or uremia)</td>
<td>Fatigue, bruising, bleeding</td>
<td>Pallor, ecchymoses, petechiae</td>
<td>Hct, bleeding time</td>
</tr>
<tr>
<td>GU</td>
<td>Obstructive uropathy, analgesic nephritis</td>
<td>Oliguria, anuria, CHF Sx</td>
<td>Rales, edema</td>
<td>BUN/Cr, UA, CXR</td>
</tr>
</tbody>
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**Intraoperative Management**

**Preoperative Preparation**
- Evaluate renal function
- Evaluate plt function
- R/O anemia

**Anesthetic Technique**
- Regional anesthesia usually preferred for immersion-type lithotriptors
- Plt dysfunction may influence decision to use regional anesthesia.
- If GA used, small TV should be used to keep stone at focal point of shock wave.
- Pt position in gantry requires care to avoid peripheral nerve damage; pt can be prone on newer lithotriptors.
- Local anesthesia, topical anesthesia (e.g., EMLA) and/or IV sedation usually sufficient with newer lithotriptors

**Monitoring**
- Easier if pt arms not immersed
- Cover ECG leads with waterproof tape

**Indications and Usual Treatment**
- Preferred choice for upper urinary tract stones <2 cm; lower stone-free rate compared to invasive methods, esp. with stones >1 cm. Absolute contraindications: Urosepsis, pregnancy, coagulopathy, uncorrected obstruction; relative contraindications: morbid obesity, renal malformations, intra-abd calcific processes (e.g., AAAs).
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**Eye Enucleation**

**Risk**
- Eye trauma
- Incidence in USA: >2 million/y; 8000–10,000 ablations/y
- Majority pts <30 y of age
- Most common cause of enucleation in children >3 y of age
- Ocular tumors <5/100,000 population

**Perioperative Risks**
- Age, co-morbidities and medications
- Hx of postop N/V (PONV)
- Assoc trauma
- Chemotherapy and/or radiation therapy
- Pt acceptance and/or psychological preparation

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<td>Sleep apnea</td>
<td>Snoring</td>
<td>Airway exam</td>
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<tr>
<td></td>
<td>Anxiety</td>
<td>Claustrophobia</td>
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<td></td>
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<tr>
<td>CARDIO</td>
<td>Htn, CAD</td>
<td>Headache, angina</td>
<td>BP</td>
<td>EKG/ECHO</td>
</tr>
<tr>
<td></td>
<td>Arrhythmia</td>
<td>Syncope, palpitations</td>
<td>Pulse rate/rhythm</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>COAD</td>
<td>Chronic cough, exercise Tolerance</td>
<td>Chest auscultation</td>
<td>O2, sat</td>
</tr>
</tbody>
</table>

**Preoperative Management**

**Preoperative**
- Age, co-morbidities and medications
- Hx of postop N/V (PONV)
- Assoc trauma
- Chemotherapy and/or radiation therapy
- Corroboration of surgical necessity by second ophthalmologist
- Pt acceptance and psychological preparation (lesser importance in pts with blind painful eye)
- Anesthesia technique discussion (GA vs. RA+MAC)
- Anxiolysis

**Intraoperative Management**

- Pt, site, and procedure verification (“TIME OUT”)
- Standard ASA monitors
- Technique: General (GA) vs. regional (RA) and monitored anesthesia care (MAC)
- Evisceration: RA > GA (usually elderly, frail, ASA Status III-IV)
- Enucleation: RA = GA (trauma/painful eye, children, ASA Status I-II)
- Exenteration: GA (extensive resection + bony orbital wall components)

**General anesthesia**
- Endotracheal tube (ETT) versus supraglottic airway (LMA)
- Deep plane anesthesia during dissection of extra-ocular muscles and optic nerve
- Optic nerve difficult to dissect and sever: Beware of bradycardia and asystole.
- Request early alert from surgeon, and deepen anesthesia and/or sedation in a timely fashion
- Venous congestion and pt positioning (head-up and/or pressure points)

**Regional Anesthesia**
- Extraconal versus intraconal block
- Local anesthetic solution (intermediate vs. long-acting agent)

**Postoperative Management**

- Analgesia: Supplement GA with regional block (esp. children)
- Emesis control (consider use > 1 agent)
- Minimize venous congestion with head-up position
- Hemorrhage and/or apply firm dressing
- Visual hallucinations
- Psychological concerns and/or professional intervention

**Worry About**
- Oculocardiac reflex (OCR)
- Co-morbid illness
- PONV
- Hemorrhage

**Overview**
- Evisceration: Removal of the contents of the globe leaving the sclera and extra-ocular muscles intact
- Enucleation: Removal of the eye from the orbit with preservation of all other orbital structures
- Exenteration: Removal of the eye, adnexa, and portions of the bony orbit.

**Etiology**
- Intraocular malignancy (choroidal melanoma, retinoblastoma, other)
- Trauma (primary or sympathetic ophthalmia)
- Blind painful eye (endophthalmitis, uveitis, neovascular glaucoma, cosmetic)
Gas Embolism

Effect
Assessment by Hx

Test


Worry About

Overview and Etiology

Immediate effect: Obstruction of blood flow and tissue ischemia; pulm Htn if venous gas

Secondary effect: Increased permeability of vascular endothelium, tissue edema (pulm edema from venous gas)

Tertiary effect: Leukocyte accumulation on vascular endothelium, resulting in release of mediators and late reduction in blood flow

While small venous gas emboli are prevented by the pulm capillary network from entering the arterial circulation, large volumes of gas can exceed its filtration capacity. Air can also enter the left heart via intrapulmonary or intracardiac right-to-left shunts, incl patent foramen ovale.

Venous gas embolism can cause sudden hypotension, hypoxemia, pulm Htn or cardiac arrest. Awake individuals may experience dyspnea, tachypnea, or cough.

Arterial gas embolism is manifested by altered consciousness, acute onset of focal neurologic deficit, arrhythmias, and ST segment elevation or depression.

Venous gas emboli can be detected by a change in the cardiac sounds as detected using a precordial Doppler monitor, an immediate reduction in $ETCO_2$, mass spectrometer or Raman detection of nitrogen in the expired gas (when the inspired gas contains no air). Bubbles can also be detected using trans-thoracic or trans-esophageal echocardiography and transcranial doppler.

When NO is used, gas in blood vessels or tissues can expand to 2—4× its original volume, compounding the original injury.

Risk

• Accidental injection of gas into a blood vessel during diagnostic or therapeutic procedures
  • Cardiopulmonary bypass
  • Cardiac catheterization, angiography
  • Hemodialysis
  • Pressurization of an IV bottle using air
  • Entrainment of air into a vein during surgical procedures in which venous pressure at the wound site is subatmospheric (wound higher than heart)
  • Sitting craniotomy
  • Spine surgery
  • Total hip replacement
  • Dental implant surgery
  • Cesarean section
  • Surgery in which gas could be injected into tissues
    • Intrauterine laser surgery
    • Laparoscopy
    • Arthroscopy
  • Other forceful instillation of air into tissues
  • Injury due to industrial compressed air
  • Blowing air intravaginally during oral sex
  • Pulm overexpansion, in which gas enters the pulm capillaries
  • Breathing or regional gas trapping during ascent from a scuba dive
  • PPV
  • Hydrogen peroxide irrigation or ingestion

ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE | Test
--- | --- | --- | --- | ---
CARDIO | Filling of cardiac chambers with air | Hypotension | Echocardiography, precordial Doppler, ↓ PETCO₂, ↑ PETN₂, ECG (ST depression)
 | ↑ Permeability of vascular endothelium | Mill-wheel murmur | |
 | Tissue edema | |
RESP | Pulm Htn | Dyspnea, tachypnea, cough | Crackles on auscultation of chest (commonly not heard) | PA pressure measurement CXR
 | Pulm edema | |
CNS | Arterial gas embolism or transpulmonary/transcardiac passage of venous gas emboli | Acute onset of focal neurologic deficit, generalized encephalopathy, seizures | Neurologic exam | Brain imaging (CT, MRI) often normal, cerebral edema may be seen

PROCEDURES

• Rarely, a “mill-wheel” murmur (a whoosh sound in both systole and diastole) can be heard by precordial stethoscope. The absence of this sign cannot be used to exclude the diagnosis.

ICD-9-CM Codes: 958.0; 999.1; 673.0

Usual Treatment

• If possible prevent further ingress of gas
• If surgical gas embolism:
  • Flood surgical field with fluid
  • Lower surgical site with respect to the heart
  • Elevate venous pressure (accomplishing this via application of PEEP can augment right-to-left shunt if the pt has an intracardiac defect, e.g., patent foramen ovale).
  • 100% inspired O₂ to enhance oxygenation of ischemic tissues and increase the nitrogen diffusion gradient from bubble into blood.
  • IV fluid administration to maintain intravascular volume
• Use hyperbaric O₂ to effect reduction in size and rapid resolution of bubbles. Hyperbaric O₂ (HBO) may also inhibit adherence of leukocytes to endothelium, thus minimizing the tertiary effects noted above. Immediate treatment with hyperbaric O₂ is most efficacious; delayed treatment can also be effective. Indications for HBO incl neurologic deficit, myocardial ischemia, residual left-sided intracardiac gas, or cardiovascular compromise. Pulm edema may also respond to HBO therapy.
• Radiographic imaging (e.g., brain CT, MRI) cannot be used to confirm the diagnosis and plays no useful role.

Perioperative Implications

• Pts at high risk for venous gas embolism should receive appropriate monitoring, which may include
  • Direct arterial pressure monitoring
  • Continuous monitoring of $PETCO_2$, $PETN_2$
  • Precordial Doppler
  • TEE

  • Consider avoiding the use of NO, and if pulm O₂ toxicity is not a concern, using 100% O₂
  • If gas embolization is suspected, NO or nitrogen and/or O₂ gas mix should be immediately D/C and replaced with 100% O₂
  • Individuals requiring surgery, who have recently suffered gas embolism due to scuba diving, or decompression sickness (in situ gas formation due to nitrogen supersaturation) from diving or compressed air exposure, should not be administered NO anesthesia.

RN Bryant W. Stolp
Richard E. Moon
Bryant W. Stolp
Gastrectomy

Risk
- Adenocarcinoma of stomach: 21,000 new cases annually; ~11,000 deaths annually
- 1/113 people will be diagnosed in lifetime; most pts diagnosed after age: 65
- M:F ratio: 2:1
- Higher incidence in first-generation Asian-Americans; higher incidence in USA black men compared to white men
- Incidence of gastrectomy has been decreasing 2nd to treatment of Helicobacter pylori infection, medical treatment of Zollinger-Ellison syndrome

Perioperative Risks
- In-hospital mortality 2–6% in more recent trials; 15–32% in slightly older trials with extended dissection; <2% for partial gastrectomy
- Worse nutritional status results in higher morbidity and mortality

Perioperative Management

Preoperative Preparation
- Assess pre-existing conditions and volume status, electrolytes consider periop beta-blockade in elderly
- Aspiration premedication with H2 blockers, metoclopramide (contraindicated in obstruction), and sodium citrate
- Preop resuscitation; correct anemia appropriately, esp. with pre-existing cardiac disease
- Consider TPN to correct malnutrition prior to OR if albumin <2.1
- Consider insertion of an epidural for periop pain control and less pulm complication postop; test prior to induction to ensure correct placement

Monitoring
- Essential to have good measurement of volume status 2nd to fluid shifts and periop hypovolemia
- Foley catheter to monitor UO
- Consider arterial line based on co-morbidities
- CVP may be helpful; PA cath is usually not needed

Anesthetic Technique/Induction
- Balanced general anesthetic; general/regional
- Consider awake techniques or rapid sequence induction with cricoid pressure for pts at high risk of aspiration
- Use forced air warmers, warmed IV fluids, and warmed humidified gases to prevent hypothermia
- NGT is often needed; discuss when to place with surgeon

Surgical Stages
- Incision: Upper midline; unilateral or bilateral substernal
- Pt is positioned supine, thoracotomy is a possibility in proximal lesions; lung isolation should be planned through discussion with surgeon
- Some surgeons use laparoscopy to assess for metastasis prior to laparotomy
- Left lobe of liver is retracted and packs may be inserted near diaphragm; watch for increased peak airway pressures
- The greater and lesser omentum are part of surgical specimen; splenectomy is occasionally performed for total gastrectomy for cancer; distal pancreatectomy if pancreas involved with tumor
- Stomach is mobilized and blood supply ligated; duodenum and esophagus are transected

Assessment by Hx
- BP ↓, HR ↑, UO ↓, skin turgor

Assessment by PE
- Hypovolemia
- N/V, diarrhea, poor oral intake
- Pulm complications in 15% of pts
- Pulm emboli: 1%
- Pneumonia: 8%
- Postop bleeding: 6%
- Abd abscess: 7%

Periop hypovolemia
- Anemia
- Large third space losses
- Malnutrition

Overview
- Resection of all or part of the stomach for either malignant or benign diagnoses
- Bleeding can occur from spleen or blood supply to stomach
- Periop hypovolemia from blood loss, poor PO intake, or GI losses
- NGT can be accidently ligated; make sure it is properly pulled back prior to resection


ICD-9-CM Codes: 151.9 (Gastric cancer); 531 (Gastric ulcer)

Indications and Usual Treatment
- Total gastrectomy
- Gastric cancer (adenocarcinoma, GIST, etc)
- Uncontrollable hemorrhagic gastritis (less commonly currently with angiography and endoscopy)
- Zollinger-Ellison syndrome (medical management more common currently with proton pump inhibitors and octreotide)
- Severe gastroparesis after previous stomach surgery
- Partial gastrectomy
- Gastric ulcers
- Gastric cancer
Gastric Bypass Stapling for Morbid Obesity

**ASSESSMENT POINTS**

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<thead>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
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<tbody>
<tr>
<td>HEENT</td>
<td>Obstructive sleep apnea</td>
<td>Snoring, dyspnena, daytime somnolence</td>
<td>Large tongue, tonsils, uvula</td>
<td>Polysomnography</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Htn, pulm Htn, CAD with possible ventricular dysfunction, LVH, RVH</td>
<td>Angina, PND</td>
<td>Pedal edema</td>
<td>Stress ECG, ECHO, dipyridamole thallium or dobutamine ECHO</td>
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<tr>
<td>RESP</td>
<td>Obstructive sleep apnea, diminished closing volume</td>
<td>Snoring, dyspnena, daytime somnolence</td>
<td>Dyspneic when supine</td>
<td>Polysomnography, PFTs, CXR, ABG</td>
</tr>
<tr>
<td>CNS</td>
<td>Neurovascular insufficiency</td>
<td>TIA, stroke</td>
<td>Cognitive impairment, neuro deficits</td>
<td>Neuro assessment</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Assess co-existing morbidities, plan to maintain homeostasis
- Consider epidural placement for open procedures for periop analgesia
- Consider inferior vena cava filter placement for superobese pts and discussion with surgeon regarding DVT for all

**Monitoring**
- Noninvasive monitoring, large-bore vascular access
- Invasive arterial pressure monitoring for pts with significant cardiac disease (LVEF < 35%, and/or proven significant CAD)
- Central venous access only if peripheral access cannot be established

**Anesthetic Technique/Induction**
- Pt carefully placed in ramped positioned for induction of general anesthesia following placement of epidural, if used
- Adequate preoxygenation using PEEP or pressure support
- All drugs dosed for total or ideal body weight, as appropriate
- Stable induction technique with full NMB achieved prior to intubation

**Surgical Stages**

**Dissection**
- Maintain adequate NMB
- Provide appropriate level of mechanical ventilation, esp. in laparoscopic procedures during pneumoperitoneum
- Provide adequate IV fluids, esp. with use of steep reverse Trendelenberg position

**Gastric Stapling**
- Remove NG tube, esophageal temp probe prior to stapling
- For gastric pouch creation, advance and remove bougie carefully without disturbing ETT position
- Carefully reinsert NG tube into gastric pouch under surgical guidance and assess staple lines for leaks

**Postoperative Considerations**
- Maintain CPAP or BiPAP, supplemental O₂ and head-up positioning in recovery and on floor
- Postop analgesia: Epidural vs. IV PCA
- Monitor IV PCA use and undesired side effects increasing airway obstruction
- Use continuous pulse oximetry postop
- Clearly identify those pts who were difficult to intubate

**Anticipated Problems/Concerns**
- Use of IV PCA can be assoc with resp depression and exacerbation of airway obstruction sympsoms requiring reintubation
- Pts may derespirate lung volume rapidly during any phase of the anesthetic care
- Controversies incl rapid sequence induction versus attempted mask ventilation after anesthetic induction; resp monitoring postop; requirement for high acuity nursing floor care

- Complication rates higher with open procedures
- Re-operation causes incl postop intra-abd bleeding, anastomotic leakage, suture line dehiscence, small bowel obstruction, deep wound infection
- DVT and pulm embolism

**Worry About**
- Pt positioning for airway management
- Maintenance of lung volume
- Anesthetic drug dosing

**Overview**
- High incidence of co-existing metabolic syndrome, obstructive sleep apnea, Type 2 diabetes, Htn, CAD
- Ease of airway management directly related to optimizing pt positioning

**Indications and Usual Treatment**
- Body mass index >35 kg/m²
- Failure of nonsurgical management of obesity
- Endocrine abn (metabolic syndrome, Type 2 diabetes) or other CV (Htn) or pulm (obstructive sleep apnea) pathophysiology present
- Restrictive (gastric banding) or malabsorptive (roux-en-y gastric bypass or sleeve) procedure to induce wt loss and aid resolution of endocrine and other abn

- Anesthesia goals are to maintain lung volume, manage airway safely, dose drugs and medications for obesity, optimize return to spontaneous ventila ation in the face of obstructive sleep apnea

- Maintain mechanical ventilatory support until extubation
- Extrabute pt from head-up position and immediately apply CPAP

- Incidence in USA: 65% of adult population (>200 million in USA)
- >300,000 deaths annually attributable to obesity
- Second to smoking as preventable cause of death
- Predominantly caused by long-term excess caloric ingestion
- Polysomnography, PFTs, CXR, Neurovascular insufficiency Assessment by Hx
- TIA, stroke
- PE
- Obstructive sleep apnea
- CV disease, renal dysfunction
- Test achieved prior to intubation
- Stable induction technique with full NMB as appropriate
- Adequate preoxygenation using PEEP or pressure support
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- Re-operation causes incl postop intra-abd bleeding, anastomotic leakage, suture line dehiscence, small bowel obstruction, deep wound infection
- DVT and pulm embolism
- Cerebral or CV Htn, pulm Htn, CAD
- Snoring, dyspnea, daytime somnolence
- Large tongue, tonsils, uvula
- Polysomnography
- Htn, pulm Htn, CAD with possible ventricular dysfunction, LVH, RVH
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**Gastrochisis Surgery**

**Risk**
- Rare abd wall abn
- Occurs in 1/20,000 births

**Perioperative Risks**
- Increased risk of infection
- 90–100% survival reported

**Worry About**
- Large fluid requirements
- Temp instability
- Cardiopulmonary compromise 2° to increase intraabd pressure
- Postop ventilation
- Postop nutrition
- Other congenital abn

**PROCEDURES**

**Monitoring**
- Avoid N₂O
- CV instability 2° to hypovolemia

**Induction**
- Muscle relaxation required
- Ventilation
- Avoid distention of bowel by bag and/or mask ventilation
- Muscle relaxation required

**Airway**
- Expect usual neonatal variants
- Endotracheal intubation
- Avoid distention of bowel by bag and/or mask ventilation
- Muscle relaxation required


**Overview**
- Extrusion of abd contents through a defect to the right of the umbilical cord. Must be differentiated from omphalocele.
- True surgical emergency
- Abd contents not covered by sac
- Abd viscera matted together and thickened 2° to amniotic fluid exposure and chemical peritonitis
- 60% of pts are premature
- Assoc abn (other than GI) rare, but their presence may affect pt morbidity and mortality
- GI abn incl intestinal atresia and stenosis.

ICD-9-CM Code: 756.7 (Congenital)

**Assessment by Hx**

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<tr>
<td></td>
<td>Hypotension 2° to ↑ intra-abd pressure</td>
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<td>Large 3rd space fluid requirements</td>
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**Postoperative Considerations**
- Need for mechanical ventilation
- Need for hyperalimentation
- In pts with silo, gradual reduction of abd contents, increased risk of sepsis, and prolonged ventilatory requirements

**Anticipated Problems/Concerns**
- Hypovolemia 2° to large third space—fluid requirements
- Instability 2° to increased intra-abdominal pressure following abd wall closure
- Decrease in lung compliance 2° to impaired diaphragmatic movement from reduced abd contents and increased intra-abd pressure
- Prolonged postop pain relief may be required
- Increased risk of necrotizing enterocolitis
- Long-term survival dependent on assoc abn
- Bowel obstruction common morbidity

**Perioperative Implications**

**Anesthetic Technique**
- GA
- Endotracheal intubation
- Avoid N₂O

**Monitoring**
- Pulse oximeters on both the right upper extremity and a lower extremity
- Adequate venous access preferably above the diaphragm
- Consider arterial catheter
- Consider central venous catheter
- Foley catheter

**Airway**
- Expect usual neonatal variants
- Endotracheal intubation
- Avoid distention of bowel by bag and/or mask ventilation
- Muscle relaxation required

**Induction**
- CV instability 2° to hypovolemia
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Geriatric Surgery

Risk
- Major complications: 20% for vascular procedures, 10% for general surgery, 3% for minor procedures.
- Periop (1 mo) mortality: About 3%

Perioperative Risk Factors
- Emergency procedures
- Increased acuity and severity of pre-existing disease
- Compromised physical status (ASA 3–4) and limited range of daily activity
- Extreme agedness (85 y or older)
- Surgery that will significantly limit postop mobility

Worry About
- Susceptibility to infection
- Fluid overload
- Renal insufficiency
- Prolonged drug effects
- Myocardial ischemia
- Disorientation and delirium
- Pressure points and tissue fragility

Overview
- Aging of healthy individuals reduces organ system functional reserve.
- Age-related disease exposes and exacerbates the decrease in reserve produced by aging.
- Polypharmacy for age-related co-morbidities further increases the risk of adverse periop outcome in older adults.
- Therefore, the severity of age-related disease is the most powerful predictor of periop complications and death in the elderly.

Choice Of Anesthetic Approach
- Anesthesia itself is not a major factor in periop morbidity and mortality.
- There is no objective evidence to support a "best" anesthetic approach for the elderly pt.
- Optimal periop outcome generally reflects aggressive preop disease management, meticulous attention to the details of intraop management, adequate control of periop pain and stress, and early ambulation.

Perioperative Management

Preoperative Preparation
- Careful clinical Hx and assessment of physical status with special scrutiny of ability to ambulate and exercise
- Limit preop "screening tests" to those areas with a clinical correlate (Hx or physical exam) that suggests presence of a definable disease process.
- Echocardiographic estimate of cardiac ejection fraction if adequacy of aerobic capacity not obvious
- Optimize control of significant pre-existing medical issues such as CAD, asthma, HTn, or diabetes.

Monitoring
- Standard ASA monitors usually adequate for short diagnostic procedures (e.g., endoscopy or cystoscopy) and surgery not involving major body cavity
- Consider arterial cannulas for pts having major surgery (i.e., intra-abdominal, -thoracic, -cranial) or for prolonged procedures requiring repeated arterial samples or continuous BP assessment
- Consider central venous catheters for pts with evidence of diffusely impaired ventricular function or those who do not have reliable peripheral IV access; consider pulm artery catheter if clear evidence of severe left ventricular compromise.

Altered Response to Anesthetics
- Prolonged circulation time dictates slow drug infusion to avoid overdosage
- Unconsciousness usually achieved with lower plasma levels of drug than in younger adults.
- Anesthetic maintenance requirements reduced 20–30% by the eighth decade of life
- Regional anesthesia is effective but slightly smaller segmental dose requirements and greater risk of sympathectomy-induced hypotension
- Expect reduced narcotic requirement and increased duration of clinical effect, but adequacy of analgesia must be carefully titrated.
- Usually little change in dosage required for NMB, but expect some prolongation of duration of clinical effect.
- Complex intraop polypharmacy may produce unpredictable emergence phenomena, esp. if it alters central cholinergic receptors.

Postoperative Considerations
- Pay careful attention to adequate hydration and control of hemodynamics to maintain metabolic homeostasis and good tissue perfusion.
- Avoid excessive sympathoadrenal stress that can increase CV demands in excess of the age-related reduction in functional reserve.
- Avoid the CNS depression and coagulopathies assoc with hypothermia.
- Anticipate high likelihood of acute postop delirium, exacerbated by poor pain control, Hx of alcohol abuse, or pre-existing cognitive impairment.

Anticipated Problems/Concerns
- 1° causes of postop morbidity and mortality in this age group are infection, myocardial ischemia, and thromboembolism.
- From the middle adult years onward, increasing age is assoc with a significant risk (5–15%) of subtle and poorly understood adult long-term postop cognitive dysfunction (POCD). The risk does not appear to be reduced with any specific anesthetic approach.
- Long-term survival in elderly surgical pts appears to be enhanced when there are minimal complications in the acute postop period and the surgery itself improves the pt’s mobility and physical independence.

Consequences of Aging

Anesthetic Implications

Clinical Adjustments and Therapies

Generalized loss of metabolically active tissue mass and reduced aerobic capacity
- Decreased cardiopulmonary reserve; reduced hepatorenal capacity
- Cautious dosing of anesthetic agents with myocardial- and respiratory-depressant properties; supplemental O2 therapy; reduced dosage of anesthetic agents requiring hepatic or renal biotransformation; avoidance of aggressive volume loading with IV fluids

Reduced autonomic and immune homeostasis
- Greater intraop variability of vital signs; increased risk of infection
- Minimize periop sympathectomy due to neuraxial anesthesia; early treatment of abn BP or HR; antibiotic prophylaxis and meticulous sterile technique for invasive procedures; periop warming

Decreased neural tissue mass and neurotransmitter activity
- Decreased anesthetic and analgesic requirement
- Age-adjusted dosing of anesthetic agents and adjuvants; allow increased time for emergence and for recovery of NM transmission; increased risk of postop delirium and/or cognitive dysfunction

Increased co-morbidities and prevalence of age-related disease and polypharmacy
- Complex periop polypharmacy; uncontrollable or inadequately-controlled cardiopulmonary or metabolic disorders; increased frequency of adverse drug interactions
- Careful preop assessment of chronic disease and functional status; full review of routine medications and adverse drug episodes; selective use of pharmacy expertise and specialist consultants

GI Endoscopy/EGD, Non-Operating Room Anesthesia

Overview
- Diagnostic and therapeutic procedures for Barrett’s esophagitis, eosinophilic esophagitis, esophageal strictures and rings, mediastinal masses, gastric bypass screening, GI bleeding, pancreatic masses and cysts, chronic pancreatitis, lower GI tract strictures, choledolithiasis and choledocolithiasis, biliary cancer, Crohn’s, ulcerative colitis, diverticulosis, colon and rectal cancer staging, polypectomies, colon cancer screening, follow-up for h/o colon polyps.
- Procedures incl esophagogastroduodenoscopy (EGD), colonoscopy, esophageal and rectal US, esophageal dilation, tissue biopsies, endoscopic retrograde cholangiopancreatography (ERCP), celiac plexus block, pancreatic cyst drainage, balloon assisted enteroscopy (BAE).

Perioperative Management

Preoperative Preparation
- NPO guidelines must be followed unless emergent.

Monitoring
- Standard ASA monitors

Anesthetic Technique/Induction
- The majority of these procedures are done on an outpatient basis. IV propofol and lidocaine provide adequate anesthetic in most cases.
- Midazolam and fentanyl should only be given in cases of extreme anxiety or opioid tolerance or withdrawal. Administering these medications indiscriminately will increase discharge times and have little added benefit.
- Intubation is generally not necessary on most cases unless ventilation becomes an issue. Prone procedures such as ERCPs can be done without an ETT in many cases, but success depends on provider experience and pt co-morbidities with obesity and airway anatomy being a major factor.
- Airway adjuncts, esp. the nasal trumpet, maintain airway patency and help avoid the need for an ETT.

Surgical Stages
- Colonoscopy: Endoscopic passage through the sigmoid and splenic flexures tend to be the most stimulating and deepening of the anesthetic should be considered.
- Upper endoscopies: Passage through the oropharynx and dilation (if applicable) are most stimulating and laryngospasm and bronchospasm are risks, esp. in smokers, so a deeper level of anesthetic is required for this population.
- Procedures can be very short or last 2 hr depending on complexity and pt disease.

Postoperative Considerations
- Recovery nearby, standard postop monitoring, recovery for some procedures are far longer than needed for anesthetic recovery.

Anticipated Problems/Concerns
- Postop pain control for some procedures
- Chest or abd pain may suggest procedure complication
- Coughing 2° to airway irritation with upper endoscopies

Blood Loss and Volume Concerns
- Significant blood loss is rare except for procedures involving varices where caution is advised and a type and cross may be prudent.
- Plavix and other anticoagulants may be of concern depending on the procedure.

Worry About
- Airway obstruction
- Bleeding
- Perforation of visceral structures

Perioperative Risks
- Risks are more dependent on the pt’s co-morbid conditions.
- Airway risk
- Vagal response with insufflation
- Perforation leading to pneumoperitoneum or pneumomediastinum: Rare
- Dental injury
- Sore throat
- Bronchospasm and laryngospasm

Risk
- Varied risks depending on disease process

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Airway obstruction and cannot intubate</td>
<td>OSA, obesity, difficult mask and intubation</td>
<td>Airway exam</td>
<td>Poor dentition</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchospasm and laryngospasm</td>
<td>Asthma and smoking</td>
<td>Auscultation</td>
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<tr>
<td>GI</td>
<td>Aspiration risk</td>
<td>Hx of emesis, gastroparesis, gastric outlet syndrome</td>
<td>Active emesis or hematemesis</td>
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<tr>
<td>HEME</td>
<td>Anemia</td>
<td>Hematemesis or melena</td>
<td>Tachycardia, pale skin and mucous membranes</td>
<td>PCV or CBC</td>
</tr>
</tbody>
</table>
Bernard Wittels

GIFT Procedure

Risk
- Infertility affects 3 million couples in USA. 1 of every 100 deliveries is the culmination of assisted reproductive technologies.
- GIFT is the preferred technique for the most complicated, high risk, infertile women, incl those with advanced age, obesity, polycystic ovarian syndrome, tobacco use, marijuana use, early stage ovarian malignancies, and hypoplastic uterus assoc with a 46,XY karyotype.

Perioperative Risks
- Periop mortality is rare
- Obstetric-related risks
  - OHSS, follicular rupture, hemorrhage, pleural effusion, ascites, thromboembolism
  - Failed fertilization 70%
  - Multiple gestation 33%
  - Ectopic pregnancy 0.4%
  - Birth defects 2%
  - Neonatal death 1%
- Anesthesia-related risks
  - Conscious sedation: Awareness, anxiety, pain, nausea (20–80%), movement

Worry About
- Potential effects of drugs on oocyte fertilization, implantation, growth, development
- CO₂ insufflation requires hyperventilation
- Residual peritoneal CO₂ causes diaphragmatic, subcapsular pain
- Laparoscopy can injure bowel, bladder, cause air embolism or subcutaneous emphysema

Overview
- Young, female outpatients have an increased risk of postop nausea, emesis.
- Laparoscopy increases ventilatory demands and may influence the choice of anesthesia.

ICD-9-CM Code 628.9 (infertility, female)

Indications and Usual Treatment
- 1° or 2° infertility
- Endometriosis
- Ovulatory disorders

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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Increased workload</td>
<td>Asthma, smoking, obesity</td>
<td>Auscultation</td>
<td>Pulse oximetry</td>
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<tr>
<td>GI</td>
<td>Adhesions</td>
<td>Previous abd surgery</td>
<td>Scar survey</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Tubal patency</td>
<td>Previous infection, surgery</td>
<td>Hysterosalpingogram</td>
<td></td>
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</tbody>
</table>


Perioperative Management

Anesthetic Techniques
- Conscious sedation….. total IV anesthesia
- Spinal, epidural and/or GA for laparoscopy or laparotomy

Monitoring
- Routine

Induction/Maintenance
- Propofol, N, O, midazolam, fentanyl, and iso-flurane do not alter success rates for pregnancy or delivery.
- Consider avoiding metoclopramide and droperidol to avoid PONV.

Surgical Stages

Trocars Introduction
- Adhesions of intestines to anterior abd wall increase risk of bowel perforation.
- Traumatic trocar placement may perforate bowel bladder, blood vessels.

CO₂ Insufflation
- Increased intragastric pressure, increased CO₂ absorption requires increased ventilation (increase rate to avoid barotraumas with increased PIP)

Postoperative Considerations
- Mild abd pain after laparoscopy; ibuprofen and fentanyl usually suffice

Anticipated Problems/Concerns
- Excessive postop pain warrants evaluation for peritoneal trauma and bleeding.
- Unexplained hypotension and tachycardia may herald endotoxic shock and sepsis that must be diagnosed and treated emergently to avoid maternal mortality.

- Male-factor infertility
- Unexplained infertility
- Specific indications for GIFT
  - Advanced maternal age
  - Donor insemination failures
  - Repeat IVF failures
  - Difficult transcervical embryo transfers
  - IVF not acceptable due to religious beliefs

Criteria
- Ovulation (serum LH, U/S)
- Adequate luteal phase (serum progesterone)
- Normal endometrium (biopsy)
- Normal TSH, T₄, prolactin
- Tubal patency (laparoscopy or hysterosalpingogram)
- Normal semen analysis
- Usual treatment
  - Ovarian hyperstimulation (clomiphene, hCG, GnRH analogs)
  - Chromotubation, tuboplasty
  - In vitro fertilization
Heart Transplant, Adult

Wanda M. Popescu

Risk

• Incidence in USA: ~14,000 candidates/y with stage D heart failure (HF); most common Dx are ischemic cardiomyopathy and idiopathic dilated cardiomyopathy.
• ~2000 orthotopic procedures/y in USA, limited by suitable donor organ availability
• Overwhelmingly male; no unambiguous racial predominance for end-stage HF

Perioperative Risks

• Early (30-d) mortality: ~8% due to surgical technique complications; fulminant rejection or infection; reperfusion injury
• Early morbidity from nosocomial bacterial infection (Pneumococcus pneumonia, Pseudomonas sepsis); later opportunistic infection with Pneumocystis carinii, Candida spp, CMV

Worry About

• Recipient heart: Low cardiac index (evaluate the current level of inotropic support); ventricular irritability; mediastinal adhesions from prior cardiac surgery; pulm Htn
• Donor heart (allograft): Function compromised after CPB by increased pulm afterload, reperfusion injury, prolonged ischemia (decreased function after 4–6 hr), and atypical drug responses

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Biventricular failure</td>
<td>Exercise intolerance</td>
<td>JVD, Liver edge ↓</td>
<td>R &amp; L heart cath</td>
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<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>Orthopnea</td>
<td>Rales</td>
<td>CXR</td>
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<tr>
<td>RENAL</td>
<td>Prenral azotemia</td>
<td>PND, nocturia</td>
<td></td>
<td>BUN/Cr</td>
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<tr>
<td>CNS</td>
<td>Poor perfusion</td>
<td>Confusion?</td>
<td>Mental status</td>
<td></td>
</tr>
<tr>
<td>HEPAT</td>
<td>Chronic congestion</td>
<td>RUQ fullness/pain</td>
<td>Liver edge</td>
<td>LFTs</td>
</tr>
</tbody>
</table>


Intraoperative Management

Monitoring

• Standard ASA monitors and arterial line cannula
• PA cath with long sheath (to facilitate withdrawal during cardiac anastomoses, use of caval snare); placement may be complicated by arrhythmias, TR
• TEE useful to optimize volume management, rule out mural thrombus and PFO pre-CPB; assist in maintaining stable hemodynamics pre-CPB, assist de-airing during CPB; evaluate RV and LV function and assist weaning off CPB

Airway

• Bacterial filter may be placed in anesthesia circuit to minimize nosocomial pneumonia risk

Blood Products

• Only CMV-neg blood products used for seronegative recipients, to avoid CMV sepsis
• Leukocyte-filtered or γ-irradiated blood unnecessary to avoid alloimmunization during transplant
• FFP and/or vit K may be required if on chronic warfarin therapy preop
• Significant bleeding and coagulopathy may occur in pts with prior cardiac procedures

Anesthetic Technique

• Judicious use of IV premedication
• Immunosuppressive drugs are often infused ASAP after arrival in OR
• Specialized antibiotic therapy
• Antifibrinolytic therapy initiated pre-CPB
• GA regimen should be least perturbing to precarious CV status; full-stomach precautions
• Phylephrine, inotrope, judicious volume infusion may then be required to optimize CO

Surgical Stages

Dissection

• Cardiomegaly and/or prior cardiac surgery increase risk of RV or innominate vein laceration
• Epicardial irritability can lead to V fib
• Redo surgery is assoc with protracted pre-CPB interval
• With LV mural thrombus, cardiac manipulation can lead to systemic embolism

Definitive Surgery

• Bivacumal cannulation for CPB mandatory due to atrial anastomoses; SVC pressure must be scrupulously monitored to avoid intracranial Htn
• Cardiectomy not performed until just before donor organ arrival
• After atrial, great vessel anastomoses, aortic cross-clamp removed ASAP to end ischemia; methylprednisolone 500 mg IV used to prevent hyperacute rejection

Weaning/Post-CPB Considerations

• Inotropic support: Usually required (in case of prolonged ischemic time or reperfusion injury)
• Chronotropic support: Due to heart denervation direct acting drugs are required (isoproterenol) to increase HR; alternatively pacing the heart at HR 90–110 beats/min

Indications and Usual Treatment

• Specific indication: Stage D HF (refractory HF requiring specialized interventions) and NYHA Class IV (severely compromised status with guarded prognosis) unimproved by maximal medical Rx
• Maximal medical Rx usually incl oral inotropes, ACE, PDE III inhibitors, diuretics, antiarrhythmics
• Absolute contraindication: Fixed pulm Htn (pulm vascular resistance >3.5 WU that does not decrease >20% with pharmacologic intervention)
• Relative contraindication: Severe pulm disease, chronic kidney failure, liver failure, malignancy within last 5 y, active infections, advanced age

Anticipated Problems/Concerns

• Atypical responses to cardioactive drugs by denervated donor heart—e.g., indirect-acting agents (e.g., atripine) fail to produce expected cardiac effects (tachycardia); thus only direct-acting agents should be used (e.g., isoproterenol); but denervation supersensitivity to catecholamines, a theoretical concern, is NOT clinically relevant
• Persistently slow functional rhythms lead to permanent pacemakers in ~5% of recipients
• Acute rejection is marked by low CO, arrhythmias; monitoring for rejection requires biopsies
• Chronic immunosuppression prone pts to infection and leads to the development of Htn, CAD, DM, kidney disease
Heart Transplant, Pediatric

Risk
- More than 8000 heart transplants have been performed in pts under 18 y of age from 1982 to 2007

Perioperative Risks
- The highest risk for death following a transplant is within the first 6 mo of transplant. Survival is also influenced by indication for transplant (worse with CHD), donor age (worse with older donors) and the volume at the transplant center.
- At risk for rejection and developing allograft vasculopathy.
- Htn is common in post-transplant pts.

Worry About
- Psychosocial difficulties from chronic illness
- Drug interaction with immunosuppressants
- Airway problems from steroid therapy
- Rejection and abn cardiac function
- Other end, organ dysfunction incl renal and 2º malignancies
- Other co-morbid conditions, particularly Htn
- Vascular access could be a challenge
- “Denervated heart,” therefore the use of direct acting agents is recommended

Overview
- Over the past 3 y, there is an average of 450 pediatric heart transplants per year. 27% of transplants are performed in infants (<1 year of age) in North America

ASSESSMENT POINTS

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<tbody>
<tr>
<td>Airway</td>
<td>Post-Tx may have difficult airway due to chronic steroid use</td>
<td>Hx of previous anesthesia</td>
<td>Airway exam</td>
<td>ECHO</td>
</tr>
<tr>
<td>CARDIAC</td>
<td>Possible need to have alternate cannulation site for CPB (i.e. femoral)</td>
<td>Functional status</td>
<td>Routine cardiac exam</td>
<td>ECHO Reports of recent cardiac evaluation</td>
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<tr>
<td></td>
<td>Slow onset of drug effects due to poor cardiac function</td>
<td>Current meds</td>
<td>Evaluate vascular access</td>
<td>MR</td>
</tr>
<tr>
<td></td>
<td>Exacerbated hemodynamic depression from sympatholytic or cardiodepressant drugs</td>
<td>Most recent cardiac evaluation (ECHO? Cath? MRI?)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Recent infections</td>
<td>Bronchodilator therapy</td>
<td>Routine resp exam</td>
<td>RSV titer if pt has active resp infection</td>
</tr>
<tr>
<td>IMMUNE (PTLD)</td>
<td>Post-Tx pts are at risk for lymphoproliferative diseases (PTLD)</td>
<td>Clinical presentation of PTLD is variable and could mimic viral illness</td>
<td>Creatinine LFT</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>Any renal or hepatic system dysfunction</td>
<td></td>
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</tbody>
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Perioperative Implications

Preoperative Preparation
- Careful review of Hx incl cardiologist’s note and recent ECHO is very important.
- Psychosocial concerns for child with Hx of frequent hospitalization necessitate possible need for premedications.
- Depending on the procedure, SBE prophylaxis may be indicated.

Monitoring
- Routine
- Unless there are signs of rejection or other major end-organ dysfunctions, invasive monitoring is not usually needed.
- If child has difficult vascular access, this may further exacerbate poor volume status from preop NPO.

Induction
- Ascertain volume status is adequate.
- Children tolerate low heart rate poorly. Bradycardia often precedes cardiac arrest.
- Pts for transplant have no cardiac reserve and onset of drug action may be slow due to poor function.
- Etomidate with small dose of opioids.
- Combination of benzodiazepines and opioids could lead to significant sympatholytic effects and unstable hemodynamics during induction.

Maintenance
- Remember to use only direct-acting agents in post-Tx pts.
- Infusion of opioid in combination with low dose of volatile anesthetics is usually well tolerated.
- Postop care should incl involvement of the cardiologist, appropriate pain management and possible ICU care for major surgery.

Anticipated Problems/Concerns
- Vascular access in pts who had multiple previous surgery
- Bleeding and hemostasis in pts who had previous surgery
- Donor heart function and ischemic time are important determinants on the success of the transplant
- Know all of the medications and their potential interactions
- Rejection and PTLD are two major causes for mortality/morbidity
Herniorrhaphy

Risk
- Groin hernias: 750,000/y
- Annual risk of hernia incarceration: 2–3/1000 pts/y.
- Incidence: Groin 75% majority of hernias
  - Incisional hernias 15–20%
  - Umbilical and other ventral hernias comprising the remainder
- Gender predominance
  - Inguinal: M:F ratio: 9:1
  - Femoral: M:F ratio: 1:3
- Abd: M:F ratio: 7:13

Perioperative Risks
- Periop mortality rare (<0.3%)
- Higher mortality/morbidity if strangulated bowel
- Risk related to co-morbidities
- Morbidity: Wound abscess, hematoma

Worry About
- Appropriateness of surgery as outpatient
- Possible strangulated bowel, sepsis
- Vagal stimulation with retraction, resultant bradycardia
- Postop urinary retention
- Spinal headache: Incidence ≤3% with 25-gauge needle

Overview
- An external hernia is an abn protrusion of intra-abdominal tissue through a fascial defect in the abd wall.
- The definitive treatment of hernia is operative repair.
- Procedure performed for repair of abd wall (epigastric, femoral, incisional, umbilical)
- Reducible hernia: Contents of sac return to abd spontaneously or with manual pressure
- Irreducible (incarcerated) hernia: Contents cannot be returned to the abd, usually trapped by a narrow neck. Incarceration does not imply obstruction, inflammation, or ischemia of the herniated organs.
- Obstructed: Lumen of a segment of bowel within the hernia sac may become obstructed; initially there may be no interference with blood supply.
- Strangulated hernia: Compromise to the blood supply of the contents of the sac (e.g., omentum or intestine) results in gangrene of the contents of the sac. The incidence of strangulation is higher in femoral than in inguinal hernias, but strangulation may occur in other hernias as well.
- Richter hernia: Uncommon and dangerous, part of circumference of bowel incarcerated or strangulated in fascial defect. Strangulated Richter hernia may spontaneously reduce and gangrenous piece of intestine may be overlooked with subsequent perforation and peritonitis:
  - May be un-complicated or may be complicated with bowel contents
  - Strangulated hernia with necrotic bowel can be assoc with sepsis syndrome
  - May require bowel resection if necrotic
  - Laparoscopic hernia repair currently performed: exact role poorly defined

ICD-9-CM Codes: 550–553.9

Indications and Usual Treatment
- Uncomplicated hernia: Elective surgery, binder, truss
- Incarcerated hernia: Urgent surgery
- Strangulated hernia: Emergent surgery
- Absolute contraindications to elective repair incl pregnancy, active infection
  - Acute repair difficult: Higher likelihood of complications
  - Incarceration: 9–20%. 10% of inguinal and 20% of femoral hernias incarcerated
  - Frequent in children <6 mo
  - Incarcerated hernias can cause intestinal obstruction or strangulation and infarction, resulting in a high incidence of infection, hernia recurrence, and operative mortality, esp. in elderly pts
  - In absence of signs of strangulation: Can usually be manually reduced followed by elective repair

Perioperative Implications

Preoperative Preparation
- Determine appropriateness of outpatient procedure.

Anesthetic Technique
- Local, regional, spinal, GA, or combined anesthetic technique
  - Local anesthesia ± sedation preferred for appropriate cases [MAC]
  - Local anesthesia permits pt to strain, cough during procedure if desired by surgeon
  - Consider local wound or nerve infiltration for postop pain control.
- Laparoscopic surgery, strangulated hernia require GA. Factors that determine choice of anesthesia incl pt and surgeon preference, type of procedure performed (open or laparoscopic), hernia characteristics (recurrent, large, slider, bilateral), and the pt’s ability to cooperate.
- GA for pts with questionable airways and those unable to cooperate (e.g., because of dementia) or for more challenging cases

Monitoring
- Routine
- Consider arterial line, CVP, PA cath if signs of sepsis

Airway
- Routine

Induction
- Require level of at least T8 for regional
- Local infiltration of ilioinguinal, iliohypogastric nerves

Surgical Stages

Dissection
- Depends on hernia site

Definitive Surgery
- Inguinal hernia most common: Transversalis aponeurosis, internal oblique fascia sutured to shelving edge of inguinal ligament
  - Prosthetic mesh can be used for all sites to relieve tension
  - Incisional hernia may require intraperitoneal approach
- EBL: 50–100 mL

Hernia Repair Options
- Open
- Laparoscopic

Postoperative Considerations
- Postop pain depends on site, use of local infiltration
  - Pain score: 3 (local)–6
  - Important to void before discharge if outpatient
  - Consider stool softener for inguinal hernias to avoid strain

Anticipated Problems/Concerns
- Bradycardia during peritoneal retraction
- Potential for necrotic bowel
  - Recurrence rates (1–10%). Nearly 40,000 inguinal hernia repairs performed annually for hernia recurrence. Nearly 90% of 1° adult hernia repairs have a prosthetic mesh implanted.
  - Vascular injuries: In laparoscopic repairs, inferior epigastric, external iliac, femoral, and testicular vessels are at risk.
  - May result in intraop hemorrhage or may present as postop hematomas
  - Incidence of postop hematoma: 1 to 8%
Hip Fracture Repair

Meg A. Rosenblatt
John G. Hagen

Risk
- Young pts: Traumatic fracture
- Elderly pts: Femoral neck, intertrochanteric, subtrochanteric, intracapsular Fx through osteoporotic bone
- Pathologic Fx
- Incidence in USA: 340,000 Fx diagnosed and treated / y
  - 22.5-23.9/100,000 at age 50
  - 630.2-1289.3/100,000 at age 80
- By 2050 expected to exceed 500,000 in USA and 7 to 21 million worldwide
- M:F ratio: 1:4–5

Perioperative Management

Preoperative Preparation
- Early surgery indicated in premorbidity fit pts (greater than 48 hr post-admission assoc with increased morbidity and mortality in low risk/healthy pts)
- Surgery delayed if correctable co-morbidities
- Treat preop pain: Consider femoral block, lumbar plexus catheter with single-shot sciatic block, fascia iliaca blocks, analgesic adjuvants (NSAIDs, opioids)

Anesthetic Technique
- No association between type of anesthesia and postop mortality after 30 d, spinal anesthesia assoc with reduction in postop confusion
- Increased use of vasopressors and arrhythmia assoc with neuraxial anesthesia
- Regional anesthesia reduces incidence of DVT, intraop blood loss and need for airway manipulations
- Isobaric spinal anesthesia may limit level of sympathetamcy, thus hypotension
- Paramedian approach to neuraxial blocks 2° to inability to reduce lumbar lordosis
- US may improve success/decrease time to perform neuraxial blocks
- Prophylactic antibiotics mandatory

Monitoring
- Consider arterial monitoring if underlying severe Htn, pulm Dx, or hypovolemia.
- PA cath if significant cardiac disease
- Urinary drainage catheters: retention vs. UTIs

Perioperative Risks
- Cardiac, CNS, accident and/or fall: Seek cause of Fx
- Periop fluid deficiency: Large volumes of blood in leg or thigh after fracture
- Geriatric pts with multiple co-morbidities

Worry About
- Fat embolism syndrome

Overview
- Commonly performed procedures
- Goal is reduction and stabilization of Fx to allow mobilization
- 30-d mortality 5–10%
- 1-y mortality 12–37%

I CD-9-CM Code: 820.x

ASSESSMENT POINTS

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<th>Test</th>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Hypotension 2° to hypovolemia vs. fat embolism</td>
<td>Tachycardia</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Fat embolism syndrome</td>
<td>Right-sided heart failure</td>
<td>ST-segment abn</td>
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<tr>
<td>HEME</td>
<td>Blood loss 2° to Fx</td>
<td>Orthostatic hypotension</td>
<td>PA pressures, ECHO</td>
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<tr>
<td>CNS</td>
<td>Senile dementia vs. fat embolism syndrome</td>
<td>Confusion, agitation, stupor, coma, cerebral edema</td>
<td>Neuro assessment</td>
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<td>GU</td>
<td>Fat embolism syndrome</td>
<td></td>
<td>CT scan</td>
</tr>
<tr>
<td>DERM</td>
<td>Fat embolism syndrome</td>
<td>Petechiae on chest, extremities, conjunctivae</td>
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</tr>
</tbody>
</table>


Surgical Stages

- Performed on Fx table: Allows manipulation of Fx and radiographic evaluation
- Arns frequently across chest or in overhead dings: avoid antecubital IVs
- Avoid compression injuries from perineal post
- Lateral position for hemiarthroplasty
- Dependent shoulder compresses brachial plexus: Place auxiliary roll
- Meticulous padding of pressure points
- Embolism of air, fat, or bone fragments during insertion of femoral prosthesis with assoc systemic hypotension and pulm Htn
- Aggressive warming to decrease blood loss
- EBL <100 to >500 mL

Postoperative Considerations

- Pain management
- Regional: Neuaxial (epidural), peripheral (lumbar plexus catheter)
- Opioids: IV PCA→PO
- Adjuvants: NSAIDs (PO/IV), Opioids
- Multimodal approach emphasizing reg + NSAID adjuvants → opioids
- Decreased hospitalization, increased joint ROM, increased analgesia
- Decreased opioid requirements
- Decreased urinary retention, decreased post-op ileus (N/V)
- Thromboembolic prophylaxis
- Recommended agents
  - Warfarin: INR of 2.0–3.0; <2.0 not effective in preventing VTE

- Non-displaced neck Fx treated with closed reduction and percutaneous pinning
- Displaced neck Fx treated with bipolar hemiarthroplasty or THR
- ORIF with dynamic hip screws or other sliding nail fixations for inter- or subtrochanteric fracture
- Cemented or uncemented hemiarthroplasty prostheses for intracapsular Fx

- LMWH: Enoxaparin: 40 mg SQ q d; dalteparin 5,000 IU SQ q d
- Fondaparinux: 2.5 mg SQ q d, superior to enoxaparin in preventing VTE; however, long half-life, lack of easy reversibility, contraindicated in pts <50 kg and renal insufficiency, ↑ risk of bleeding in pts >65
- ASA/low-dose UFH: Not to be considered as sole agents
- Mortality rate in assoc with poorly controlled systemic disease, cognitive disorders, and absence of DVT prophylaxis
- Daily protein supplementation for malnourished pts
- Pressure ulcer prevention: Frequent turning, visual inspection, and topical duoderm treatment
- Pressure-decreasing mattresses
- Multidisciplinary approach using skilled medical, nursing, and paramedical care to maximize rehabilitation potential

Anticipated Problems/Concerns

- Be wary of elderly pts with normal-range Hcts.
- Fat embolism syndrome may be 2° to direct release of fatty acids, which cause capillary endothelial breakdown. Pericapillar hemorrhagic exudates are found in lungs and brain. Require supportive care, often incl mechanical ventilation, and vigilant fluid management.
Hypospadias Repair

**Risk**
- 1 in 250 live male births
- Hypospadias found in 6–8% of fathers of affected boys
- Hypospadias found in 14% of sibling of affected boys
- 7–9% assoc with cryptorchidism
- 9–16% assoc with inguinal hernia and hydrocele
- Classification
  - 50% distal hypospadias
  - 30% mid shaft hypospadias
  - 20% proximal hypospadias

**Perioperative Risks**
- Complication rates
  - 5% for distal repairs
  - 10% for mid shaft repair
  - 15–20% for proximal repair
- Postop complications
  - Bleeding and/or hematoma (most common)
  - Wound infection
  - Repair breakdown

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>ENDO</td>
<td>Intersex state with adrenal dysfunc</td>
<td>Family Hx of consanguinity, unexplained infant deaths, females who are infertile or have amenorrhea</td>
<td>Micropenis, cryptorchidism, proximal hypospadias</td>
<td>If high suspicion, karyotype US abd and pelvis Assessment of adrenal steroids Electrolytes, glucose</td>
</tr>
<tr>
<td>HEME</td>
<td>Bleeding</td>
<td>Recent NSAID ingestion? Family Hx of bleeding disorder?</td>
<td>Bruising Petechiae</td>
<td>Coagulation test if positive family or positive physical exam</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- May have clear fluids up to 2 hr before surgery
- Normal child does not require laboratory work.
- Mostly done as outpatient surgery
- Discuss with surgeon the need to obtain graft from other sites, e.g., buccal mucosa
- Explain risk and benefits to parents if regional analgesic technique, esp. if caudal, is used

**Monitoring**
- Routine ASA monitors plus temp monitor

**Anesthetic Technique/Induction**
- General anesthesia
- Have available thermoregulation capabilities, e.g., Bair hugger, overhead warming lights, and heating blankets
- Usually inhalational induction with $\text{N}_2\text{O}/\text{O}_2$/sevoflurane
- Intubate/LMA with positive airway ventilation/spontaneous breathing depending on length of operation
- May need antibiotic coverage esp. if urethral catheterization
- Discuss with surgeon regarding administration of IV ketoroloc because this may increase the risk of bleeding postop.
- Supplement with regional analgesic technique:
  - Caudal for proximal hypospadias repair or distal/midshaft hypospadias repair with assoc orchiopexy/inguinal hernia repair
  - Dorsal penile nerve block, SQ penile ring block or caudal for distal or mid shaft hypospadias repair.
  - No epinephrine additive for dorsal penile nerve and SQ ring block

**Surgical Stages**
- Operative time 1 hr for distal 2–3 hr for proximal repairs
- Minimal blood loss intraop

**Indications and Usual Treatment**
- Correction of deformities that interferes with the function of urination and/or procreation
- Usually operated at 6–12 mo of age
- Surgical treatment dependent on classification of hypospadias.
- Usually one-stage approach for distal and mid-shaft hypospadias
- May require two or more stages for proximal hypospadias

**Etiology**
- Multifactorial
- Insufficient androgen production
- Limited androgen sensitivity in target tissues of developing genitalia
- Premature cessation of androgenic stimulation

**Postoperative Considerations**
- Pediatric age group at risk for N/V
- Postop pain relief with caudal 0.25% bupivacaine + epinephrine 1:200,000 at 1 mL/kg will last for about 4–6 hr. May repeat half dose at lower concentration, single shot caudal at end if surgery lasted 2 hr or more.
- If catheter in situ, may need oxybutynin to relieve bladder spasms.
- Discharge per post anesthetic criteria
Hysterectomy, Vaginal

Risk
- Second most common female surgery after C-section
- Incidence in USA: 600,000 hysterectomies performed in 2003
- More than 120,000 vaginal hysterectomies performed in USA in 2003

Perioperative Risks
- Cystotomy: 1.5%
- Ureteric injury: <0.1%
- Vesico-vaginal fistula: <0.1%
- Bowel injury: 0.6%
- Hemorrhage causing return to OR: 3.1%
- Reoperation: 0.4%
- Transfusion: 1.8%
- Hematoma: 0.5%
- Jejun/obstruction: 0.2%
- Infection: 1.9%
- Thromboembolism: <0.1%
- ICU admission: 0.7%
- Readmission: 1.1%

Worry About
- Indication for procedure
- Operative diagnosis
- Lithotomy positioning

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<tbody>
<tr>
<td>CARDIO</td>
<td>Dehydration, electrolyte abn from bowel prep</td>
<td>CHF, CAD, SOB, exercise tolerance</td>
<td>Cardiac exam</td>
<td>Basic chemistry</td>
</tr>
<tr>
<td>PULM</td>
<td>Pleural effusion or other lung pathology in cancer pts</td>
<td>Orthopnea, SOB, Cough</td>
<td>Chest exam</td>
<td>CXR, O₂ sat</td>
</tr>
<tr>
<td>HEME</td>
<td>Consider anemia in pts w/Hx of abn bleeding</td>
<td></td>
<td></td>
<td>CBC</td>
</tr>
</tbody>
</table>


Perioperative Implications

Monitoring
- Standard ASA monitors
- Foley catheter may be needed if neuroaxial blockade is performed.
- May require arterial line and/or central venous access if significant blood loss is anticipated or if the pt has major medical co-morbidities.

Airway
- Increased aspiration risk from increased abd pressure and Trendelenberg
- May require orogastric suctioning to minimize aspiration

Anesthetic Technique
- General anesthesia is most common with standard induction.
- Spinal, epidural, combined spinal-epidural is appropriate for euvolemic pts with a simple hysterectomy
- Will need a sensory level of T8–T6
- Pencil-point needle is preferred to reduce incidence of postdural puncture headache

- CO₂ insufflation for laparoscopic-assisted vaginal hysterectomy (LAVH)
- Possible hidden and/or unrecognized blood loss

Overview
- Preferred over abd approach because 2–4 x less risk of morbidity and mortality, though the vaginal approach is limited by pelvic adhesions, size of uterus, pelvic anatomy, and location of gynecological cancers that require abd approach
- Laparoscopic assistance offers improved surgical exposure and visibility of pelvic anatomy, and ability to remove adhesions
- Robotic assistance enables three-dimensional visualization, improved surgical articulation, and magnification
- Bilateral salpingo-oophorectomy may be performed in addition to hysterectomy in pts >45 y for ovarian cancer prophylaxis.

ICD-9-CM Codes (2010)
- 681.1 (Uterine prolapse); 625.3 (Dysmenorrhea); 215.6 (Uterine myoma);
- 617.9 (Endometriosis); 233.2 (Endometrial hyperplasia); 182.0 (Endometrial cancer);
- 179.0 (Uterine cancer)

Indications
- Uterine myoma
- Pelvic relaxation syndrome
- Pelvic pain 2° to endometriosis or adhesions
- Abn bleeding incl dysfunctional uterine bleeding
- Dysmenorrhea
- Endometrial hyperplasia
- Gynecological cancers incl uterine cancer

Contraindications
- Uterine enlargement
- Nulliparity and minimal uterine descent
- Need for oophorectomy
- Previous abdominopelvic surgery and extra-uterine surgery
- Cardiac or pulm co-morbidities complicated by CO₂ insufflation and Trendelenberg position

Usual Treatments
- Medical treatments may incl oral contraceptive pills, gonadotropin releasing hormone analogues, and progesterin inhibitors.

- Extra-abdominal insufflation with sufficient amounts may cause cardiopulmonary arrest or SQ air compromising the airway
- Volume overload is a risk when fluids are used to manage hypotension from the general anesthetic and intraperitoneal insufflation.

Extubation
- Awake

Adjuncts
- High risk of PONV can be treated with intraop ondansetron, metoclopramide, dexamethasone

Postoperative Period
- Shoulder pain from diaphragmatic irritation by CO₂
- Increased risk of postop N/V secondary to female gender and genitourinary surgery
Hysteroscopy

**Risk**
- Pt population incl women of all ages.
- Overall rate of complications is 0.01% for diagnostic hysteroscopy.

**Perioperative Risks**
- Operator technique
- Hemorrhage: 1.2%–3.5%
- Uterine perforation: 0.8%
- Bowel and/or bladder injury
- Infection
- Air or gas embolism
- Hyponatremia
- Hypo-osmolarity
- Pulm edema
- Cervical lacerations
- Nerve injury related to lithotomy position

**Worry About**
- Unrecognized preop acute/chronic anemia
- Potential injury related to positioning
- Potential complications related to chosen distention media

**Overview**
- Allows for direct visualization of uterine cavity through distention
- Typically performed in ambulatory and office-based settings
- Has allowed for reduced hospital stays, earlier recovery periods and lower postop morbidity
- Each distention media has its own inherent risks
- With appropriate monitoring, those risks can be minimized

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<tr>
<td>CARDIO</td>
<td>Obesity/comorbidities, Trendelenburg position</td>
<td>Exercise tolerance, SOB, Hx of cardiopulmonary Dz</td>
<td>Auscultation</td>
<td>ECG, stress test</td>
</tr>
<tr>
<td>RESP</td>
<td>If obese/Trendelenburg</td>
<td>Orthopnea</td>
<td>Auscultation</td>
<td>O₂ sat</td>
</tr>
<tr>
<td>HEME</td>
<td>Chronic/acute blood loss</td>
<td>Fatigue, syncope</td>
<td>Orthostatic changes</td>
<td>Hgb/Hct</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Assess co-existing morbidities
- Consider preop transfusion if pt symptomatic
- Consider pretreatment of pt with gonadotropin-releasing hormone (GnRH) agonists to reduce endometrial vascularity

**Monitoring**
- Routine
- If CO₂ insufflation used, monitor ETCO₂
- Monitor peak airway pressures if Trendelenburg position utilized.
- Avoid steep Trendelenburg position
- With significant CV disease, consider invasive monitoring because of assoc risks with use of fluid distention media.

**Anesthetic Technique/Induction**
- Local anesthetic: Paracervical versus intracervical block
- Monitored anesthetic care: Narcotic-propofol infusion versus bolus technique
- Regional/general anesthetic: Consider LMA unless Trendelenburg position required because of increased risk of gastric aspiration. Induction should be tailored appropriately if pt significantly anemic.
- Tailor the anesthetic to accommodate the ambulatory, office-based nature of the procedure.

**Postoperative Considerations**
- Monitor for excess bleeding.
- Monitor the irrigation fluid administered. If greater than 500 mL deficit, measure serum electrolytes. If greater than 1000 mL deficit, administration furosemide and measure serum electrolytes. If greater than 2000 mL deficit, terminate procedure
- Pt population prone to PONV: Recommend anti-emetic prophylaxis
- Pain score: 1–3; NSAIDs useful as an adjuvant for opioid-sparing effect and reducing PONV

**Indications and Usual Treatment**
- Abnormal bleeding
- Intra-uterine adhesions
- Endometrial polyps and submucosal fibroids
- Uterine septum
- Endometrial resection/ablation
- Sterilization
- Missed IUCD (Intra-uterine contraceptive device)
- Infertility evaluation
- Contraindicated for pregnancy, genital tract infection and uterine carcinoma

**ICD-9-CM Code:** 621.3 (Endometrial hyperplasia); 626 (Disorders of menstruation); 628 (Infertility, female)

**Anticipated Problems/Concerns**
- To help prevent gas embolization, gaseous insufflation equipment must limit flow rate to 100 mL/min and deaerate the supply tubing by flushing with CO₂.
- If glycine is used as the fluid distention media, monitor for possible electrolyte imbalance (hyponatremia, hypokalemia, hypocalcemia, and hyponatremia) and development of female TURP syndrome
- If sorbitol and mannitol solution is used, there is still a risk of developing dilutional hyponatremia and female TURP syndrome.
- If dextran 70 is used, monitor for acute anaphylactic shock as well as pulm edema and coagulopathy.
- If physiologic media is used, monitor for fluid overload esp. in pts with cardiac and renal insufficiency. Typically, electrolyte imbalance not a problem.
- Altered levels of consciousness must be evaluated, incl electrolyte determination, particularly if large volumes of distending media were used.
Ileostomy

**Risk**
- Relatively common procedure for adults ages 20–65 y
- There is no gender predominance, although more women receive the procedure for inflammatory bowel disease and more men for colon cancer.

**Perioperative Risks**
- Periop mortality is low (<1%) and is generally assoc with co-existing disease.
- Procedural morbidity incl ileus 5%, wound infection <5%, intestinal obstruction 2–3%, fistula formation 1–3%, and ostomy necrosis <0.5%.
- Higher risk in pts with strangled bowel and cancer-related indications.
- Non-procedural risks incl: Aspiration, electrolyte abn, blood loss, postop pain, nutritional compromise, adrenal suppression from chronic steroid use, infection, and sepsis.
- Blood loss usually less than 200 cc

**Worry About**
- Pulm aspiration if bowel obstruction present
- Periop intravascular volume deficiency
- Electrolyte abn
- Bradycardia from vagal response due to bowel manipulation
- Hypotension and vagal response to insufflation and positioning (laparoscopic procedures)
- Anemia from chronic and acute blood loss
- Hypothermia due to prolonged procedure and radiant/evaporative losses
- Postop pulm atelectasis

**Overview**
- Diverting ileostomy is common procedure to manage intestinal obstruction or trauma to large bowel or lower small intestine or other intraabd process
- Pts with IBD have usually failed chronic steroid and immunosuppressant therapy and often show signs of those interventions.
- Long-term surgical risks incl disease recurrence, adhesions, kidney stones, and gallstones.

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<tr>
<td>CARDIO</td>
<td>Hypovolemia from ↓ fluid intake, diarrhea, bowel prep, third spacing during procedure, acute and chronic blood loss</td>
<td>PO status, vomiting, bowel prep, bleeding, UO</td>
<td>Poor pulses, hypotension with tachycardia, orthostatic BP, skin turgor, dry mucous membranes</td>
<td>BUN/Gr, UO, electrolytes, PCV</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp insufficiency from abd distention/ spliniting and reduced FRC</td>
<td>Dyspnea</td>
<td>Tachypnea, rales, absent BS, abd distention and rigidity</td>
<td>Pulse oximetry</td>
</tr>
<tr>
<td>GI</td>
<td>Possible ↑ intragastric pressure, volume, acidity</td>
<td>Abd pain</td>
<td></td>
<td>X-ray—dilated bowel</td>
</tr>
<tr>
<td>HEME/IMMUNO</td>
<td>Hemoconcentration from dehydration, Blood loss</td>
<td>GI losses</td>
<td>See under CV</td>
<td>Cort stim Albumin, TP</td>
</tr>
<tr>
<td></td>
<td>Immune suppression from chronic steroid therapy</td>
<td>Abn bleeding</td>
<td>PCV, plt, electrolytes PCV</td>
<td>WBC with diff, lactate</td>
</tr>
<tr>
<td></td>
<td>Potential malnutrition from malabsorption</td>
<td>Hemodynamic instability</td>
<td>Febrile, hypotensive</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bacteremia and sepsis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Possible hypokalemic, hypochloremic metabolic alkalosis with vomiting, lyte abn from lower GI losses/bowel prep, hemoconcentration Assoc renal insufficiency in elderly</td>
<td>Vomiting, bowel prep</td>
<td>See under CV</td>
<td>Electrolytes, ABG</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>BUN/CR</td>
</tr>
</tbody>
</table>

**Premedication**
- Consider acid reducer and oral nonparticulate antacid (full stomach or obstruction)
- Consider metoclopramide: Contraindicated if obstruction present
- Consider steroid stress dose if on chronic steroid therapy
- Consider NSAIDs to reduce incidence of mesenteric traction syndrome

**Monitoring**
- Consider arterial catheter if hemodynamically unstable or severe cardiopulmonary disease
- Foley catheter to monitor fluid administration and renal perfusion
- Consider CVP, PA catheter, or TEE to monitor volume status if pt has renal insufficiency or significant cardiac dysfunction.

**Airway**
- Generally routine; consider awake intubation, rapid-sequence or modified rapid-sequence intubation if increased aspiration risk present
- Consider preop NG tube placement and decompression prior to induction if upper obstruction and/or vomiting present

**Induction**
- Consider pre-induction intravascular volume expansion with crystalloid or colloid (RBCs if anemic) for hypovolemia.
- Use reduced dosage of sedative hypnotic agent if significant hypovolemia or myocardial dysfunction present.
- Muscle relaxant chosen for induction technique (RSI or routine) and perceived airway difficulty

**Maintenance**
- May be performed under regional, general, or combined anesthetic techniques depending on surgical approach and pt factors
- NG tube placed to suction reduces gastric distention
- Moderate muscle relaxation usually required to facilitate procedure
- Extra attention to fluid replacement often required
- Convective air and fluid warming indicated to maintain normothermia and reduce infectious complications
- Consider IV lidocaine infusion (2 mg/min) during and after procedure to reduce anesthetic requirements, improve return of bowel function and reduce LOS

**Emergence**
- Extubation depends on usual criteria with emphasis on normovolemia and normothermia
- Moderate postop pain; consider epidural infusion/PCA or IV opiate PCA.

**ICD-9-CM Codes:** 555.9 (Crohn’s disease), 556.5-556.9 (Ulcerative colitis), 560.9 (Intestinal obstruction)

**Indications and Usual Treatment**
- Most common indications are intestinal obstruction or perforation, abd abcess formation, intractable IBD (Crohn’s and ulcerative colitis), abd cancer, adhesions, and intestinal trauma.
- Ileal disease: IBD, small bowel obstruction, volvulus, intussusception, mesenteric vascular occlusion, radiation enteritis, intestinal fistulae, small bowel tumors, Crohn’s disease, trauma
- Large bowel disease: Proctocolectomy for IBD, Crohn’s disease, ulcerative colitis, familial polyposis, neoplasm, trauma
- Surgical approach commonly involved laparoscopic assistance to reduce complications and length of stay
- Surgical alternatives incl both incontinent and continent ileostomy (Kock pouch)

Imperforate Anus Repair

**Risk**
- Incidence: 1 in 500 (minor defect); 1 in 5,000 (major defect) live births
- High lesions (superlevator lesions, when rectum ends above levator muscles) are more common in males (2:1)
- Low lesions (infralevator lesions, when rectum ends below levator muscles) occur with equal frequency in both sexes.
- Runs equally through all racial, cultural, and socioeconomic groups.

**Perioperative Risks**
- The higher the anatomic relation of the terminal bowel to the puborectalis sling of the levator muscle, the greater is the incidence of associated anomalies.
- Additional anomalies present in one-third of pts; incidence higher with suprarelevator lesions.
- Part of VACTERL: Vertebral defects, anal atresia, cardiac anomalies, tracheoesophageal fistula, esophageal atresia, renal anomalies, limb anomalies.
- Gastrointestinal: 10–20% have another GI lesion (e.g., esophageal atresia, intestinal atresia, malrotation, annular pancreas, omphalocele).
- Cardiovascular: 7% have associated CV lesions.
- Vertebral defects: 6% have skeletal lesions such as spina bifida or agenesis of the sacrum; 24% have tethered spinal cord.
- Genitourinary: 25–50% with associated GU anomalies such as urological anomalies, duplicate uterus, septate vagina, and vaginal atresia.

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<tr>
<td>CARDIO</td>
<td>Cardiac anomalies</td>
<td>Cyanosis, F/T</td>
<td>Abnormal heart sounds</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Tracheoesophageal fistula</td>
<td>Excessive salivation, drooling, cyanotic spells, coughing relieved by suctioning</td>
<td>Inability to pass a catheter down the esophagus into the stomach</td>
<td>Plain radiograph of the chest and abdomen reveals air or gas bubbles in the stomach and intestines that have entered through the fistula</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal anomalies</td>
<td></td>
<td></td>
<td>Renal US, voiding cystourethrogram, intravenous pyelogram</td>
</tr>
<tr>
<td>CNS</td>
<td>Vertebral anomalies, spina bifida, tethered cord</td>
<td></td>
<td></td>
<td>Lumbosacral films</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Assess for co-existing anomalies.
- Invertogram: After sufficient time for a transit of gas (>12 hr after birth), the child is placed in an upside-down position for 3 min, after which a lateral view of the pelvis is obtained.
- Cardiac echo to assess for cardiac anomalies.
- Lumbosacral films to evaluate for vertebral anomalies.
- MRI of the spine to look for a tethered cord.
- Renal US, voiding cystourethrogram, and IV pyelogram to evaluate for urinary tract anomalies.

**Monitoring**
- Standard ASA monitoring
- Invasive monitors such as an arterial catheter and a CVP catheter generally are reserved for those with marked cardiorespiratory instability
- Attempt to place IV catheters and pulse oximeter on the upper extremities because pt is often situated at the far end of the OR table.
- Most frequent defect in female pts is vestibular fistula, followed by vaginal fistulas.

**Worry About**
- Abd distention from intestinal obstruction
- Accompanying congenital malformations that affect anesthetic management

**Overview**
- The hindgut comes in contact with the cloacal membrane during the sixth week of fetal development. At this time, the hindgut is divided into a ventral urogenital and dorsal rectal component. By the eighth week, the dorsal half perforates to the exterior. In imperforate anus, the process is arrested during this critical period.
- There are many anatomic variants of imperforate anus. Treatment and prognosis depends on anatomic classification: Suprarelevator (high) and translevator (low). Level of the lesion is determined by abd radiograph or perineal US.
- Marked abdominal distension 2 hours after birth, the child is placed on a horizontal surface. If the child develops air under the skin, the child is placed in an upright position with the abdomen down.
- There are many anatomic variants of imperforate anus. Treatment and prognosis depends on anatomic classification: Suprarelevator (high) and translevator (low). Level of the lesion is determined by abd radiograph or perineal US.
- Presenting forms: Absent or diminished bowel sounds; abdominal distension; vomiting; failure to pass meconium; constipation; dyspnea; cyanosis; signs of sepsis.
- Evaluate for lumbosacral neurologic function. Anal wink can usually be elicited because a vertiginous external anal sphincter is present in most cases.

**Anesthetic Technique/Induction**
- Intubation of the trachea of infants with an apparently normal upper airway can be accomplished with a rapid sequence technique. If the anesthesiologist suspects that the upper airway will be difficult to visualize, the usual airway precautions should be followed. Supplemental support systems for the difficult airway (e.g., neonatal bronchosopes, light wand, LMA) should be available.
- Anesthesia requirements vary depending on the severity of the abd distention and complexity of the surgery: e.g., simple perineal anoplasty, temporary colostomy, or extensive abdominoperineal repair.
- Anesthetic considerations for neonates with intestinal obstruction incl assessment of fluid status (anticipate insensible fluid loss of 7–10mL/kg/d during major portions of the procedure), correction of electrolyte disturbances, treatment of sepsis, and cardiorespiratory evaluation.
- Marked abd distension 2" to intestinal obstruction can impede diaphragmatic excursion and impair ventilation.
- Anesthesia during surgery can incl potent inhalation anesthetics, narcotics, or both. NO should be avoided because of the risk for increased bowel distention. Intermediate- or long-acting nondepolarizing muscle relaxants often improve surgical conditions at lower inhaled anesthetic concentrations.
- If early postop extubation is planned, narcotics should be judiciously administered, but in many cases postop mechanical ventilation is required.
- During surgery, the management of fluids, blood replacement, and electrolyte delivery is important. A major challenge is maintaining an adequate intravascular volume. The presence of radiopaque contrast agents, bowel manipulation, and peritonitis increases third-space fluid requirements. In such cases, 10 mL/kg/hr or more of isotonic saline solution or colloid is frequently...
needed intraop. Monitoring UO, quality of heart tones, HR, and BP is a basic requirement to assess continuing fluid needs.

### Surgical Stages

- Perineal signs in low malformations that will not need a colostomy are: Meconium in perineum, bucket-handle defect, anal membrane, and anal stenosis. These infants can be managed with a perineal anoplasty during the neonatal period with an excellent prognosis.
- High lesions require an emergent temporary diverting colostomy. After sufficient growth of the child, a pull-through procedure with a Peña posterior sagittal anorectoplasty (PSARP) is performed at 3–9 mo of age. The procedure usually begins in the prone position; some surgeons will turn the child and complete the procedure in the supine or lithotomy position. The colostomy is closed after the anoplasty has healed and any necessary 2° dilatation has been completed.
- Laparoscopic-assisted anorectal pull through using laparoscopy and muscle electrostimulation (LAARP) has been developed.
- EBL: Usually 10–20 mL
- Regional anesthesia required for abd surgery: Caudal or lumbar epidural catheter preferred.

### Postoperative Considerations

- Many infants require postop ventilatory support, total parenteral alimentation, CV support, and treatment of sepsis. The function and recovery of the GI system vary enormously among infants with imperforate anus and seem to be related to whether the lesion is isolated and whether the complications of total bowel obstruction have developed.
- Complications of surgery incl stricture of the anocutaneous anastomosis, rectourinary fistula, mucosal prolapsed, constipation, and incontinence.
- After surgery, followup with anal dilatation helps minimize the risk of stricture formation and helps the newly constructed canal to become functional.
- Continence can be attained in 90% if pts have low lesions.
- <50% of pts with high lesions are continent before school age, but most continue to improve and achieve continence by adolescence.
- Highest incidence of incontinence occurs in males with fistula between rectum and bladder.

### Anticipated Problems/Concerns

- Constipation, incontinence
- Management of assoc CHD or other existing medical problems
Implantable Cardioverter Defibrillators (ICDs), Implantation

Nabil Elkassabany
Sanjay Dixit

**Risk**
- Incidence in USA: Each year 400,000 to 460,000 people die of unexpected sudden cardiac arrest (SCA).
- Over 127,000 ICDs implanted in 2008 (data from the national ICD registry).
- The average age for pts undergoing ICD insertion is 68.1 ± 12.8 y.
- M:F percentage is 74/26.
- ICDs were inserted for 1° prevention in 78% of all pts.
- Assoc comorbidities: CAD, 66%; Htn, 74%; diabetes, 36.5%; and chronic lung disease, 22.7%.

**Perioperative Risks**
- Procedure related risks incl cardiac perforation, pericardial effusion, hematoma at the site of insertion, infection, lead dislodgment, hemor- and/ or pneumothorax, and death. Radiation exposure poses another hazard for the health care providers and the pt.
- Pt related risk factors: Pts requiring ICDs often have impaired cardiac function and HF which can place them at higher risk for development of periop complications.
- Anesthesia-related factors incl the risks assoc with delivering anesthesia in a remote location, usually the electrophysiology (EP) lab, and caring for pts who are sicker and have multiple co-morbidities.

**Worry About**
- Myocardial stunning and hemodynamic instability: Low CO, ↓ BP during defibrillation threshold testing (DFT). DFT is defined as the minimal energy necessary for successful defibrillation of induced ventricular fibrillation (VF). This is usually done at the time of the implant to ensure adequate safety margin in the ICDs ability to successfully treat this condition.
- Increase DFT: Sedative, narcotics, antiarrhyth- mics (class IA, B, C—incl lidocaine, propranolol, amiodarone, verapamil); halogenated hydrocarbons; hypothermia; myocardial ischemia; acidosis
- Limited access to the airway of the sedated pt because of the tight space and bulky x-ray equipments.
- Hypoventilation and hypercarbia in sedated pts.
- Side effects of antiarrhythmic medications: e.g., pulse toxicity in amiodarone
- Pts receiving antiocoagulants
- Arrest and cerebral function; some authors found a negative relationship between the number of circulatory arrest periods and the EEG recovery time.

**Overview**
- Advances of the ICD technology resulted in the development of transvenous systems, which can be implanted using percutaneous vascular access in the EP lab, where the pt receives monitored anesthesia care (MAC).
- Components of the ICD system are the generator canister (can), and the lead, which incorporates the shocking coils as well as bipolar for sensing/pacing. The programmer, a stand-alone unit, is used to communicate with the ICD via radio frequency (RF) link. The generator canister is made up of a low-voltage battery unit, capacitor, and circuitry.
- The basic function of an ICD is to detect tachyarrhythmias by determining the cycle length of the sensed signal and treat these if they fall under the category of VT or VF.
- The ICD therapies are of two types: Antitachycardia pacing (ATP), which is reserved only for tacharrhythmias that falls under the category of VT during which the device can deliver a short burst of overdrive pacing at a rate faster than the arrhythmia. This therapy is typically painless. Shock which can be programmed anywhere from 1 Joule (J) to the the maximum device capacity (3J–4J).
- ICDs also have the capability of bradytherapy and so can serve as pacemakers.
- ICDs also have a diagnostic algorithm that can allow them to distinguish SVT events in order to minimize inappropriate therapy.

**ICD-9-CM Codes**: 427.1 (VTach); 427.41 (VFIB)

**Indications and Usual Treatment:**
- Survivors of cardiac arrest due to VF or hemodynamically unstable sustained VT after evaluation to define the cause of the event and to exclude reversible causes.
- Nonischemic dilated cardiomyopathy who have an LVEF less than or equal to 35% and who are in NYHA functional Class II or III.
- LV dysfunction due to prior MI who are at least 40 d post-MI, have an LVEF less than 30%.
- Structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable
- Syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study
- Nonsustained VT due to prior MI, LVEF less than 40%, and inducible VF or sustained VT at electrophysiological study (EPS)
- Unusual forms of genetically determined inherited arrhythmia syndromes such as long QT; Brugada, catecholaminergic polymorphic VT, hypertrophic cardiomyopathy, etc.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Myocardial ischemia LV dysfunction Rate, mechanism of VTach</td>
<td>Angina symptoms Exercise tolerance, DOE S$_{3}$, rales</td>
<td>ECG, pharmacologic or exercise stress testing ECHO, cardiac cath EPS, ambulatory ECG, Holter monitor</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pt co-morbidities may incl COPD, obstructive sleep apnea (OSA), and amiodarone toxicity</td>
<td>Exercise tolerance, DOE</td>
<td>CXR, PFTs, ABGs</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Edema</td>
<td>BUN, Cr</td>
<td></td>
</tr>
<tr>
<td>NEURO</td>
<td>CV disease</td>
<td>Stroke, TIAs</td>
<td>Carotid duplex</td>
<td></td>
</tr>
<tr>
<td>LYTEs</td>
<td>Reversible causes of arrhythmias (VTach/VFIB)</td>
<td>Diuretic Rx</td>
<td>Serum K$^{+}$ and Mg$^{2+}$</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Anesthetic Technique**
- Monitored anesthesia care during device insertion.
- Local anesthetic infiltration of the insertion site with IV sedation is employed.
- The choice of agent for sedation depends on the discretion and judgment of the anesthesia provider. Midazolam and fentanyl are commonly used.
- Induction of general anesthesia for DFT testing after insertion is completed. Commonly used drugs are propofol and etomidate. Choice and dosage of the induction agent depend on the pre-existing myocardial dysfunction and the amount of sedation the pt received during device insertion.
- Support of ventilation and oxygenation usually provided with a portable anesthesia (mapleson) circuit if the EP lab is not equipped with an anesthesia machine.

**Monitoring**
- ASA standard monitors
- Invasive BP monitoring takes place if an arterial line was placed for either pt specific indication or if it is performed as part of the procedure.

**Airway**
- Support with a facemask attached to a portable anesthesia circuit. Airway instrumentation equipments should be readily available during sedation and before induction of general anesthesia for DFT testing.

**Surgical Stages**
- Surgical preparation and draping of the operative site, usually the left pectoral region.
- Infiltration of the skin with local anesthetic.
- Obtaining venous access through the subcla- vian or cephalic vein. Confirm venous access can-nulation using fluoroscopy.
• Insertion of transvenous defibrillation lead system with or without additional leads for atrial and/or coronary sinus pacing
• Creation of the SQ or submuscular pocket at the site of venous access
• Connect the lead to the generator can.
• Insert the device into the pectoral pocket.
• DFT testing of implanted ICD

Postoperative Considerations
• Blood loss and volume shifts are minimal

• Postop pain is treated with IV and/or oral narcotics, and NSAID drugs.
• Monitor your pt in the PACU until satisfying the discharge criteria established by the specific institution.
• CXR to validate lead position and rule out pneumothorax
• Restricted movement of the arm (in a sling) on side of the ICD implant for 24-48 hr

Anticipated Problems/Concerns
• Bleeding at the access site with or without pocket hematoma formation
• Pneumothorax and/or hemothorax during venous access
• Cardiac perforation and pericardial effusion may lead to tamponade physiology.
• Failure to resuscitate from induced VF at the time of DFT testing
Inguinal Herniorrhaphy

Risk
• Incidence in USA: 700,000 groin hernias repaired per year
• Congenital: 1–5% of newborns, approx 10% of premature births
• Age predominance: Congenital; may manifest at any age, incl elderly
• Sex: 80–90% of adult repairs in males; incidence in congenital hernias quoted as 4–10x higher in males

Perioperative Risks
• Mortality: <0.01% elective repairs; higher in emergency cases and high risk/elderly pts
• Morbidity: Hematoma, 2–3%; infection, 1–2%; entrapment of ilioinguinal or genitofemoral nerve with neuralgia; ischemic orchitis, 0.03–0.5% in primary hernias; recurrence, 0.5–15%

Worry About
• Straining or bucking with emergence may damage repair
• Bowel obstruction with incarcerated hernia
• Apnea risk in formerly premature infants

Overview
• Groin hernias represent defect of transversalis fascia
• Classification incl location (direct hernia arises medially to the inferior epigastric vessels; indirect hernia develops at the internal ring; femoral hernia) and size; also sliding, recurrent, or incarcerated
• Chronically increased abd pressure thought to be predisposing factor, as in obesity, COPD, prostatic hyperplasia, ascites, pregnancy, constipation, colonic stenosis
• Most inguinal hernias can be diagnosed by palpation: Sensitivity and specificity of physical exam have been reported as 75% and 96%, respectively
• Ultrasound exam, or other imaging modalities such as MRI, may be useful in pts with symptoms but no physical signs

ICD-9-CM Code: 550.9

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Ischemic heart disease prevalent in elderly</td>
<td>Exercise tolerance&lt;br&gt;Chest pain/discomfort</td>
<td>Evaluate for signs of CHF</td>
<td>ECG &gt;50 y or with Hx&lt;br&gt;Testing for myocardium at risk if hx suggests</td>
</tr>
<tr>
<td>RESP</td>
<td>Obstructive pulm disease may predispose to hernia</td>
<td>Dyspnea, wheezing</td>
<td>Auscultation (forced exhalation; wheeze)&lt;br&gt;Chest diameter&lt;br&gt;Clubbing, cyanosis&lt;br&gt;Periodic breathing</td>
<td>O₂, sat&lt;br&gt;CXR if infection suspected&lt;br&gt;PFTs if etiology unclear or to evaluate Rx</td>
</tr>
<tr>
<td>GI</td>
<td>Bowel obstruction if hernia incarcerated</td>
<td>Postconceptional &lt;56 wk Hx&lt;br&gt;apnea; caffeine Rx</td>
<td>Abd distention</td>
<td>KUB&lt;br&gt;Electrolytes</td>
</tr>
<tr>
<td>CNS</td>
<td>Ability to tolerate procedure under local anesthesia</td>
<td>Orientation/cooperation</td>
<td>Mental status exam</td>
<td></td>
</tr>
<tr>
<td>SOCIAL</td>
<td>Most procedures done on outpatient basis</td>
<td>Adequate home support for elderly</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
• Consider caffeine citrate, 20 mg/kg in ex-premature infants at risk for apnea
• Avoid sedatives in premature infants having procedure under spinal anesthesia
• Fluid resuscitation and aspiration prophylaxis if bowel obstruction present

Anesthetic Technique
• Local infiltration with sedation (nursing sedation vs. monitored anesthesia care) effective for open repair in adults; potentially fewer side effects than field block/nerve block
• General anesthesia with mask airway or laryngeal mask airway for open repair
• General anesthesia with ET for laparoscopic repair (and for open repair in pt with risk factors for aspiration or in case of surgeon request for muscle paralysis)

• Rapid sequence intubation may be indicated in case of very large or obstructed hernia
• Spinal or epidural (T8) may be used for open repair; one retrospective study raises question of higher mortality in elderly pts with regional techniques
• Paravertebral block (T10–L2) for open repair

Surgical Stages

Skin Incision
• Above inguinal ligament (open) or at site of ports (laparoscopic)

Dissection
• To identify type of hernia and vital structures

Definitive Surgery
• Management of peritoneal sac; repair of fascial defect

Postoperative Considerations
• Minimal blood loss; minimal fluid shifts unless incarcerated hernia with bowel obstruction
• Pain management: Local infiltration; nerve block; caudal (pediatric); NSAIDs
• Pain score: 4–5

Anticipated Problems/Concerns
• Potential for rare complications of laparoscopic insufflation with laparoscopic repair
• Occasional vagal response to traction (notify surgeon, consider treatment with atropine or glycopyrrolate)
• Possibility of femoral nerve palsy with leg weakness after blind ilioinguinal block
• Recurrence in 0.5–15% cases; increased risk with smoking, steroid use, and obesity
Intestinal Obstruction

Risk

- Operations counted in millions if all etiologies included
- Small bowel obstructions predominate by 60–80%
- Etiology of most cases is adhesions from factors such as previous surgery, inflammatory processes, and endometriosis. Other etiologies incl neoplasms, hernias ± strangulation, volvulus, foreign body, and treatment with NSAIDs.
- Race and gender predilection: None

Perioperative Risks

- Apache II scores > 8 correlate with increasing risk
- Mortality: SBO assoc with adhesions 5–10%. SBO assoc with cancer or bowel gangrene and LBO 15–28%.
- Increased risk (bowel factors): Strangulation, malignancy, high obstruction, delay in treatment >24 hr, nonviable strangulation, bowel resection
- Increased risk (general): Sepsis, CV instability (esp hypovolemia and hypotension prior to surgery), extremes of age, co-existing disease, suboptimal nutritional state

Perioperative Implications

Preoperative Preparation

- Restoration of intravascular volume
- Correction of acid-base and lyte abn
- Decompression of stomach
- Antibiotic coverage

Anesthetic Technique

- Usually GA
- Hemodynamic concerns mandate careful selection of anesthetic, relaxant, and analgesic medication

Monitoring

- Required: Routine + Foley catheter
- Consider: Arterial line, CVP, PAC, TEE

Induction/Maintenance

- Rapid-sequence
- CV instability 2° to volume status
- Avoidance of NO
- Muscle relaxation
- Large-bore IV access

Surgical Stages

- Laparoscopy in a selected population
- Laparotomy accompanied by more profound physiologic changes
- Large fluid shifts on opening of abd
- Tumors or mass manipulation may cause hemodynamic alterations
- Restriction of pulm function or cardiac performance 2° to placement of surgical retractors
- Release of hemodynamically active substances when the bowel is manipulated
- Relaxation until abd closed
- Body heat and fluid loss due to exposed bowel
- Blood loss ranges from minimal to significant depending on etiology
- Abd closure may be difficult or contra-indicated

Indications and Usual Treatment

- Any intrinsic or extrinsic lesions that obstruct the intestinal lumen or strangulate the vascular supply require urgent surgery
- Prophylactic surgery may be indicated if an abn that predisposes to obstruction is detected.
- If Dx is uncertain, water-soluble contrast mate

Overview

- Indications for surgery: Strangulation of vascular supply or complete obstruction of lumen. Functional or partial obstructions may be amenable to conservative management.
- Pts may need aggressive management prior to surgery (see Worry About)

ICD-9-CM Code: 560.9

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypotension</td>
<td>Orthostasis, edema</td>
<td>BP (positional)</td>
<td>As indicated by pre-existing disease and age as well as present condition</td>
</tr>
<tr>
<td></td>
<td>Tachycardia</td>
<td></td>
<td>Skin color</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Poor peripheral perfusion</td>
<td></td>
<td>Capillary refill</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Venous return impeded</td>
<td></td>
<td>Pulse quality, HR</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive defect</td>
<td>SOB (positional)</td>
<td>Resp rate/pattern, skin color</td>
<td>ABGs, pulse oximetry</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Resp work/effort</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Loss of fluids, lytes, or</td>
<td>I/O, vomiting (amount,</td>
<td>ABD scars</td>
<td>Abdom CT ± enhancement</td>
</tr>
<tr>
<td></td>
<td>blood</td>
<td>description)</td>
<td>Mass</td>
<td>NG drainage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BM (timing, character)</td>
<td>Rectal tenderness</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abd pain ± distention</td>
<td>Abd girth</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prior operations</td>
<td>Bowel sounds</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hernias</td>
<td></td>
</tr>
<tr>
<td>RENAL/</td>
<td>UO</td>
<td>I/O</td>
<td>Skin turgor</td>
<td>BUN, Cr, Na+, K+, CF,</td>
</tr>
<tr>
<td>HYDRATION</td>
<td>Other fluid</td>
<td></td>
<td>Dry mouth</td>
<td>HCO₃⁻, UA</td>
</tr>
<tr>
<td>IMMUNO</td>
<td>Contamination of GI flora,</td>
<td>Fever, chills</td>
<td>Temp (see above, CV and GI)</td>
<td>CBC with differential</td>
</tr>
<tr>
<td></td>
<td>sepsis or peritonitis</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>


Postoperative Period

- Delay extubation if pulm and CV status in question
- Continued fluid and lyte abn
- ARDS a potential if large fluid volumes were required
- Pulm atelectasis, pneumonia, or aspiration
- DVT ± pulm emboli
- Infection: General or local
- Pain management may be vital to ensure deep breathing and coughing
- PCA for 3–5 d (pain score: 2–9)
- Coagulopathies
- Renal failure
- Malnutrition
- Antiemetics

Anticipated Problems/Concerns

- Hemodynamic, fluid, lyte, pulm alterations
- Sepsis
Intra-Aortic Balloon Counterpulsation (IABCP)

**Overview**
- The device consists of a polyethylene or polyurethane balloon placed in the proximal portion of the descending aorta typically 2 cm distal to the left subclavian artery and proximal to the origin of the renal arteries. Helium is used to inflate the balloon because of its low density allowing faster inflation/deflation cycles.
- It is designed to inflate during diastole after closure of the aortic valve and the onset of isovolumic relaxation of the left ventricle and deflate near the onset of ventricular depolarization (using an electrocardiogram), anticipating ventricular systole.
- The inflation of the balloon causes a displacement of a volume of blood (equal to that of the balloon volume), typically 30 mL to 50 mL. This augments diastolic blood flow and velocity proximal and distal to the balloon and serving as a “diastolic pump.” This increases vital diastolic inflow pressure and velocity to the coronary arteries, improving O₂ delivery to the myocardium and the vascular beds of other organs.
- Typically the hemodynamic response seen is an augmentation of diastolic BP. Also there is a lowering of systolic BP caused by the active deflation of the balloon that decreases afterload and increasing forward blood flow during systole. Think of it as creating a suction effect during systole “sucking” blood out of the left ventricle while the aortic valve is open.
- Other positive hemodynamic effects incl increases in stroke volume, reduction of left ventricular end diastolic pressure, left atrial pressure, pulm artery pressures, myocardial O₂ consumption and left ventricular stroke work index. Systolic BP could increase because of improvement in left ventricular systolic function and augmented coronary blood flow.

**Indications**
- Cardiogenic shock that is not promptly reversed with pharmacologic intervention
- Acute severe mitral regurgitation causing hemodynamic instability
- Acute ventricular septal rupture
- Unstable angina and severe coronary occlusion in pts undergoing percutaneous coronary intervention and/or coronary surgery. Mechanical support of the left ventricle as a bridge to transplant.

**Contraindications**
- Aortic valve insufficiency
- Aortic dissection and aortic aneurysm. Pts with thoracic aortic graft (may be considered 12 mo after graft placement).

**Risks**
- Placed in approx 37,000 pts in USA (2004).

**Perioperative Risks**
- Most common risks of device placement incl injury of the femoral and iliac arteries and aorta.
  - Including dissection, perforation, pseudoaneurysm formation, and critical bleeding.
- Can be a nidus for clot formation, which causes thrombosis of the same vessels listed beforehand and vessels which supply the viscera.
- Embolism also as a result of clot formation can lead to severe ischemic injuries of the leg and viscera.
- Incorrect placement can lead to ischemia of the left arm caused by left subclavian artery occlusion.
- Paraplegia results from the mechanisms listed above and physical occlusion of the artery of Adamkowitzy.

**Worry About**
- Incorrect placement and incorrect use caused by faulty timing of balloon inflation.
- Overinflation (causing intimal damage) and underinflation (causing of the balloon, bleeding and limb and visceral ischemia.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Dysrhythmias, volume status BP, bleeding, inspect distal extremities, UO</td>
<td>Pulses</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Inspect site</td>
<td>CVP, PAP, PCWP, CI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CT (retroperitoneal bleed)</td>
<td>Doppler of limbs, ECHO</td>
</tr>
</tbody>
</table>


**Perioperative Management**
- Optimal placement from the leg involves insertion below the inguinal ligament and above the bifurcation of the femoral artery.
- Optimal position is a point 1 cm to 2 cm distal to the subclavian artery (can be estimated by placing the balloon tip on the pt’s chest at the angle of Louis and marking the distal portion of the catheter where insertion will take place at the thoracic level). Left radial arterial pressure monitoring can help assure that flow to the left arm is adequate. Fluoroscopy is highly recommended to assure ideal placement between the left subclavian artery and the renal arteries.
- Fluoroscopy should be used to ensure ideal position but if not available transesophageal echocardiography is an acceptable alternative.
- Size of balloon placed varies according to the pt’s height with a general recommendation of a 25 cc balloon for pts less than 5 ft in height and 50 cc for pt’s greater than 6 ft tall.
- The balloon diameter is general designed to inflate to 80–90% of the interak diameter of the descending aorta.
- Timing of balloon inflation and deflation is critical. The goal is to optimize afterload reduction during systole and optimize diastolic BP, flow and velocity without interference with systolic ejection.
- Early inflation results in increased afterload and the augmented pressure wave imposed on the systolic component of the pressure tracing.
- Late inflation and early deflation lead to reduced augmentation of diastolic flow.
- Late deflation causes the augmented pressure wave to be seen extending into the systolic component of the pressure wave.
- Ideally an electrocardiogram is used for timing inflation and deflation. The descending slope of the T wave correlates with the onset of diastole and inflation and the R wave, which marks the onset of electrical systole, is used to trigger deflation.
- If an aortic pressure tracing is used then the dicrotic notch (aortic valve closure) is used to trigger inflation and end of diastole triggers deflation.
- The operating settings allow for augmentation of every heart beat (1:1) every other heart beat (1:2) and depending on the manufacturer as low as every eighth heart beat (1:8). There is probably little benefit beyond a low setting of augmentation of every fourth heart beat (1:4). These lower settings are used during the weaning process. A nonfunctional or stopped intra-aortic balloon pump should be removed within 20 min or less. Volume weaning, by slowing reducing the volume of the balloon inflation, can also be used.
- The physiologic goals during therapy with the device and the weaning process incl a cardiac index greater than 2.2 L/min/m², acceptable BP with no ongoing signs of organ under-perfusion (acceptable UO, no signs of visceral ischemia, good mental status, normal pH and other ABG parameters, with low lactate levels). Constant physiologic monitoring of pulm capillary wedge pressure, central venous pressure and pulm artery pressures is advisable (a drop in these pressures accompanied by a rise of cardiac index are good indicators that myocardial work has been decreased with the onset of therapy).
- Monitoring with frequent echocardiograms can also be useful.
- Unless there is a risk of major acute bleeding anticoagulation with heparin is commonly used to reduce the risk of clot formation on the balloon and in areas of stagnant or low flow caused by the insertion of the device (femoral artery). A partial thromboplastin time (PTT) of 1.5 to 2 times baseline is maintained while the device is needed and then allowed to wear off sufficiently to prevent major bleeding complications usually (4 hr).
- Constant vigilance for signs and symptoms of major bleeding (following all hemodynamic parameters and hemoglobin levels) and limb and organ ischemia is important. Assessment of pulses in all extremities but esp. the affected limb is critical. Retroperitoneal bleeding is a potential complication that may not be evident immediately so be observant of subtle signs of ongoing bleeding. A non-contrast computerized axial tomography scan can be useful to diagnose this complication.

**Anticipated Problems/Concerns**
- Timing may be difficult in pts with variable heart rates such as atrial fibrillation. May be difficult to allow for adequate balloon inflation in heart rates greater than 120 beats/min (if heart rate cannot be slowed then consider augmentation of every other heart beat with a 1:2 setting).
Intussuscepted Bowel Repair

Assessment by Hx

- NMB
- RSI

Induction/Maintenance

- ASA routine monitors
- UO
- Invasive for sepsis or hemodynamic instability

Monitoring

- ASA routine monitors
- UO
- Invasive for sepsis or hemodynamic instability

Preoperative preparation

- Hydration
- Transfusion
- NG drainage
- Antibiotics for sepsis

Anesthetic technique

- General anesthesia preferred

Surgical Stages

Pediatric

- Laparoscopy 1° approach
- May need extension of incision for reduction or resection
- Resection 25%

Adult

- Laparoscopy primary approach
- May need extension of incision for reduction or resection
- Resection >90%

Diagnosis: CT most sensitive
- Colonoscopy for subacute or chronic
- Lead points malignant: Carcinoma, lymphoma, melanoma, sarcoma
- Lead points benign: Endometriosis, diverticulum, strictures, adhesions, Meckel’s, celiac, Crohn’s, lipoma, adenoma, heterotopic pancreas, HIV, infection

ICD-9-CM Code: 560.0

Indications and Treatment

Pediatric

- Surgical consultation regarding signs of peritonitis
- Enema reduction: Air versus liquid or contrast
- Enema perforation: –1%
- Position: Prone
- Dx: US
- Sedation/GA lower reduction rates
- Recurrence rate: –10%
- Surgery: Laparoscopy 1°, bowel resection increases with age
- Surgery indications: Perforation, failed enemas, pathologic lead point
- Enema reduction may be attempted for idiopathic small bowel.
- Delayed repeat enema beneficial

Adult

- Enema reduction rarely indicated
- Surgery: Laparoscopy 1°, bowel resection common

Overview

- Intussusception: Telescoping of bowel into itself, usually distal.
- Lead point: Anatomic stimulus for invagination

Worry About

- Sign of advanced disease: Lethargy, fever, dehydration, changes in mental status, tachypnea
- Check Hct, electrolytes
- Contraindications to non-surgical reduction: Perforation, strangulation, sepsis, peritonitis, CV instability

Risk, Adult

- 5–10% of intussusception
- <5% adult bowel obstruction
- Major pelvic ileum
- Bowel abscess common
- Male predominance overall
- Appendiceal intussusceptions more common in adult women
- Retrograde jejunal-gastric intussusceptions after gastric bypass, gastrectomy, gastrostomy tubes, or repair duodenal atresia
- Idiopathic 10%
- Colon: Majority malignant lead point
- CT imaging reveals intussusception images in ~0.1% of scans
- Non-surgical intussusceptions >10× more frequent than surgical
- Periop risk, pediatric: Mortality with treatment 1–3%
- Mortality 100% untreated ≤5 d
- Bowel perforation during enema

Risk, Pediatric

- Most common cause of pediatric small bowel obstruction
- 25% of children ≤2 y
- Rare <3 mo
- M:F ratio: 3:1 overall
- Incidence increases with age
- Recurrence 3–11%
- Chronic course 5%
- 80–90% ileocolic
- 90% idiopathic
- Lead point ½ children >2

Assessment Points

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GL</td>
<td>Venous congestion</td>
<td>Pain</td>
<td>Mass</td>
<td>US</td>
</tr>
<tr>
<td></td>
<td>Bowel edema</td>
<td>N/V</td>
<td>Tenderness</td>
<td>CT</td>
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<tr>
<td></td>
<td>Ischemia/infarction</td>
<td>BM + blood</td>
<td>Rectal prolapse</td>
<td>Enema</td>
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<tr>
<td></td>
<td>Perforation</td>
<td>Diarrhea</td>
<td></td>
<td>Abd x-ray</td>
</tr>
<tr>
<td>RENAL/HYDRATION</td>
<td>Dehydration</td>
<td>Vomiting</td>
<td>Urine amount/color</td>
<td>Electrolytes</td>
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<tr>
<td></td>
<td></td>
<td>I/O</td>
<td>Fontanelle</td>
<td>BUN, Cr</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sunken eyes</td>
<td>UA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Skin turgor</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Anemia</td>
<td>Lethargy</td>
<td>Tachycardia</td>
<td>Hgb/Hct</td>
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<tr>
<td></td>
<td>Sepsis</td>
<td>Mental status</td>
<td>Hypotension</td>
<td>WBC</td>
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<tr>
<td></td>
<td>Shock</td>
<td>Fever</td>
<td>Pallor</td>
<td>Lactate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Capillary ref</td>
<td></td>
</tr>
</tbody>
</table>

Joint Replacement Cementing (Methylmethacrylate Cementing)

**Risk**
- True incidence of bone cement implantation syndrome (BCIS) is unknown.
- BCIS primarily a problem associated with hip replacement, although it has been described during other cemented procedures such as knee arthroplasty and vertebroplasty.
- Pt risk factors for BCIS incl pre-existing pulm Htn, and significant cardiac disease (New York Heart Association Class 3 or 4).
- Surgical risk factors for BCIS incl pathologic fracture, inter-trochanteric fracture, and long-stem arthroplasty.

**Perioperative Risks**
- Mortality after cemented and uncemented 1st total hip replacement (THR) is 2.3% and 1.6% respectively.
- Incidence of intraop mortality during cemented total hip replacement (THR) is 0.11%.
- DVT
- Fat embolism syndrome (resp, neurologic, cutaneous, hematologic symptoms) <1%
- Pulm embolism 1.8–3.4% (with no prophylaxis)

**Overview**
- Methylmethacrylate (MMA) is used as a bone cement in orthopedic surgery
- Bone cement implantation syndrome (BCIS) occurs in pts undergoing cemented bone surgery, and is characterized by signs such as hypotension, hypoxia, arrhythmias, increased pulmonary vascular resistance (PVR), loss of consciousness, and cardiac arrest.
- BCIS can occur around the time of cementation, prosthesis insertion, reduction of the joint, or deflation of a limb tourniquet.
- Different models exist to explain BCIS, but many probably play a role.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>BCIS can cause ↓ MAP, ↓ SV, ↓ cardiac output, ↑ PVR, ↓ PAP, ↓ RV ejection fraction</td>
<td>ECG, TEE</td>
</tr>
<tr>
<td>Pulm Htn → Heart failure</td>
<td>CVP, PA catheterization</td>
<td></td>
</tr>
<tr>
<td>PFO → risk for paradoxical emboli</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>MMA activates coagulation cascade and mediates plt aggregation → ↑ thrombosis</td>
<td>Venography, duplex US</td>
</tr>
<tr>
<td>CNS</td>
<td>Confusion, stroke (from paradoxical emboli)</td>
<td>Baseline neuro exam</td>
</tr>
<tr>
<td>RESP</td>
<td>↑ Pulm pressure, pulm emboli → V/Q mismatch</td>
<td>PA catheterization</td>
</tr>
<tr>
<td>If pt awake, dyspnea may be early sign of BCIS</td>
<td>ABG</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ETCO2, TEE</td>
<td></td>
</tr>
</tbody>
</table>

**Intraoperative Management**

**Monitoring**
- Consider invasive monitoring in pts with baseline cardiopulmonary disease or in pts at high risk for BCIS
- CVP monitoring may aid volume optimization but changes in CVP may correlate poorly with changes in PAP in BCIS
- Increased vigilance during times at which BCIS can occur
- If BCIS occurs, consider arterial line, PA catheter or TEE to guide management (intravascular volume, RV function).
- Exhaled gas analysis: Decreased ETCO2, if significant pulm embolism; increased end-tidal N2 if air embolus
- Esophageal Doppler may detect impending BCIS earlier than standard monitors.

**Worry About**
- Increased PVR leading to decreased right ventricular EF, leading to reduced left ventricular EF, which leads to decreased CO
- Clinical features (may incl one or more):
  - Hypotension, hypoxia, loss of consciousness, cardiac arrhythmias
  - Pulm embolism 1.8–3.4% (with no prophylaxis)

**Surgical Stages**
- Pre-cement
- THR: Insidious blood loss due to irritation
- TKR: Tourniquet-induced Htn (tourniquet time >30–60 min)
- Maintain intravascular volume
- Consider avoiding N2O to prevent exacerbat ing air embolism
- Cementing
- If GA, D/C N2O
- FIO2 100% to combat increased PVR from emboli
- Hypotension in BCIS may be due to decreased SVR, decreased CO, or both
- CV collapse may be treated for RV failure with inotropes, vasodilation with alpha agonists, insufficient preload with fluid

**Anticipated Problems/Concerns**
- Monomer-mediated model: MMA monomers lead to vasodilation in vitro, but this hypothesis is not supported in vivo in animal studies.
- Embolic model: MMA undergoes an exothermic reaction and expands, resulting in intramedullary Htn which forces emboli into the circulation. Embolic debris can be composed of fat, marrow, cement, air, bone, plt and fibrin aggregates.
- PVR may be due to mechanical obstruction of pulm circulation, and endothelial damage that results in release of inflammatory mediators.
- Intraop emboli detected on TEE in 90–98% of THR pts, but embolization not always assoc with hemodynamic changes
- Increased embolic load with cemented versus uncemented arthroplasties

ICD-9-CM Code: 820.x (Hip fracture)

**Indications and Usual Treatment**
- Disabling arthritis, femoral fractures

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Kasai Procedure

Risk
- Biliary atresia occurs in 1/10,000 live births in Japan, and 1/15,000 live births in USA
- 10–15% of pts have assoc abn referred to as Biliary Atresia Splenic Malformation Syndrome (BASM). Polysplenia, asplenia, preduodenal portal vein, situs inversus, absent inferior vena cava, intestinal malrotation, and cardiac anomalies
- Affects girls more than boys
- Affects Asians and African-Americans more than Caucasians

Perioperative Risks
- Impaired hepatic function (generally a latter manifestation): Coagulation disorders, drug metabolism
- Vitamin deficiencies (K and E) and malnutrition may result in coagulopathy

Worry About
- Preoperative: The differential diagnosis of conjugated jaundice in an infant incl cholestochal cyst, inpsissated bile, neonatal hepatitis, alpha 1 antitrypsin deficiency, CMV hepatitis, cystic fibrosis, and Alagille’s syndrome (arteriohepatic dysplasia) in which jaundice is assoc with hypercholesterolemia, pulm stenosis, elfin facies. Also Dubin Johnson syndrome (DJS) is a rare autosomal recessive condition with normal liver transaminases, a unique pattern of urinary excretion of heme metabolites (coproporphyrins), and the deposition of a pigment that gives the liver a characteristic black color.
- Postop: Cholangitis, and cholestasis (malnutrition, pruritus)

Overview
- Surgical procedure to correct biliary atresia
- Infants present from 1–6 wk of age with persistent jaundice and acholic stools.

ASSESSMENT POINTS

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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>DERM</td>
<td>Jaundice</td>
<td>Progressive increase</td>
<td>Deep greenish-bronze color</td>
<td>Conjugated hyperbilirubinemia</td>
</tr>
<tr>
<td>GI</td>
<td>Cirrhosis</td>
<td>Abd discomfort</td>
<td>Firm, enlarged liver</td>
<td>Hypoalbuminemia</td>
</tr>
<tr>
<td></td>
<td>Ascites</td>
<td>White or clay-colored</td>
<td>Splenic enlargement Abd distention</td>
<td>Aortic aneurysm</td>
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<tr>
<td>CARDIAC</td>
<td>CHD (pulm stenosis)</td>
<td>Alagille’s syndrome</td>
<td>Murmur</td>
<td>ECHO</td>
</tr>
<tr>
<td>RENAL</td>
<td>Urine</td>
<td>Greenish color</td>
<td>Bile stained</td>
<td>Vitamin deficiency, abn PT/PTT</td>
</tr>
<tr>
<td>NUTRITIONAL</td>
<td>Wt loss</td>
<td>Poor appetite</td>
<td>FTTT</td>
<td>Vitamin deficiency, abn PT/PTT</td>
</tr>
<tr>
<td>CNS</td>
<td>Vit E deficiency</td>
<td>Ataxia</td>
<td>Hyporeflexia</td>
<td>Ophthalmoplegia</td>
</tr>
<tr>
<td>HEME</td>
<td>Blood loss</td>
<td>Weakness</td>
<td>Bloody stool</td>
<td>Endoscopy for esophageal varices, anemia</td>
</tr>
</tbody>
</table>

Indications and Usual Treatment
- Biliary atresia
- Neomycin, metronidazole and lactulose may be given 24–48 hr prior to surgery.
- Hepatic portoenterostomy (Kasai procedure) is usually performed before 4 mo of age.
- Intraop cholangiogram may be performed.

Surgical Stages
- Dissection
  - Right subcostal incision extended to the left
  - Cholangiogram and wedge biopsy of the liver to confirm diagnosis
  - Liver mobilization and resection of atretic biliary system at porta hepatitis
  - Preparation of jejunum for roux-en-Y
  - For biliary drainage the antimesenteric side of the jejunum is sutured to the porta hepatitis

Postoperative Considerations
- NG tube for stomach decompression
- Supplementation of fat and fat-soluble vitamins if there is impaired absorption due to poor bile flow
- Prednisone 2 mg/kg/d may be given for its choleretic effect

Anticipated Problems/Concerns
- Cholangitis, portal Htn, and fat-soluble vitamin deficiency
- Approx 10–30% have long-term normal liver biochemistry. 70–90% go on to require liver transplantation.

Perioperative Management

Preoperative Preparation
- Check PT, PTT, hemoglobin, and plt. Abn not common but a type and screen should be performed and blood and FFP made available
- NG tube
- Broad-spectrum antibiotics
- Consider epidural block if coagulation is normal
- Consider central line if vascular access is difficult

Monitoring
- A large bore (22–20 g) IV in upper limb
- Arterial line and central venous pressure monitors rarely used unless other co-existing conditions (pneumonia, sepsis, cholangitis, and sever cirrhosis) or redo procedure
- All pressure points need to be padded and reassessed
- Temp monitoring: Forced warm air device, warming lamps

Anesthetic Technique/Induction
- If IV access is available, induction with an hypoxic agent like propofol (2–3 mg/kg) or pentothal (4-6 mg/kg) along with a muscle relaxant (cisatramium 0.2 mg/kg)
- If IV access unavailable, inhalation induction with O₂, N₂O, and sevoflurane. Once adequately anesthetized, an IV catheter is inserted and muscle relaxant can be used to facilitate ET intubation. Anesthesia is maintained with O₂-air-isoflurane/sevoflurane mixture with IV opioids.
- Avoid the continued use of N₂O due to bowel distention
- An isotonic IVF (preferably 0.9% NS or plasmalyte) should be used and serum glucose should be monitored
- An epidural can be placed for postop pain provided there is no coagulopathy.

Kidney Transplantation

Risk
- Incidence in USA: As of 2006, the prevalence of end-stage renal disease (ESRD) was 506,256 pts with a median age of 58.8 y.
- As of 2007, 48,773 active pts and 94,741 total pts were on the renal transplant waiting list.
- Etiologies of ESRD on the waiting list: Diabetic nephropathy, 28%; hypertensive nephrosclerosis, 22%; glomerular disease, 21%; and other renal pathologies (SLE, polycystic kidney disease, vasculitides, etc.).
- Renal transplant demographic data from 2008 indicate:
  - 61.4% male; 38.6% female
  - 4.7% less than 18 y/o, 80.1% 18–64 y/o, and 15.2% 65 y/o or greater
  - 53.7% Caucasian, 24.5% African American, 14.8% Hispanic, and 7% minority (i.e., Asian, Native American, Pacific Islander, etc.)
- Of the 16,520 kidneys transplanted in 2008, 10,552 were from deceased donors and 5,968 were from living donors.
- As of 2004, the median time to transplantation was 3.3 y.
- As of 2006, the 3-mo pt survival rate post-transplant was 99.3% (living donor) and 97.9% (deceased donor).

Perioperative Risks
- Cardiac events, pulmonary edema, hemorrhage from vascular anastomoses, infection, vascular thrombosis, and graft rejection (non-functioning)

Worry About
- Interval between last dialysis and renal transplant
  - Volume and electrolyte status (esp. serum K+ level)
  - Coagulopathy
  - Proper pt positioning
  - AV graft and/or fistula
  - Pad graft and/or fistula
  - Prevent heat loss (place heat pad on graft/fistula)
- Periodically palpate and/or auscultate to check breath sounds
- IV access while preserving dialysis access
- Renal metabolism and/or excretion of drugs
- Maintaining adequate allograft perfusion
- Ischemia and/or reperfusion injury of leg and renal graft
- Potential for rapid blood loss during reperfusion
- Antirejection regimen
- Hypotension, allergic reaction, increased bleeding and/or palm edema may be seen with use of monoclonal anti-CD3 antibodies (i.e., alemtuzumab)

Overview
- 3–5 hr procedure
- Supine position
- Heterotopic renal graft implantation in extra-peritoneal iliac fossa
  - No need for removal of recipient’s kidneys
- Main anesthetic goal is to ensure graft perfusion and forced diuresis
  - Perfusion pressure (dopamine)
  - Volume expansion
  - Diuretics (furosemide and mannitol)

Indications and Usual Treatment
- Treatment of choice for ESRD (improves life expectancy and quality of life)
- Contraindications to renal transplant include:
  - Active infection
  - Continued illicit drug abuse
  - Complete thrombosis of IVC and iliac veins
  - Metastatic disease

ICD-9-CM Code: 55.69

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</tr>
</thead>
<tbody>
<tr>
<td>PULM</td>
<td>Pulm edema, effusions, pulm Htn, pneumonia, OSA</td>
<td>SOB, DOE, orthopnea, cough</td>
<td>Crackles, rales</td>
<td>CXR, CT chest, ECHO, RHC, sleep study</td>
</tr>
<tr>
<td>CARDIO</td>
<td>CAD, cardiomyopathy, Htn, hyperlipidemia, PVD</td>
<td>Angina, claudication, CVA/TIA</td>
<td>S3/S4 murmurs, JVD, carotid bruits, weak peripheral pulses, edema</td>
<td>EKG, ECHO, DSE, cardiac cath, carotid Doppler, ABI, C-reactive protein, daily weights</td>
</tr>
<tr>
<td>GI</td>
<td>Gastropareis</td>
<td>Bloating, early satiety, GERD</td>
<td>Gastric emptying study</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>ESRD (usually on dialysis)</td>
<td>N/V, altered mental status</td>
<td>Electrolytes, BUN, Cr, Cr clearance</td>
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<tr>
<td>ENDO</td>
<td>Hyperglycemia, hyperparathyroidism</td>
<td></td>
<td>Blood glucose, Ca, Phos</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, coagulopathy, plt dysfunction</td>
<td>SOB, easy bleeding</td>
<td>Capillary refill, pallor</td>
<td>CBC, PT/INR, PTT, plt function assays</td>
</tr>
<tr>
<td>NEURO</td>
<td>Neuropathy</td>
<td>Numbness, orthostasis, syncope</td>
<td>Decreased sensation, orthostatics</td>
<td>Tilt table testing</td>
</tr>
</tbody>
</table>


Perioperative Management
- Epidural or paravertebral catheter can be used for postop analgesia
- Coagulation status usually normal under scheduled hemodialysis
- Consider arterial line and central line with aseptic technique
- Consider pre-treatment with a non-particulate antacid (gastroproxis)
- Remember preop antibiotic prophylaxis
- Consider rapid sequence induction with cricoid pressure (full stomach/gastroproxis) using rocuronium (succinylcholine is best avoided due to hyperkalemic disease)
- Consider cisatracurium for maintenance (avoid pancuronium).
- Use of sevoflurane is controversial.
- Avoid large doses of morphine and meperidine.
- Maintain normovolemia or slight hypervolemia
- Adjuvants: Furosemide (0.25–1 mg/kg), mannitol (0.25–1 g/kg), and/or low dose dopamine (2–5 µg/kg/min) are often administered shortly prior to and following allograft reperfusion.
- An immunosupressant (methylprednisolone 10 mg/kg) is typically administered prior to graft reperfusion
- An induction immunosuppressant (OKT3 or alemtuzumab) may be used in the preop or intraop period.

Surgical Stages
- Lateral oblique skin incision in lower abd
- Extraperitoneal space (less fluid loss)
- Iliac vessel clamping during anastomoses (graft renal artery to iliac artery and graft renal vein to iliac vein)
- Donor ureter attached to recipient’s bladder
- Maintain normal to elevated MAP and CVP to preserve graft perfusion
- Chest x-ray (to R/O pneumothorax, pulm edema)
- Monitor fluid status and UO closely
- If stable, transfer to a monitored floor bed
- Potential for delayed graft function (ECD kidney)
- May consider postop hemodialysis
- Consider ICU transfer
- Use PCA and/or epidural or paravertebral block for postop analgesia

Postoperative Considerations
- Maintain normal central venous pressure and cardiac output
- Monitor fluid status and UO closely
- If stable, transfer to a monitored floor bed
- Potential for delayed graft function (ECD kidney)
- May consider postop hemodialysis
- Consider ICU transfer
- Use PCA and/or epidural or paravertebral block for postop analgesia
Knee Arthroscopy

Risk
- Incidence in USA: >1.5 million/y
- Incidence of arthroscopic anterior cruciate ligament (ACL) repair in USA: >100,000/y
- Pts primarily ≤60 y; related to athletic injury
- No racial or gender predominance

Perioperative Risks
- Mortality rate < 1:100,000
- Morbidity is rare
- Neurologic injury: Direct trauma, compartment syndrome, tourniquet-related, and dysfunction due to Complex Regional Pain Syndrome I (CRPS I). Femoral nerve most vulnerable.
- Vascular injury more likely during meniscectomy repair (popliteal artery)
- Tourniquet: Temporary paralysis after prolonged inflation
- Infection (esp. with allografts)
- Deep vein thrombosis/pulmonary embolism rare

Monitoring
- Potential length of procedure

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Risk of bradycardia/arystole due to tourniquet deflation, neuraxial anesthesia, emotional reaction</td>
<td>Hx of athletic state</td>
<td>Slow resting HR</td>
<td>ECG</td>
</tr>
<tr>
<td>HEME</td>
<td>Bleeding diathesis contraindication to regional anesthesia</td>
<td>Bleeding or easy bruising with injury or tooth extraction</td>
<td>Bruises</td>
<td>PT, APTT, plt count, bleeding time as indicated by Hx</td>
</tr>
<tr>
<td>GU</td>
<td>Postop urinary retention</td>
<td>Nocturia Hx of prior catheterization</td>
<td>Size of prostate</td>
<td>Bladder US if no void and if long-acting spinal used</td>
</tr>
<tr>
<td>CNS</td>
<td>Risk of bradycardia/hypotension and nausea with sight of blood/surgery</td>
<td>Fainting or stress with prior surgery Rx with anaxiolytic medication</td>
<td>Visibly anxious</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Issues
- Pt may have expectation to watch procedure
- Focus on early discharge
- Potential length of procedure

Monitoring
- Routine

Anesthetic Technique
- Local infiltration of portals + intra-articular injection of 20–30 mL 0.25% bupivacaine (often requires additional sedation). Inadequate for ACL repair or if thigh tourniquet is used.
- Femoral nerve block: Limited by tourniquet use and surgery in back of knee
- Epidural, spinal, or combined spinal-epidural: Issues incl PDPH risk, duration of surgery, rate of onset and resolution of blockade.
- Choice of local anesthetic for spinal
  - Controversy exists with off-label use of preservative-free, isotonic 1.5% mepivacaine and 2% 2-chloroprocaine for spinal anesthesia, although evidence suggests they are efficacious for this procedure.
  - Lidocaine (risk of TNS)

Postoperative Considerations
- Control of pain and side effects can lead to early discharge
- PONV and sedation limit discharge, esp. after deep sedation, GA, narcotics
- Options: Intra-articular drugs, e.g., bupivacaine ± morphine
- Local infiltration of portals with bupivacaine
- Femoral nerve blocks particularly helpful after ACL repair. Requires knee brace for discharge.
- IV or PO NSAIDs reduce narcotic requirement
- Urinary retention usually not limiting factor in young pts
- Follow-up phone call helps define and treat problems.

Anticipated Problems/Concerns
- Postop back pain (with or without regional anesthesia)
- PDPH: Should be followed and treated
- Postop pain usually controlled with oral medication and hypothermia
- Late urinary retention
- TNS can be managed with NSAIDs
- Local anesthetic or epinephrine-containing solution: Toxicity with infiltration
**Labor, Epidural Block**

**Risk**
- Incidence in USA: Approx 65% get an epidural block for pain relief during labor at hospitals that perform more than 1500 deliveries per year.

**Preoperative Risks**
- Edema offers technical difficulty while placing the epidural catheter.
- Obesity is another factor making epidural catheter placement a challenge.
- Hypovolemia, from significant bleeding or dehydration.
- Other causes of hypotension incl sepsis

**Perioperative Risks**
- No pain relief, also called block failure, occurs in about 1 in 20. Additionally another 15% experience partial failure, and have only partial pain relief.
- Intravascular injection (catheter displacement is uncommon, less than 1 in 300) or migration (about 1 in 10,000 insertions) with possible arrhythmias and seizures. This also results in block failure.
- Catheter misplacement into the subarachnoid space (less than 1 in 1000), with possible subsequent spinal administration of large doses of local anesthetics.
- Level and degree of sympathetic blockade will determine the effect on the CV system (less incidence of hypotension in women who received 1 liter of Ringers lactate versus those who did not receive prehydration).

**Postoperative Risks**
- Postdural puncture headache

**Worry About**
- CV collapse due to accidental venous injection of the local anesthetic
- Hematoma in pts with underlying coagulopathies
- Increased incidence of instrumental delivery
- Alterations in uteroplacental perfusion and acidosis of the fetus due to the placental transfer of local anesthetics and subsequent hypoxia
- Epidural abscess in pts with sepsis
- Total spinal (high level of anesthesia) with loss of airway
- Hypoxemia and acidosis can develop in labor and cardiopulmonary resuscitation might be difficult.

**Overview**
- Epidural analgesia is a safe and effective way to manage pain in labor with minimal motor block.
- The decrease in uterine activity following the administration of epidural block is transient. However studies have suggested a better fetal outcome when epidural analgesia has prolonged the second stage of labor.
- Recent data suggest that there is no difference between the administration of early epidural block (dilation 3–4 cm) versus late epidural block (dilation greater than 5 cm) regarding the occurrence of dystocia or necessity for C-section (controversial). Combined epidural and spinal anesthesia does not seem to negatively influence the progress of labor, it might even speed up labor in early primiparous pts. Controversy remains regarding the effect of more concentrated local anesthetics solutions in the progress of labor.

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<tbody>
<tr>
<td>CARDIO</td>
<td>Aortocaval compression</td>
<td>Dizziness, syncope, decrease FHR</td>
<td>Hypotension in supine position</td>
<td>Assess UO provide left uterine displacement</td>
</tr>
<tr>
<td>RESP</td>
<td>Decreased FRC increases minute ventilation and O₂ consumption</td>
<td>Rapid development of hypoxemia</td>
<td>Tachypnea</td>
<td>Avoid high spinal blockade, provide supplemental O₂</td>
</tr>
<tr>
<td>CNS</td>
<td>Decreased MAC epidural vein distention</td>
<td>Exaggerated effect of local anesthetics</td>
<td>Sedation, increased sensory and motor block</td>
<td>Assess dermatomal blockade</td>
</tr>
<tr>
<td>COAGULATION</td>
<td>Decreased plt count abn function</td>
<td>Abn hemostasis</td>
<td>Oozing at IV site, bruising</td>
<td>Assess plt count fibrinogen, FSPs, check bleeding time</td>
</tr>
<tr>
<td>FETAL HR</td>
<td>Sensitive to maternal hypotension</td>
<td>Bradycardia, decreased heart rate variability</td>
<td>Decreased movement, HR variability, meconium</td>
<td>Phonocardiogram, fetal scalp blood sampling</td>
</tr>
</tbody>
</table>

**Perioperative Management**

**Monitoring**
- Noninvasive BP measurement and continuous fetal and maternal HR.
- Ensure drugs and equipment are available to access the airway.
- An obstetrician should be available to manage obstetric complications during induction and maintenance of epidural analgesia.
- Prehydration with Ringers lactate

**Induction**
- The uterus should be displaced left to improve maternal venous return, hypotension should be treated with additional IV boluses of crystalloid solution and/or administration of small IV doses of a vasopressor such as ephedrine (5–10 mg IV).
- The most commonly used intermediate-acting local anesthetic via the epidural route with catheter insertion is 2% lidocaine. Adding epinephrine to the solution (1:200,000) prolongs the duration of action by 40–60%.

**Dilute Techniques**
- Primigravida in early labor or slowly progressing multipara: Use bupivacaine 0.05–0.0625% (10 mL) by bolusing 5 mL at a time. Additive effect can be obtained with 50–100 mcg of fentanyl, or 100 mcg of hydromorphone. A continuous infusion of bupivacaine 0.0625–0.125% with 2–3 mcg/mL of fentanyl, or 3 mcg/mL of hydromorphone at 5–15 mL/hr are good choices for maintenance. Alternatively a combined spinal-epidural provides advantages for pain control esp. in early labor. A combination of 12.5–25 mcg of fentanyl with 2.5 mg bupivacaine intrathecally may be used for induction, followed by an epidural continuous infusion of bupivacaine at 0.0625–0.125%.
- Neuraxial analgesia in early labor does not increase the rate of cesarean delivery, and provides better analgesia. It also results in a shorter duration of labor than systemic analgesia.
- The use of pt controlled epidural analgesia with bupivacaine, 0.0625–0.125% + fentanyl 2–3 mcg/mL, is another possibility.

**Indications**
- Significant pain resulting in maternal stress
- Vaginal delivery of twins
- Preterm infant delivery (more control of delivery)

**Contraindications**
- Uncooperative pt
- Inability to increase cardiac output in response to sympathectomy
- Anatomic abn of the spine
- Certain neurologic diseases that can be further exacerbated by the block
- Inability to communicate with the pt
- Previous back surgery
- Significant blood loss


It can be given at 5 mL/hr basal rate, bolus of 5 mL with 5–6 min lockout, and 1-hr limit of 15–20 mL.

**Standard Techniques**
- Local anesthetic only: Induction with ropivacaine 0.2% (8–10 mL), followed by continuous infusion of ropivacaine 0.1% at 8–10mL/hr.
- Local aesthetic plus opioid: Induction with bupivacaine 0.125–0.25% (8–10 mL/hr) plus fentanyl 50–100 mcg. After epidural block is established, an infusion can be started with bupivacaine 0.0625–0.125% plus fentanyl 1.6–2 mcg/mL, at a rate of 8–10 mL/hr. Ropivacaine 0.1% mixed with fentanyl 1.6–2 mcg/mL, at 8–10 mL/hr can be used as well.
- The infusion can be maintained for pain relief during episiotomies and to facilitate the removal of the placenta. If a C-section is needed, the epidural block can be extended with lidocaine 2% plus epinephrine 1:200,000.
Concentrated Techniques
• Rapidly progressing multipara in first stage of labor: In these cases it is important to achieve fast pain relief. Lidocaine 1.5% or 2% can be used with epinephrine 1:200,000 (10 mL bolus) in 5 mL increments combined with fentanyl 50–100 mcg. If necessary, a continuous infusion of bupivacaine at 0.125% with fentanyl 2–3 mcg/mL at rate of 5–15 mL/hr may be initiated to maintain analgesia.

Special Situations
• For women in labor with a Hx of mild to moderate aortic or mitral stenosis, narcotics can be used alone. Fentanyl 1–4 µg/mL/hr or sufentanil 0.03–0.05 µg/mL/hr are good options in these instances.

Second stage arrest: Low concentrations of local anesthetics preserve the urge reflex to push with minimization of the laxity of the perineal musculature. However, one must consider that better pain relief is achieved with higher concentrations of local anesthetics. Alternatively fentanyl 12.5–25 mcg with 2.5 mg bupivacaine can be used intrathecally, and can be repeated after 1.5–2.5 hr.

Anticipated Problems/Concerns
• Difficulties in placing the catheter: Obese pts, presence of edema, repetitive attempts, back pain, and paresthesias.

• Fetal compromise: Decreased uteroplacental perfusion with fetal hypoxia, neonatal exposure to opioids and local anesthetics, with dose-dependent CNS damage.
• Unwanted effects: Motor blockade, excessive relaxation of the perineum, decreased ability to push, necessity of oxytocin augmentation
• Neuraxial analgesia in early labor does not increase the rate of cesarean delivery, and it provides better analgesia and results in a shorter duration of labor than systemic analgesia.
Perioperative Risks
- Periop morbidity/mortality from anesthetic technique: rare
- **Worry About**
  - Accidental IV injection
  - Hypotension (sympathetic nerve block)
  - Fetal bradycardia (paracervical block)
  - Neuropathy (nerve damage)
  - Infection
  - Hematoma formation (LSB)
  - Total or high spinal very rare with lumbar sympathetic block

Overview
- Lumbar sympathetic block (LSB): Analgesia for the first stage of labor. Blocks visceral efferents as they join sympathetic chain.
- Paracervical block, Frankenhauser’s plexus: Lower uterus and cervix—analgesia for the first stage of labor.
  - Pudendal block: Analgesia for the second stage of labor by blocking distribution of sacral nerves 2, 3, and 4 (lower vagina and perineum). Useful for forceps, vacuum or episiotomy.
  - Perineal infiltration: Supplemental analgesia for the second stage of labor

ICD-9-CM Code: V22.2 (Pregnancy)

Perioperative Management

**Monitoring**
- BP, pulse, O sat, FHR

**Anesthetic Technique**
- Lumbar sympathetic block
  - Sitting position
  - Bilateral L1 or L2 transverse process
  - Needle advanced approx 9 cm to anterolateral surface of L1 or L2 vertebral body
  - Inject bilaterally 10 mL of local anesthetic (with or without epinephrine or narcotic)
  - Will block approx 4–6 dermatomes
- Analgesia for 2–3 hr with bupivacaine or ropivacaine
- Paracervical block
  - Modified lithotomy position
  - 3–5 mL of LA each side, Iowa trumpet (needle guide) is very useful
  - First injection: Lateral fornix of vagina at 4-o’clock position, 0.5 cm deep
  - Second injection at 8-o’clock position
  - Epinephrine with LA may increase incidence of fetal bradycardia
  - Analgesia for 2–3 hr depending on local anesthetic
- Pudendal block
  - Transvaginal approach usual with Iowa trumpet needle guide
  - Bilateral (10 mL) local anesthetic injections 1 cm posteromedial to ischial spines
  - Failure rate high and perineal infiltration often needed
  - Perineal infiltration
  - Supplemental analgesia for vaginal delivery, episiotomy, forceps or vacuum delivery
  - Infiltration of several milliliters of LA into the posterior fourchette

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypotension (LSB)</td>
<td>Patient response</td>
<td>Peripheral vasodilation</td>
<td>BP</td>
</tr>
<tr>
<td></td>
<td>Hematoma</td>
<td>Vessel puncture</td>
<td>Swelling</td>
<td>MRI</td>
</tr>
<tr>
<td></td>
<td>Cardiac depression, arrest</td>
<td>IV injection</td>
<td>Unresponsive</td>
<td>ECG</td>
</tr>
<tr>
<td>CNS</td>
<td>Horner syndrome (LSB)</td>
<td>Subarachnoid injection</td>
<td>Eyelid droop, facial warmth</td>
<td>P exam</td>
</tr>
<tr>
<td></td>
<td>Total spinal anesthesia</td>
<td></td>
<td>Loss of consciousness</td>
<td>EMG</td>
</tr>
<tr>
<td></td>
<td>Nerve damage</td>
<td></td>
<td>Neuropathy</td>
<td></td>
</tr>
<tr>
<td>FETAL</td>
<td>Bradycardia (PCB)</td>
<td>Fetal absorption</td>
<td>Poor aseptic technique</td>
<td>MRI</td>
</tr>
<tr>
<td>INFECTIOUS</td>
<td>Retropsoas abscess</td>
<td></td>
<td>Fever, pain</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Subgluteal abscess</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Laparoscopy, Gynecologic

Susan Chan

Risk
- Incidence in USA: >400,000 pts undergoing gynecologic laparoscopy yearly; female > male (50:1)
- Most common gyn surgical procedure

Perioperative Risks
- Mortality: 1.6–11/100,000
- CV complications (e.g., air embolus): 1–10/100,000
- Intra-abdominal complications: 1%
- Postop pain necessitating hospitalization: 0.5–2%

Worry About
- Hypercarbia, resp acidosis, hypoxemia, pulm Htn, systemic vasodilation
- Pressure of pneumoperitoneum
- Hypothermia
- Pulm: Atelectasis, decreased FRC, high peak airway pressure, CO₂ embolus
- CV: Decreased venous return, decreased cardiac output, cardiac dysrhythmia
- Gastric reflux: esp. in pts with gastroparesis, hiatal hernia, obesity, or gastric outlet obstruction

Overview
- Endoscopic technique to visualize pelvic structure
- Adhesions and endometriosis can be treated endoscopically
- A small incision below umbilicus is made to insufflate CO₂ and two or more slightly larger incisions for insertion of visualization devices and instruments

Duration of hospital stay significantly reduced. Most performed on outpatient basis, but duration of procedure may exceed that for open technique
- Surgical complications incl misplacement of Veress needle or trocar resulting in acute hemorrhage; bowel, bladder, uterus perforation; SQ emphysema

ICD-9-CM Code: 54.21

Indications
- Tubal ligation, ectopic pregnancy, vaginal hysterectomy, PID, infertility
- Question of PID versus appendicitis

Perioperative Management

Monitoring
- Postop course more benign than in open procedure, yet intraop period may have much physiologic derangement
- Routine with ETCO₂ waveform

Airway
- Increased risk of passive regurgitation and aspiration due to Trendelenburg position and increased intra-abdominal pressure. GA with ET intubation most common.
- Laryngeal mask airway satisfactory alternative in nonobese pts undergoing relatively short procedures

Anesthetic Technique
- No advantage of less physiologic stress has been shown by using regional anesthesia
- With increased intra-abdominal pressure, increased ventilatory pressures required to ventilate
- NG tube recommended to minimize gastric reflux
- Complete relaxation of abd muscle

Anticipated Problems/Concerns
- Hypercarbia and acidosis most common physiologic complications when CO₂ used
- SQ emphysema, pneumothorax, pneumopericardium, pneumomediastinum, gas embolism less common
- Hypothermia and cardiac arrhythmia may be due to increased ventricular irritability, decreased venous return, decreased cardiac output, hypoventilation, gas embolism, or profound vagal response.
- Blind insertion of the Veress needle or trocar assoc with injuries to hollow viscera, major vessels, abd wall vessels

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CV arrhythmia; ↓ venous return; ↑ level of stress hormones</td>
<td>Chest pain, SOB, Hx of CAD, DM, arrhythmia</td>
<td>CV exam</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>If pre-existing lung disease, hypercarbia, acidosis, hypoxia not tolerated well</td>
<td>SOB, ↓ exercise tolerance</td>
<td>Chest exam</td>
<td>O₂ sat</td>
</tr>
<tr>
<td>GI</td>
<td>↑ Intra-abdominal pressure Trendelenburg position may ↑ chance of aspiration</td>
<td>DM, gastroparesis, hiatus hernia, obesity, gastric outlet obstruction</td>
<td>Airway exam</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Minimal blood loss normally</td>
<td>Hx of anemia, exercise tolerance</td>
<td>Vital signs</td>
<td>Hct</td>
</tr>
</tbody>
</table>

Laryngoscopy

**Risk**
- Direct laryngoscopy is performed for most tracheal intubations and to visualize pharynx and larynx for diagnostic and/or therapeutic purposes in pts with upper airway pathology
- All age groups

**Perioperative Risks**
- Depends on nature of complaint and underlying disease
- Htn and tachycardia common because of pain and stimulation of airway reflexes
- Tracheal intubation is assoc with higher incidence of postextubation airway obstruction requiring reintubation compared to other approaches of airway management such as supraglottic airway devices

**Indications and Usual Treatment**
- Routinely applied for tracheal intubation in the OR and outside of the OR
- In combination of esophagoscopy and bronchoscopy, is applied for diagnosis and staging of oropharyngolaryngeal malignant lesions

<table>
<thead>
<tr>
<th>ASSESSMENT POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>System</strong></td>
</tr>
<tr>
<td>--------------------</td>
</tr>
<tr>
<td>HEENT</td>
</tr>
<tr>
<td>CARDIO</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>RESP</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>AIRWAY</td>
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</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Closely monitor pts with lower airway obstruction
- Communicate with endoscopist, as airway is shared with endoscopist
- Check equipment that may be needed in case of failed rigid laryngoscopic intubation

**Monitoring**
- Routine

**Anesthetic Technique**
- Consider slow inhalation induction of general anesthesia maintaining spontaneous ventilation if symptoms of compromised airway are present
- Short-acting IV analgesics attenuate CV response to rigid laryngoscopy

- Short-acting muscle relaxant such as succinylcholine should be considered when difficult intubation is expected.
- Ventilate with 100% O₂ before intubation is attempted.

<table>
<thead>
<tr>
<th>Surgical Stages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt supine with head and neck in sniffing position</td>
</tr>
<tr>
<td>Laryngoscope is entered through right side of the mouth advancing midline until epiglottis is exposed.</td>
</tr>
<tr>
<td>With curve blade the tip is advanced in the vallecula lifting epiglottis indirectly to expose the glottis opening. With straight blade the tip of blade is placed under the tip of the epiglottis lifting it up to expose the vocal cords.</td>
</tr>
<tr>
<td>Steroid considered if airway is compromised or if manipulation was extensive</td>
</tr>
</tbody>
</table>

**Postoperative Concerns**
- Confirm position of the ETT by auscultation and CO₂ monitor.
- Secure the ETT with tape.
- Continue observation of airway.

**Anticipated Problems/Concerns**
- Unanticipated difficult or failed intubation
- Accidental extubation
- Blockage of the ETT
- Severe Htn and tachycardia in pts with CAD
- Danger of spinal cord injury in pts with unstable cervical spine
- Undiagnosed esophageal intubation will be lethal
**Laser Surgery of Airway**

**Risk**
- Incidence in USA: 3000–5000/y
- Race and gender predominance: none
- Laryngeal papilloma is more common in children ≤ 4 y than adults

**Perioperative Risks**
- Airway compromise (preop and postop)
- Airway fires (5–70 reported/y)

**Worry About**
- Loss of patent airway
- Displacement or ignition of ETT
- Postop laryngospasm

**Overview**
- Focused, coherent far-infrared (CO2) laser light can precisely vaporize superficial tissue lesions at a distance through free air (no contact)
- Shorter wavelength (i.e., Nd:YAG) laser light can coagulate and necrose deeper lesions

**ICD-9-CM Code:** 478.4 (Laryngeal polyp); 478.74 (Laryngeal stenosis)

**Indications**
- Many heterogeneous conditions, incl laryngeal papilloma, tracheal scarring, webs or synechiae, vascular malformations, neoplasms, idiopathic subglottic stenosis

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**ASSESSMENT POINTS**

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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Glottic or tracheal stenosis</td>
<td>DOE</td>
<td>Stridor</td>
<td>Indirect laryngoscopy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mallampati exam</td>
<td>PFTs (flow-volume loop)</td>
</tr>
<tr>
<td>RESP</td>
<td>Neoplasia (V/Q mismatch)</td>
<td>Hemoptysis</td>
<td>Auscultation</td>
<td>CT or MRI of chest ABGs</td>
</tr>
</tbody>
</table>


---

**Perioperative Implications**

**Preoperative Preparation**
- Consider antisympathetics
- Eye protection for OR personnel appropriate to laser wavelength

**Anesthetic Technique**
- GA
- FIO2, ≤40% to retard combustion, consider FIO2 of 21%
- N2O is contraindicated as it supports combustion
- Profound NMB

**Monitoring**
- Routine

**Airway**
- Use laser-safe ETTs, either commercial or smoothly wrapped with metal foil
- For surgical access, use smallest diameter tube consistent with adequate ventilation, i.e., 5.5–6.5 mm outside diameter for adults
- Jet ventilation may spread virus throughout resp tree

**Maintenance**
- Maintain close communication with surgeon as procedure may conclude without significant lead time
- Be ready to emergently extubate trachea and provide mask ventilation in case of airway fire

**Extubation**
- Despite suctioning, pharynx may contain blood that may promote laryngospasm.

---

**Anticipated Problems/Concerns**

- Residual NMB
- Misdirected laser beam igniting drapes
- Infection of OR personnel by vaporized but intact human papillomavirus (HPV)

- Some surgeons have strong preference for deep extubation following vocal cord surgery in order to avoid cough-induced glottic injury.

**Postoperative Period**
- Stridor, excess coughing, or bronchospasm warrants immediate investigation.

**Adjuncts**
- Topical lidocaine ointment on ETT and/or saline in cuff may retard ignition.
- Surgeons may place moist pledgets in airway—be sure to retrieve them.

---

**Anticipated Problems/Concerns**

- Airway fire: Clamp ETT and remove; then reintubate with new ETT
Liver Resection

Risk
- Liver resection is most commonly performed as part of the management of malignant disease.
- 95% of hepatic tumors are metastatic.
- 5% are 1st, hepatocellular carcinomas (HCC) being the most common.
- Incidence: 4/100,000 but rising
- M:F ratio: 2:1
- Racial predominance: Asian

Perioperative Risks
- 1–10% mortality dependant on institutional, surgical and anesthetic expertise combined with pt co-morbidities.
- Cirrhotic pts undergoing general surgery have an increased risk of mortality assoc with male gender, high Child-Pugh score, presence of ascites, raised plasma creatinine, Dx of COPD, preop infection, preop GI bleeding, high ASA status: presence of intraop hypotension

Worry About
- Extended surgical time esp. prolonged Pringle maneuver (clamping of hilar structures)

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Possible hyperdynamic circulation if cirrhotic</td>
<td>Fatigue, SOB</td>
<td>CV exam</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Possible diaphragmatic splinting due to ascites, pleural effusions</td>
<td>SOB</td>
<td>Resp exam</td>
<td>PFTs, ABGs</td>
</tr>
<tr>
<td>GI</td>
<td>Cirrhosis, ascites</td>
<td>↑ Abdominal girth</td>
<td>Hepatomegaly</td>
<td>Paracentesis</td>
</tr>
<tr>
<td>RENAL</td>
<td>Hepatorenal syndrome</td>
<td>Urine production and use of diuretics</td>
<td>UO, BUN, Cr</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulopathy</td>
<td>Bleeding Hx</td>
<td>PT, PTT, TEG</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
- Blood products must be made readily available.
- Coagulopathy should be corrected.
- Regional anesthesia may be inappropriate in the presence of coagulopathy or predicted extensive resection.

Monitoring
- Although massive blood loss may not be expected, it is necessary to prepare as if it were.
- Reliable, large-bore venous access must be obtained.
- Consider intra-arterial monitoring in long cases.
- Central venous access is highly desirable.
- Aim for a CVP ≤ 5 cm H2O to reduce blood loss during dissection.
- Air embolism is a possibility if the CVP falls too low or if there is sudden hypovolemia.

Induction
- No one technique has been shown to be better than another.
- Consider a rapid sequence induction esp. in cases complicated by ascites.
- Need for vena-cava cross clamp
- Blood loss, adequacy of vascular access and availability of blood products

Overview
- Liver resection surgery has become more common and in many pts may be considered to be curative. This has been largely due to improvements in preop investigations, pt selection and operative technique.
- Some pts undergoing liver resection will exhibit features of chronic liver disease.
- The extended heptectomy is usually a long case complicated by the potential for massive blood loss and fluid shifts. Hemodynamic instability from surgical interruption of the blood supply can cause significant problems.
- Segmentectomies may be performed with little physiological disturbance and resection is also possible laparoscopically.

ICD-9-CM Code: 155.0 (Malignant neoplasm of liver and intrahepatic bile ducts); 197.7 (Secondary malignant neoplasm of respiratory and digestive systems)

Indications and Usual Treatment
- Benign tumors: Hepatic adenoma, hemangioma, focal nodular hyperplasia
- Malignant tumors: Primary-HCC, cholangiocarcinoma, hepatoblastoma, angiosarcoma, lymphoma
- Metastatic: Breast, lung, pancreas, stomach, large intestine, ovary
- Other: Hydatid disease, living donor transplantation
- Depending on the nature and location, it may be possible to use radiofrequency or ethanol to ablate the lesion.
- Techniques for resection inc wedge, left lateral segment, right/left lobe and trisegmentectomies.

Adrian Hendrickse

Adrian Hendrickse
Liver Transplantation

**Risk**
- Incidence in USA: ~16,000 pts on waiting list
- 6319 transplants in 2008; M:F ratio 2:1; 9.7% <18 y.
- 6070 from deceased donors and 249 from living donors
- No significant difference in 5- pt or graft survival with LDLT or DDLT

**Perioperative Risks**
- MELD score: Implemented in 2002; predictor of mortality on the waiting list; 10% decrease in wait-list mortality since implementation
- MELD score = 9.6 × Loge (creatinine mg/dL) × 3.8 8 pt Loge (bilirubin mg/dL) + 11.2 × Loge (INR) + 6.4
- Increased MELD score a/w increased need for pressors, ventilator support, and transfusions.
- MELD modifiers for HCC, HRS, PPHTN (add 20 points to calculated MELD); HCC modifier has resulted in 5-fold increase in transplantation for HCC.

**Overview**
- Remove native liver and replace with whole or partial new liver
- Increased CIT is a/w increased short-term mortality, ICU LOS, postop biliary complications
- Technique of hepatectomy can impact renal outcome.

**Indications and Usual Treatment**
- Noncholestatic cirrhosis (HCV, alcoholic, HBV, cryptogenic, postnecrotic, neoplasm)
- Cholestatic cirrhosis (Primary sclerosing cholangitis or primary biliary cirrhosis)
- Biliary atresia
- Fulminant hepatitis (drug induced, viral, pregnancy)
- Inborn errors of metabolism; amyloidosis, hemochromatosis, etc.
- Contraindications: Infection, obesity (BMI >35), portal and mesenteric thrombosis

**Anesthetic Technique/Induction**
- GI prophylaxis
- Cautious anxiolysis
- RSI

**Surgical Stages**

**Preanhepatic**
- Replace blood loss with PRBC, factors
- Correct acidosis, electrolyte abn., treat increased glucos, consider fluids
- Prepare for hepatectomy tech.

**Anhepatic**
- Piggyback: Occlusion of portal V., hepatic A.&V.
- TVO: Supra and infrahepatic IVC clamping-60–70% decrease in preload. Pretreat with volume loading.
- VVBP: Less hemodynamic changes. Increased risk of clotting in the circuit, air embolus, increased surgical time.
- Increased requirement for Ca
- Correct lytes, Hb, coags in preparation for reperfusion
- Flush donor liver with saline, Albumin, blood.

**ASSESSMENT POINTS**

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<tbody>
<tr>
<td><strong>CARDIO</strong></td>
<td>↑ CO, CI, HR, CVP, SvO₂, ↓ SVR, ↓ N/P VPR and PCWP, cardiomypathy, CAD</td>
<td>Poor exercise tolerance, fatigue, DOE, orthopnea</td>
<td>↑ JVD, HJR, LE edema,</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td><strong>RESP</strong></td>
<td>Restrictive pattern, atelectasis, plural effusion, HPS (PaO₂ &lt;70 mmHg or A-a gradient &gt;20 mmHg), PPHTN (MAP &gt;25 mmHg)</td>
<td>SOB, DOE,</td>
<td>↓ SaO₂, BS, ↑ RR, clubbing, platypnea, orthodeoxia</td>
<td>RA-SaO₂ (supine &amp; standing), CXR, ABG, contrast ECHO, IC-macroaggregates lung scanning, pulm angiography</td>
</tr>
<tr>
<td><strong>GI</strong></td>
<td>Portal Htn, varices, GI bleed, ascites, delayed gastric emptying, cholestasis</td>
<td>Hematemesis/malena, water retention, N/V, itching, confusion</td>
<td>Icterus, spider angioma, ascites, hepatosplenomegaly</td>
<td>↑ Transaminases, bili, INR, ↓ Albumin, upper GI, CT scan</td>
</tr>
<tr>
<td><strong>HEME</strong></td>
<td>Anemia, ↑ plts, coagulopathy, hypercoagulopathy</td>
<td>Bleeding diathesis, portal and mesenteric thrombosis, DVT, PE</td>
<td>Easy bruisability</td>
<td>CBC, plt count and function, INR, PT, PTT, fibrinogen, TEG</td>
</tr>
<tr>
<td><strong>RENA</strong></td>
<td>Oliguria, HRS</td>
<td>Nephrotoxic drugs (NSAIDs, ABx, IV contrast) aggressive diuresis, hypotension</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>CNS</strong></td>
<td>Encephalopathy, ↑ ICP in ALF</td>
<td>Confusion, mental status changes, ↓ memory, precipitating factor</td>
<td>Lethargy, coma, papilladema</td>
<td>ICP monitor, Transcranial Doppler</td>
</tr>
<tr>
<td><strong>INFECTION</strong></td>
<td>SBP, sepsis</td>
<td>Onset of fever</td>
<td>Fever</td>
<td>WBC w/diff, acitic fluid exam</td>
</tr>
<tr>
<td><strong>NUTRITION</strong></td>
<td>Malnutrition, folate, Zn, vit.B &amp; fat-soluble vit. deficiency, osteopenia</td>
<td></td>
<td></td>
<td>Glucose, Na, K, Ca</td>
</tr>
</tbody>
</table>

Reperfusion
- Postreperfusion syndrome: Decreased MAP; severely decreased SVR, HR; increased CVP, PAP; decreased contractility; may progress to asystole; PE.
- Treat coagulopathy and electrolyte imbalance
- Worry about fibrinolysis and overcorrection of coagulopathy.

Postoperative Considerations
- Continue correcting bleeding and lytes abn.
- Consider early extubation, if appropriate.
- Watch for graft function with improving lactate, coagulopathy.

Anticipated Problems/Concerns
- Massive hemorrhage
- Cardiopulmonary events
- Hypercoagulopathy, low flow state and portal venous thrombosis; Hepatic A. thrombosis.
Liver Transplantation, Pediatric

Christopher Karsanac

Risk
- Incidence in USA: 15,807 pts are on the waiting list for liver transplantation
- 341 are under the age of 18
- Children <18 y of age underwent 483 transplantations in 2009 (OPTN data)

Perioperative Risks
- Global physiological derangements
- 2–5% mortality
- Reoperation risk of 18%

Worry About
- Encephalopathy requiring ICP monitoring
- Hepatopulmonary syndrome causing hypoxia
- Electrolyte and metabolic derangements
- Renal failure 2° to hepatorenal syndrome
- Air embolisms
- Hemodynamic instability
- Coagulopathies
- Hypothermia
- Immunosuppression

ASSESSMENT POINTS

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>CARDIO</td>
<td>Hyperdynamic circulatory state (↑ CO, ↓ SVR), Cardiomyopathy, ↓ CO with palp Htn</td>
<td>SOB, fatigue, symptoms of CHF</td>
<td>CV exam, anasarca</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxia 2° to pleural effusion/ pulm edema, atelectasis due to ascites, ↓ FRC, V/Q mismatch, intrapulmonary shunting, pulm Htn, hepatopulmonary syndrome, subglottic stenosis 2° to previous prolonged intubation</td>
<td>Dyspnea, SOB</td>
<td>↓ SpO₂, tachypnea, auscultation</td>
<td>CXR, ABGs</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal dysfunction due to prerenal azotemia Hepatorenal syndrome</td>
<td>UO</td>
<td></td>
<td>BUN, Cr, electrolytes, CrCl if indicated</td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulopathy, anemia, thrombocytopenia, DIC</td>
<td></td>
<td>Bleeding time, PT, APTT, thrombin time, FDPs</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalopathy, abn blood-brain barrier, cerebral edema in fulminant hepatic failure</td>
<td>Sx of ↑ ICP</td>
<td>Ammonia level, ↑ ICP in fulminant failure, head CT (to R/O acute bleed)</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>GI varicies, delayed gastric emptying</td>
<td>Edema</td>
<td>Bilirubin, serum albumin, SGOT, SGPT</td>
<td></td>
</tr>
<tr>
<td>FLUID, LYTE, ACID-BASE STATUS</td>
<td>Intravascular volume depletion Hypokalemia, hyponatremia (hyperkalemia in hepatorenal syndrome), metabolic acidosis, ↓ glycogen metabolism</td>
<td>Symptoms of hypoglycemia (lethargy, somnolence, irritability)</td>
<td>ABG, electrolytes, glucose</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Room preparation may take up to 1 hr: incl fluid warmers, forced air warmers, emergency drugs, blood and/or blood products, US for IV access, multiple cuffed tubes
- IV premedication as appropriate

Anesthetic Technique
- Rapid-sequence or modified rapid-sequence IV induction
- Volatile agent maintenance while avoiding N₂O, muscle relaxation and fentanyl 10–50 mcg/kg

Monitoring and Positioning
- Standard ASA monitors
- CVP from central line via internal jugular or subclavian plus two large-bore IVs above the diaphragm
- Right and left arterial cannulation for BP monitoring and blood specimen collection

Surgical Stages

Preamhepatic Phase
- Skin incision to remove recipient’s liver
- Copious blood loss is possible if portal Htn or adhesions from previous surgeries

Overview
- Most common cause for pediatric transplantation is biliary atresia (~40%).
- Other pathologies incl: TPN related (15%), acute hepatic necrosis (10–15%), metabolic diseases (10%) and hepatoblastoma (3%)
- Demand out numbers supply with the added disparity of donor-recipient organ size mismatch
- Due to shortage of organs, recent increases in living-related and split-liver transplantation

Indications and Usual Treatment
- Indications for transplantation: Progressive primary liver disease, metabolic diseases of the liver, fulminant hepatic failure, hepatic tumors, or retransplantation for graft failure

- Peripheral nerve injuries due to positioning
- Anemia
- Massive fluid shifts
- Pain scores of 10 out of 10 common
- Blood loss is replaced by PRBCs and FFP
- Frequent coagulation testing; avoid a hypercoagulable state for fear of future graft anastomosis thrombosis
- Massive blood transfusion assoc with increased citrate load, decreased calcium, increased potassium
- Hourly blood labs are a minimum; may need q15 min labs during hemodynamic instability
- UO maintained
- Maintain adequate BP with lowest CVP to decrease bleeding

Anhepatic Stage
- Starts with clamping of hepatic vessels
- IVC may be clamped (drastically decrease preload), but usually not in a “piggy back” technique
- Donor liver may decrease core temp by 1.5° or more
• Drastically decrease clearance of hepatically metabolized drugs, heavily decreased glycogen formation
• Decrease volatile, 100% FIO₂ and ready emergency drugs for reperfusion stage

**Post Perfusion Stage**
• Reperfusion of donor liver after unclamping portal vein and IVC
• Anticipate initial hemodynamic instability, dysrhythmias, lactic acidosis
• Rapid increase in K⁺ level may cause cardiac arrest

• Split liver grafts may bleed more upon reperfusion
• Improvement in coagulation, decrease in lactic acid, and normalization of acid-base status and lytes
• Biliary system will then be constructed

**Postoperative Period**
• Transferred to PICU intubated
• Hepatic functions monitored and PT maintained 1.5–2.0 times normal to avoid hepatic artery thrombosis

**Anticipated Problems/Concerns**
• Discuss administrative timing of antirejection drugs with team prior to surgery
• Bleeding
• Portal vein thrombosis and/or hepatic artery thrombosis
• Primary graft nonfunction and/or rejection
• Renal failure
• Electrolyte abn
• Pulm complications
Lumbar Laminectomy

Moustafa Ahmed

**Risk**
- 2% of adult population suffer from sciatica due to lumbar disc herniation
- 90% improve with nonoperative care (6–12 wk)
- Neurologic complication range from 1–33%
- The reported success rate of lumbar disc surgery varies from 60–90%

**Perioperative Risks**
- Preop mortality is low and assoc with preop co-morbidity
- Continuation or recurrence of lower back pain range from 30–40%
- Recurrent disc herniation range from 4–8%
- Major complication such as pulm embolism, hemorrhage from major vessels injury is fatal but rare

**Worry About**
- Intrapar blood loss
- Be aware that sudden drop in the BP may be a venous air embolism (in prone position). It is rare but possible because the venous system above the level of the heart.
- Be aware of sudden collapse of the ventilator billows after positioning the pt in prone position or during surgery may be due to accidental extubation.
- Decrease lung compliance because of chest wall and abd compression in prone position
- Complication of prone position, direct pressure over bony prominences may lead to tissue necrosis, direct pressure on the orbit may lead to blindness due to ION, stretching or direct pressure on the nerves may lead to nerve injury, and hyperextension of the neck may lead to Horner's syndrome.
- Irreversible pyramidal tract damage during decompressive laminectomy in pts with spinal stenosis.
- Epidural hematoma postop is rare but devastating if it happens, the risk increases with pts who require multilevel lumbar procedures and/or have coagulopathy. Immediate Dx and treatment of postop epidural hematoma are crucial.

**Overview**
- Symptomatic lumbar disc herniation assoc with sciatica occurs in approx 2% of the adult population.
- 90% of the pt cured with nonoperative care
- Acute surgical emergency occur with cauda equine syndrome when the back pain assoc with saddle anesthesia, urinary retention, and multiple nerve root involvement
- Another indication for surgery is intractable pain not improve by nonoperative care or progressive neurologic deterioration

ICD9-CM: 722.10 (Lumbar intervertebral disc without myelopathy)

### ASSESSMENT POINTS

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<tbody>
<tr>
<td>CARDIO</td>
<td>Decomposition</td>
<td>↓ Exercise tolerance—difficult to assess</td>
<td>Heart murmur, gallop</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Restrictive diaphragm or other rib cage movement</td>
<td>Smoking Hx, pulm disease</td>
<td>Lung exam—signs of failure?</td>
<td>PFTs if severe lung disease</td>
</tr>
<tr>
<td>CNS/PNS</td>
<td>Peripheral neuropathy, paraplegia, myelopathy or sensory deficit, compromised by prone position</td>
<td>Pain, inability to ambulate, bowel or bladder dysfunction</td>
<td>Sensory deficit, ?motor defect</td>
<td>Preop evoked potentials, EMG, or MRI/CT</td>
</tr>
<tr>
<td>MS</td>
<td>Skeletal metastasis from primary cancer</td>
<td>Primary tumor from breast, lung, kidney, thyroid, prostate</td>
<td>Chemotherapy Hx</td>
<td>?CXR, electrolytes (Ca++), bone scan</td>
</tr>
<tr>
<td>PSYCH</td>
<td>Chronic pain, possible substance abuse (opioids)</td>
<td>Multiple medications (narcotics, NSAIDs, antidepressants)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Management**
- Intravenous prophylactic antibiotics

**Anesthetic Technique/Induction**
- Most common practice using general anesthesia, it can be done by neuroaxial (spinal or epidural) anesthesia, the broad use of GA is not based on any scientific or clinical evidence but on surgeon preference, the arguments against neuroaxial anesthesia is airway complications in a sedated prone positioned pt and possibility for neural injury if the awake patient moves or shifts during the decompressive procedure.
- Muscle relaxant is given to facilitate intubation and muscle dissection, avoid succinylcholine if severe neurological deficit is present (paraplegia)
- TIVA is preferred with SSEPs and EMG for better monitoring

**Monitoring**
- Standard ASA monitors
- Monitoring for possible venous air embolism in prone position using Precedrill Doppler/estimal T, or TEE
- Invasive monitoring such as intra-arterial and/or CVP based on associated co-morbidity and neurological deficits
- SSEPs and EMGs for neurologic monitoring especially with patient suffering from spinal stenosis

**Airway**
- Securing the airway is crucial since the patient is in prone position, confirm ET position above the carina after change patient to prone position
- Be aware of facial edema and possible laryngeal edema in prolonged prone position (multiple level laminectomy), if no cuff look consider postop ventilation for the next 24 hr

**Indications and Usual Treatment**
- One of the most common back surgeries world-wide and still controversial among neurosurgeons and orthopedic surgeons.
- Indications categorized to four groups: degenerative, traumatic, neoplastic and infectious
- May affect one level or multiple levels, 90% of disc prolapses involve the L4-S and L5-S1 segments, decompressive laminectomy with or without fusion and/or instrumentation and fixation indicated in lumbar spinal stenosis, degenerative spondylolisthesis and/or stenosis, degenerative scoliosis and/or stenosis, lumbar vertebra fractures, tumors and low back pain
- Lumbar stenosis, either congenital-achondroplastic dwarfs and manifested as kyphosis, bulging discs and short pedicles or acquired, which is the most common form due to disc degeneration, leads to hypertrophy of both bone and ligaments, thereby compressing the neural elements.
- Recommend psychological consultation before any elective lower back surgery even when there are clinical indications especially if assoc with obvious psychosocial issues
- Nonop care: Brief period of bed rest, use of NSAIDs and active physical therapy as tolerated, the efficacy of epidural steroids or even oral steroids has not been convincingly demonstrated in randomized prospective studies, the use of Transcutaneous Electrical Nerve Stimulation (TENS) is controversial.

**Positioning**
- Patient placed in prone, lateral or in knee-to-chest position (prone-sitting frame)
- Padding all pressure points, no pressure applied to the eyes and nose during the length of surgery
- Be aware to free the male genitalia
- Maintenance. Balanced anesthesia, maintaining euvoletic status to minimize facial edema and possible laryngeal swelling, if the spinal cord compromised with neurologic deficits, maintains spinal cord perfusion pressure by maintaining MAP as preoperative or higher.

**Procedures**

Surgical Stages

**Dissection**
- Different technique used, posterior approach, minimally invasive techniques, minimvasive extra-peritoneal approach.
- The skin incision in general is approximately 3–5 cm in the midline over the spinous process, dissection is carried through the subcutaneous fat to the lumbar dorsal fascia, midline is easily identified by palpating the groove over the spinous processes, on the side of disc prolapsed the fascia divided and the lateral spinal dissection then follows, the ligamentum flavum is gently dissected free and removed, then laminotomy is performed, the affected nerve root is swept medially and the disc fragment visualized the annulus is then incised and the fragment removed. The nerve should be inspected to be certain that no residual disk compression remains. The wound should be closed in appropriate layers.

**Postoperative Considerations**
- Early neurological assessment and frequent examination for early detection of any neurological deficits
- Postoperative pain can be controlled using PCA, intraoperative intrathecal morphine or placement of epidural catheter at the end of surgery before closing (can be placed by surgeon or anesthesiology)
- No postoperative orthosis is required
- The necessity and duration of activity restrictions after lumbar disc surgery is still controversial.

**Anticipated Problems/Concerns**
- Disease of elderly (CAD, COPD, CKD, DM)
- Postoperative neurological deterioration may be caused by extruded disc fragments, mechanical damage to nerve root, epidural hematoma, infections, epidural abscesses.
- Problems with prone positioning
- Reoperation, recurrence of lumbar disc prolapsed range from 3–12% in patients who underwent disc surgery for first time
- Remote cerebellar hemorrhage (rare)
Lung Transplantation

**Risks**
- Worldwide: >2000 lung transplants per year for end-stage lung disease (bilateral/single, 2:1)
- Etiology of underlying lung disease: COPD (50%), IPF (19%), CF (17%), idiopathic PAH (5%)
- Recipient age varies with underlying lung disease, although peak presentation in sixth decade
- Co-morbidities vary with underlying disease process
  - COPD: Smoking related vascular disease
  - Cystic fibrosis (CF): Diabetes mellitus, GERD, malnutrition
  - Pulmonary arterial Htn (PAH): Typically anticoagulated with coumadin

**Perioperative Risks**
- Depends on underlying lung disease and other co-morbidities
- 30-d mortality: 4–15%
- Primary graft dysfunction (PGD): Incidence 10–65% depending on definition. Severe PGD assoc with prolonged mechanical ventilation, increased ICU length of stay, increased 30-d mortality and increased incidence of bronchiolitis obliterans
- Myocardial ischemia and/or infarction: Incidence not well-characterized
- Acute kidney injury: Reported incidence 50–60% within 2 wk, dialysis in up to 7.5%

**Perioperative Management**

**Preoperative Preparation**
- Assess pattern of underlying ventilatory defect (obstructive vs. restrictive), baseline SpO2
- Assess co-existing morbidities, esp. pulm Htn +/- RV dysfunction, LV dysfunction, DM, GERD, anticoagulation status
- Consider epidural catheter placement (or paravertebral catheter for single lung transplant)

**Monitoring**
- Invasive arterial pressure monitoring and central venous access essential
- PA catheter routinely used—may guide selective use of inhaled vasodilators (NO, prostacyclin)
- Intraop TEE allows assessment of RV and LV function, intravascular volume state, patent foramen ovale (PFO), pulm venous anastomoses
- Consider depth of anesthesia monitoring (e.g., BIS)

**Anesthetic Technique/Induction**
- No single technique demonstrated to be superior, hemodynamic stability is the goal
- Beware gas trapping/dynamic hyperinflation/pneumothorax with PPV and subsequent hemodynamic instability or collapse

**Assessment by Hx**
- Blood glucose, HbA1c
- Creatinine, creatinine clearance
- Dynamic instability or collapse
- Pneumothorax with PPV and subsequent hemo.

**Overview**
- Anesthesia goals: Maintain adequate oxygenation with one-lung ventilation (OLV), minimize pulm vascular resistance (PVR), optimize coronary perfusion pressure to reduce risk of acute RV failure

**ASSESSMENT POINTS**

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<tbody>
<tr>
<td>HEENT</td>
<td>Need to secure airway with double lumen ETT</td>
<td>Previous anesthesia difficulties, GERD</td>
<td>Clinical assessment of airway</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Pulm Htn with RV dysfunction +/- TR, CAD with LV dysfunction</td>
<td>Angina, orthopnea</td>
<td>Elevated JVP, peripheral edema</td>
<td>ECG, ECHO, dipyridamole thallium, coronary angiography, right heart cath</td>
</tr>
<tr>
<td>RESP</td>
<td>COPD/CF/interstitial lung disease/PAH</td>
<td>Functional capacity, productive cough, hemoptyis, Supplemental O2</td>
<td>Bronchospasm, wheeze, hyperinflated, cyanotic</td>
<td>CXR, ABGs, PFTs, V/Q split function testing</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Htn, diabetes</td>
<td>-</td>
<td>Creatinine, creatinine clearance</td>
</tr>
<tr>
<td>ENDO</td>
<td>Pancreatic insufficiency (CF), steroid use</td>
<td>Diabetes, medications</td>
<td>-</td>
<td>Blood glucose, HbA1c</td>
</tr>
</tbody>
</table>


**Indications and Usual Treatment**
- Depends on underlying disease process and may vary between pts
- In general: Age >65 y, NYH III or IV but ideally still ambulatory; 6-min. walk test >600 feet
- Contraindications: Significant non-pulm vital organ dysfunction, active infection with HIV, hepatitis B or C, active cigarette smoking or substance abuse, medical non-compliance, lack of social support
- Allocation system based on clinical urgency rather than time accrued on wait list

**Surgical Stages**

**Dissection**
- OLV required, may see refractory hypoxemia +/- hypercapnia. Consider INO or prostacyclin if usual measures fail
- Blood and fluid loss may be significant if extensive adhesions present
- May require vasopressors and judicious IV fluid replacement to support hemodynamics

**Pulmonary Artery Clamping**
- Ensure PA catheter withdrawn from PA prior to clamping. Consider heparin 2500–3000 U.
- Monitor PA pressure and acute RV failure (rising CVP, distending RV, acute TR on TEE)
- May require vasopressors, inotropes, iNO to support RV function and systemic hemodynamics. Consider CPB if necessary.

**Allograft Reperfusion**
- Anticipate hemodynamic instability due to volume loss into new lung, washout of metabolites and pneumoplegic solution, marked systemic vasodilation.
- Anticipate systemic air from allograft which may enter right coronary artery producing acute ischemia, RV dysfunction, hypotension, and arrhythmias
- Support with vasopressors, volume resuscitation, defibrillation if required.

**Postoperative Considerations**
- Change to single lumen ETT if double-lumen in place. Bronchoscopy to provide pulm toilet and assess bronchial anastomoses, optimize re-expansion and V/Q matching with lung recruitment and PEEP.
- Protective ventilation to minimize volu- and baro-trauma is not well studied in humans. Animal studies suggest improved graft function with these strategies. Reduce FIO2 as tolerated

**Cardiopulmonary bypass (CPB):** Used in 15–40% of cases, typically due to severe pulm Htn, acute RV failure, hemodynamic instability or refractory hypoxemia

ICD-9-CM Code: 33.50 (Lung transplantation, not otherwise specified)
• Early tracheal extubation if allografts function well and analgesia adequate. May reduce incidence of ventilator-induced lung injury and ventilator-assoc pneumonia.
• Epidural infusion of local anesthetic may necessitate judicious IV fluid and vasopressor support to maintain hemodynamic stability. Systemic ketamine or dexmedetomidine may be considered as adjuncts for improved postop analgesia and/or opioid-sparing effects.

Anticipated Problems/Concerns
• Intraop use of epidural local anesthesia may increase hemodynamic instability
• Difficult to maintain normothermia due to large body surface area exposed, large incision, prolonged surgical time and potential for large fluid requirements
• Controversies: Potential for anesthetic techniques to impact reperfusion injury remains unclear, e.g., fluid management, preemptive iNO.
Early versus late ECMO for severe PGD
Lung Volume Reduction Surgery (LVRS)

Swaminathan Karthik
Brett A. Simon

Risk
- Severe, activity-limiting pulmonary emphysema; given prevalence of emphysema (2 million Americans affected with 90,000 deaths annually), the number of potential candidates for this operation is enormous.
- Average preop FEV₁, 25–30% of predicted early studies had contradictory results, and variable outcomes.
- The National Emphysema Treatment Trial [NETT], a prospective randomized trial was conducted to compare medical and surgical treatments with a 3-5 year follow-up.

Perioperative Risks
- LVRS not recommended if FEV₁ <20% and either DLCO <20% or diffuse pattern of emphysema seen on CT scan.
- In-hospital mortality: 5.5%
- LVRS most beneficial in pts with both upper lobe predominant disease and low exercise tolerance. LVRS is not superior to medical therapy if only one of these two characteristic is present.
- Highest mortality and cardiopulmonary complications occurred in pts with non-upper lobe predominant disease and good exercise tolerance.
- 25% morbidity incl prolonged air leaks, resp failure, pulm embolism, pneumonia
- Greatly increased risk of complications in pts with reactive airway disease, CAD, pulm Htn

Worry About
- Hypotension due to air trapping with controlled ventilation
- Difficulty with ventilation and oxygenation (less so) during one-lung ventilation
- Exaggerated resp depress effects of narcotics (IV and neuraxial)
- Minimizing airway pressures and smoothly expirating spontaneously ventilating pt in OR to avoid creating or worsening air leaks

Overview
- Palliative procedure for severe, activity-limiting emphysema with 20–30% of lungs resected to reduce lung volume and reshape diafragm and chest wall
- Bovine pericardial or Gore-Tex strips used to reinforce staple lines and reduce air leaks
- Variety of unilateral, bilateral, open, and VATS techniques used. Open, bilateral lung volume reduction via median sternotomy is the original approach. The NETT study showed no difference in mortality or morbidity between the median sternotomy approach and VATS. However, median sternotomy was assoc with longer hospital stays and higher costs.
- Benefit thought to result from improving mechanical function of chest wall and diaphragm by reducing total lung volume, combined with reduction and reshaping of lung tissue; results in increase in lung recoil at this lower volume and increased expiratory flows
- Pts typically require a great deal of attention to keep them extubated during first several hours postop
- Successful outcome requires team approach incl experienced pulmonologists, pulm rehab, thoracic surgeons, anesthesiologists, pain service, chest PT, ICU physicians. Sophisticated pulm function testing and lung imaging facilities should be available.
- Endoscopic LVRS is an evolving alternative. Methods under investigation incl one-way endobronchial valves or airway bypass stents (drug eluting-paclitaxel)

ICD-9-CM Code: 492.8 (Emphysema)

Indications and Usual Treatment
- Alternative to lung transplantation for pts with primarily pure emphysema that significantly limits their activity
- Exclusion criteria incl pulm Htn (mean PAP >35 at rest), bronchospasm, LV dysfunction, bronchitis or excessive sputum production, persistent smoking, previous thoracotomy or pleurectomy, obesity, or cachexia
- NETT pts required to undergo at least 6 wk of preop pulm rehab, with supplemental O₂ if necessary. Poor results expected if cannot perform at least 800 ft in standard 6-min walk test.
- Successful procedures typically result in 60–70% increase in FEV₁, by 3 mo sustained at least 1 y; decreased TLC and residual volume, improved exercise tolerance, and significant reductions in O₂ requirements at rest and during exercise. Data suggest improvements are sustained for at least 2–3 y.

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<tbody>
<tr>
<td>CARDIO</td>
<td>LV/RV dysfunction</td>
<td>CHF, PND, palpitations</td>
<td>Peripheral edema, JVD, S₁</td>
<td>MUGA, as ECHO may be unreliable with COPD</td>
</tr>
<tr>
<td>RESP</td>
<td>Emphysema</td>
<td>SOB, exercise tolerance and performance with rehab, bronchospasm, sputum production</td>
<td>Wheezing</td>
<td>PFTs, CT, plethysmography, quantitative V/Q scan</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
- Successful completion of pulm rehab program
- Maximize bronchodilator therapy, smoking cessation, O₂ therapy, and influenza and pneumococcal vaccinations

Anesthetic Technique
- Thoracic epidural placement at T₄ interspace for intraop and postop use; test and verify onset of segmental block prior to induction
- Minimize narcotics because of risk of resp depression
- Goal of anesthetic is to maximize possibility of extubation in OR
- Chest tubes placed to water seal only, unless suction required

Monitoring
- Arterial line required
- Consider central line for intraop infusions and postop fluid management

Airway
- Double-lumen tube required

Induction/Maintenance
- Anticipate hypotension due to air trapping with chest closed
- Ventilate with low pressures and low rates; tolerate hypercapnia if necessary
- Maintain on low-dose inhaled agent or propofol infusion
- Use of epidural depends on hemodynamic stability; consider waiting until chest open before dosing; use of strait local may require phe-nylephrine infusion for BP maintenance; if narcotics used, limit dose
- Continue bronchodiator therapy in OR if necessary

Surgical Stages
- Bronchoscopy: Flexible bronchoscopy
- Resection: Better side, usually right, resected first to improve tolerance of one-lung ventilation when second side resected (for bilateral procedure)
- Emergence: Consider switch to single-lumen tube, LMA, or mask while pt is deep to facilitate emergence
- Elevate head; optimize pain control; suctioning; bronchodilators for emergence
- Pts require up to 30–90 min observation, encouragement, and fine-tuning in OR prior to transport to ICU; first ABG in ICU has Pco₂ >70 mmHg in >50% of pts

Fluid Considerations
- Typically run dry
- Significant blood loss requiring transfusion unusual

Postoperative Considerations
- Make every effort to extubate in OR and avoid reintubation and ventilation; if required, use minimum pressure support without mandatory breaths if possible
- Use epidural infusion (½–¾% [0.0625–0.125%] bupivacaine ±1–3 μg/ml fentanyl) supplemented with non-narcotic pain relievers
- Pain score: 6–8

Anticipated Problems/Concerns
- Extremely marginal pts susceptible to even mild postop insults (pneumonia, pulm embolism, oversedation, pneumothorax, bronchospasm)
- Reintubation and mechanical ventilation assoc with high morbidity
Mastectomy

Risk
- Overall incidence of breast cancer is 127.8 cases per 100,000 women. Median age 61.
- In 2009, 192,000 new cases w/62,000 Carcinoma in situ diagnosed
- Rates less in African-Americans, with greater mortality rates
- Age, alcohol, obesity, hormonal influences increase risk.
- BRCA1/2 genes ~5–10%
- 5-y survival rates 83–90%
- Death rates 22% to 40% nationwide

Perioperative Risks
- Basically dependent on co-morbidities
- Postop complications greater in smokers, age >65, and obesity; with increased risk of failure of reconstruction in smokers, obesity, Htn.
- Usual risks of field avoidance, positioning

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Airway</td>
<td>Sore throat prior ops, Hx of difficult intubation</td>
<td>Usual A/W exam</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Circulatory integrity</td>
<td>Activity level</td>
<td>Examine/listen</td>
<td>EKG/CXR as appropriate</td>
</tr>
<tr>
<td>RESP</td>
<td>Ventilatory sufficiency</td>
<td>Smoking, bronchitis/RAD</td>
<td>Clinical assessment</td>
<td>CXR/PFTs</td>
</tr>
<tr>
<td>RENAL</td>
<td>Fluid balance</td>
<td>Use of diuretics/prior insults</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Anxiolysis</td>
<td>Neuro-psych Hx</td>
<td>Behavior w/interview</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>CV stability</td>
<td>Hx of anemia</td>
<td></td>
<td>Hct/T&amp;S as indicated</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
- Optimize co-morbidities; current consults with perioperative recommendations
- Initiate N/V prophylaxis, hydration, timely anxiolysis, prophylactic antibiotics, thromboembolic precautions.
- Confirm D/C of aspirin, anticoagulants, and supplements.

Monitoring
- Routine ASA; place IV in contralateral arm/neck/foot; EKG leads out of the field.
- Invasive as indicated by co-morbidities

Anesthetic Technique/Induction
- MAC: Local per surgeon with sedation as appropriate.

Worry About
- Choice of anesthesia
- Need for anxiolysis; no sedation prior to needle localization, or prior to plastic surgeon marking, if combined procedure
- Co-morbidities
- Positioning (brachial plexopathies reported)
- Field avoidance (eye protection, NIBP position, SpO₂ sensor position, IV integrity)

Overview
- Majority of procedures involve skin, SQ tissue, breast tissue; few involve muscles (true radical or reconstructive technique).
- Some surgeons concerned for long thoracic nerve integrity and may wish to use nerve stimulator (hampered by muscle relaxants).
- Anesthesia goals are to provide excellent operating conditions for surgical team, optimization of co-morbidities, pain and nausea-free PACU experience with proper anxiolysis throughout.

Indications and Usual Treatment
- Breast cancer diagnosed by mammography, needle aspiration, excisional biopsy.
- Less commonly, severely symptomatic fibrocystic breast disease
- Procedures range from simple lumpectomy, partial mastectomy, simple mastectomy, modified radical mastectomy, to (rarely) radical mastectomy.
- Increasingly common for plastic surgeon to be involved in closure, or first stage reconstruction procedure after mastectomy performed.

Postoperative Considerations
- Pain and nausea issues in PACU
- Adequate perfusion of flaps in closure or reconstruction (wound complications #1 morbidity)
- Attention to co-morbidities (morbid obesity correlates highest with postop complications)

Anticipated Problems/Concerns
- Prompt attention to nausea and analgesia problems may avoid admission.
- Early ambulation and return to activity will facilitate discharge.
- Psychiatric problems common postop, handle with care, provide appropriate support.
Meningomyelocele Repair

Cynthia Tung

Risk
- Incidence: 2–5/1000 live births
- Results from failure of neural tube to close; most commonly occurs along thoracic or lumbosacral region, can also occur along cervical region

Perioperative Risks
- Bacterial contamination leading to infection or sepsis
- Poor autonomic control below level of lesion, must maintain core body temp
- Potential for blood loss, hypotension
- Assoc with Arnold-Chiari malformations, hydrocephalus
- No assoc with congenital cardiac anomalies

Perioperative Management

Preoperative Preparation
- Assess for other congenital malformations, document any preop neurologic deficits.
- Antenatal Hx, birth Hx, other co-morbidities
- Latex precautions

Monitoring
- Depends on co-morbidities; usually standard ECG and non-invasive BP monitoring, pulse oximeter; arterial line placement based on other co-morbidities
- Adequate IV access or umbilical lines for fluid replacement and possible blood transfusion

Overview
- Occurs at 28 d gestation, failure of neural tube to close
- Antenatal diagnosis with US or amniocentesis
- Assoc with Arnold-Chiari malformations, hydrocephalus, neurologic deficits
- Definitive treatment is surgical

ICD-9-CM Code: 741.9 (Without mention of hydrocephalus)

Indications and Usual Treatment
- Surgical repair within first day of life to prevent infection
- Defect covered to protect from infection and fluid losses prior to surgery

ASSESSMENT POINTS

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<tbody>
<tr>
<td>CARDIO</td>
<td>Congenital cardiac anomalies</td>
<td></td>
<td>Murmur, cyanosis</td>
<td>ECHO, pulse oximeter</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp failure/RDS</td>
<td>Premature birth Location of defect; cervical, thoracic, or lumbosacral</td>
<td>Resp insufficiency</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Congenital anomalies</td>
<td>Abd exam</td>
<td>Abd US</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Cord compression, paralysis Arnold-Chiari malformations Hydrocephalus</td>
<td>Neuro exam</td>
<td>CT/MRI</td>
<td>US</td>
</tr>
</tbody>
</table>


Anesthetic Technique/Induction
- Depending on anticipated difficulty of airway, inhalation or IV induction with muscle relaxant. If awake intubation required, preoxygenate and premedicate with atropine.
- Monitor blood and fluid losses, replace as needed.
- Small defects: Minimal volume offluid losses
- Large defects: Large volume of fluid losses
- Maintain body temp: Keep neonate warm prior to surgical draping and at end of procedure, use forced hot air warmer intraop, humidified gases.

Surgical Stages
- Usually minimal blood loss
- Prone position

Postoperative Considerations
- Postop apnea, CSF leakage, neurologic deficits
- Recover prone

Anticipated Problems/Concerns
- Prematurity: Resp insufficiency, apnea, cardiac anomalies
- Other congenital anomalies or co-morbidities
- Postop neurologic deficits
- Future development of hydrocephalus requiring ventriculoperitoneal shunt
Mitral Valve Replacement

Risk
- Mitral stenosis
  - Rheumatic disease (predominant cause in adults); M:F 1:2. Less commonly systemic disease states or senile annulus calcification
  - Unrepaired 10- y survival: 15% if symptomatic, 80% if asymptomatic
  - Congenital abs of the mitral valve (children)
- Mitral regurgitation, organic
  - Dysfunction of mitral leaflets or chordae
  - Leaflet prolapse, endocarditis, rheumatic valve disease, connective tissue disorders and mitral annular calcification
- Mitral regurgitation, functional
  - Structurally normal leaflets and chordae tendineae
  - Ischemic heart disease
  - Idiopathic dilated cardiomyopathy and mitral annular calcification
- Mitral regurgitation unrepaird 5- y survival 27–97% because of variability of causes

Perioperative Risks
- Depend on pt characteristics such as age, functional status, and other co-morbid conditions
- Mitral stenosis surgical mortality
  - Less than 5% in younger pts with few co-morbidities
  - 10–20% in elderly pts with severe symptoms and preexisting medical problems

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</tr>
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<tbody>
<tr>
<td>CARDIO</td>
<td>MS: RV failure, pulm Htn, atrial fibrillation</td>
<td>MS: Fatigue, chest pain, hemoptysis, palpitations, dyspnea, pulm edema, hoarseness</td>
<td>MS: Opening snap, mid-diastolic, rumbling murmur</td>
<td>EKG, ECHO, cardiac catheterization</td>
</tr>
<tr>
<td></td>
<td>MR: LV dysfunction, atrial fibrillation</td>
<td>MR: Fatigue, dyspnea, pulm edema, orthopnea, PND</td>
<td>MR: Pan-systolic murmur to axilla, +/- 3rd heart sound; cardiomegaly if chronic</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pulmonary vascular congestion</td>
<td>Dyspnea, pulm edema</td>
<td>Edema</td>
<td>CYR, pulm vascular congestion</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency due to CHF</td>
<td>Renal vasoconstriction, sodium and water retention</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Systemic thromboembolism</td>
<td>Stroke, TIA</td>
<td>Focal deficits</td>
<td>Non contrast head CT, MRI</td>
</tr>
</tbody>
</table>

Anesthetic Technique/Induction
- Large IV access
- Maintain stable hemodynamics and rhythm; may need to defibrillate new onset AFib

Mitral stenosis
- Preload: Normal to slightly increased
- Afterload: Normal
- Heart rate: Normal, avoid tachycardia

Mitral regurgitation
- Preload: Increased
- Afterload: Decreased
- Heart rate: Mild tachycardia

Indications and Usual Treatment
- Mitral stenosis
  - Normal valve area 4–6 cm²
  - If valve area 1.5-2.5 cm², symptoms with moderate exertion
  - If valve area <1.5 cm², symptoms typically occur at rest
  - Symptoms incl dyspnea, palpitations (atrial fibrillation), or chest pain
- Mitral regurgitation
  - Acute MR often results in increases in left atrial pressures leading to pulm edema and biventricular failure
  - Ruptured papillary muscle is an indication for emergency surgery
  - Severe MR with evidence of left ventricular dysfunction (EF<60% m ESLV diameter >45 mm) should be referred to surgery
  - Asymptomatic pts without LV dysfunction may be surgical candidates if the effective regurgitant orifice is >40 mm² or there is evidence of atrial fibrillation, VTach, or pulm Htn.

Perioperative Preparation
- Dental evaluation to R/O acute inflammation/ infections and airway mgmt
- Optimize hemodynamics with medical management

Monitoring
- EKG for rate, rhythm, and ischemia
- Pre-induction invasive arterial pressure catheter
- Pulm artery catheter or central venous catheter
- Transesophageal ECHO

Preoperative Preparation
- Transesophageal ECHO
- Pulm artery catheter or central venous catheter
- Transesophageal ECHO
- Optimize hemodynamics with medical management

Monitoring
- EKG for rate, rhythm, and ischemia
- Pre-induction invasive arterial pressure catheter
- Pulm artery catheter or central venous catheter
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Surgical Stages
- Precordial pulmonary bypass
- TEE to measure valve motion, pressure gradients, orifice diameters and cardiac function

Surgical Repair (minimal versus open)
- Mitral stenosis
  - Mitral valve replacement (mechanical vs. tissue)
  - Percutaneous mitral commissurotomy
  - Surgical commissurotomy
  - +/- Maze procedure if chronic atrial fibrillation

- Mitral regurgitation
  - Mitral valve replacement (mechanical vs. tissue)
  - Mitral valve repair: Often involves resection of a portion of the mitral valve leaflet and at times an annuloplasty ring

**Postcardiopulmonary Bypass**
- Maintain sinus rhythm (please)
- RV dysfunction
  - Systemic vasodilators (milrinone, dobutamine, isoproterenol)
  - Inhaled vasodilators (NO or iloprost)
- LV dysfunction: Inotropic support, afterload reduction, IABP

**Postoperative Considerations**
- Irreversible pulm Htn or LV dysfunction
- Possible atrioventricular block requiring pacing
- Renal insufficiency or failure
- CNS events

**Anticipated Problems/Concerns**
- Pulm Htn and RV strain may necessitate post CPB inotropic and vasodilatory support
- Rhythm disturbances can be key
- Mitral valve replacement can result in perivalvular leak
- Mitral valve repair can result in systolic anterior motion of mitral valve with LVOT obstruction in 4–5% of pts
- Maintain eucarbia or slight hypcarbia
**Muscle Biopsy for Undiagnosed Myopathy**

**ASSESSMENT POINTS**

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<tbody>
<tr>
<td>HEENT</td>
<td>Visual and/or hearing loss (MM)</td>
<td>Coughing, choking, recurrent aspiration pneumonia</td>
<td>Sialorrhea</td>
<td>Swallow study</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cardiomyopathy (MM, NMD)</td>
<td>Sx CHF, Palpitations</td>
<td>Murmur, gallop, crackles, irregular rhythm</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td>Conduction defects (MM, NMD)</td>
<td></td>
<td></td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>Restrictive chronic lung disease</td>
<td>Hypoventilation, hypoxia</td>
<td>↓ Baseline SpO₂</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>(muscular involvement/scoliosis)</td>
<td>Ventilatory support (CPAP)</td>
<td>↓ Resp effort</td>
<td>PFTs ideal</td>
</tr>
<tr>
<td></td>
<td>Reactive airway (chronic aspiration)</td>
<td>Wheezing</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(MM, NMD, CM)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Chronic diarrhea (MM)</td>
<td>Episodes of dehydration</td>
<td>Hydration status</td>
<td>Electrolytes</td>
</tr>
<tr>
<td>ENDO/METAB</td>
<td>Lactic acidosis (MM)</td>
<td>Fasting times</td>
<td>Hyperventilation</td>
<td>Serum lactate</td>
</tr>
<tr>
<td></td>
<td>Hypoglycemia (MM)</td>
<td></td>
<td>Lethargy</td>
<td>Serum glucose</td>
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<td>GU</td>
<td>Nephropathy (MM)</td>
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<td></td>
<td>BUN/Cr</td>
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<tr>
<td>CNS</td>
<td>Encephalopathy (MM)</td>
<td>Developmental delay</td>
<td>Focal neurologic deficits</td>
<td>Head CT</td>
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<tr>
<td></td>
<td>Seizures (MM)</td>
<td>Poor coordination</td>
<td></td>
<td>MRI</td>
</tr>
<tr>
<td></td>
<td>Ataxia (MM)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PNS</td>
<td>Peripheral neuropathy (MM)</td>
<td>Muscle weakness</td>
<td>Hypotonia</td>
<td>Serum CK and K+</td>
</tr>
<tr>
<td>MS</td>
<td>Hypotonia (MM, NMD, CM)</td>
<td>Muscle weakness</td>
<td>Hypotonia, scoliosis</td>
<td></td>
</tr>
</tbody>
</table>

MM = Mitochondrial Myopathy, NMD = Neuromuscular dystrophy, CM = Central core myopathy.


**Perioperative Management**

**Preoperative Preparation**

- Assess for cardiac and resp involvement.
- Avoid prolonged fasting and dehydration which can worsen acidosis.
- Administer preop anticholinergic if oral secretions are copious.

**Monitoring**

- Routine assuming no cardiomyopathy or CHF.
- There is no optimal anesthetic as all carry some risk for complications. Choice should be dictated by most likely Dx. If feasible regional anesthesia may be best option. Beware central neuraxial blockade if pt has preoload dependent cardiomyopathy.
- Avoid use of lactated solutions (MM).
- Avoid use of succinylcholine (DMD and CM, unclear in MM).
- Avoid volatile agents (CM and DMD).
- Prolonged propofol infusion may promote metabolic crisis. Consider avoiding completely (MM)
- All pts: Increased sensitivity to inhaled, opioid and NM agents. Titrate accordingly.

**Surgical Stages**

- Muscle biopsy is a short duration procedure (30–45 min) but is often combined with gastroscopy tube and MRI.

**Postoperative Considerations**

- Local anesthesia or regional anesthesia (peripheral nerve blocks) recommended for postop pain control.
- Continue close postop monitoring: resp function, risk of MH up to 4–5 hr after anesthesia, rhabdomyolysis and cardiac arrest described in NMD pts in postop period.
- Very careful titration of opioids in monitored setting.

**Anticipated Problems/Concerns**

- Risk of propofol infusion syndrome (brady-cardia, metabolic acidosis, rhabdomyolysis, lipo-demia) in pts with mitochondrial myopathy due to impaired fatty acid utilization.
- Risk of MH (metabolic acidosis, rhabdomyolysis, myoglobinuria, hypotension, and cardiovascular collapse) in core myopathies. Estimated risk for NMD 1.09%.

- 70% of pts with NMD have some degree of cardiomyopathy.
- Core myopathies usually involve only skeletal muscles. Resp involvement is due to muscle weakness and NM scoliosis. Cardiac involvement is rare and is 2° to severe restrictive lung disease.
- Core myopathies (central core, multiminicore and nemaline rod myopathies) and King congenital myopathy are the only known clinical entities assoc with MH. However, the 1% risk for rhabdomyolysis or MH in NMD pts following a volatile anesthetic warrants strong consideration for using other techniques or agents.

**ICD-9-CM Code:** 995.86 (Malignant hyperthermia)

**Indications and Usual Treatment**

- Muscle biopsy is indicated for definitive Dx.
- Many pts have a putative Dx prior to muscle biopsy based on clinical, laboratory, and genetic testing.
Myringotomy and Tympanostomy

**Risk**
- Most common operative procedure in children
- Age 6 mo to 3 y is the peak incidence
- Higher incidence in children in large daycare settings
- Higher incidence of eustachian tube dysfunction in cleft palate pts

**Perioperative Risks**
- Trisomy 21 and other craniofacial syndromes may have smaller ear canals and be more difficult and surgery may take longer

**Overview**
- Most common childhood surgery
- Airway maintenance issues in small children
- Postop pain management

**Indications and Usual Treatment**
- Recurrent otitis media
- Persistent middle ear fluid, glue ear
- Hearing loss
- Speech delay

**ICD-9-CM Code:** 20.0 (Myringotomy); 20.1 (Removal of tympanostomy tube)

**PERIOPERATIVE MANAGEMENT**

**Preoperative Preparation**
- Evaluate airway with mallampati score.
- Consider preop Tylenol for postop pain relief
- Preop fever common in acute otitis and is not necessarily a contraindication to anesthesia

**Monitoring**
- Routine ASA monitors

**Anesthetic Technique/Induction**
- Mask Inhalational induction with sevoflurane, \( N_2O, O_2 \) induction
- 2.0 mcg/kg fentanyl intranasal, up to 50 mcg total dose
- Consider oral airway or LMA as needed
- Continue use of \( N_2O \) to facilitate myringotomies

**Surgical Stages**
- Cleaning ear canal
- Myringotomy
- Tympanostomy tube placement
- Irrigation of middle ear
- Eardrops

**Postoperative Considerations**
- Transport to PACU on \( O_2 \)
- PO pain meds
- Emergence agitation after sevoflurane anesthesia, decreased with intranasal fentanyl

**Anticipated Problems/Concerns**
- Airway obstruction when head turned for myringotomy tube placement
- Laryngospasm without IV in place
- Postop resp insufficiency

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</tr>
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<tbody>
<tr>
<td>HEENT</td>
<td>Airway obstruction</td>
<td>Trisomy 21 or craniofacial syndromes</td>
<td>Mallampati score</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Resp insufficiency</td>
<td>Sleep apnea Hx or severe snoring</td>
<td>Snoring and airway obstruction</td>
<td>Sleep Study</td>
</tr>
</tbody>
</table>

Nephrectomy/Radical Nephrectomy

**Risk**
- 85–90% of radical nephrectomies performed for renal cell carcinoma.
- Incidence: 51,000 cases/y.  Mortality: 13,000 deaths/y.
- M:F ratio: 1.6:1
- Urban > rural
- 5–10% of lesions extend into IVC

**Assessment by Hx**
- Kidney and pedicle removed in simple nephrectomy
- Kidney, adrenal, perinephric fat, and Gerota’s fascia removed en bloc in radical nephrectomy
- Monitoring requirements depend upon IVC involvement and pt co-morbidities
- Significant bleeding may occur if IVC involved
- CPB indicated if large atrial thrombus. Right heart catheterization to be avoided, echocardiography preferred for monitoring.
- Venous return impeded by tumor thrombus in IVC can lead to hypotension and falsely elevated CVP
- Pulm embolization may occur during mobilization of tumor thrombus

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<tbody>
<tr>
<td>CARDIO</td>
<td>Thrombus extending into IVC and right atrium</td>
<td>SOB</td>
<td>Lower limb edema</td>
<td>MRI</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm metastases</td>
<td>Pleural effusion</td>
<td>Pulm embolus</td>
<td>Auscultation</td>
</tr>
<tr>
<td>HEPAT</td>
<td>Venous occlusion (Budd-Chiari syndrome)</td>
<td>Abd pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Brain metastasis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Bone metastasis</td>
<td>Pain</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Preoperative Preparation**
- US, CT, MRI, metastatic work-up
- Cardiorespiratory, renal evaluations

**Postoperative Considerations**
- Pain score: 5–10 for open cases
- Epidural analgesia improves resp function, shortens hospital stay. IV PCA can be used as an alternative.
- At risk for DVT or pulm embolism
- Atelectasis due to splinting
- Head and neck edema from head-down positioning
- Shorter length of stay if laparoscopic approach

**Anticipated Problems/Concerns**
- Blood loss, pulm embolism, pneumothorax, atelectasis

Neuroprotection

Risk
- CNS injury can be the result of ischemia, hypoxia, stroke, hemorrhage, trauma (surgical or otherwise), or infections, drugs (incl. chemotherapeutics, i.e., 'chemo brain'), toxins, neurodegeneration, sepsis, or immune/inflammatory insults.
- Procedures most commonly assoc with risks of CNS injury incl:
  - Neurosurgical resection for aneurysm, AVM or tumor
  - Spinal cord tumor
  - Thoracic or less commonly abd aortic aneurysm resection (incl TEVAR)
  - Carotid endarterectomy
  - CABG, including valve repair, aortic arch procedures, DHCA
  - Interventional neuroradiology incl thrombolysis, stents, coiling. Potentially any surgical procedure that involves massive blood loss, sitting position, or prolonged hypotension.

Perioperative Risks
- Bleeding
- Ischemia and hypoxia
- Increases in ICP
- Edema
- Cranial nerve and brainstem injury
- Epidural, subdural, or parenchymal hematoma
-Transient and permanent brain deficit
-Seizures

Worry About
- Hyper- and hypoventilation and effects on CBF
- Cerebral metabolic rate
- Hypoxia
- Hypercarbia
- ICP changes

Overview
- Refers to preventing and/or minimizing the effects of 2nd or 3rd injury once the initial insult has ended.
- In anesthetic practice it can also mean the ability to provide protection to the CNS (or to other organ systems as well) prior to the stressor. Anesthetic neuroprotection can incl drug delivery, gene expression, or other cellular manipulation, but also anesthetic management of physiologic variables to ensure oxygenation, ventilation, perfusion, and fluids (MAP, CBF, ICP, O2, CO2) appropriately matched to the clinical circumstances. Neuroradiology outside of anesthesia practice incl treatment after stroke, TBI, or SCI.
- Numerous failed human clinical trials in neuroprotection after stroke, despite animal models showing efficacy at several different targets. Early work focused on the possible benefit of thiopental induced burst suppression and hypothermia. Recent work has investigated multiple additional targets and pathways, incl ischemic preconditioning and identification of hypoxia inducible factors.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Airway, need for fiberoptic intubation for controlled induction, C-spine cleared if trauma</td>
<td>Hx of difficult ventilation or intubations, Hx of trauma</td>
<td>Airway exam, other head and neck injuries</td>
<td>CT</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Htn, carotid or vertebral disease, cardiac failure or dysfunction, bradycardia</td>
<td>TIA, stroke, MI, IDDM, Htn, syncope, angina</td>
<td>BP, HR, peripheral edema, JVD, angina</td>
<td>EKG, ambulatory?, exercise tolerance</td>
</tr>
<tr>
<td>RESP</td>
<td>Apnea, COPD, asthma</td>
<td>Difficult weaning from prior intubation, Cushing reflex</td>
<td>Need for O2 at rest, obesity, wheezing, rales</td>
<td>Able to lie flat, O2 rarely PFTs, able to vigorously 'blow out candle', apnea</td>
</tr>
<tr>
<td>RENAL</td>
<td>CRI</td>
<td>UO</td>
<td>BUN/Cr</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Increased ICP, neuro deficits, change in consciousness</td>
<td>N/V, lethargy, alert and oriented</td>
<td>Lethargy, focal deficits, alert and oriented, neuro exam</td>
<td>Neuro exam, CT, MRI</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
- ICP, Lixoc or ventriculostomy in situ?
- CT showing edema or midline shift
- Fluid and volume resuscitated
- Mannitol, HTS, hyperventilation if ICP increased
- Avoid long-acting sedative medications
- Control airway, avoid hypoxia and hypercapnia
- Consider steroids, dilantin, H blocker

Monitoring
- STD monitors plus arterial line for most cases (CVP for major vascular and sitting procedures)
- EEG, SSEP, MEP monitoring, incl in Interventional radiology for coils, embolization
- Lumbar drain for surgical exposure
- Consider processed EEG in the absence of other neuromonitoring if burst suppression is a consideration

Anesthetic Technique/Induction
- Controlled induction with IV agents: Propofol (or thiopental), NMB, short duration opioids
- Attention to physiology, avoid hypoxia and hypercapnia and swings in BP avoid change in wall stress in aneurysms.
- Intraop
- Tight BP control
- Brain relaxation (~1 MAC inhalational agent, consider propofol infusion)
- Burst suppression (not convincing evidence but surgeons may request)
- Surgical retraction can cause local hypoperfusion and postop edema
- Avoid muscle relaxation during MEP or EMG monitoring
- Maintain steady-state level of anesthesia during neuroradiology

Postoperative Considerations
- Extubation some prefer deep, rapidly awake for neuro exam
- Older pts with delayed emergence common
- Frequently get CT scan if not able to do immediate neuro exam

Anticipated Problems/Concerns
- Postop brain edema
- Increased seizure risk (procedures involving cortex)
- Airway edema if unusual positioning (e.g., prone)
## Neuroradiology

### Types of Patients
- Young children
- Pts with disorders causing uncontrolled movements
- Pts with whom communication is impossible (e.g., language barrier, obtundation, mental retardation)
- Pts who are very ill or in severe pain

### Preoperative Implications

#### Anesthetic Technique
- Bring well-stocked anesthesia cart
- Conscious sedation, MAC, GETA or general anesthesia with spontaneous ventilation
- Again ensure a reliable venous access, esp. in young children

#### Monitoring
- Routine; may need to add temp
- BP, pulse oximetry, capnography, electrocardiogram

#### Positioning
- Airway and ventilation must be maintained
- Generally need long circuits, long IV tubing
- Watch for increasing fluid volumes in pediatric pts
- May need closed circuit television with intercom if direct visualization is not possible

#### Post-Procedure Considerations
- Make sure to use standard discharge criteria.
- Have warm blankets available for pts after their test.

### Procedure

#### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Patient Type</th>
<th>Anesthesia Type</th>
<th>Concerns</th>
<th>Monitoring</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT</td>
<td>Pediatric—less than 3 mo—with contrast</td>
<td>Deep sedation, MAC, TIVA (total IV anesthesia) or GA</td>
<td>Keep pt warm to prevent hypothermia (neurologic dysfunction ↑ thermoregulation problems)</td>
<td>BP, pulse oximeter, ETCO₂, ECG, consider temp monitoring</td>
<td>Position sedated child to avoid airway obstruction; mobile cart equipped for emergencies</td>
</tr>
<tr>
<td></td>
<td>Pediatric—less than 3 mo—no contrast</td>
<td>No sedation—have infant suck on bottle of formula or allow to sleep</td>
<td>As above</td>
<td>No monitoring required</td>
<td>As above</td>
</tr>
<tr>
<td></td>
<td>Pediatric—under age 8 y, and/or mental handicap and/or contrast</td>
<td>Deep sedation—MAC, TIVA, or GA</td>
<td>If oral contrast given, treat pt as with full stomach</td>
<td>As above</td>
<td>As above</td>
</tr>
<tr>
<td></td>
<td>Pediatric—over age 8 y, and/or no contrast</td>
<td>Often no sedation required, if required give conscious sedation, TIVA, or GA if unable to hold still</td>
<td>Maintain thermal stability</td>
<td>For conscious sedation: BP, pulse oximeter, ECG; consider temp monitoring</td>
<td>As above</td>
</tr>
<tr>
<td></td>
<td>Adult—healthy</td>
<td>No sedation</td>
<td>If oral contrast is given, treat pt as with full stomach</td>
<td>None</td>
<td>If suspected ↑ in ICP, use caution to avoid ↑ arterial O₂, which can further ↑ ICP</td>
</tr>
<tr>
<td></td>
<td>Adult—difficulty holding still</td>
<td>Conscious sedation or MAC/GA</td>
<td>If oral contrast given, treat pt as with full stomach</td>
<td>BP, ECG, pulse oximeter, ETCO₂, FIO₂</td>
<td>Positioning of gantry or table during procedure may result in kinking or disconnection of anesthesia circuit</td>
</tr>
<tr>
<td>RADIOThERAPy</td>
<td>Pediatric—esp. under age 8 y</td>
<td>Sedation, MAC or TIVA use short-acting, easily titratable drugs; for repeated treatments, pt needs central indwelling catheter</td>
<td>Temp maintenance Airway management and ventilation Positioning of the gantry during procedure may result in kinking or disconnection of the anesthesia circuit Observe pt prior to each treatment for sepsis or ↑ ICP</td>
<td>ECG, ETCO₂, pulse oximeter, BP Make sure closed-circuit TV shows pt and monitors</td>
<td>Immobility is the primary goal of anesthesia; using TIVA may avoid transporting agent vaporizers and other bulky equipment</td>
</tr>
<tr>
<td></td>
<td>Adult</td>
<td>None, conscious sedation or easily titratable drugs</td>
<td>Same as above</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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**Key Reference:** Porche VH. Anesthetic considerations in radiologic procedures performed outside the operating room. *Int Anesthesiol Clin* 1998;36:9–19.
Off Pump and Minimally Invasive Cardiac Procedures

**Risk**
- Increased risk with advanced age and male gender
- Family Hx of CAD
- HTN, hypercholesterolemia, diabetes, cigarette smoking

**Perioperative Risk**
- Operative mortality for bypass procedures is double for women compared to men
- Off-pump coronary artery bypass (OPCABPOS)
  - Mortality 0–5%
  - Pulm insufficiency 1–5%
  - Reoperation for rebleeding 1–4%
  - MI 0–4%
  - Mediastinal infection 1–2%
  - Neurologic complications 1–2%
  - Renal dysfunction 1–2%
- Minimally invasive cardiac procedures
  - Mortality 1–3%
  - Arrhythmias 10%
  - Conversion to sternotomy 4%
  - MI 0–4%
  - Reoperation for rebleeding 3%
  - Stroke 2%

**ASSESSMENT POINTS**

<table>
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<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Family Hx, previous MI, ventricular dysfunction, valvular dysfunction</td>
<td>Htn, DM, hyperlipidemia, smoking, level of activity</td>
<td>Murmurs, pulm rales, lower extremity edema, intolerance to lying flat</td>
<td>ECG, ECHO, stress testing, coronary angiography</td>
</tr>
<tr>
<td>PULM</td>
<td>COPD, emphysema, chronic bronchitis</td>
<td>Smoking Hx, use of supplemental O₂</td>
<td>Decreased breath sounds, sputum production</td>
<td>CXR, PFTs, ABG</td>
</tr>
<tr>
<td>CNS</td>
<td>Carotid disease</td>
<td>Previous stroke, TIA</td>
<td>Carotid bruits, residual deficits</td>
<td>Carotid US, carotid angiography</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency, renal failure</td>
<td>Htn, Hx of dialysis</td>
<td>Pulm rales</td>
<td>Creatinine, potassium levels</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Preoperative Preparation**
- Baseline cardiac function incl regional wall motion, right heart function, and valvular function

**Monitoring**
- Arterial line
- ECG monitoring particularly lead II and V5
- CVP
- PA catheter for multiple grafts and decreased cardiac function
- Continuous cardiac output monitoring
- TEE is most sensitive and specific for early detection of ischemic events
- Minimally invasive cardiac procedures
  - Right and left radial lines should be placed
  - Right radial line monitors endoaoartic clamp migration
- TEE essential for placement
- Endoaortic clamp

**Cardiovascular**
- Venous drainage catheter
- Endosinus catheter
- Endopulmonary vent

**Anesthetic Technique/Induction**
- Adequate preoxygenation
- Preinduction arterial line for pts with decreased LV function
- External defibrillator pads prior to induction
- Avoid hypothermia, particularly for OPCAB
- Heat lost cannot be restored as easily as compared to on pump procedures
- Warm OR prior to pt arrival
- Use warming pads/mattresses on OR table
- Use forced air warming devices over head and exposed areas
- Minimize time pt is uncovered
- Warm IV fluids
- Use fresh gas flows <2.5 L/min
- Displacement of heart or placement of stabilizing devices can cause significant decreases in BP

**CNS**
- Stroke 2%
- Reoperation for rebleeding 3%
- MI 0–4%
- Conversion to sternotomy 4%
- Arrhythmias 10%
- Mortality 1–3%
- Renal dysfunction 1–2%
- Neurologic complications 1–2%
- MI 0–4%
- Pulm insufficiency 3–5%
- Mortality 0–5%

**Pulm**
- Port access cardiac surgery
- Aortic dissection
- Aortic valve trauma
- Coronary sinus trauma
- Right ventricular perforation
- Endoaortic balloon migration can compromise cerebral perfusion

**Worry About**
- OPCAB
  - Multiple grafts require repeated occlusions and can affect hemodynamics.
  - Displacement of the heart, esp. for PDA and circumflex grafts cause labile hemodynamics.
  - Ischemia caused by the occlusion of the proximal coronaries must be recognized and treated early.
  - Possibility of conversion to traditional on-pump CABG
  - Minimally invasive cardiac procedures
  - CO₂ insufflation necessary for visualization can hinder heart filling and depress contractility

**ICD-9-CM Code:**
- 410 (Acute myocardial infarction); 414 (Coronary arteriosclerosis)

**Overview**
- Maintain cardiac output to sustain coronary flow
- Early recovery desired
- OPCAB
  - Goal is to provide adequate revascularization but avoid pulm dysfunction, coagulopathy, and CNS injury caused by CPB
- Minimally invasive cardiac procedures
  - Requires good communication between anesthesiologist and surgeon

**Indications and Usual Treatment**
- OPCAB is recommended for pts with severe vascular disease (esp. severe aortic atherosclerosis), stroke, pulm disease, and renal dysfunction.
- Surgeon preference and experience determine procedure.

**Postoperative Considerations**
- Complete TEE examination incl function, regional wall motion abn, and valvular function should be assessed.
- Continue active warming
- Should evaluate for possible extubation after considering adequate hemostasis, stable hemodynamics, and pain control as well as attainment of extubation criteria
Office-Based Anesthesia

Patrick Guffey

Risk
- Approx 10 million cases performed in office based setting
- 10% of all surgeries
- Wide range of surgical subspecialties perform procedures in office settings
- Full complement of anesthesia agents may be utilized

Perioperative Risks
- Pt’s underlying medical condition
- Procedure-specific risks
- Anesthetic emergencies without full complement of backup equipment
- Low number of trained responders
- Immediate transfer to hospital may not be available

Overview
- Procedures performed in a physician’s office with no direct connection to a hospital or ambulatory surgical center
- Anesthetic techniques range from monitored care to general anesthesia
- Allows for potential decrease in costs as compared to a hospital or surgical center
- May be useful in a setting where the majority of pts do not require anesthesia and capital equipment costs are high.

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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Difficult airway management</td>
<td>Hx of difficult mask or intubation, mouth or neck abn (mass, prior radiation), sleep apnea</td>
<td>Mallampati classification, thyromental distance, mouth opening</td>
<td>Transfer to higher level of care if imaging warranted</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Poor CV function or reserve</td>
<td>CAD, Htn, diabetes, prior MI, TIA, heart failure. Assess exercise tolerance</td>
<td>Chest auscultation, Assessment of pedal edema and JVD</td>
<td>ECG, stress test</td>
</tr>
<tr>
<td>RESP</td>
<td>Difficulty with ventilation and oxygenation</td>
<td>Asthma, COPD, active or recent URI</td>
<td>Chest auscultation and percussion</td>
<td>CXR if low suspicion, transfer if high suspicion</td>
</tr>
<tr>
<td>RENAL</td>
<td>Metabolic derangement</td>
<td>Renal failure, diuretic use</td>
<td>Basic metabolic panel, BUN/Cr</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Postop agitation, exacerbation of pre-existing condition</td>
<td>Hx of Parkinson’s disease, psychiatric disorders</td>
<td>Resting tremor, preop assessment</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Aspiration and nausea</td>
<td>Hx of GERD, Hx of postop nausea</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
- Complete preanesthetic evaluation by anesthesiologist
- Special attention to preparation for anticipated complications

Monitoring
- ASA standard monitors

Anesthetic Technique/Induction
- Full complement of anesthesia techniques can be utilized with preference to short acting agents.

Worry About
- Small number of trained responders for a crisis
- Emergency equipment (adequate dantrolene if triggering agents used)
- How to transfer pt to higher level of care
- Lack of oversight and little to no regulation

Indications and Usual Treatment
- Procedure and anesthetic must be within the capabilities of the facility, incl complications requiring immediate response.
- ASA classification I & II for low-risk procedures
- Pt who desires privacy that cannot be provided in a larger setting
- May offer a level of convenience not possible in a hospital

Emergence
- Minimize long-acting agents during the case, consider regional anesthesia to decrease anesthetic requirements and facilitate timely discharge

Postoperative Monitoring
- Until pt is hemodynamically stable, able to void, pain controlled, and nausea resolved with ability to tolerate PO fluids

Transfer pts to higher level of care if unable to discharge in a timely manner
- A physician should be immediately available until all pts are discharged.

Anticipated Problems/Concerns
- Rarely a pt will require hospitalization. Plans should be in place to facilitate this.
- Few, if any trained responders will be available for an intraop anesthetic complication
- PACU capabilities limited compared to surgical center or hospital. Plan for pts to be stable upon leaving operating suite.
Omphalocele Surgery

Risk
- Incidence varies from 1/6000 live births
- Racial predominance: none

Perioperative Risks
- Possibility of impaired blood supply to the herniated organs
- Intestinal obstruction
- Significant heat losses
- Major fluid shifts
- Infection
- Risks due to assoc genetic, cardiac, urologic and metabolic abn

Worry About
- Blood glucose, esp. with possibility of Beckwith-Wiedemann syndrome

ASSESSMENT POINTS

<table>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Beckwith-Wiedemann syndrome:</td>
<td>Head exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>macroglossia, microcephaly</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>CHD: tetralogy of Fallot, ASD</td>
<td>CV exam</td>
<td>Transthoracic ECHO</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>If assoc with prematurity,</td>
<td>Gestational age</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>may have immature lungs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Gastric and intestinal distention,</td>
<td>Size of omphalocele</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>small abd cavity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Possibility of Beckwith-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Wiedemann syndrome</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL/H</td>
<td>Immaturity of hepatic and renal</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>HEPAT</td>
<td>systems possibility for hepatic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>blood flow and impaired renal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>perfusion post closure</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- IV access and restoration of intravascular volume
- Fluid losses tend to be isotonic; therefore, balanced salt solutions often used (lactated Ringer’s or 5% albumin)
- Multiple fluid boluses of up to 20 mL/kg of fluid is often necessary initially
- Maintain normothermia by wrapping abd in moist, warm, sterile dressing; lower body can be placed in plastic bag
- Decompression of the stomach to prevent regurgitation/aspiration
- Antibiotics to prevent sepsis

Monitoring
- Consider arterial line depending on extent of defect and ventilatory status
- Consider CVP if defect is large
- Adequate temp and glucose monitoring

Airway
- After decompression of the stomach, rapid sequence induction or awake intubation should carried out.
- ETT should be secured in a manner suitable for prolonged postop ventilation

Maintenance
- NO usually avoided
- A suitable mixture of O2 and air to produce adequate oxygenation (Pao2 50–70 mmHg), SaO2 97–98% for term infants, 87–92% for preterm infants); will vary as surgeons attempt to replace bowel in abd
- Maximal muscle relaxation facilitates reduction of the eviscerotized organs and bowel.
- Primary closure may lead to significant increases in intra-abdominal pressure which leads to decreased organ perfusion (intestinal, renal, and hepatic) will result in impaired organ function and altered drug metabolism and decreased ventilatory reserve due to significantly decreased diaphragmatic function and bilateral lower lobe atelectasis.
- Ability to tolerate primary closure assessed by the ability to measure BP and/or SaO2 in the lower extremities
- Placing bowel in a silo assoc with higher infection rate

Exsufflation
- Postop care varies with magnitude of defect, type of repair, and assoc congenital anomalies
- Healthy pts with a small I° closure often tolerate exsufflation in OR

- In pts with large defects, who undergo 1° closure postop intubation, ventilation, and maximal muscle relaxation should be continued until abd pressure results in little resp or circulatory compromise
- In pts with a large defect treated with a silo, although exsufflation might be tolerated, postop intubation should be continued due to repeated trips to the OR and muscle relaxation will facilitate the abd cavity accommodation of the increasing mass.

Anticipated Problems/Concerns
- Ventilatory care: Similar to that for other neonates with resp distress
- Fluid requirements: May remain high until abd venous pressure decreases, at which time fluid restriction and diuresis probably indicated
- FIO2: Adjust to maintain a normal Pao2
- PEEP: Appropriate levels used to increased FRC
- Nutritional status: Because bowel function is usually compromised and slow to resume, TPN requirements are often extended.
- Circulatory, renal, hepatic, and intestinal dysfunction common
- Infection: Common, esp. if silo used instead of 1° closure

Etiology
- A failure of the gut to migrate from the yolk sac into the abd during gestation
- Amniotic sac present, although it may have been ruptured during birth or shortly thereafter

Indications
- Prompt surgical repair, either I° or staged, depending on size

ICD-9-CM Code: 756.72

Overview
- Congenital abd wall defects that result in herniation of the intestine into base of umbilical cord
- The herniated viscera are covered with a membranous sac. The bowel is morphologically and usually functionally normal
- Vary in size: May contain only small bowel or may contain liver, spleen, stomach, and other abd organs

System Effect Assessment by Hx PE Test
HEENT Beckwith-Wiedemann syndrome: Head exam Head
FACE: macroglossia, microcephaly CV exam Transthoracic ECHO
CARDIO CHD: tetralogy of Fallot, ASD
RESP If assoc with prematurity, Chest exam and signs of resp distress O sat
Gestational age If available, predeelivery
Gastric and intestinal distention, lecithin/sphingomyelin ratio
small abd cavity
RENAL/ HEPAT Possibility of Beckwith- Blood glucose
HEPAT Wiedemann syndrome: Blood glucose

**Risk**
- Premature infants: Risk is 30% for one or both testicles to be undescended
- Full-term infants: Risk is 3%
- 60% of undescended testicles found in inguinal position; 8% intra-abdominal; only 24% in the easily operable low inguinal/high scrotal position
- Progressive injury occurs when testicle is left undescended: Decreased sperm production after age 6 y, impaired hormonal production, increased risk of malignant degeneration
- Risk of malignant degeneration may not be improved following orchiopexy, but self-examination becomes more reliable

**Perioperative Risks**
- Periop mortality rare in term infants (<0.01%)
- Risks in ex-premature children dependent upon co-existing morbidity (e.g., bronchopulmonary dysplasia, reactive airway disease, subglottic stenosis, hydrocephalus and seizure due to intraventricular hemorrhage, GI dysfunction due to necrotizing enterocolitis, malnutrition, anemia, RV hypertrophy/failure, poor IV access)

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<tbody>
<tr>
<td>HEENT</td>
<td>Subglottic stenosis</td>
<td>Stridor, wheezing, croup</td>
<td>CXR, bronchoscopy</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Pulm Htn, PDA, RV hypertrophy</td>
<td>FTT, ↑ S, murmurs</td>
<td>ECG Cardiac ECHO/catheter</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchopulmonary dysplasia, blebs</td>
<td>Asthma, O₂, Apnea monitor alarms</td>
<td>CXR</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Nephrocalcinosis</td>
<td>Htn</td>
<td>BP, electrolytes, BUN, Cr</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Intradural hemorrhage, Seizures, hydrocephalus</td>
<td>Mental status, Development, Seizure type, frequency, Ventriculoperitoneal shunt</td>
<td>Shunt evaluation, Anti-epilepsy drug levels</td>
<td></td>
</tr>
</tbody>
</table>


### Perioperative Management

**Preoperative Preparation**
- May have clear liquids up to 2 hr before induction
- No lab work necessary if otherwise normal
- If uncomplicated, may be done as outpatient procedure

**Anesthetic Technique**
- Combined general and regional anesthetic in open case
- Usually regular mask or laryngeal mask airway (LMA) and caudal injection of local anesthetic (1mL/kg 0.25% bupivacaine or 0.2% ropivacaine) or ilioinguinal/iliohypogastric nerve block for analgesia intra- and postop
- Combined technique in laparoscopic procedures but with trachea intubated (usually; LMA now being used by some)

### Monitoring

- Routine

**Induction**
- Inhalational mask induction, with sevoflurane, then maintenance with sevoflurane or isoflurane and NO
- IV, then single-injection caudal placed following induction of anesthesia

### Surgical Stages

**Dissection**
- Caudal usually not effective in blocking visceral pain that occurs with pulling on spermatic cord; may require increasing volatile anesthetic concentrations temporarily
- Possible resp compromise if laparoscopic procedure
- Minimal blood loss

- Operative risks of testicular atrophy or hypopotrophy: 8% for those beyond external ring, 13% when canalicular, 26% for intra-abdominal locations

**Worry About**
- Co-morbidity assoc with prematurity
- Cryptorchidism a/w congenital anomalies in up to 4.4% of pts: Smith’s lists cryptorchidism as frequent in 53 known syndromes/sequences, occasional in another 31 syndromes
- Venous air embolism, aspiration, diaphragmatic embarrassment if laparoscope utilized for repair
- Intravascular injection of local anesthetic

**Overview**
- Testicle(s) located and spermatic cord and accompanying vasculature freed and mobilized so that testicle can be relocated within hemiscrotum. Spermatic vessels may be sacrificed, with vasculature of vas deferens supplying collaterals.

**Indications and Usual Treatment**
- Testicle will not descend beyond 1 y of age
- 2% risk per mo of germ-cell depletion for unoperated testes and later sperm counts/motility increased when operated on in first year of life. Average age of operations has decreased to 6–12 mo of age
- Some evidence that hCG may promote testicular descent, so this may be attempted prior to operation

**Postoperative Considerations**
- If 0.025% bupivacaine used for caudal, 4–6 hr of analgesia usual
- Most children need enteral opioids for 2–3 d
- Incidence of emesis postop 45%; usually self-limited

**Anticipated Problems/Concerns**
- Ex-premature children have an increased incidence of wheezing and desaturation intra- and postop. May need to be observed overnight.
ORIF of Hip

Nina Singh

Regional versus GA: Regional may reduce incidence of DVT, MI, confusion, EBL, postop hypoxemia, pain, 1 mo mortality (no difference at 2 mo). GA may reduce operative time, CVA, intraop hypotension.

ICD-9-CM Code: 733.14 (Pathologic fracture of neck of femur)

Indications and Usual Treatment

• Femoral neck fx: Percutaneous, THR, or hemiarthroplasty. THR and/or hemiarthroplasty may increase length, invasiveness, EBL, greater HD changes (esp. if cementing); may require lateral decubitus.
• Intertrochanteric fx: Closed reduction, followed by ORIF or IM nailing. Muscle relaxation imperative to optimize manipulation, allow for ideal femur alignment prior to fixation or nailing.

Perioperative Management

Preoperative Preparation

• Optimize co-morbidities. Abn cardiac testing rarely changes before ORIF, esp. in intermediate risk pt. No advantage, possible worse outcome from PCI (restenosis, thrombosis from D/antiplalet Tx). LOC causing fall often warrants cardiac, neuro testing.
• Dehydration, hemoconcentration common; consider rehydration, pRBCs.
• Consider neuraxial with pt, surgeon, anticoagulation, expected EBL, risk of delirium, mechanism for postop follow up (pain management, catheter removal, management of complications).
• Consider Gram (-) Abx if S/Sx of UTI, to reduce implant infection.

Monitoring

• Consider A-line if preop hypoxemia, co-morbidities, expected EBL, controlled hypertension
• CVP if poor IV access, revision repair, large expected EBL, co-morbidities
• Cell saver: Leukocyte removal filter shown to reduce transfusion of fat

Anesthetic Technique/Induction

• Regional versus GA: Reduced EBL at same MAP as GA, likely from vasodilation of venous, arterial vasculatures leads to redistribution of blood flow. Consider sedation to tolerate positioning, Ex table.

• B-blockers: Important to continue periop administration if previously taking agents. Control of heart rate important.

Surgical Stages

Dissection

• Muscle relaxation: NMBD versus neuraxial dense motor block
• Controlled hypotension: Balance risks (inadequate organ perfusion [SBP >80 mmHg 90% delirium]) versus benefits (reduced EBL, dry bone surface [improves prosthetic cementing and reduces duration of surgery]).
• Normothermia may reduce delirium, arrhythmic, low BP, bleeding, transfusion, infection, poor wound healing.

Reaming/cementing

• Cement implantation syndrome (CIS): Increased intramedullary pressures generated during implant insertion, are accentuated by cementing, and can cause emboli of fat, marrow, debris to pulm vasculature that are hemodynamically significant

Postoperative Considerations

• Pain: Intrathecal morphine; consider reduced dosage for elderly, appropriate postop monitoring. Inadequately treated pain can increase cardiac O₂ demand, stress response, delirium. Delirium also seen from opiates.

• Delirium: May be reduced with regional, early ORIF.
• DVT: Regional may reduce; low dose anticoagulant prophylaxis, SCDs, early ambulation.
• Fat emboli syndrome (FES): Clinical Dx with classic triad of hypoxemia, delirium, petechial rash. Typically presents 24-72 hr after Fx. Early immobilization, ORIF may prevent; therapy is supportive (oxygenation/PEEP/IV fluids); steroids controversial.

Anticipated Problems/Concerns

• Emboli: Fat, FES, CIS, DVT
• Delirium: Increased hospital LOS (urinary incontinence, feeding problems, decubitus ulcers), mortality, likelihood of being placed in nursing home for first time.
• Long term: 25% full recovery, 40% nursing home, 50% cane or walker, up to 30% mortality in 1 yr.


PROCEDURES

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CAD + O₂, supply/demand imbalance (HR, Htn, anemia, fluid shifts, emboli); arrhythmia as cause of fall</td>
<td>Angina, SOB, palpitations, LOC as cause of fall</td>
<td>Vital signs, pitting edema, irregular pulse, diaphoresis</td>
<td>12-lead EKG, telemetry, stress test, ECHO, cardiac enzymes</td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoxemia/PE</td>
<td>Dyspnea, wheezing</td>
<td>RR, accessory muscle</td>
<td>CXR, ABG, continuous pulse ox</td>
</tr>
<tr>
<td>RENAL</td>
<td>Dehydration, UTI</td>
<td>Tachycardia, hypotension, altered mentation, dysuria</td>
<td>Vital signs, cap refill, mucous membranes</td>
<td>Cr, BUN, sodium, UA/culture</td>
</tr>
<tr>
<td>CNS</td>
<td>TIA as cause of fall; Delirium</td>
<td>Sensory/motor/speech deficit; Hx of LOC</td>
<td>Neuro deficit; mentation, fluctuating course, disorganization</td>
<td>DSM-IV-TR, CAM, MMSE, CT, MRI</td>
</tr>
</tbody>
</table>

DSM-IV-TR Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision; CAM Confusion Assessment Method; MMSE Mini-Mental State Examination
Pacemaker Implantation for Sick Sinus Syndrome

**Overview**
- SSS, also called sinus node dysfunction, is a group of arrhythmias presumably caused by a malfunction of the sinus node, the heart’s normal 1° pacemaker.
- SSS is divided into three types: Simple sinus bradycardia, sinus arrest or sinus node block with or without sinus bradycardia, and bradycardia with paroxysmal tachycardia (i.e., tachy-brady syndrome).
- Tachycardia may be caused by atrial fibrillation and/or flutter.
- Syncope or severe lightheadedness results from prolonged sinus or atrial pauses following termination of tachycardia; atrial pause frequently caused by sinus exit block.
- Sinus or atrial pauses may rarely be up to 15 sec in duration.
- SSS may be assoc with high-degree AV block (pan-conduction defect).
- Stroke risk is increased in pts who are paced ventricularly (VVI) and thus more prone to develop atrial fibrillation with subsequent thrombus formation/propagation due to atrial stasis.
- Where once surgeons were primarily responsible for PPM implantation, the procedure now is typically performed by cardiologists. For most pts, PPM insertion is performed on an out pt basis in the cardiac catheterization suite. For high risk pts however, anesthesiologists may be called upon to provide monitoring, administration of analgesics and sedatives, and resuscitation should any complications occur.

**Etiology**
- SSS is usually caused by degenerative, sclerotic, or fibrotic changes of the sinus node.
- SSS can be a manifestation of 1° cardiac disease: e.g., myocardial ischemia/infarction, pericarditis, cardiomyopathy.
- SSS may also be 2° to cardiac involvement by other diseases (typically infiltrative): e.g., muscular dystrophy, collagen disease, hemochromatosis, amyloidosis, metastatic disease.

**Indications And Usual Treatment**
- PPM placement is indicated in symptomatic pts or in asymptomatic pts who need β-blockers or antiarrhythmic drugs.
- Bradycardias are typically well controlled with PPMs, while tachyarrhythmias respond well to medical therapy. However, because both bradyarrhythmias and tachyarrhythmias may be present, drugs to control tachyarrhythmia may exacerbate bradyarrhythmia. Therefore, PPM implantation is indicated prior to implementation of drug therapy for tachyarrhythmias.
- For previously undiagnosed disease, temporary transvenous pacing may be appropriate.
- Sinus rate and atrial conduction may be enhanced by stimulation with β-adrenergic agonists (isoproterenol, epinephrine, ephedrine) and parasympathetic blocking agents (atropine, glycopyrrolate).

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Bradycardia, tachycardia</td>
<td>PPM implantation</td>
<td>Low (or high) HR</td>
<td>ECG, electrophysiologic testing</td>
</tr>
<tr>
<td>CNS</td>
<td>Unexplained episodic lightheadedness, confusion, syncope</td>
<td>Holter</td>
<td>CT scan</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Monitoring**
- Basic ASA monitors; ECG, BP, pulse oximetry
**Anesthetic Technique**
- Conscious sedation in the cardiac catheterization laboratory
- Monitored anesthesia care in the OR
- Regional anesthesia; local anesthesia at insertion site, field block of the supraclavicular nerves, interscalene block at C4, and interscalene block at C6

**Maintenance**
- Volatile agents, esp. enflurane, may suppress SA function.

**Extrusion/Emergence**
- Emergence excitement may contribute to development of paroxysmal tachycardia.

**Adjuvants**
- Calcium-channel blockers, esp. verapamil, are contraindicated in the absence of pacemaker capability due to risk of inducing severe bradycardia.
- With a PPM present, paroxysmal tachycardia may be treated acutely with IV verapamil; digoxin may be added for longer-lasting rate control.

**Anticipated Problems/Concerns**
- Severe brady- and/or tachyarrhythmias 2° to anesthetic agents or autonomic imbalance are common in the periop period. Relative parasympathetic predominance in particular may be caused, for example, by high-dose opioid therapy or by maneuvers that increase vagal tone. Hence, in the anesthetic management of pts with SSS presenting for PPM implantation, drugs such as atropine, glycopyrrolate and isoproterenol should be immediately available should a sudden decrease in HR compromise hemodynamics before the new PPM is functional.
Pancreas Transplantation

**Risk**
- 35,000 new type 1 diabetics diagnosed yearly
- Transplant recipients usually <55 y old
- 1200–1300 transplants annually

**Overview**
- Primary indication for transplant is insulin-dependent DM (Type I diabetes)
- Pts often have co-existing ESRD
- Simultaneous kidney and pancreas (SKP) transplant done in more than 80% of cases
- Pancreatic graft survival 86% at 1 y and 53% at 10 y when done with concomitant renal transplant
- Can lead to complete insulin independence

**Etiology**
- End-organ damage from longstanding diabetes
- Macrovascular atherosclerosis leading to coronary artery and PVD states
- Microvascular damage of kidneys, heart, retina, and extremities
- Autonomic neuropathy of GI, cardiac, and urinary systems
- Collagen cross-linking defects

**Usual Treatment**
- Type I diabetics with renal failure most common, usually SPK transplant
- 15% transplanted after renal transplant
- 10% without renal disease, but with labile and difficult diabetic state

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<th>Test</th>
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</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Stiff joint syndrome</td>
<td>Neck and/or finger stiffness</td>
<td>Neck extension, failure to oppose palms (prayer sign), abn palm print</td>
<td></td>
</tr>
<tr>
<td>PULM</td>
<td>Decreased elasticity</td>
<td>Co-morbid risk factors</td>
<td>Auscultation of lung fields</td>
<td>PFTs</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Accelerated atherosclerosis CAD PVD</td>
<td>Functional capacity</td>
<td>Assess heart rhythm and rate</td>
<td>ECG, exercise or pharmacologic stress, ECHO, Holter monitoring</td>
</tr>
<tr>
<td>RENAL</td>
<td>Chronic failure leading to dialysis Anemia</td>
<td>Dialysis history UO Fatigue</td>
<td>Conjunctiva, mucous membrane pallor; functional murmur</td>
<td>Electrolytes BUN/creatinine CBC</td>
</tr>
<tr>
<td>NEURO</td>
<td>Autonomic dysfunction Peripheral neuropathy</td>
<td>Orthostatic hypotension, neurogenic bladder</td>
<td>Decreased peripheral sensation</td>
<td>Sitting/standing BP, tilt table test</td>
</tr>
<tr>
<td>ENDO</td>
<td>Pancreatic insufficiency</td>
<td>Hyper- and hypoglycemia</td>
<td>Hyperglycemia</td>
<td>Insulin therapy Blood glucose</td>
</tr>
<tr>
<td>GI</td>
<td>Diabetic gastroparesis Delayed gastric emptying Decreased lower esophageal tone</td>
<td>GERD NPO status</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Aspiration prophylaxis with sodium citrate, metoclopramide, or H2
- Consider epidural placement for intra- and postop pain control

**Monitoring**
- Central venous catheter for access, CVP monitoring, vasopressor administration
- Arterial catheter for BP and frequent blood sampling
- Pulm artery catheter if warranted if pt has severe cardiac disease

**Airway**
- Intubations in diabetics can be problematic.
- Increased risk of difficult laryngoscopy due to stiff joint syndrome

**Induction**
- Avoid succinylcholine with hyperkalemia
- Minimize hypo- and Htn, tachycardia in pts with CAD
- Cisatracurium or mivacurium with severe renal disease

**Maintenance/Intraoperative Management**
- Antibiotic coverage for streptococci and gram-negative bacilli
- Judicious fluid administration in pts with renal disease
- Immunosuppressive medication administrated intraop

**Exsufflation**
- Often done in ICU in a controlled setting
- Minimize BP and HR extremes to avoid cardiac events
- Avoid severe Htn to preserve vascular anastomoses

**Postoperative Period**
- Immediate and late complications can incl bleeding, rejection, infection, pancreatitis, and graft thrombosis.
- Hyperglycemia is preferred over tight glucose control
- Transplanted organ begins functioning within hours, therefore avoid giving insulin and glucose containing solutions after reperfusion for better evaluation of organ function.
**Parathyroidectomy**

**Risk**
- Primary hyperparathyroidism 50–100/100,000 (USA); in women >45 incidence is 1/500
- Females > males
- Caused 90% by benign adenoma; 85% by single adenoma; 2–3% by 2 or more adenomas; 10–15% by hyperplasia (all 4 glands); <1% by carcinoma (rare)
- 10% of parathyroid hyperplasia cases may exist as part of MEN 1 or MEN 2 syndrome (MEN 2 assoc with pheochromocytoma)

**Perioperative Risks**
- Moderate to severe hypercalcemia (serum calcium >11.5–13 mg/dL), may cause abn in cardiac conduction, arrhythmias, Htn; muscle weakness, nausea, depressed mental status
- Periop mortality rare
- Postop morbidity inc the following risks: Hypoparathyroidism <5%, injury to recurrent laryngeal nerve/vocal cord paresis <2%, hypocalcemia <1%, neck hematoma <1%

**Worry About**
- Symptomatic elevated serum calcium should be treated and controlled before elective parathyroidectomy (volume expansion, correction of electrolyte abn, furosemide diuresis, bisphosphonates, calcitonin/glucocorticoids)

**Preinduction/Induction**
- Choices for minimally invasive technique incl local and/or MAC, GA, GA with EMG ETT
- Careful positioning important in osteopenic pts at risk for fracture. Arms at sides, neck extended with head on donut
- Protect eyes
- After induction, baseline PTH level is drawn. It’s helpful to have access to hand and/or foot for blood draws intraop 50% drop in PTH level 5 min after adenoma removal confirms hypersecretory gland successfully removed.

**Maintenance**
- Avoid acidosis (acidosis, incl resp acidosis, will increase serum calcium level)
- Avoid muscle relaxant during maintenance if using EMG/NIM ETT, deeper anesthetic depth required to avoid bucking on ETT with tracheal manipulation.
- Expect intraop frozen sections to positively identify parathyroid tissue, and rule out carcinoma (rare).

**Indications and Usual Treatment**
- Surgery is treatment of choice for symptomatic hypercalcemia (or Ca > 11 mg/dL), also indicated if evidence of nephrolithiasis or renal impairment, decreased bone density
- Single adenoma verified by localization studies can be treated with minimally invasive surgery technique (local/MAC or GA), advantage is shorter operative time and hospital stay, lower cost, better cosmetic result, possibly less PONV and pain, (unilateral small incision/endoscopy assisted) and similar success rate to conventional parathyroidectomy
- Multiple adenoma or parathyroid hyperplasia is treated with conventional collar incision, bilateral neck exploration, all four parathyroid glands identified, diseased glands removed, part of a single gland is left, or reimplanted in forearm to preserve some parathyroid function.

**ASSESSMENT POINTS**

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Conduction abn (short QT; prolonged P-R intervals assoc with hypercalcemia)</td>
<td>ECG</td>
<td>Serum calcium level</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Htn</td>
<td>NIBP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Assoc with MEN1 (hyperplasia) (insulinoma/pancreas tumors, gastrinomas) hypercalcemia</td>
<td>CT scan/MRI</td>
<td>EGD/endoscopic US</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Hypercalcemia effects Neuromyopathic symptoms</td>
<td>Depression, memory problems, lethargy</td>
<td>Serum PTH by ria</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperparathyroidism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Nephrolithiasis (60–70%) Renal insufficiency</td>
<td>Flank tenderness</td>
<td>X-ray to look for stone</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Flank pain Polyuria Polydipsia</td>
<td></td>
<td>GFR serum, BUN/creatinine</td>
<td></td>
</tr>
<tr>
<td>NM</td>
<td>Peripheral muscle weakness</td>
<td>Extremity exam</td>
<td>Ca²⁺ level</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Easily fatigued</td>
<td>Skeletal exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Osteitis fibroa cystica Osteopenia</td>
<td>Frequent fractures Bone pain</td>
<td>Bone density studies</td>
<td></td>
</tr>
</tbody>
</table>

**Emergence**
- Control BP, minimize coughing, bucking, or increased venous pressure in the neck to decrease the chance for postop neck hematoma.
- Evaluate immediately after extubation for signs of airway obstruction or compromise (edema, hematoma, vocal cord paresis). Increased risk of aspiration with vocal cord paresis.

**Anticipated Problems/Concerns**
- Potential for postop airway obstruction from intraop injury to recurrent laryngeal nerve with resultant VC paresis, neck hematoma, glottic edema
- Monitor serum calcium levels postop: Decrease will occur within 24 hr if adenoma successfully removed, calcium nadir 3–7 d. Also monitor serum phosphate, magnesium, PTH
- Untreated hypocalcemia may cause tetany, laryngospasm, seizure (treat with IV calcium)
- Clavostek's sign (facial muscle contraction with facial nerve tap) and Trouseau's sign (carpopedal spasm with application of BP cuff) are classic signs warning of the potential for tetany from hypocalcemia.
### Patent Ductus Arteriosus (PDA), Ligation of

#### Overview
- Ductus arteriosus (DA) is essential to maintaining fetal systemic perfusion
- DA functionally closes within 8 hr after birth when smooth muscle constricts in response to increased O₂ tension (from 18–28 mmHg in utero to 40–60 mmHg postnatally)
- Anatomic closure of DA with intimal remodeling and loss of smooth muscle occurs over next several days
- DA sensitivity to increased O₂ tension is diminished in preterm infant.
- Fluid overload, RDS or severe illness (e.g., acidosis, hypoxia) may re-open a functionally closed DA prior to anatomic closure.
- Patent ductus arteriosus (PDA) leads to L→R shunt that results in:
  - Pulm overload: CHF, tachypnea, dyspnea on exertion or with feeding, atelectasis, recurrent resp infections, FTT, and feeding intolerance

#### Perioperative Risks
- Periop mortality rare (approaches zero)
- Hemorrhage due to vascular injury
- Recurrent laryngeal nerve (RLN) injury
- Inadvertent ligation of aorta, pulm artery or subclavian vessels

#### Worry About
- Hypothermia
- Uncontrolled hemorrhage
- Vagally mediated reflex bradycardia
- Desaturation with lung retraction

### ASSESSMENT POINTS

<table>
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<tr>
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<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Shunt-(usually L→R)</td>
<td>Exercise intolerance</td>
<td>Continuous machinery murmur</td>
<td>ECHO</td>
</tr>
<tr>
<td></td>
<td>Diastolic runoff</td>
<td></td>
<td>Hyperactive precordium</td>
<td>NIBP</td>
</tr>
<tr>
<td>RESP</td>
<td>CHF</td>
<td>Diuretic dependence</td>
<td>Rales/crackles</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Bowel ischemia</td>
<td>Poor feeding</td>
<td>Abdominal distension</td>
<td>Abdominal X-ray</td>
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<tr>
<td></td>
<td>Renal ischemia</td>
<td>Poor UO</td>
<td>Fluid overload</td>
<td>Creatinine</td>
</tr>
</tbody>
</table>

#### Key References:

### Indications and Usual Treatment
- Medical management: Fluid restriction (120 mL/kg/day) and diuretics
- Pharmacologic closure for neonates: NSAIDs (indomethacin, ibuprofen) inhibit prostanlagent production and are effective in many cases.
- Surgical options for failed pharmacologic closure in neonates (usually after two doses) or for older pts with PDA:
  - Open thoracotomy (most common): Usually a clip for premature infants; otherwise ligation and division
  - Video-assisted thoracoscopic clip ligation
  - Transcatheter closure in select pts. Coil for ≤4 mm in diameter; Amplazer® PDA device if >4 mm diameter

### Perioperative Management

#### Preoperative Preparation
- Warm the OR prior to pt arrival
- Have RBC products available in the OR prior to incision
- Clear bubbles from all IV tubing
- Ensure peripheral or central vascular access large enough for red cell transfusion
- Check for assoc congenital anomalies of airway
- Pt transport is major risk for premature neonates; consider performing procedure in the neon ICU if feasible

#### Anesthetic Technique
- General anesthesia
- NARCotic and/or ketamine induction preferred in neonates
- May require fluid resuscitation at induction because of prep fluid restriction and diuretics
- Avoid high FIO, if possible; this could increase shunting to pulm vasculature
- Need to check vocal cord function at end of case if at risk of RLN injury

#### Surgical Stages

- **Incision**
  - Thoracotomy incision; side depends on aortic arch orientation (normally left)

- **Dissection**
  - Lung retraction may produce hypoxia and hypercarbia

- **Ligation**
  - PDA is usually ligated (suture or clip); may or may not be surgically divided
  - Although blood loss usually minimal, must be prepared for hemorrhage due to vessel injury or inadequate ligation
  - Watch for intraop Htn after ligation; risk of stroke in neonates

#### Postoperative Considerations
- Postop CXR to check for pneumothorax
- Many centers now use small-tube thoracostomy or no thoracostomy tube
- Optimal analgesia is essential for recovery
- IV PCA (with or without intercostal nerve blocks)
- Continuous neuraxial analgesia in select pts
- Neonates may require prolonged intubation and ongoing postop resp management
- Premature infants may develop postop hypotension

#### Anticipated Problems/Concerns
- CV collapse at ligation: Undiagnosed anomalous coronary artery or pulm Htn
- Residual ductal patency may result in intra-vascular hemolysis and ongoing hemodynamic symptoms
- Vocal cord/RLN palsy may occur after ligation (even after transcatheter device techniques)
- Risk of thoracic duct injury and chylothorax

#### ICD-9-CM Code: 747.0 (Patent ductus arteriosus)
**Pituitary Resection, Transsphenoidal Approach**

**Risk**
- Pituitary adenoma: 14–20/100,000
- Males > females, 1:2

**Perioperative Risks**
- <15% immediate periop mortality
- 8–15% morbidity (transient–1 to 3 d–DI most frequent); hypopituitarism in large resections; CN II – VI damage
- Microadenomas of all types can have up to 90% cure rates in some surgical series
- 70% of untreated acromegalics die before age 50 y
- Untreated Cushing’s disease has a 50% 5-y mortality

**Worry About**
- Increased ICP in large tumors; anemia preop
- Airway in acromegalics and Cushing’s syndrome
- Intraop injection of epinephrine-containing local anesthetics to vasocostric nasal mucosa

(precipitate dysrhythmias or myocardial ischemia); severe Htn if β-blocker present
- Hemorrhage intraop (cavernous sinus intrusion or carotid artery injury)
- Air embolism reported
- Saddle deformity of nose postop
- Blood in airway at end of procedure
- Corneal abrasion if exophthalmos in Cushing’s
- DI or SIADH postop

**Overview**
- Pituitary microadenoma usual indication for surgery; most common complaint is headache. Any tumor may cause hyperprolactinemia 2° to loss of tonic inhibition

**Etiology**
In descending order of frequency, tissue types/most common presenting symptoms are:
- Nonsecreting adenoma/visual field defect, headache, CN II–VI may be affected by pressure
- Prolactin-secreting adenoma/amenorrhea; galactorrhea, lost libido, infertility
- ACTH-secreting tumor/Cushing’s disease; obesity, Htn, LVH, diabetes, sleep apnea
- Growth hormone–secreting tumor/acromegaly, Htn, LVH, cardiomyopathy, diastolic dysfunction, diabetes, sleep apnea, hypertropic mandible/facial bones, laryngeal stenosis
- Thyrotropic adenomas (rare): Signs and sx hyperthyroidism, palpitations, tremor, wt loss, sweating

ICD-9-CM Codes: 253.0 (Acromegaly); 255.0 (Cushing’s syndrome)

**Assessment Points**

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<tbody>
<tr>
<td>HEENT</td>
<td>Airway in acromegalic; glottic fixation</td>
<td>Hoarseness, sleep apnea?</td>
<td>Airway</td>
<td></td>
</tr>
<tr>
<td></td>
<td>or narrowing in GH excess</td>
<td>Tongue size, stridor/DOE?</td>
<td></td>
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<tr>
<td></td>
<td>Airway in Cushing’s CN III–VI impingement</td>
<td>Visual disturbance, field cut</td>
<td>Visual field</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Htn in acromegaly and Cushing’s</td>
<td>CV status, LVH</td>
<td>Exercise tolerance</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chest pain, sleep apnea?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>↓ FRC in obese</td>
<td>SOB, DOE</td>
<td></td>
<td>ABG’s, SpO₂</td>
</tr>
<tr>
<td>ENDO</td>
<td>DM</td>
<td>Glucose intolerance</td>
<td></td>
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<tr>
<td></td>
<td>Hypercortisolism</td>
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<tr>
<td></td>
<td>Hypopituitarism</td>
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<tr>
<td>RENAL</td>
<td>Hypertensive or diabetic kidney disease</td>
<td></td>
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</tr>
<tr>
<td>CNS</td>
<td>↑ ICP in severe suprasellar extension</td>
<td>Headache, N/V</td>
<td></td>
<td>Visual field</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Funduscopic exam</td>
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</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Evaluate for significant CAD (Hx, ECG, exercise tolerance); may suffer myocardial stress from exogenous epinephrine, sleep apnea?
- Case requires oral ET tube; plan fiberoptic intubation if indicated.
- Surgeon may request lumbar subarachnoid drain to manipulate CSF (inject saline or remove CSF) or inject air intraop to outline suprasellar extension and monitor progress of resection. Postop, CSF catheter may be placed to drain if CSF leak anticipated.

**Anesthetic Technique**
- GA required

**Monitoring**
- Routine + air embolism (ETCO₂)
- A-Line if cardiomyopathy/CHF in acromegalics/Cushing’s

**Airway**
- In acromegalics, hypertrophy of facial bones, jaw, nose, turbinates, soft palate, tonsils, epiglottis, and larynx may occur; Mask fit/intubation may be difficult. Fiberoptic intubation may be indicated.

**Induction/Maintenance**
- Rapid-acting induction agent acceptable
- Maintain anesthetic with narcotic, volatile anesthetic ± N₂O and relaxant
- Target Paco₂ = 34–38 mmHg
- Subarachnoid drain prior to final positioning
- Surgical positioning: Semi-sitting 5–35° head-up
- Placement of tongs: Watch for adrenergic/hypertensive response
- Injection of epinephrine containing local anesthetic to vasoconstrict: Watch for dysrhythmias, Htn, myocardial ischemia
- Transnasal approach, endoscopic endo nasal more common, or nasal septal and sublabial incision (blood loss)
- Placement of transsphenoidal speculum or endoscope: Bone work, needs fluoroscopic control to ensure midline approach (high anesthetic requirement for this stage)
- Adenoma removal under direct visualization with microscope
- If suprasellar extension, surgeon may want saline or air injected: if air, D/C N₂O
- If lateral extension of tumor occurs, excessive bleeding may ensue from invasion of cavernous sinus or carotid artery. Induced hypotension via high concentration volatile anesthetic may improve visualization and allow adequate hemorrhage control.
- Rebuilding sella turcica (part of nasal septum used)
- If CSF leak with valsalva, pack with fat pad (abd wall donor site)
- Close, EBL: 150–400 mL

**Exubation**
- Aim to have pt awake, comfortable, and cooperative prior to extubation
- Mouth and pharynx packed with gauze to prevent blood in stomach or airway; ensure its removal at end of case

**Adjutants**
- PONV prophylaxis, be ready to Tx Htn (labetolol)

**Postoperative Period**
- Headache, pain score: 2–3
- Assess CN II – VI function
- Disorders of H₂O balance: DI or SIADH in up to 25% of cases
- DI in 8–15%, usually transient. Dx via analysis of high volume (3–6 mL/kg/h) of dilute urine
Procedures

(<200 mOsm/L, specific gravity = 1.001–1.005). May require desmopressin (DDAVP) 0.1 mg po or 1 mcg SQ q4–6h if serum Na+ >145 meq/L. Replace urinary losses. If serum >320 Osm/L, replace H2O loss.

• Delayed hyponatremia (SIADH). Serum Na+ <135 mEq/L. Confirm high urine Na+ (>20 mEq/L) + euvolemia. Tx: Restrict H2O. If Na+ <120 consider slow hypertonic saline

• CSF rhinorrhea: Lumbar CSF drain to reduce CSF pressure

• If excessive packing needed to control cavernous sinus bleeding, CN II, IV, or VI compression can occur. Impingement of cavernous internal carotid can result in carotid spasm.

• If air has been injected subarachnoid, tension pneumocephalus can occur

• Hypopituitarism postop
Pneumonectomy

**Risks**
- Primarily for bronchogenic CA; median age 73 y
- ≤20% of non-small cell CA surgeries; lobectomy often as effective
- Benign: Mycobacteria, fungus, infection/necrosis, trauma

**Perioperative Risks**
- Mortality up to 12% within 30 d
- Cardiac morbidity significant
- Morbidity and/or mortality higher after right pneumonectomy, more complex procedure, some benign diseases
- Mortality for trauma and/or massive hemoptysis >33%

**Perioperative Implications**

**Preoperative Preparation**
- Bronchodilators
- If sputum: Antibiotics, hydration, mobilization
- Prophylactic digoxin not warranted

**Monitoring**
- Arterial line; CVP not routine intraop, but may be useful postop
- PA: Place on nonop side; fluoroscopy helpful; consider TEE

**Airway**
- If difficult airway or unable to intubate orally; bronchial blocker, nasal intubation
- Aspiration devastating

**Induction/Maintenance**
- Lateral decubitus positioning: Check ear and eye on down side, axillary roll, arm positioning
- Potent inhalational agents bronchodilate but ↓ LV function, attenuate hypoxic vasoconstriction; ↑ in Qs/Qt not clinically significant at 1.0 MAC

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Recurrent laryngeal nerve involvement</td>
<td>Hoarseness</td>
<td>HEENT exam</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>RV dysfunction due to PA Hm LV function, valvar disease Arrhythmias</td>
<td>Chest pain/SOB Exercise tolerance, palpitations</td>
<td>CV exam</td>
<td>ECG, possible ECHO, Doppler studies, PA catheterization</td>
</tr>
<tr>
<td>RESP</td>
<td>Sputum, bronchospasm; ability to tolerate loss of lung</td>
<td>SOB, exercise tolerance, sputum, smoking Hx</td>
<td>Resp exam</td>
<td>Clubbing</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypercalcemia; SIADH → hyponatremia; Cushing's syndrome</td>
<td>Somnolence, anorexia, N/V, wt loss, signs of water intoxication</td>
<td></td>
<td>Chest CT; ABGs; PFTs; FEV₁, DLco Quantitative VQ scan; VO₂max; See note</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, polycythemia Migratory thrombophlebitis</td>
<td>Hx of thrombophlebitis</td>
<td></td>
<td>Hct</td>
</tr>
<tr>
<td>NM</td>
<td>Eaton-Lambert syndrome (E-L) Polymyositis</td>
<td>Muscle wasting</td>
<td>E-L: Sensitivity to nondep muscle relaxants</td>
<td></td>
</tr>
</tbody>
</table>

**Worry About**
- Pulm reserve and/or pulm Hm, edema, after resection
- Concomitant CV disease
- Cardiac arrhythmias common postop
- Periop thromboembolic events in 26%
- Benign diseases: Neovascularization, high-pressure bronchial system bleed; soiling of contralateral lung

**Overview**
- Mortality of untreated non–small cell lung CA is 100%; treatment is surgery
- Paraneoplastic syndromes not a contraindication
- Effective for drug-resistant TB
- Last resort for traumatic injury: Resection in hypovolemic shock leading to persistent high

**Indications and Usual Treatment**
- Non–small cell lung CA (T2) with hilar involvement, no distant mets; mainstem bronchus involvement or crossing major fissure
- T3 lesions: Plus resection of involved chest wall, diaphragm, mediastinal pleura and/or pericardium
- Sleeve pneumonectomy: resection of carina, ipsilateral lung, and bronchial tree; anastomosis of contralateral mainstem bronchus to distal trachea

**Prophylaxis**
- Acceptable risk: DLCO >60%; FEV₁ >60% predicted if no pulm Hm; quantitative V/Q scans predict postop DLCO >40% FEV₁ > 40% or VO₂max > 15 mL/kg/min. Test values are used to define risk to the patient to aid in informed decision-making, not as rigid limits prohibiting attempted surgical cure.


**Anticipated Problems/Concerns**
- Hypotension from unrecognized blood loss; cardiac tamponade; MI/ischemia with low CO
- Postop pulm edema: R/O myocardial dysfunction; volume overload; treat dysrhythmias, hypoa- lbuminemia; atelectasis, pneumonitis
- DVT and pulm embolism common (20%)
- Persistent air leak
- Excess mediastinal shift life-threatening
- Cardiac herniation (mortality >50%); after R pneumonectomy, cardiac torsion→shock, SVC syndrome; after L resection, pericardial compression→ischemia, arrhythmias, outflow tract obstruction; RX: Full lateral with op side up; surgical emergency
- Empyema in 5%
- Bronchopleural fistula in 4%

**ICD-9-CM Codes:** 162.2–9 (Primary lung cancer); CPT code 32.5 (Pneumonectomy)
Pregnant Surgical Patient

Ihab Kamel

Risk
- Incidence in USA: 75,000 pregnant pts/year undergo non-delivery procedures
- Most common: Trauma-related procedures, cervical suture, appendectomy, biliary tract disease-related procedures, breast biopsy, ovarian cystectomy
- Major procedures such as liver transplantation, cardiopulmonary bypass and craniotomy have been performed during pregnancy with good outcomes to the mother and fetus.
- Surgery is performed in pregnancy in about 1–2% of pts. The number is increasing due to laparoscopic procedures.

Perioperative Risks
- ↑ Maternal anesthetic risk for hypoxemia
- ↑ Maternal risk pulm aspiration due to failed ET intubation (increased mucosal vascularity and weight gain distortion of pharyngeal anatomy)

Overview
- If surgery must be performed during pregnancy, 2nd trimester is preferred period, since organogenesis is complete and risk of preterm delivery relatively low

Worry About
- Maternal airway precautions
- Gastric chemophraphylaxis
- Prevention and treatment of maternal hypoxemia
- Avoidance of aortocaval compression (after 20 wk of gestation) and hypotension (uterine blood flow is not autoregulated)
- Detection and treatment of preterm labor

Perioperative Management

Anesthetic Technique
- Regional anesthesia: ↓ Risk of maternal airway problems, ↑ risk of hypotension (compared with GA). Regional anesthesia might be considered the first choice where appropriate.
- General anesthesia: Inhalation agents are tocolytic—may prevent contractions in OR; however, preterm labor may occur in recovery period. Endotracheal intubation and rapid sequence induction after 14–18 wk of gestation.

Monitoring
- Viable-gestational-age fetus (18–22 wk): Consider pre-, intra-, postop fetal heart tone and uterine activity monitoring.
- Loss of beat-to-beat variability (present by 25–27 wk) is normal with anesthetics.
- Fetal heart rate decelerations are abn and should be managed by improving maternal oxygenation, raising BP and increasing left uterine displacement

Induction
- Regional anesthesia: Spinal, epidural, or other block may be appropriate depending on location of surgical site
- General anesthesia: Full stomach—awake intubation vs. denitrogenation followed by rapid-sequence induction

Maintenance
- Maintain left uterine displacement

ASSESSMENT POINTS

<table>
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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Engorged, fragile mucosa, difficult intubation</td>
<td>Airway exam</td>
<td>Mallampati class</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td>Supine hypotensive syndrome</td>
<td>Nausea, diaphoresis while supine</td>
<td>Assess for hypotension, bradycardia while supine</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>↑ O₂ consumption; ↓ FRC</td>
<td>Ambulatory: ↑ Pao₂, ↓ Paco₂</td>
<td>ABGs (if indicated)</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Full stomach, decreased LES tone</td>
<td>Reflux symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>↓ MAC, ↓ intraspinral local anesthetic requirements</td>
<td></td>
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</table>


Indications and Usual Treatment
- Cervical suture placement for prevention of preterm delivery due to cervical incompetence (performed at 12–16 wk of gestation)
- Other procedures performed only when risks of postponement outweigh benefits of avoiding increased maternal anesthetic risk and potential fetal harm

Anticipated Problems/Concerns
- Nonviable-gestational-age fetus: Pre- and postop fetal heart tone documentation; consider pre-, intra-, and postop uterine activity monitoring
- Obstetric consultation highly recommended; pediatric notification indicated if delivery of a viable fetus is possible

Exubation
- After pt awake, able to protect airway

Postoperative Considerations
- Pain: Consider IV or epidural PCA (opioids should not be withheld). Avoid nonsteroidal anti-inflammatory drugs.
- Left uterine displacement in PACU
- Document fetal viability
- Consider monitoring for preterm labor

Acute exposure to anesthetic agents has not been assoc with fetal malformations at birth
- Depressant effects of anesthetic agents are mainly of concern if fetus is delivered periop

ICD-9-CM Code: V22.2 (Pregnancy)
Pyloric Stenosis Repair

J. Lance Lichtor
Ulrike Berth

**Risk**
- 1–4/1000 births
- Highest concentration in whites of northern European ancestry, lower incidence in African-Americans, rare in Asians
- M:F ratio: 4:1
- Tends to run in families (children of affected parents have higher incidence [20% of male and 10% of female descendants of mother who had pyloric stenosis])
- 2.5–5.5 times higher incidence for firstborn
- Increased incidence with types B and O blood groups
- Average age of onset is 1–2 mo, though can occur as early as first week of life and as late as the fifth month.
- Etiology unknown, increased incidence after repair of congenital abn (e.g., esophageal atresia, omphalocele, hyperplasia or agenesis of inferior labial frenulum)

**Overview**
- Gross thickening (hypertrophy) at circular smooth muscle of pylorus resulting in gradual obstruction of gastric outlet
- Nonbilious projectile (no projectile initially but usually progressive) vomiting occurring immediately after feeding; usually 2–4 wk; can have severe dehydration and acid-base abn
- Surgical treatment is curative
- Not a surgical emergency

**ICD-9-CM Code: 750.5**

**Indications and Usual Treatment**
- Persistent vomiting usually after or toward the end of a feed. After vomiting, infant is hungry and wants to feed again, until dehydration profound, when the infant becomes lethargic.
- Pyloromyotomy is treatment of choice
- No successful medical therapy

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>Pyloric thickening</td>
<td>Vomiting</td>
<td>Olive-size mass, upper abd</td>
<td>US or upper GI series</td>
</tr>
<tr>
<td>HEME</td>
<td>Hemoconcentration</td>
<td></td>
<td>Volume status measures; orthostatic vital signs Vasoconstriction</td>
<td>Hct</td>
</tr>
<tr>
<td>LYTE</td>
<td>Dehydration</td>
<td>Acid-base abn</td>
<td>Volume status measures; orthostatic vital signs; area with cold skin</td>
<td>Electrolytes Hypokalemia, hyperkalemia</td>
</tr>
<tr>
<td>RENAL</td>
<td>Alkaline urine (initial)</td>
<td>Persistent vomiting</td>
<td>Volume depletion</td>
<td>UO</td>
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<tr>
<td></td>
<td>Acid urine (late)</td>
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**Perioperative Implications**
- Volatile anesthetic, consider desflurane (rapid awakening, decreased episodes of apnea)
- Local anesthetics using local infiltration of skin with bupivacaine (maximum dose 1 mL/kg of 0.25% bupivacaine) useful in combination with general anesthesia

**Postoperative**
- Apnea (continue close monitoring)
- Hypoglycemia
- Continue volume and fluid resuscitation
- Consider acetaminophen for pain control (40 mg/kg initial dose followed by 20 mg/kg every 6 hr rectally or 10–15 mg/kg by mouth every 4–6 hr for a total 24-hr dose of 100 mg/kg)

**Surgical Stages**

**Dissection**
- Pylorus delivered into wound (open procedure); incision through serosa and extended through the length of hypertrophic pylorus; hypertrophied muscle split bluntly

**Anticipated Problems/Concerns**
- Hypotension due to altered volume status
- Hypoglycemia and convulsions, possibly due to cessation of IV glucose and depletion of liver glycogen
- Severe metabolic disturbance (correct preop)
- Apnea, possibly related to cerebrospinal fluid alkalosis
- Vomiting on induction, postop vomiting if early feeds
- Wound infection not uncommon
- Duodenal perforation may occur during myotomy; not a life-threatening problem if recognized intraop
Radical Neck Dissection

Risk
• More than half a million cases of new head and neck cancers occur every year
• Risk of head and neck cancers assoc with long-term smoking, alcohol abuse, and viral infection with HPV
• M:F ratio: 3:1

Perioperative Risks
• Periop morbidity and mortality assoc with multiple comorbidities
• Risk of MI, CVAs as per CV comorbidities related to smoking and/or alcohol abuse
• Risk of cranial nerves and major vessels injury, venous air embolism and pnx depend on site/level of surgery and technique used
• Malnutrition
• Presence of tumor may affect airway patency (obstruction/compression/obstruction)
• Previous radiation therapy can cause difficult airway management (edema, fibrosis)
• Long-term smoking in these pts frequently assoc with COPD
• Alcohol abuse linked to liver failure, anemia, neurologic impairment, postop withdrawal, DT
• Malnutrition

Overview
• The term radical neck dissection implies removal of all cervical lymph node groups that are ipsilateral to the cancerous lesion, from level I to V. The spinal accessory nerve (SAN), sternalcleidomastoid muscle (SCM) and the internal jugular vein (IJV) are also removed

ASSESSMENT POINTS

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<tbody>
<tr>
<td>HEENT</td>
<td>Tumor impact on airway</td>
<td>Dyspnea, dysphagia, dysarthria</td>
<td>Direct and FO airway exam</td>
<td>CXR, CT/MRI, flow-volume loops</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Smoking and alcohol-related co-morbidities</td>
<td>SOB, exercise tolerance, Hx of palpitations and/or chest pain</td>
<td>CV exam</td>
<td>CXR, ECG, stress test echocardiogram/angio</td>
</tr>
<tr>
<td>RESP</td>
<td>Smoking related</td>
<td>SOB, cough, sputum</td>
<td>Chest auscultation</td>
<td>CXR, PFTs, consider ABG</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, coagulation abn</td>
<td>Fatigue, bleeding, easy bruising</td>
<td>Inspection of skin, mucosas</td>
<td>CBC, PT, PTT, LFTs</td>
</tr>
<tr>
<td>CNS</td>
<td>Alcohol withdrawal Nutritional deficiencies</td>
<td>Anxiety, tachycardia, diaphoresis, peripheral neuropathy</td>
<td>Neuro exam</td>
<td>Based on Hx and PE</td>
</tr>
</tbody>
</table>


Perioperative Management

Preoperative Preparation
• Prepare for table rotation at 180° (head of pt 180° away from anesthesia provider) with extensions on IV lines and ventilator circuit
• 2 large-bore peripheral IVs required also considering arms tucked and 180° rotation (consider CVC if peripheral access not adequate; femoral line preferred, as neck is site of surgery)
• Prepare blood infusion set connected to fluid warmer

Monitoring
• Standard + Temperature measuring urinary catheter + A-line +/- CVC for invasive BP/CVP monitoring
• Consider minimally invasive hemodynamic monitors based on beat-by-beat derivation of hemodynamic variables from A-line waveform
• Close monitoring of NMB: Complete reversal of muscle relaxation must be obtained to allow nerve monitoring by surgeon

Anesthetic Technique/Induction
• General anesthesia with ETT inserted orally (DL or FO technique), unless elective awake tracheostomy is required to secure airway

Maintenance
• General inhalational/IV anesthesia + opioid continuous infusion (infusion pump required)
• Avoid peripheral vasoconstrictors if flap reconstruction is part of the procedure
• Cautious fluid management is recommended, better if goal directed, as guided by hemodynamic monitoring
• Administration of LMWH is suggested to prevent venous thrombosis

Surgical Stages
• Neck incision and dissecting technique may vary
• Large vessels and nerves can be damaged with risk of significant blood loss and nerve paralysis
• Occasionally carotid artery is sacrificed when invaded by tumor
• Modified neck dissections preserve SAN, SCM and/or IJV. Selective neck dissections preserve one or more lymph node groups, depending on the location of the tumor

ICD-9-CM Code: 40.40 (Radial neck dissection, not otherwise specified)

Indications and Usual Treatment
• Neck dissections are the procedures of choice to control the spreading of metastases from cancers of the upper airway, thyroid, parotid glands, and upper digestive tract
• Often are performed in combination with the resection of the 1° tumor
• Surgical defect may need repair with flap and/or skin graft

• Duration: 4–8 hr (mono or bilateral). If combined with tumor resection and flap reconstruction, may take up to 20 hrs or more.

Postoperative Considerations
• Pts that have been intubated and have not had a tracheostomy during the case may be extubated immediately after surgery (esp if monolateral neck dissection) or kept intubated overnight (bilateral neck dissection, combined tumor resection/flap reconstruction)
• Pts with tracheostomy/stoma may also be allowed to wake up immediately after surgery (calm, optimal pain control) or kept sedated overnight.

Anticipated Problems/Concerns
• Preventing coughing and retching at emergence is of paramount importance to avoid straining of sutures and rebleeding.
• Adequate nutritional support, pulm toilet and treatment of potential alcohol withdrawal symptoms are critical points in the postop management of these pts.
Radical Prostatectomy (Retropubic)

Overview
- Prostate, bladder, and seminal vesicles are removed
- Significant blood loss assoc with transection of dorsal vein
- Regional anesthesia may be assoc with lower blood loss and lower incidence of DVT
- Laparoscopic procedure rarely being performed
- Although autologous predonation common, becoming less widely used
- Consider hemodilution techniques to ↓ blood loss
- Myocardial ischemia ↑ if Hct <28%
- Epidural analgesia with general anesthesia and use of NSAIDs prior to operation may reduce cancer recurrence rates compared to general anesthesia and postop opioids

ICD-9-CM Code: 185.0 (Cancer of prostate)

Indications and Usual Treatment
- The following conditions should be met:
  - Isolated and localized prostatic malignancy
  - Anticipated life expectancy of ≥10 y
  - Good general health
  - Absence of indication of metastatic disease by work-up
  - Robotically assisted prostatectomy becoming more common
  - If co-existing disease, orchietomy and hormonal therapy 1° treatment
  - Radiation therapy has similar 5-y survival rates
  - “Smart bomb” radiation therapies also have similar 5-y survival rates with less surrounding tissue toxicity than classic radiation therapy
  - Proton therapy newer option without known success rate
  - Herbal therapies are alternative—8 hydroxy compounds in green teas have effectiveness in experimental animal models

Perioperative Implications

Anesthetic Technique
- Can be performed under regional, general, or combined anesthetic techniques

Monitoring
- Large-bore IV lines for blood loss
- Consider arterial line depending on extent of surgery
- Consider CVP, PCWP, or TEE if co-existing disease
- Consider monitoring for air embolism
- Consider aspirin or other NSAID prior to induction

Airway
- None

Induction
- Requires level of at least T8 for regional

Postoperative Considerations
- Significant postop pain
- Epidural may lead to fewer complications
- Pain score: 4–8
- IV PCA or epidural PCA for 2–3 d; newer modalities with education are assoc with discharge on postop day 2 or 3
- May develop pulmonary embolism or DVT; use aspirin or other NSAID preop and continue postop daily for at least 3 wk
- May develop peroneal nerve injury from lithotomy position

Anticipated Problems/Concerns
- Air embolism may occur because of large open veins and pt position

Risk
- 50,000 pts/y but decreasing with alternative therapies (randomized outcome data shows requires about 48 ops to save 1 life with this procedure) Often done robotically now.
- Racial predominance: None

Perioperative Risks
- Periop mortality rare (<1%)
- Increased risk of DVT, pulm embolism
- 25% risk of impotence with nerve-sparing procedure; 75% risk without nerve-sparing procedure

Worry About
- Venous air embolism
- Massive blood loss
- Nerve injury from position and surgical manipulation
- Often in flexed position with head down, increasing risk of aspiration

System Effect Assessment by Hx PE Test
RESP Pulm metastases SOB Auscultation CXR, CT scan
MS Skeletal metastases Bone pain Palpation X-ray, bone scan, prostate-specific antigen (PSA)

ASSESSMENT POINTS

**Risk**
- Incidence: 3% of vaginal deliveries in developed countries
- Risk increases with Hx of high parity, uterine injury and/or anomaly, preterm labor, and induction of labor

**Perioperative Risks**
- Second most common cause of postpartum hemorrhage
  - 10% mortality if left untreated
- Extraction with curettage increases risk of uterine perforation
- Umbilical cord traction may lead to uterine inversion

**Worry About**
- 6% of placentas requiring manual extraction are placenta accretas
- Blood loss is often much greater than that visually estimated
- Pt is at high risk for difficult intubation and pulm aspiration if general anesthesia indicated

**Overview**
- 1° causes incl: Placenta adherens, myometrium underlying placenta fails to contract; trapped placenta, detached placenta is trapped behind a closed cervix; and partial accreta, a small area of accreta prevents detachment
- Significant increase in risk of postpartum hemorrhage when the third stage of labor is greater than 30 min

**Indications and Usual Treatment**
- Manual removal is effective in all causes of retained placenta
- Medical management may be attempted for placenta adherens and trapped placenta
  - 20% of placenta adherens respond to intraumbilical oxytocin (30 IU in 30 mL saline)
  - Trapped placenta may respond to IV (50 to 100 μg) or sublingual (1 mg) nitroglycerin
- If third stage of labor exceeds 60 min, manual exploration and extraction is indicated

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIRWAY</td>
<td>Airway edema</td>
<td>Snoring, stridor</td>
<td>MP classification</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Hemorrhage</td>
<td>EBL</td>
<td>BP</td>
<td>Hgb, Hct</td>
</tr>
<tr>
<td>GI</td>
<td>Delayed gastric emptying</td>
<td>NPO status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Coagulopathy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UTERUS</td>
<td>Placenta adherens</td>
<td>Fundus soft</td>
<td>Fundus contracted; edge of placenta palpated through tight cervical os</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Trapped placenta</td>
<td>3rd stage &gt;30 min</td>
<td>3rd stage &gt;30 min</td>
<td>US</td>
</tr>
</tbody>
</table>

**Perioperative Management**

**Preoperative Preparation**
- Prepare for postpartum hemorrhage: adequate multiple large bore IV access, type and cross

**Monitoring**
- Standard monitors incl non-invasive BP and pulse oximetry
- Consider invasive BPs and Foley catheter if blood loss is significant

**Monitored Anesthesia Care/Sedation**
- May be sufficient with trapped placenta
- Analgesia achieved with 10 mg ketamine boluses (not exceeding 0.5 mg/kg) or 50 to 100 μg fentanyl
- Nitroglycerin (50 to 100 μg) provides reliable, rapid (60 sec) smooth muscle relaxation with a short duration (1 to 3 min)

**Neuraxial Anesthesia**
- Addition of local anesthetic through existing epidural catheter or initiation of spinal
- Provides analgesia, not uterine relaxation
- Severe hypotension may occur in the setting of a postpartum hemorrhage

**General Anesthesia**
- Indicated in the presence of hemorrhage or the need for additional uterine relaxation
- 1.5 MAC of sevoflurane or desflurane decreases uterine contractility by 50%
- Increased risk of difficult intubation and pulm aspiration

**Surgical Stages**
- Blind identification of the interface between the uterus and placenta and gentle manual dissection
- Uterine atony may lead to additional blood loss following removal of placenta
- Ergometrine, which causes a powerful, continuous uterine contraction, may close the cervix at the same time as placental detachment occurs, thus trapping the placenta behind a closed cervix

**ICD-9-CM Code. 666-0 (Retained placenta with hemorrhage)**
Retinal Buckle Surgery

Dhamodaran Palaniappan
Steven Gayer

Risks
- Jeopardy of vision loss
- Incidence in USA: <100,000 cases/y
- Racial predominance: None

Perioperative Risks
- Airway obstruction and resp depression from oversedation during MAC
- Blindness from increased IOP 2° to interaction of N2O with intravitreal gas (SF6, C3F8)
- Bleeding 2° to antplatelet/anticoagulant therapy
- Increased risk for globe puncture in severe myopic pts during needle blocks

Worry About
- Oculocardiac reflex (OCR)
- IOP elevation

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effects</th>
<th>Assessment by HX</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
</table>
| HEENT  | Axial length of eyeball
              Claustraphobia
              Deafness | Hx of myopia | Eyeglass prescription | US globe to determine axial length |
| CARDIO | Htm, CAD, Arrhythmias | Exercise tolerance/angina/ palpitations | Pulse rate, rhythm/BP Auscultation for heart murmur | EKG/ECHO |
| RESP   | COPD, OSA, Interstitial lung disease | Chronic cough
              Snoring, home O2, CPAP, exercise tolerance | Chest exam
              Airway exam | CXR/O2 sat |
| HEME   | Bleeding 2° to anticoagulants/antplatelet agents | Easy bruisability
              Hx of DVT | Bruise marks | PT/PTT/INR |
| ENDO   | Hypo/hyperglycemia
              Autonomic instability | Glucose control/antidiabetic medications
              Orthostatic hypotension | Fasting Glucose | |
| RENAL  | Impairment 2° to DM | UO | | BUN/Cr |

Perioperative Management

Preoperative Preparation
- Identical for local and general anesthesia
- Preop fasting as per ASA guidelines
- Anxiolysis and reassurance are crucial
- Ability to lie supine for prolonged duration

Intraoperative

Monitoring
- Routine ASA monitors

Anesthesia Techniques
- RA with MAC, GA, combined RA and GA
- RA: Peribulbar, retrobulbar, sub-tenon’s block
- Often requires MAC sedation
- Peribulbar (extraconal) preferred over retrobulbar (intraconal) block if axial length of globe is unknown
- RA preferred over GA
- Decreased requirement for periop systemic analgesia
  - Decreased incidence of OCR
  - Decreased incidence of PONV
  - Avoidance of IOP alterations 2° to airway instrumentation

MAC Sedation for RA
- Midazolam, fentanyl, remifentanil infusion
- Propofol infusion
- Dexmedetomidine infusion
- Avoid oversedation

- Use O2 by nasal prongs monitor nasal ETCO2
- Elevate drapes at head to prevent CO2 rebreathing
- Consider forced air or active suction cannula to dissipate CO2
- Consider precordial stethoscope

General Anesthesia

Induction
- Intravenous agents
- LMA, ETT (Oral RAE preferred for field avoidance)

Maintenance
- Inhalation agents or TIVA
- N2O: Preferable to avoid, if used D/C 15 min before intravitreal injection of gas (air, SF6, C3F8)
- Muscle paralyis: Usually not required
- Ventilation: PEEP, maintain eupcapia
- Consider IV dexamethasone for multimodal PONV prophylaxis
- Supplementary intraop sub-tenon’s block provides excellent postop pain relief

Emergence
- Beware of premature emergence
- Avoid bucking or coughing and consider deep extubation (IOP)
- Consider IV NSAID for postop pain management if supplemental block not administered intraop

- Consider antiemetic prophylaxis before emergence

Postoperative Considerations
- Pain: 3-5, NSAIDs usually adequate, opioids may be required (beware of PONV)
- PONV: 5-HT3 receptor antagonists preferred
- EBL: Nil

Surgical Considerations
- OCR: Vigilant monitoring for bradycardia and asystole during insertion of RA block, intraop muscle traction
- OCR: Avoid hypercapnia and light plane of anesthesia
- OCR: Stop precipitating surgical stimulus
- OCR: Anticholinergics may be considered for rescue or persistent OCR
- IOP management: Avoid hypercapnia and light plane of anesthesia
- IOP management: Mannitol or acetazolamide may be required to decrease IOP
- N2O: Intravitreal injection of oil or gas (air, SF6, perfluorocarbons)

Etiology
- Diabetes mellitus
- Myopia
- Eye trauma
- Marfan’s syndrome
- Idiopathic

Usual Treatments
- Cryotherapy, laser photocoagulation, plombage
- Scleral buckling or banding (‘Explant’)
- Vitrectomy
- Intravitreal injection of oil or gas (air, SF6, perfluorocarbons)

ICD-9-CM Code: 361.9 (Retinal detachment)
Retropharyngeal and Peritonsillar Abscess Drainage in Adults

Michael F. Roizen

Risk
• Rare without other debilitating conditions, such as alcohol abuse, immune compromise, or dental disease

Perioperative Risks
• Losing airway: No perfect approach (see below), all have risks and benefits

Worry About
• Airway compromise, esp. if severe enough to cause pt to drool and lean forward

Overview
• Complications of acute tonsillitis in which the infection has spread deep to the tonsillar capsule. Pus forms between the tonsillar capsule and the superior constrictor of pharynx, and the tonsil is displaced medially. Uvula becomes edematous, marked trismus and pain occur (head usually tilted toward site of abscess).
• Local infection with systemic implications due to airway compromise and potential for sepsis. Usually a β-hemolytic strep or anaerobe.

ICD-9-CM Code: 478.24 (Retropharyngeal abscess)

Indications and Treatment
• Depends on degree of airway compromise: Goal is 48 hr of antibiotic prior to incision and drainage

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>ENT</td>
<td>Can be massive swelling and edema that inhibits airway and mouth opening and access</td>
<td>Ability to lie flat</td>
<td>Airway exam</td>
<td>Lateral neck x-ray</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Drooling</td>
<td>Voice distortion</td>
<td>&quot;Hot potato&quot; voice</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Sepsis Hypovolemia</td>
<td>Hx of symptoms of infection</td>
<td>HR</td>
<td>ECG ?CVP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>BP</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Temp</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Management

Anesthetic Technique
• Alternatives based on two factors:
  * Most important: How to secure airway
  * Presence of sepsis

Monitoring
• Without sepsis: Usual
• With sepsis: CVP, and consider arterial line

Airway
• Difficult choices depending on severity and judgment of individual anatomy
• Topical and awake I & D if pt can tolerate is usual first choice

• Awake trach preferable if no other easy means and sepsis does not involve that area of neck
• Awake fiber optic risks abscess contamination or accidental I & D and severe lung infection or losing airway

Induction
• After airway secure

Surgical Stages

Induction
• After airway secure

Surgery
• Usual I & D and return at another time for tonsillectomy, etc.
• Pus cavity and site of infection can be difficult to find

• Airway patency
• Lung infection
• Underlying problem
• Sepsis management usually easier after abscess drained

Anticipated Problems/Concerns

• Airway compromise
• Underlying illness, incl drug withdrawal
### ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>If RA or connective tissue disease, may have limited ROM of head/neck</td>
<td>Limited ROM head/neck or neck</td>
<td>Airway exam</td>
<td>C-spine x-ray</td>
</tr>
<tr>
<td>CARDIO</td>
<td>If RA or connective tissue disease, may have valvular heart disease/conduction defects</td>
<td>Angina/PND/orthopnea</td>
<td>Chest exam</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Palpitations/CHF</td>
<td>Exam of peripheral pulses and vital signs</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>If RA, may have pulm fibrosis or pleural effusion</td>
<td>SOB</td>
<td>Chest exam</td>
<td>CXR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Exercise tolerance</td>
<td></td>
<td>ABGs</td>
</tr>
<tr>
<td>HEME</td>
<td>Hx of NSAID use—may affect coagulation</td>
<td>Hx of medications, bleed/bruise</td>
<td>Inspect for evidence of same</td>
<td>CBC and differential</td>
</tr>
<tr>
<td>IMMUNO</td>
<td>If RA, may be immunocompromised</td>
<td>Hx of medications, infectious disease</td>
<td></td>
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</tr>
</tbody>
</table>

See Rheumatoid Arthritis if appropriate


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### Surgical Stages

- Positioning either in lateral decubitus or in beach chair position, beach chair position is convenient to convert from arthroscopic to open procedure
- **Arthroscopic Repair**
  - Need multiple portals: Posterior portal, anterior, lateral, and anterolateral
  - Posterior portal is located about 2 cm inferior and 2 cm medial to the posterolateral margin of the acromion within the posterior soft spot of the GH joint. The anterior portal is placed lateral to the coracoids in line with the AC joint. The lateral portal is placed 3 cm lateral to the acromion in line with the posterior border of the clavicle. An accessory anterolateral portal is created for instrumentation and suture management.
  - Examination and evaluation of the GH joint and subacromial space, probing the undersurface of the rotator cuff to its insertion on the greater tuberosity to note and evaluate the location, size, and character of any tears as well the amount of cuff retraction and the quality of the tissue then subacromial decompression to increase in working space for cuff repair, avoid injury to the suprascapanular nerve and vessels located 1 to 2 cm medial to the lateral edge of the glenoid.

### Open Repair

- Mini-open or traditional open approach to rotator cuff repair
- Incision: Oblique over acromion, part of the deltoid muscle detached to reach rotator cuff. In mini-open repair no deltoid detachment
- After acromioplasty, visualize and mobilize the tear off the bursa, deltoid and corachohumeral ligament using blunt and sharp dissection, 1st repair of the soft tissue component followed suture anchors. Careful repair of deltoid if detached and close the wound.

### Postoperative Considerations

- A sling and 30-degree abduction pillow are used for a week and placed before awakening; supervised graded passive range of motion is initiated as soon as possible
- Pain control with ISB using long acting LA
- Augmenting ISB with NSAIDs
- Inpatient may use PCA

### Anticipated Problems/Concerns

Disease of elderly (CAD, COPD, CKD, DM)
- Be aware that sudden drop in the BP may be venous air embolism
- Axillary and musculocutaneous nerve injury with open procedures and aggressive dissection
**Overview**

- Most commonly coronal and/or sagittal plane deformity correction is performed through a posterior approach during which instrumentation (pedicle screws and/or hooks and rods) is placed and deformity correction is achieved through various deformity correction maneuvers.

- Occasionally, and an anterior approach may be utilized instead or in addition to the posterior approach; typically this is done through a trans-thoracic approach (i.e., thoracotomy), a thoraco-abdominal approach, or an abd (typically retroperitoneal) approach.

- If both a posterior and anterior approach are necessary, the procedure may be performed at the same operation or in a staged manner.

### Perioperative Management

**Preoperative Preparation**
- Pt should be instructed how to correctly use incentive spirometry (IS) and PCA.
- Discuss risks with pt and family concerning blindness, awareness, PE, paraplegia, MI, CVA, left intubated to ICU, etc.
- Warn pt of possibility of intraop wake-up test.
- Ask pt to practice wake-up test at home and in the preop holding area.

**Monitoring**
- Routine ASA monitors
- Consider arterial line

### Key References:
PROCEDURES

• CVP line useful to assess volume status intraop and provide TPN postop
• Spinal cord monitoring (SCM) incl SSEPs, MEPs (NMEP and TcCEMEP) and EMG
• BIS monitor
• Estimate blood loss from suction canister contents, cell saver device and sponges
• Urinary catheter to assess OU and monitor core temp (normothermia is essential)
• Consider O$_2$ sats pulse oximetry on big toes during anterior exposure of the lower lumbar spine to assess amount of iliac artery compression

Induction/Maintenance
• Induction (either IV anesthesia or inhalation techniques) is guided by pt’s airway and medical conditions.
• Consider awake fiberoptic intubation if indicated.
• Inhalational agents may interfere with SSEP or MEP monitoring.
• After adequate bag-mask ventilation, a short-acting muscle relaxant (NMBA) is administered for intubation. No NMBA is used during the operation if TcCEMEP or EMG is used. Sux is contraindicated in pts with various dystrophic and myopathic conditions and spinal cord injury due to the potential for rhabdomyolysis, hyperkalemia, and cardiac arrest.
• Maintenance with TIVA (continuous infusion of IV propofol and remifentanil preferred) and midazolam 1 mg/hr. IV anesthetic agents have less effect on SSEPs or MEPs.

Surgical Stages
• Posterior approach
  • Pt placed prone with abd and axillae allowed to hang free. Recommend adequate padding and make sure that eyes are protected and not compressed (use an appropriate face pillow or use Gardner-Well tongs or Mayfield head holder).
• Maintain temp using a forced-air warming device and warming IVF and blood products.
• After the spine is exposed, instrumentation is placed, and deformity correction is performed. This is then followed by decortication, bone grafting, and wound closure.
• Substantial blood loss can occur during the procedure given the large surgical area and decortications of bone.
• Adequately resuscitate the pt using blood products. Use both colloids (albumin, voluven, or hespan) and crystalloids to maintain intravascular volume and reduce postop edema.
• Monitor SSEPs or MEPs. Significant intraop neurophysiologic changes occur if SSEP amplitude decreases by 50% or latency increases >10%, or TcCEMEP amplitude decreases by 75% from baseline. Systematic approach to evaluate for potential causes of neurologic injury.
  • Step 1: R/O technical error or electrical interference (artifact or cautery)
  • Step 2: R/O anesthesia-related factors (hypotension, hypoxemia, hypothermia, high concentrations of inhalational agents, and whether using muscle relaxant or not)
• Step 3: R/O surgical factors (modifying or removing instrumentation or distraction forces)
• Step 4: Prepare to perform wake-up test.
• At conclusion, pt placed supine. Pt should be fully awake for neurologic exam (wake-up test) prior to extubation.
• Consider postop pain management with IV PCA
• Anterior approach
  • Pt placed in a lateral decubitus position for a transthoracic or thoracoabdominal approach and supine for an abdominal approach. Recommend adequate padding and an axillary roll if pt is placed in a lateral decubitus position.
• After the spine is exposed, instrumentation is placed, and deformity correction is performed. This is then followed by decortications, bone grafting, and wound closure. If the thorax was violated during the surgery, a chest tube is typically placed.
• Anesthetic issues are similar during an anterior approach, except that massive bleeding can be encountered if the greater vessels are lacerated during the surgery.

Anticipated Problems/Concerns
• Soft tissue: Hematoma, seroma, skin necrosis and most commonly, wound infection. Monitor BIS and BP to make sure neither is too low (as guide to protect somewhat against blindness)
• Continued pain from either unresolved, incompletely resolved, or recurrent postop pain.
• Pulm problems incl: Atelectasis, pneumonia, and PE; postop ventilation may be required in pts with severe resp impairment or NM scoliosis.
• Neurologic compromise or defects (acute vs. delayed): epidural hematoma or abscess, spinal cord ischemia 2° to anterior vascular injury by direct injury with spinal instrumentation or stretch or spinal artery compression after coronal and sagittal plane correction.
• GI: There are many causes of postop ileus, incl pain and the use of narcotics for analgesia, electrolyte imbalances, and manipulation of the bowel during surgery; in addition superior mesenteric artery syndrome may occur particularly in pediatric pts with extensive deformity correction.
Seizure Surgery

Overview
- Seizure: Abnormal electrical activity in the brain resulting in paroxysmal change in motor activity or behavior. Focal or generalized.
- Status epilepticus is defined as more than 30 min of continuous seizure activity or two or more sequential seizures without full recovery of consciousness between the seizures.
- First surgical resection for epilepsy performed in 1886.
- Seizure surgery: Identify and localize epileptogenic focus, then determine whether the pt has adequate functional reserve for safe resection.
- 50–90% pts undergoing seizure surgery have significant decrease in Sz frequency or complete resolution of Sz.
- Surgical procedures: Focal resection, corpus callosotomy, hemispherectomy, and vagus nerve stimulation.

ICD-9-CM Code: 790.3

Etiology
- Seizures occur in ~10% children but most are provoked by somatic disorders outside of brain such as fever, infection, head trauma, hypoxia, or cardiac arrhythmias; less than 30% of Sz in children are caused by epilepsy.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by History</th>
<th>Physical Exam</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Facial trauma; gingival hyperplasia; wt gain or poor airway</td>
<td>Hx of fall with Sz</td>
<td>Facial bruising, etc.</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Hyperventilation preop; risk of aspiration pneumonia</td>
<td>Review medications</td>
<td>Rales, wheezing</td>
<td>CXR, ABG</td>
</tr>
<tr>
<td>GI</td>
<td>Liver toxicity from AEDs; increased hepatic enzyme metabolism</td>
<td></td>
<td></td>
<td>Liver function tests</td>
</tr>
<tr>
<td>ENDO</td>
<td>Bone marrow suppression, coagulopathy, plt dysfunction; metabolic acidosis/ketosis for pts on ketogenic diet hyponatremia</td>
<td>Bleeding, infection</td>
<td>Bruising</td>
<td>CBC, BMP, coags</td>
</tr>
<tr>
<td>CNS</td>
<td>Cognitive dysfunction; Sz, personality changes</td>
<td></td>
<td></td>
<td>CT, MRI, EEG, SPECT scan, Wada test</td>
</tr>
<tr>
<td>MS</td>
<td>Trauma from Sz, bone fragility from AEDs</td>
<td></td>
<td></td>
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</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Clear communication with surgeon regarding procedure and surgical approach as this will dictate anesthetic technique.
- Evaluate if pt is appropriate candidate for awake craniotomy.
- Determine baseline neurologic status; review preop radiographic and neuropsychological diagnostic evaluation.
- Identify specific anticonvulsant regimen and potential implications for anesthetic.
- Phenytion: Leukopenia, anemia, hepatitis
- Valproate: Pancreatitis, hepatic failure, coagulopathy, plt dysfunction
- Carbamazepine: Aplastic anemia, cardiotoxicity, hypothyroidism, hyponatremia

- Other SE include: Nephrolithiasis, electrolyte abn, ataxia, paresthesias, wt changes; chronic polytherapy for refractory cases leads to drug-drug interactions
- Presence of concomitant medical problems assoc with epilepsy
- Avoid premedication with benzodiazepine unless activation of epileptogenic foci not necessary in which case it is acceptable to administer
- Preop laboratory evaluation on case-specific basis; type and cross-matched blood esp. for pediatric pts

Monitoring
- Standard ASA monitors; ETCO2 via nasal cannula for sedation.
- Large-bore IV access prior to surgical start, consider central venous line.

Risk
- The lifetime risk of single seizure: ~10%; cumulative lifetime incidence of epilepsy: 3% with more than half of cases beginning in childhood.
- An estimated 2.5 million Americans and 30 million persons worldwide have epilepsy; ~20% of which are refractory to medical treatment.
- Although 13% of medically intractable pts are considered candidates for surgery, only 1% undergo surgery.

Perioperative Risks
- 3% or less risk of major morbidity (i.e., hemorrhage, infection, stroke, memory, language, or hemianopic visual field deficit) incurred by surgery.
- Cognitive dysfunction, more profound in adults than children.
- Combined morbidity and mortality for epileptogenic focus resection 5–6%, corpus callosotomy <11%, functional hemispherectomy <17%.
- Infants are at higher risk for periop morbidity and mortality than any other age group.

Worry About
- Seizures and/or status epilepticus.
- Loss of airway with sedation.
- Increased ICP with mass lesions.
- Hemodynamic instability: Acute and severe bradycardia, sinus arrest; pediatric population with age <2 y have lower autoregulatory reserves and can be at greater risk of cerebral ischemia.
- Effects of specific anticonvulsant medications
**Procedures**

- Arterial catheter for direct BP monitoring and blood sampling
- Consider PICC line for pediatric cases performed in two stages (grid and strip placements)

**Airway**
- Case dependent
- Risk of airway compromise due to sedation, seizure, or positioning

**Maintenance**
- Choice of anesthetic agent and technique based on two factors: (1) is intraop electrocorticography (ECoG) necessary and (2) should the craniotomy be performed with general anesthesia/intubation or with local anesthesia/conscious sedation
- If GA without ECoG, anesthetic designed to maintain suppression of seizure activity and provide optimal operating conditions
- If intraoperative ECoG used, anesthetic should permit activation of ECoG: AVOID benzodiazepines, and volatile anesthetics
- Conscious sedation: Fentanyl, remifentanil, dexmedetomidine, benadryl intraop fluid and electrolyte management – maintain normovolemia
- Frequent monitoring electrolytes and hemoglobin esp. in infants

**Special Considerations**
- Awake craniotomy: Need cooperative and motivated pt
- Grids and/or strips: Two-stage procedure. Craniotomy for grid placement; monitoring for seizures over following days or week, then return to OR for grid removal and resection of seizure focus. AVOID administration of NO until dura opened because intracranial air can persist for up to 3 wk following a craniotomy and NO can cause rapid expansion of air cavities and result in tension pneumocephalus. Pts can be somnolent for few days postop.
- Resection: Discussion of modality and type of neurophysiological monitoring to be used is critical, specifically ECoG and EEG.
- Corpus callosum: Intraop EEG not required; any anesthetic regimen can be utilized. Postop lethargy and somnolence are common increasing the risk of aspiration and airway problems; leave intubated until fully awake.

**Extubation**
- Aim for rapid smooth emergence.
- Consider leaving intubated if concern for postop somnolence.

**Postoperative Period**
- Close observation in ICU with serial neurologic exams
- Seizures common
- PONV, fluid and/or electrolyte management, pain
- High infection risk in staged procedures

**Anticipated Problems/Concerns**
- Cognitive dysfunction
- New neurologic deficits
- Seizures and/or status epilepticus

**Extracranial Surgery**
- Vagus nerve stimulator: Thought to inhibit seizure activity at brainstem or cortical levels. Device is placed in SQ pocket under left anterior chest wall and bipolar stimulating electrode coils are implanted around L vagus nerve. Side effects of VNS involve vocal cord paralysis, bronchoconstriction, bradycardia, and asystole.
Spinal Fusion

Risk
- Incidence in USA: About 50,000 surgeries/y.
- 328,468 spinal fusions performed from 2002-2006
- Indications: Congenital, oncologic, traumatic, degenerative or infectious

Perioperative Risks
- Periop mortality rare (0-0.5%)
- Venous air embolism (VAE) can occur and produce CV collapse
- Postop neurologic deficit (0.7-5%)
- Massive blood loss possible (1-5%)
- Pneumothorax (1-5%)
- Permanent postop visual loss (POVL) is rare but devastating (0-1%)

Worry About
- Underlying pathology; scoliosis, spinal stenosis, herniated disc, trauma, infection, tumor
- Large intraop blood loss
- Difficult airway with trauma and cervical cases
- Potential for VAE
- Problems of prone position

- POVL due to ischemic optic neuropathy (Risk factors: Surgery duration >6.5 hr, and blood loss >44.7% of estimated blood volume. Intraop hypotension and anemia may contribute).
- Postop resp insufficiency

Overview
- Scoliosis is a rotational abn of spine and ribs that may be assoc with restrictive lung disease
- Unstable cervical fracture (trauma) or severe compression due to spinal stenosis or tumor lead to neurologic symptoms and the potential for difficult airway.
- Evoked potential monitoring can limit choice of anesthetic technique.
- An intraop wake-up test may be requested by operating surgeon
- Intraop VAE is possible

ICD-9-CM Code: 737.30

Indications and Usual Treatment
- Adolescent idiopathic scoliosis
  - Skeletally mature
    - Observation for curves <45°
    - Fusion for curves >50°
  - Skeletally immature
    - Observe for curves <25°
    - Braces for curves 25°-40°
    - Fusion for curves >45°
    - Curves >50° or if a lesser curve with resp compromise, pain, or likelihood of progression to 50° surgery is absolutely indicated.
    - Failure to correct significant curve results in a doubling of mortality for age, potential for progressive back pain, and progressive pulm dysfunction
    - Spinal stenosis, herniated disc, tumor, infections
    - Laminectomy is indicated to decompress the spinal cord and/or spinal nerve roots in pts with intolerable pain, progressive neurologic symptoms and weakness to prevent further progression and irreversible neurologic deficit.
    - Fusion is needed to stabilize the spine after laminectomy.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Cor pulmonale possible if significant lung disease</td>
<td>Exercise tolerance</td>
<td>ECG</td>
<td>ECHO</td>
</tr>
<tr>
<td>RFSP</td>
<td>Pulm dysfunction occurs with significant thoracic curves</td>
<td>Exercise tolerance testing</td>
<td>PFTs corrected for height</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Preop neurologic dysfunction unusual; should lead to further assessment and diagnostic tests</td>
<td>Complete neurologic exam</td>
<td>?CT ?</td>
<td>MRI</td>
</tr>
<tr>
<td>MS</td>
<td>Careful exam may lead to an alternative Dx, e.g., Marfan's, Ehlers-Danlos, Goldenhar's syndromes; syrinx</td>
<td>Careful airway exam. Identify pts with difficult airway</td>
<td>CBC</td>
<td>Blood type and cross-match</td>
</tr>
</tbody>
</table>


Perioperative Implications

Anesthetic Technique
- Performed utilizing general ET anesthesia in prone position
- Awake fiberoptic intubation for unstable cervical spine cases
- Awake fiberoptic intubation or inline stabilization for trauma cases
- Controlled hypotension used to limit blood loss
- Potential for interference with evoked potential monitoring influences anesthetic agent choice. May contribute to POVL if prolonged and assoc with anemia.

Monitoring
- Arterial line useful
- Consider CVP line to monitor volume and perhaps to treat VAE
- Somatosensory evoked potentials (SSEP) and motor-evoked potentials (MEP) are often used to monitor the integrity if the neural elements throughout the procedure.
- Foley catheter may facilitate adequate volume resuscitation

Induction/Maintenance
- Narcotics do not interfere with SSEPs or MEPs
- <1 MAC inhalation agent exerts only minimal effect on potential monitoring
- Muscle relaxants will interfere with MEP
- Consider total IV anesthetic technique (TIVA) for maintenance
- Anticipate need for midprocedure wake-up test
- Controlled hypotension accomplished with β-blockade and IV vasodilators

Preoperative Management
- Large-bore IV lines because of anticipated blood loss
- Discuss possible complications with the pt incl POVL
- Discuss with surgeon the possibility of staging the procedure if expected to be >6 hr
- Blood availability for extensive lumbar and thoracic cases.

Surgical Stages

Positioning
- Pt positioned prone on a frame that allows abl to be free from external compression to reduce venous pressure
- Pressure points padded carefully
- Head and neck must be midline and in neutral position. There must be no pressure on ocular structures or ear cartilage
- Shoulder abduction should be less than 90° to avoid brachial plexopathy in the prone surrender position.

Dissection/Definitive Surgery
- Prior to skin incision, surgical field is often infiltrated with dilute solution of epinephrine.
- Steady bleeding during dissection and decortication
- Hypotension may be due to blood loss or VAE
- After instrumentation in place, distraction is performed
- After distraction complete, a wake-up test may be performed
- Approx duration: 4-8 hr

Postoperative Considerations
- Pain score: 6-9
- Postop resp failure unusual but should be considered a possibility in scoliosis pts with pre-existing restrictive lung disease
- Neurologic exam after wake-up. New neurologic injury is an emergency and requires urgent removal of all hardware.
- Early postop gross check of vision
Anticipated Problems/Concerns

• Potential for massive blood loss and rapid development of hypotension
• Modest risk of VAE because of open epidural veins and prone position

• Periop neurologic injury mainly due to instrumentation. Perform postop neurologic exam.
• Occasional reports of neurologic injury in spite of normal evoked responses support considering continued use of wake-up test

• Potential for POVL, avoid anemia and prolonged hypotension. Surgeon to consider staging long (>6.5 hr) procedures. Perform a gross visual exam after emergence.
## Splenectomy

### Risk
- No age or sex predilection

### Perioperative Risks
- Mortality rate 0–3%
- Overall complication rate is 11.8% assoc with pulm complications, DVT

### Worry About
- Potential for major blood loss requiring transfusion
- Trauma of pancreatic tail, stomach, lineal flexure of colon, left hemidiaphragm, left suprarenal gland, upper pole left kidney

### Overview
- Spleen most commonly injured organ in blunt trauma
- Splenomegaly affects 3% of full-term newborns, and 10% of healthy children
- Splenomegaly common complication of sickle cell disease (African or Mediterranean descent)
- Splenic trauma frequently assoc with other intra-abdominal injuries

### ICD-9-CM Codes: 865.10 (Splenic trauma); 289.4 (Hypersplenism)

### Etiology
- Always required
  - Cancer
  - Hereditary splenocytosis (HS): Absence of specific protein in RBCs that leads to fragile cells
- Usually required
  - Idiopathic thrombocytopenic purpura (ITP)
  - Trauma S/P blunt or penetrating abd trauma requiring emergency operation (14%)

### ASSESSMENT POINTS

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<thead>
<tr>
<th>System</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Cardiotoxicity, dyshrhythmias, CHF</td>
<td>Chemotherapeutic agents—doxorubicin (dose &gt; 550 mg/m²)</td>
<td>ECG, ECHO, MUGA → determine LV function</td>
</tr>
<tr>
<td>RESP</td>
<td>Pleural effusions; left lower lobe atelectasis if splenomegaly; pulm fibrosis</td>
<td>Chemotherapeutic agents—bleomycin, methotrexate, cytarabine</td>
<td>CXR</td>
</tr>
<tr>
<td>GI</td>
<td>Hepatotoxicity</td>
<td>Chemotherapeutic agent—methotrexate</td>
<td>LFTs</td>
</tr>
<tr>
<td>HEME</td>
<td>Splenomegaly, cytopenia</td>
<td>Hematologic disease</td>
<td>CBC with differential Plt count, Bleeding time</td>
</tr>
<tr>
<td>GU</td>
<td>Renal insufficiency</td>
<td>Chemotherapeutic agents—methotrexate, cisplatin</td>
<td>BUN, serum Cr, UA, electrolytes</td>
</tr>
<tr>
<td>CNS</td>
<td>Neurologic deficits Peripheral neuropathies</td>
<td>Chemotherapeutic agents—vinblastine, cisplatin</td>
<td></td>
</tr>
</tbody>
</table>

### Key References:

### Perioperative Implications

#### Preoperative Implications
- Ensure polyvalent pneumococcal vaccine (1 mo prior to surgery, if possible; Pneumovax, Pro-Immune 23 and Menomune-A/C/Y/W-135)
- NG decompression
- Stress steroids (100 mg IV hydrocortisone q8h), if received in past
- Treat infection
- Correct abn in blood coagulation

#### Monitoring
- Routine
- Large-bore IV access

#### Airway
- Trauma pts: rule out cervical instability

#### Induction
- Routine

#### Maintenance
- Prevent hypothermia

#### Extubation
- Routine

#### Adjuvants
- Open or laparoscopic approach
- Combined general/epidural (1.5–2% lidocaine with 1:200,000 epinephrine)
- Muscle relaxants required
- Minimize sedatives: ↑ likelihood of postop resp depression

### Postoperative Period
- Postsplenectomy sepsis: Due to encapsulated organisms (e.g., pneumococci)
- Booster pneumococcal vaccine 5–10 y post splenectomy

### Anticipated Problems/Concerns
- Bleeding
- Subphrenic abscess
- Bronchopneumonia
- Thrombotic complications
- Atelectasis (left lower lobe)
- Complications related to underlying cause for splenectomy
Split-Thickness Skin Graft

Robert Gaiser

Uses
- Burns: 2 million people injured/y
- Wounds: Trauma, diabetic foot ulcers, post-traumatic skin breakdown, sacral decubitus, melanoma excision

Perioperative Risks
- Periop mortality: Rare
- Large areas may involve significant blood loss
- May be performed by plastic, general, orthopaedic, and ENT surgeons

Worry About
- Airway involvement in burns that may make intubation difficult
- Blood loss if large areas to be debrided and grafted
- Pain at donor site
- Postop infection at donor or recipient site

Overview
- Split-thickness skin graft (STSG) consists of epidermis and only a portion of dermis. STSGs categorized as thin (0.005–0.012 in.), medium (0.012–0.018 in.), or thick (0.018–0.028 in.).
- Must consider both donor and recipient sites: Addition of epinephrine will decrease bleeding at donor site without affecting survival of STSG
- Involves the harvesting of only the epidermal layer and part of the dermis
  - Since dermal cells are left in situ from the donor site, may heal by 2° intention

Nutritional status of pt
- Cleanliness of donor and recipient sites
- Suitable locations for application of monitors and IV access
- Loss of joint mobility due to scarring

Addition of epinephrine to local anesthetic is not contraindicated.
- Donor site assoc with significant pain; plan should incorporate means for postop pain management
- ENLA cream has been used for anesthesia for donor site
- Application of lidocaine spray to donor site ↓ postop pain
- Acute phase: Anticipate hemostatic problems if large area is to be grafted; consider electrolyte abn and volume status
- Chronic phase: Scarring may cause loss of function and difficulty with positioning of the pt

Addition of epinephrine to local anesthetic may cause bleeding; hemostasis important for graft survival
- Craft placed on the wound, sutured around the periphery, and dressed. Main object of the dressing is to ensure contact between graft and host bed. Dressing left in place for ~7 d, at which time sutures can be removed.
- Reasons for graft failure: Inadequate graft bed (poor vascularity), hematoma, movement, infection, technical errors
- EBL: Depends on extent of grafting, minimal to 250–500 mL

Indications and Usual Treatment
- Used to cover granulating wound: Tolerates less vascularity than full-thickness skin graft
- Disadvantage: Abn pigmentation, contraction
- Donor site: Any area of body incl scalp and extremities; depends on resulting skin match and appearance of the donor scar

ICD-9-CM Codes: Procedure Code: 151.00; Diagnosis Code (dependent upon location): 707.00 (Decubitus ulcer); 707.10 (Ulcera on LE); 873.40 (Ulcera on face); 884.00 (Ulcera on UE); 941.00 (Burn on face); 942.00 (Burn on trunk); 944.00 (Burn on UE); 945.00 (Burn on LE)

Perioperative Management

Preoperative Preparation
- Determine donor and/or recipient sites
- Stabilize hemodynamics
- IV access: Large-bore IVs if anticipate large blood loss
- Warm room and all fluids

Monitoring
- Donor and/or recipient sites limit locations available for application of monitors: Secure leads and pulse oximeter away from sites used
- May use lower extremity for BP monitoring
- Temp monitoring

Airway
- For burn or postradiation pt, determine if airway involved. Involvement of face may make intubation more difficult: Consider fiberoptic intubation/consider collaborative, consultative airway management.

Induction/Maintenance
- Avoid succinylcholine, as may precipitate hyperkalemia response in pts with burns
- No generally accepted preferred agent or technique
- Regional anesthesia is an option
- Consider lateral femoral cutaneous nerve block if donor site is lateral thigh
- If using local anesthetic, keep amount within recommended limits.

Anticipated Problems/Concerns
- Surgical debridement may cause bleeding; hemostasis important for graft survival
- Craft placed on the wound, sutured around the periphery, and dressed. Main object of the dressing is to ensure contact between graft and host bed. Dressing left in place for ~7 d, at which time sutures can be removed.
- Reasons for graft failure: Inadequate graft bed (poor vascularity), hematoma, movement, infection, technical errors
- EBL: Depends on extent of grafting, minimal to 250–500 mL

Postoperative Considerations
- Activity level maintained at a minimum for first 2 d after surgery
- Monitor CV status
- Heat loss in transfer to and from OR

ASSESSMENT POINTS

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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>DERM</td>
<td>Donor/recipient site</td>
<td>Discussion with surgeon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEENT</td>
<td>Burns or radiation</td>
<td>Review medical record</td>
<td>Airway exam</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Hypotensive shock, arrhythmias</td>
<td>Dyspnea, orthopnea</td>
<td>Vital signs</td>
<td>CXR, ECG, orthostatic vital signs</td>
</tr>
<tr>
<td>RESP</td>
<td>ARDS</td>
<td>Dyspnea, SOB, chest pain, mental status change</td>
<td>Chest exam</td>
<td>CXR ABGs</td>
</tr>
<tr>
<td>GI</td>
<td>Debilitated pt</td>
<td>Bed-bound</td>
<td></td>
<td>Albumin</td>
</tr>
<tr>
<td>HEME</td>
<td>Possible large blood loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>ATN, myoglobinemia</td>
<td>Massive tissue destruction</td>
<td>Oliguria, anuria</td>
<td>BUN, serum Cr Cr clearance; serum/urine myoglobin</td>
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<tr>
<td>NEURO</td>
<td>Compartment syndromes</td>
<td>Hx of circumferential extremity burns</td>
<td>Weak pulses</td>
<td>Transduce compartment pressures</td>
</tr>
</tbody>
</table>

Stereotactic Neurosurgery

Risk
- Often performed as an awake procedure, esp. for DBS and brain tumor surgery
- Skill of anesthesiologist required keeping pt comfortable, responsive, and cooperative over a long period of time under optimal surgical conditions while rapidly diagnosing and treating potential complications
- Major risks: Hemorrhage, seizures

Perioperative Considerations
- Planned approach to select suitable pts, develop an anesthetic plan and deal with problems

Worry About
- Contraindications
  - Coagulation disorders
  - Coagulopathies: Bleeding diatheses, iatrogenic (heparin or coumadin)
  - Low plt count: PC <50,000/mL absolute contraindication, desirable to get PC <100,000/mL

Overview
- More appropriately called image-guided stereotactic surgery because coordinate system relates to specific points in CT/MRI/angiogram
- Most commonly used for biopsies of deep-located lesions and movement disorders, in which STN, Gpi, or VIM of the thalamus are targeted
- Mechanism of beneficial effect for movement disorders not fully understood, in high-frequency stimulation interference with several points along a pathway of synchronized oscillations in incriminated neuronal circuits
- “Awake” surgery in eloquent areas
  - For electrophysiological monitoring with MER (microelectrode recording) to guide device placement (unique pattern of spontaneous firing in specific brain areas), which make anesthetics undesirable and limit the options

Indications and Usual Treatment
- Biopsy, in areas near eloquent brain, also brain stem lesions, multiple small lesions (e.g., in some AIDS pts)
- Catheter placement for drainage of deep colloid cysts and abscesses, catheter placement for intratumoral chemotherapy, radiation brachytherapy, and hunt placement
- Placement of deep brain stimulation electrode for epilepsy, chronic pain, essential tremor
- Lesion generation for movement disorders: Parkinsonism, dystonia, hemiballismus, chronic pain, and epilepsy
- Others, frequently evacuation of intracerebral hemorrhage, stereotactic ‘radiosurgery’, locating a lesion for open craniotomy, transoral biopsy of C2 lesions, and experimental or unconventional applications such as aneurysm clipping

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<th>Test</th>
<th>Key References</th>
</tr>
</thead>
<tbody>
<tr>
<td>COAG</td>
<td>PC &lt;50,000 mL</td>
<td>Easy bruising, Hx of postop bleeding, anticoagulants</td>
<td>Petechia, bruises</td>
<td>Pt clout</td>
<td>Poon CCM, Irwin MG. Anaesthesia for deep brain stimulation and in patients with implanted neurostimulator devices. BJ/A. 2009;103:152–165.</td>
</tr>
<tr>
<td>CNS</td>
<td>Ability to cooperate</td>
<td>Medication, appearance</td>
<td>Anxiety, agitation</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Anesthetic Technique in General Anesthesia

• Pts who do not require awake technique or are uncooperative due to severe dyskinesia, pain, and anxiety
• Asleep-awake-asleep variant, preferably with LMA, alternatively spontaneous ventilation with or without nasal airway or ETT to increase pt tolerance, secure the airway and use hyperventilation
• Might be started before initial frame placement and imaging or afterwards with ↑ risk for intubation difficulties, consider rigid or flexible fiberoptic intubation
• TIVA or inhalational technique comparable
• Use of drugs according to possible testing, e.g., omitting muscle relaxants for motor testing
• General considerations and precautions for craniotomies

Surgical Stages

Localization with Imaging
• In or outside the OR
• Attachment of localizing device, e.g., frame, or less frequently frameless device
• Radiological imaging: Usually MRI, or CT, to identify targets

Orientation and Targeting
• In scan suite or OR
• Orientation within the same coordinate system and evaluation of the images

Image Guided Neurosurgery
• Usually in the OR
• Via burr hole or (mini-) craniectomy
• Uni- or bilaterally
• Passing microelectrodes, biopsy needles etc. along their trajectory towards the target

• In certain awake procedures clinical testing for side effects, improvement of symptoms, and neurologic exam and/or electrophysiological recordings to fine-tune the device placement with oscilloscope and audio monitors in superimposing scaled drawings on a brain atlas at each depth
• Securing device, connection to tunnelled leads to implanted generator in chest or abd, potentially performed as a second-stage surgery and closure

Postoperative Considerations
• Invariably radiologic confirmation with CT scan
• 30° head up position to improve ventilation, reduce edema and facilitate cerebral venous and CSF drainage
• Postop monitoring, usually on ICU or OU for neurologic testing, also after ‘awake’ procedures
• Antiparkinson medication ASAP to avoid motor fluctuation and resp problems
• Avoid sedative opioids (such as morphine or hydromorphone) for pain relief, use fentanyl or codeine instead
• Specific screening for potential CNP (cranial nerve palsies), e.g., nerve X with vocal cord palsy, worsening of OSA, which might cause breathing difficulties, increasing risk of aspiration

Anticipated Problems/Concerns
• Resp complications in “awake” procedures
  • Resp depression due to oversedation
  • Mechanical obstruction with difficulties to access the airway or shifting of the body which might require release from the frame
  • LOC with resp depression due to acute intracranial hemorrhage or seizures
  • Pre-existing resp depression, e.g., due to stiffness of resp muscles in Parkinson pts, restrictive pulm disease, and ↑ sensitivity to drugs
  • CV complications in “awake” procedures
  • Arterial hypotension during positioning (orthostasis) and induction of GA
  • Higher incidence of intracranial hemorrhage with high intraop BP
  • Neurologic complications in “awake” procedures
  • Sudden onset of focal deficits, usually not requiring anesthesiologic intervention
  • Seizures, mostly focal, rarely tonic-clonic
  • Agitation and anxiety
  • Excessive pain and discomfort
  • Acute LOC
  • Air embolism (rare)
  • N/V
  • Pharmacologic complications due to drug-drug interactions with antiepileptic medication, altered sensitivity to drugs in Parkinson pts, and LA toxicity
  • Specific demands for skills to communicate well with pt and surgical team esp. in awake procedures and to titrate anesthetics to keep pt comfortable and communicative under optimal surgical conditions and monitoring
Strabismus Repair

Overview

- Strabismus is the misalignment of the visual axes.
- Repair involves manipulation of extraocular muscle lengths or insertion sites to improve visual alignment.
- Adjustable sutures are sometimes placed in adults to allow for minor corrections later.

Anesthetic Goals

- General goals are to provide adequate analgesia, prevention of PONV, safely manage the oculocardiac reflex, and provide sufficient analgesia.
- Oculocardiac reflex (OCR)
  - Usually causes sinus bradycardia +/- hypotension, but can lead to more serious arrhythmias (junctional, ectopic beats, PVCs, ventricular fibrillation, asystole).
- Iatrogenic ocular injury
- Strabismus can be a sign of an as yet undiagnosed underlying myopathy

Overview

- Strabismus is the misalignment of the visual axes.
- Repair involves manipulation of extraocular muscle lengths or insertion sites to improve visual alignment.
- Adjustable sutures are sometimes placed in adults to allow for minor corrections later.
- Anesthetic goals are to provide adequate aikeness, prevention of PONV, safely manage the oculocardiac reflex, and provide sufficient analgesia.
- Oculocardiac reflex (OCR)
  - Usually causes sinus bradycardia +/- hypotension, but can lead to more serious arrhythmias (junctional, ectopic beats, PVCs, ventricular fibrillation, asystole).

Indications and Usual Treatment

- Diplopia
- Marked esotropia
- Compensatory head posture
- Surgical repair most successful when undertaken in early childhood to prevent development of amblyopia

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</tr>
</thead>
<tbody>
<tr>
<td>HFENT</td>
<td>Assoc syndromes with possible difficult airway</td>
<td>Snoring/sleep apnea</td>
<td>Airway</td>
<td>Hx</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Assoc congenital heart disease possible</td>
<td>SOB, poor growth, birth, and gestational Hx</td>
<td>CV exam</td>
<td>ECHO may be indicated</td>
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<tr>
<td>RESP</td>
<td>Bronchopulmonary dysplasia if premature</td>
<td>Birth and gestational Hx</td>
<td>Chest exam, feeding Hx</td>
<td>O₂ saturation</td>
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<tr>
<td>GI</td>
<td>PONV</td>
<td>Past surgical history</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NM</td>
<td>Myopathy</td>
<td>Fatigue, weakness</td>
<td>Weakness, spasticity</td>
<td></td>
</tr>
</tbody>
</table>

Perioperative Management

Preoperative Preparation

- Assess for congenital abn or co-existing morbidities
- Be aware of systemic effects of topical ophthalmic drugs
- Standard NPO guidelines
- IM atropine decreases incidence of OCR from 90% to 50%

Monitoring

- Routine
- Close attention to ECG for OCR

Anesthetic Technique/Induction

- GA for pediatric pts
- For cooperative adults, consider local + regional (peri/tetrobular blocks)
- Oral RAE ETT required in most cases; may be performed with LMA
- Hypercarbia and hypoxia increase incidence and worsen severity of OCR
- Position and secure ETT or LMA with care: pt turned 90° away from anesthesiologist

Surgical Stages

Induction

- Sevoflurane inhalational induction for children, or propofol induction in older pts or those with IV access
- Succinylcholine, which causes sustained extraocular muscle contraction, is relatively contraindicated due to potential interference with surgeon’s forced duction test
- Ondansetron, dexamethasone, metoclopramide, or droperidol (with due consideration of black-box warning), or a combination of these, for PONV prophylaxis
- Maintain adequate muscle relaxation for duration of surgical manipulation, to prevent eye injury from pt movement
- Maintenance with propofol assoc with less PONV

Postoperative Considerations

- Pain is variable
- Analgesia with ketorolac, acetaminophen, or narcotics (recognizing that narcotics may contribute to PONV)
- Treat PONV with ondansetron or metoclopramide
- Usually outpatient surgery, so ambulatory practices and goals are recommended

Anticipated Problems/Concerns

- Must communicate effectively with surgeon preop regarding anesthesia plan, and inraop regarding effects of oculocardiac reflex
- PONV causes significant pt discomfort and dissatisfaction, and can lead to dehydration, delayed discharge, or unanticipated admission.

- Don’t treat phenylephrine-induced Htn with beta-blockers, which are assoc with development of pulm edema and cardiac failure; rather treat by deepening anesthesia.
- Almost no blood loss or fluid shifts

Testicular Torsion Surgery

Risk
- Incidence 1/160 males; adolescents, less commonly in neonatal period
- May follow testicular trauma or strenuous physical activity (20%)

Perioperative Risks
- Dependent only on baseline health of pt

Worry About
- N/V from severe pain and opioid administration
- Full stomach: Aspiration of gastric contents during induction of general anesthesia

Testicular Torsion Surgery

Overview
- Presents as acute scrotal pain and results from twisting of spermatic cord with vascular compromise of testicle
- Temporizing treatment involves manual detorsion by a urologist, which may alleviate ischemia, but surgical orchidopexy still required
- Often confusing diagnosis; color Doppler US considered to be test of choice

Overview
- Testicular ischemia depends on time from diagnosis to surgery: 6 hr critical time frame
- Pt anxiety

Indications and Usual Treatment
- Treatment is surgical de-torsion and subsequent orchidopexy; highly successful if performed within 6 hr of onset of pain
- Success less than 5% if performed after 24 hr

ASSESSMENT POINTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Minimal</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Systemic signs of severe pain</td>
<td>Systemic</td>
<td>Htn, tachycardia</td>
<td>Vitals assessment</td>
</tr>
<tr>
<td>RESP</td>
<td>Minimal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL/GU</td>
<td>Minimal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Systemic signs of severe pain</td>
<td>Dizziness or nausea</td>
<td>n/a</td>
<td>US can show decreased blood flow to affected testes.</td>
</tr>
</tbody>
</table>


Anesthetic Technique/Induction
- Rapid sequence induction for general endotracheal anesthesia
- Spinal anesthesia also appropriate if pt prefers
- Local infiltration with minimal systemic sedation possible in selected cases
- Scrotum innervated by inferior pudendal branch (long scrotal nerve) of posterior femoral cutaneous nerve (from the sacral plexus), and the medial and lateral posterior scrotal branches of peroneal nerve (from the pudendal nerve)

Surgical Stages
- Small incision in scrotum performed
- Testis is isolated and untwisted; removed if found to be without blood flow by intraop flow Doppler study
- Bilateral orchidopexy usually performed to prevent recurrence

Postoperative Considerations
- Minimal pain after torsion corrected
- Underlying pt condition will play a larger role in determining postop care

Anticipated Problems/Concerns
- Full stomach: Risk of pulm aspiration with induction of GA
- Testicular ischemia if duration of torsion prolonged (>6–8 hr)
Tetralogy of Fallot, Correction of

Veronica C. Swanson
Norah Janosy

Risk
- CHD incidence < 1% of live births
- TOF accounts for 10–15% of total CHD

Perioperative Risks
- Operative mortality <5% for children without RV failure or pulm Ateria
- Major perioperative risk factors after repair: Major pulm artery anomaly, other major cardiac defect, very young age, increased Hct, absent pulm valve, major AP collaterals
- 85% 10-y survival with surgery
- 90% mortality by the age of 21 y without surgery

Worry About
- Degree of R → L shunt and hypercyanosis (end-organ damage)
- Arterial hypoxemia
- Venous air embolus and ↑ likelihood of paradoxical embolism
- Polyctheremia and thrombotic events

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
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<tbody>
<tr>
<td>CARDIO</td>
<td>R → L shunt</td>
<td>Cyanotic spells, relation to crying or exercise</td>
<td>Observe for clubbing, squatting during tet spell, and cyanosis</td>
<td>ECHO, catheterization pulse oximetry (O₂ sat 80–90% room air)</td>
</tr>
<tr>
<td></td>
<td>RV failure</td>
<td>Exercise intolerance, SOB, syncope</td>
<td>↑ JVP, tachypnea, hepatosplenomegaly (late)</td>
<td>ECG: RV hypertrophy, right axis deviation</td>
</tr>
<tr>
<td>Polycythemia, prior palliative shunt</td>
<td>CVA (increased risk)</td>
<td>Neurologic exam, look for scars, absent peripheral pulses</td>
<td>Hct</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Decreased pulm blood flow</td>
<td>Exercise intolerance</td>
<td>Tachypnea, clubbing</td>
<td>Catheterization, ECHO, expect low O₂ sat</td>
</tr>
<tr>
<td>OTHER</td>
<td>Developmental and growth delay</td>
<td>Developmental and growth delay</td>
<td>Small size, test for developmental delay</td>
<td>Variety of measures</td>
</tr>
<tr>
<td></td>
<td>Assoc congenital anomalies</td>
<td>Thorough Hx of all systems</td>
<td>Thorough exam of pertinent systems</td>
<td>Variety of tests</td>
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Perioperative Management

Monitoring
- Arterial catheter: If pt has a palliative systemic artery-to-pulm shunt, arterial line should go to other side
- TEE for evaluation pre- and post-repair
- Central venous line for inotropes and pressure monitoring; Scrupulous attention to avoid air bubbles in lines
- Standard ASA monitors

Airway
- Avoid excessive airway pressures (may ↑ PVR)
- Airway concern if velocardiofacial syndrome

Induction
- Premedication is important and may incl mida- zolam, morphine, and ketamine.
- Ketamine a good choice: Does not ↑ PVR. Also, ↑ SVR effect overshadows any ↑ in contractility (as in infundibulum), therefore improving cyanosis.
- Fentanyl a good choice: Slow HR and decreased catecholamine release.
- Sevoflurane for inhalation induction; use caution to avoid ↓ in SVR assoc with inhaled agents—less of a problem in younger pts.
- Ensure adequate hydration: Special risk in severely polycthermic children
- Anesthetic technique

Choice of maintenance drugs should seek to avoid ↑ HR and myocardial contractility, and ↓ SVR
- Specific drug choices not important. Vigilance and responsiveness is.
- Phenyolphrine diluted and ready for possible tet spells.
- Heparin drawn up in the event the tet spell does not reverse with treatment—urgent crash onto bypass.

Post Cardiopulmonary Bypass
- Assess components of repair: Regurgitant flow through the pulm valve, residual VSD via TEE
- Assess RV function: RV pressure < or = ½ LV pressure.
- For struggling RV: Fluid load, inotropic support, afterload reduce pulm circulation (milrinone)
- The greater the RVH and RV failure preop, the higher the inotropic requirements and filling pressures postop.
- Common arrhythmias: Functional ectopic tachycardia (JET)—cooling, overdrive pacing, ↑ depth of anesthesia, amidarone—and heart block—temporary or permanent pacemaker.

Postoperative
- Ensure adequate RV function and low PVR
- Early extubation if possible

Anticipated Problems/Concerns
- Persistent RV dysfunction
- Persistent RV outflow tract obstruction
- Persistent VSD
- Postop bleeding
- Persistent R → L shunt (VSD)
- Most postop mortality found in pts with acute RV failure

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## Thoracic Aortic Repair

### Risk
- Incidence in USA: 3.4% incidence of aortic aneurysm; 26% involve thoracic aorta (TAA)
- Untreated TAA greater than 10 cm confers a 2-y survival rate of <30%; ½ of these deaths are due to aneurysm rupture
- Increased incidence with Htn
- M:F ratio: 2.9:1
- Peak incidence 50–70 y

### Perioperative Risks
- Htn, coronary and carotid vascular disease
- Untreated dissection: 25–35% mortality within 24 hr, 90% in 3 mo
- Surgical repair carries 10% mortality. Causes of postop death are hemorrhage (29%); cardiac events—Ischemia, MI, CHF (26%); and multiorgan system failure (22%).
- Traumatic disruption immediately fatal in 85%.

### Worry About
- Elevated HR and BP promote dissection
- Acute dissection can lead to compromised blood flow (coronary, renal, splanchnic, spinal cord), pericardial tamponade, acute aortic valvar insufficiency, or bleeding
- Assoc injuries (lungs, heart, head, abd) with traumatic rupture
- Major blood loss; need for rapid transfusion intraop
- Acute dissections frequently brought for repair with minimal preop preparation

### Overview
- Primary risk factors are Htn, tobacco use, and atherosclerosis
- Thoracic aortic aneurysms 2–3 × more likely to dissect than abd
- Group B (type III) dissections are medically managed; when operation is indicated (>10 cm diameter, progressive enlargement, or producing symptoms), left thoracotomy and one-lung ventilation employed
- Traumatic aortic dissections are treated surgically regardless of symptoms

### Endovascular stenting is gaining in popularity for elective TAA repair; this approach is assoc with fewer short term complications (myocardial ischemia, renal failure, resp complications); long-term outcomes still being assessed.

### Indications and Usual Treatment
- Group A (types I and II) dissections are surgically repaired immediately via median sternotomy using cardiopulmonary bypass (CPB) and deep hypothermic circulatory arrest (DHCA)
- Group B (type III) dissections are medically managed; when operation is indicated (>10 cm diameter, progressive enlargement, or producing symptoms), left thoracotomy and one-lung ventilation employed
- Traumatic aortic dissections are treated surgically regardless of symptoms

### Overview
- Group B (type III) dissections are medically managed; when operation is indicated (>10 cm diameter, progressive enlargement, or producing symptoms), left thoracotomy and one-lung ventilation employed
- Traumatic aortic dissections are treated surgically regardless of symptoms

### Procedures

#### Locations and DeBakey/Stanford Types

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<tr>
<th>Location</th>
<th>DeBakey</th>
<th>Stanford</th>
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<tbody>
<tr>
<td>Ascending and descending aorta</td>
<td>Type I</td>
<td>Group A</td>
</tr>
<tr>
<td>Ascending aorta only</td>
<td>Type II</td>
<td>Group A</td>
</tr>
<tr>
<td>Distal to left subclavian</td>
<td>Type III</td>
<td>Group B</td>
</tr>
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</table>

### Perioperative Implications

#### Preoperative Preparation
- IV sodium nitroprusside with β-blockers (consider calcium channel blockade if β-blockers contraindicated)
- Preop statins and discontinuation of tobacco use if elective
- Premedication to prevent anxiety and pain
- Early, prompt reduction of shear stress (dP/dt) indicated to reduce chance of rupture

### Monitoring
- Invasive arterial BP—left radial for group A, right radial for group B, and femoral or dorsalis pedis for distal aortic pressure measurement during crossclamping
- PA catheter and/or TEE
- SSEPs not consistently reliable guide to spinal cord ischemia

### Airway
- Difficult airway if compression or deviation of tracheobronchial tree
- May interfere with placement of double-lumen ET tube

### Induction/Maintenance
- High-dose narcotic useful to blunt intubation response
- Inhalation agent useful to ↓ myocardial contractility, provide amnesia
- Avoid N₂O during one-lung ventilation and to prevent expansion of air emboli

### ASSESSMENT POINTS

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<tbody>
<tr>
<td>HEENT</td>
<td>SVC syndrome, Recurrent laryngeal nerve compression</td>
<td>Dyspnea, Hoarseness</td>
<td>JVD edema</td>
<td>Flow-volume loop, Indirect laryngoscopy</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Myocardial ischemia, LV dysfunction, Aortic root dilatation, Valvular disease, Venous compression</td>
<td>Angina, Dyspnea, Dyspnea</td>
<td>S, gallop, Friction rub, Early diastolic murmur, Muffled heart sounds, Pethora</td>
<td>ECG/Echo, Stress testing, TTE/TEE, Coronary angio</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchial/tracheal compression, Recurrent pneumonia, Pulm compression</td>
<td>Dyspnea, Cough</td>
<td>Wheezing, Tracheal deviation, Hemoptysis</td>
<td>ABGs, Flow-volume loop, CXR, Chest CT or MRI</td>
</tr>
<tr>
<td>GI</td>
<td>Mesenteric ischemia</td>
<td>Abd pain, Bloody diarrhea</td>
<td>Tenderness</td>
<td>Colonoscopy, Angio</td>
</tr>
<tr>
<td>RENAL</td>
<td>↓ Renal perfusion</td>
<td>Oliguria</td>
<td>Cr, Cr clearance</td>
<td>MRI, EMG, Carotid duplex</td>
</tr>
<tr>
<td>CNS</td>
<td>Spinal cord ischemia, Carotid stenosis</td>
<td>Weakness, Paraplegia</td>
<td>MRI, EMG, Carotid duplex</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Back or chest pain</td>
<td></td>
<td>Chest CT or MRI</td>
<td></td>
</tr>
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</table>

### Key Reference
Extubation
- Usually keep sedated and mechanically ventilated until warmed and stable
- Neurologic status important to assess following deep hypothermic circulatory arrest
- Prevent hemodynamic response to extubation

Adjuvants
- β-blockers, nitroprusside, nitroglycerin continued periop
- Maintain adequate hydration
- Questionable renal benefit: Mannitol 0.5 g/kg well prior to crossclamping; “renal dose” dopamine, fenoldopam

- Maintenance of aortic pressure distal to cross-clamp; mild hypothermia; CSF drainage; steroids, intrathecal papaverine may limit spinal cord ischemia (3–7% incidence)

Anticipated Problems/Concerns
- Aortic cross-clamping causes ↑ LV afterload, LV wall tension, and O2 consumption. Vasodilators useful, but distal hypotension may induce spinal cord ischemia. Vascular shunting or partial bypass during cross-clamp may overcome these effects.
- Aortic unclamping results in ↑ stroke volume but profound ↓ in SVR and significant metabolic acidosis if no shunt used. Fluid management, α-agonists (phenylephrine), and sodium bicarbonate may be useful.
- CPB and DHCA are assoc with increased rates of complications incl coagulation abn, paraplegia, CNS injury, stroke, myocardial ischemia, renal failure, and resp complications.
Thyroidectomy (Open or Minimally Invasive) for Hyperthyroidism

Michael F. Roizen

Overview
- Major goal is to avoid thyroid storm; if not euthyroid prior to surgery, try to delay operation
- If emergency operation, use β-blockers and iodides to ↓ periop effects of released thyroid hormones and ↓ further synthesis and release of hormones; keep in ICU until risk of storm has passed
- Done in young adults with hyperthyroidism, or normal thyroid function with a cold nodule, or with a goiter that is bothersome physiologically or psychologically
- Hyperthyroidism is an endocrinopathy with CV disease—tachycardia (commonly idiopathic if no prior Dx of hyperthyroidism), CHF, dysrhythmias (AFIB)—as a major manifestation
- Other target systems of hyperthyroidism are resp and CNS (↓ drive to breathe, anxiety, psychoes) and metabolic

ASSESSMENT POINTS

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<tbody>
<tr>
<td>HEENT</td>
<td>Weakened tracheal rings, distorted/displaced trachea Ophthalmonaphy Large tongue if associated with goiter or amyloidosis</td>
<td>Snoring, hoarseness, neck pain</td>
<td>Ask to vocalize “e”; examine airway and neck, look at eyes</td>
<td>CXR (PA and lat), lat neck films, CT scan of neck, US of neck</td>
</tr>
<tr>
<td>CARDIO</td>
<td>CHF, cardiomyopathies Sinus tachycardia, mitral valve prolapse, AFib</td>
<td>DOE, orthostatic SOB palpitations, ↑ HR during sleep</td>
<td>Standard exam</td>
<td>Rhythm strip or full ECG</td>
</tr>
<tr>
<td>GI</td>
<td>Wt loss, diarrhea, dehydration</td>
<td>Dizziness on arising, Hx of diarrhea, constipation</td>
<td>Skin turgor, orthostatic VS</td>
<td>↑ Serum alkaline phosphatase</td>
</tr>
<tr>
<td>HEME</td>
<td>Mild anemia, thrombocytopenia Agranulocytosis 2° to propylthiouracil or methimazole</td>
<td></td>
<td>Skin/mucous membranes for infection/petechie</td>
<td>CBC with plt count, differential</td>
</tr>
<tr>
<td>CNS</td>
<td>Shaking, anxiety, emotional lability Hypothyroid goiter assoc with slow thought processes</td>
<td></td>
<td>Reflex speed, tremor, nervousness, mental status</td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Need to assess if euthyroid Malnourished</td>
<td>Reflex speed, tremor, heat intolerance, fatigue, weakness; wt loss, anorexia, or ↑ appetite</td>
<td>Reflex speed, HR</td>
<td>Free T₄</td>
</tr>
</tbody>
</table>


Perioperative Implications

Anesthetic Technique
- No one best technique

Airway
- Occasionally distorted anatomy 2° to goiter, tracheal ring involvement, inflammation 2° to thyroiditis
- Consider awake fiberoptic intubation
- Consider armored tube or equivalent if tracheal rings are affected

Preinduction/Induction
- Prehydrate if CV status tolerates
- Routine unless abn airway or CV system or noneuthyroid condition

Monitoring
- Temp (also place cooling blanket on OR table to treat thyroid storm if it occurs)
- Consider invasive monitoring if CV system severely affected
- If considerable head-up position, consider air embolus monitoring and therapy strategies
- If done with minimally invasive surgery, can monitor for pneumothorax with entidial CO₂ and with portable ultrasound

Induction/Maintenance/Exubation
- Exubate with optimal conditions for reintubation

Surgical Stages

Initial Dissection
- Can approach from axilla if minimally invasive approach or robotic minimally invasive approach (majority at Cleveland Clinic for Hyperthyroidism are now done with this approach)
- Transverse collar incision if open approach
- Thyroid lobe freed from strap muscles with securing of superior thyroid vessels; these are clamped after ensuring localization and preservation of recurrent laryngeal nerve and parathyroid glands in either open or minimally invasive approach

Thyroid Removal
- Following division of middle and inferior thyroid vessels, thyroid lobe is retracted medially and liberated

ICD-9-CM Codes: 242.9 (Hyperthyroidism [thyrotoxicosis]); 242.0 (Graves’ disease); 245 (Thyroiditis); 193 (Malignant thyroid disease)

Etiology
- Multinodular diffuse enlargement (Graves’ disease)
- Thyroid adenoma—toxic multinodular goiter (firm gland) later in life and almost never malignant; unilateral solitary nodule with autonomous function earlier in life almost always benign
- Cold nodules assoc with radiation therapy of other diseases as well as idiopathic
- Goiter assoc with iodine deficiency

Adjuvants
- Usually no requirement for NMB
- Can be done with regional: Superficial and deep cervical plexus blocks and infiltration

Postoperative Considerations
- EBL: 50–150 mL
- Pain score: 2–4
- Usually can be treated with NSAIDs or occasionally with PCA

Anticipated Problems/Concerns
- Thyroid storm is life-threatening illness manifested by hyperpyrexia, tachycardia, striking alterations in consciousness
- Bleeding can compromise airway function
- Recurrent laryngeal nerve injuries damage abductor fibers, resulting in hoarseness. Bilateral injury results in fixed narrow opening to glottis with inspiratory airflow obstruction (stridor), inability to vocalize, aspiration risk, and immediate need for tracheal intubation.
TMJ Arthroscopy

Overview
- Used to evaluate and treat pain or lack of motion in TMJ
  - TMJ anatomy: Joint divided into superior and inferior articular cavities by articular disk
  - Approaches: Inferolateral, posterolateral, anterolateral
  - Structures to watch: facial nerve, superficial temporal branch of auriculotemporal nerve, maxillary artery, superficial temporal artery and vein, parotid gland

ICD-9-CM Code: 524.6

Perioperative Risks
- Periop mortality exceedingly rare
- Epistaxis

Worry About
- Co-existing diseases (e.g., rheumatoid arthritis)
- Establishing and maintaining airway
- Medications

Indications and Usual Treatment
- Diagnosis of TMJ pain
- Surgery
  - Biopsies
  - Debridement and lavage
  - Incision of adhesions
  - Restoration of disk mobility and position
  - Instillation of medication
  - Capsular or disk attachment scarification/plication
- Usual treatment
  - Behavior modification
  - Pharmacotherapy
  - Physical therapy
  - Appliance therapy
  - Occlusive therapy

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Perioperative Management

Preoperative Preparation
- IV sedation if needed
- Standard

Induction/Maintenance
- After standard induction, prepare nostrils with 4% cocaine or phenylephrine and dilate with lubricated nasal airways of increasing diameter
- Nasotracheal intubation (soften NT tube in warm H₂O; may need Magill forceps)
- Cover eyes with moistened eye pads (possible laser use)
- Some surgeons request IV administration of dexamethasone (10 mg)

Surgical Stages
- Injection of local anesthesia: Usually 1% lidocaine with 1:100,000 epinephrine
- Incision over superior joint space, insertion of obturator and/or sheath, removal of obturator, camera attachment
- Use of helium or Nd:YAG laser
  - Protective eyewear for pt and staff
  - Wet towels surrounding operative area
  - Water readily available
- Complications (<1%)
  - Hemorrhage (usually superficial temporal artery or vein)
  - Joint damage
  - Perforation into middle cranial fossa
  - Damage to middle ear ossicles
  - Injury to auriculotemporal nerve
- Minimal blood loss
- Some surgeons inject intra-articular steroids or local anesthetics (bupivacaine 0.5% with 1:200,000 epinephrine) for anti-inflammatory and analgesic effects

Postoperative Considerations
- Pain score: 0–5
- IV or PO narcotics/anti-inflammatory medications in PACU

Anticipated Problems/Concerns
- Epistaxis with intubation or extubation
- Postextubation pulm edema reported
- IV injections of local anesthetic

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Perioperative Risk
- Periop mortality exceedingly rare
- Epistaxis
Tonsillectomy and Adenoidectomy

**Risk**
- One of the most common surgical procedures in children
- Incidence varies by region and country; continues to decrease since 1970
- Racial and gender predominance: None
- Bimodal peaks at ages 4 and 16–17
- Most available data is in pediatric pts

**Perioperative Management**

**Perioperative Preparation**
- Avoid preanesthetic sedatives if Hx of sleep apnea or very large tonsils
- Consider moderate dose of anxiolytic to ease induction
- Evaluate for bleeding abn; stop meds that interfere with coagulation
- Pre- or interop acetaminophen

**Monitoring**
- IV prior to induction if Hx of significant airway obstruction

**Airway**
- Preferred RAE ETTs fit best into groove of mouth gag
- Use cuffed tubes when possible
- LMA may also be used in older children and adults

**Induction**
- Consider atripine (0.02 mg/kg, max 1 mg) during induction

**Surgical Stages**
- Placement or removal of mouth gag may dislodge or obstruct ETT
- Assoc URI increases the risk of resp complications and bleeding
- Obstructive sleep apnea (OSA) severity (as measured by a formal sleep study or overnight oximetry) predicts the nature of periop resp complications
- Bleeding, airway obstruction, apnea, and pain postop

**Indications and Usual Treatment**
- Obstruction of nasal or pharyngeal airway, esp. when assoc with anatomic or physiologic disturbances
- Adenotonsillectomy is currently the treatment of choice for surgical treatment of OSA in children, although there is no strong evidence to support this.
- Chronic or recurrent infection of adenoids (also ears or sinuses) or tonsils despite adequate antibiotic therapy
- Acute peritonsillar abscess

**Assessment by Hx**
- Placement or removal of mouth gag may dislodge or obstruct ETT
- Consider atropine (0.02 mg/kg, max 1 mg) during induction
- LMA may also be used in older children and adults
- Use cuffed tubes when possible
- Most available data is in pediatric pts

**Overview**
- Adenotonsillectomy and tonsillectomy usually performed together, but consideration given to the specific risk-benefit ratio for each procedure
- Bleeding may occur in first 24 hr (1°) or 7–10 d (2°)
- Age and co-morbidities of pts dictate postop care

**Worry About**
- Indication for procedure: Usually airway obstruction (81% of pts <3 y) or recurrent infection
- Several operative methods exist; each is assoc with different risks of postop pain and hemorrhage
- ICD-9-CM Codes: 474.0 (Chronic tonsilitis); 474.1 (Hypertrophy of tonsils and adenoids)

**Assessment Points**

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<td>Chronic nasal obstruction assoc with abn facial growth</td>
<td>Snoring, emesis, speech disorders; cognitive deficits</td>
<td>Adenoid facies, mouth breathing, obesity</td>
<td>Ask child to breathe with mouth closed</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Chronic airway obstruction may lead to pulm Htn and right heart failure</td>
<td>Exercise intolerance</td>
<td>Cardiac exam: Loud 2nd heart sound</td>
<td>ECHO, ECG, CXR if positive history/exam</td>
</tr>
<tr>
<td>RESP</td>
<td>Tonsillar hyperplasia may result in sleep apnea and CO₂ retention</td>
<td>Disturbed sleep, daytime sleepiness</td>
<td>Airway exam, tonsil size</td>
<td>Polysomnography, O₂ sat</td>
</tr>
<tr>
<td>HEME</td>
<td>Pts with pre-existing bleeding disorders are at greater risk of postop hemorrhage</td>
<td>Patient or family Hx of bleeding, bruising, or aspirin (check OTC meds)</td>
<td>Multiple bruises above the knees</td>
<td>PT, PTT, plt count, bleeding time if positive Hx</td>
</tr>
<tr>
<td>NEURO</td>
<td>Brainstem dysfunction may amplify sleep apnea with moderate tonsillar hyperplasia</td>
<td>Cerebral palsy, Arnold-Chiari malformation</td>
<td>Polysomnography, O₂ sat</td>
<td>Polysomnography, O₂ sat</td>
</tr>
<tr>
<td>SYSTEMIC</td>
<td>Craniofacial abn (e.g., trisomy 21, Treacher Collins may have pre-existing airway narrowing), NM disorders, mucopolysaccharidoses, obesity</td>
<td>General exam</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total Abdominal Hysterectomy

**Risk**
- Hysterectomy is the second most commonly performed surgical procedure in USA (cesarean delivery is first)
- In 2003, a rate of 5.38/1000 females for benign disease (~550,000, total ~600,000 with all diagnoses)
- By age 40.9, 25% of American women will undergo hysterectomy
- Racial predominance: None
- Age: 30 + y

**Perioperative Risks**
- Overall mortality ~0.1%; lowest mortality in women ≤55 y, greatest mortality in women >75 y
- Periop morbidity rare; overall incidence of 7.5% (fever and wound infections most common)
- Reduce surgical site infection with preop (within 60 min of incision) IV antibiotics
- Recommend routine VTE prophylaxis

**Worry About**
- Femoral nerve injury
- Bladder and/or ureter injury

### Assessment Points

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Dehydration 2° to bowel prep Blood loss from primary problem</td>
<td>Menorrhagia</td>
<td>Orthostasis</td>
<td>High/Het Electrolytes</td>
</tr>
<tr>
<td>RESP</td>
<td>Rule out effusion or metastases</td>
<td>SOB, bronchospasm</td>
<td>Auscultation</td>
<td>CXR ABGs + PFTs as indicated</td>
</tr>
</tbody>
</table>


**Perioperative Management**

**Anesthetic Technique**
- Regional, general, or combined techniques are options. Regional and/or combined techniques allow for postop regional analgesia.

**Monitoring**
- Routine
- Consider arterial line, central monitoring for extensive oncologic procedure if indicated by surgical plan and/or skill or by co-existing disease

**Induction/Maintenance**
- GA: Abdominal manipulation makes ET intubation preferable to LMA or mask airway
- Regional anesthesia: Dense T3–T4 level necessary for pt comfort. Avoid in obese pts or those who might otherwise not tolerate high regional levels.

### Indications and Usual Treatment
- Most common indications are fibroids and dysfunctional uterine bleeding
- Indications for hysterectomy incl:
  - Uterine, cervical, ovarian cancer
  - Pelvic relaxation syndrome, fibroids, abs bleeding, endometriosis, or other benign disorders (usually via vaginal approach)
- Route of hysterectomy may be influenced by uterine or adnexal size, presence of adhesions, malignancy, pt body habitus, and surgeon skill relative to the technique
- In 2003, abd hysterectomy was performed in ~66% of cases. This percentage is decreasing rapidly as the use of robotic technology and improved surgical techniques with both laparoscopic and robotic techniques improve.

### Anticipated Problems/Concerns
- Older pts, obese pts and oncology pts at greater risk for DVT
- Bladder and/or ureteral damage may prolong urethral catheterization and/or hospitalization
- Recognition of femoral nerve injury may be delayed by use of regional anesthesia—consider epidural opioid analgesia without local anesthetic postop

**Surgical Stages**

**Incision**
- Pfannenstiel or low-transverse incision: Limited access to upper abdomen
- Midline incision extending from ~4 cm above symphysis pubis to umbilicus offers greater exposure, important for oncologic procedure

**Inspection**
- Avoid excessive bowel manipulation
- Self-retaining retractor may be used

**Dissection**
- Intraperitoneal hysterectomy
- Intimate proximity of uterus to ureters makes ureteral identification and dissection important
- Bladder must be carefully advanced down lower uterine segment
- EBL: <1500 mL

**Postoperative Considerations**
- Moderate-severe postop pain (pain score 5–7)
- Single-dose spinal opioid followed by IV PCA for 12–24 hr (36–48 hr for oncologic procedures)
- Nononcologic pts usually taking oral medication on operative day or PO day 1
- High incidence of postop N/V

**ASSESSMENT POINTS**

- Hemorrhage requiring transfusion
- VTE

**Overview**
- Choice of TAH, total vaginal hysterectomy (TVH), laparoscopic or robotic surgery made according to pelvic anatomy, uterine size, adenexal or other assoc disease (i.e., malignant versus benign)
- Technique for TAH varies according to indication for operation; may incl oophorectomy, lymph node dissection, omentectomy, or tumor debulking
- Choice of anesthetic will vary with indication for surgery as well as presence of co-existing disease

**ICD-9-Codes:** 826.8 (Dysfunctional uterine bleeding); 179 (Uterine neoplasm, malignant); 219.9 (Uterine neoplasm, benign); 218.9 (Uterine fibroid); 180.9 (Cervical neoplasm, malignant); 219.0 (Cervical neoplasm, benign); 220.x (Ovarian neoplasm, benign); 614.6 (Pelvic adhesions); 617.9 (Endometriosis)
Total Anomalous Pulmonary Venous Return Correction

**Risk**
- 1–3% of all congenital CV abn
- Marked male predominance

**Perioperative Risks**
- Periop mortality is rare (<3%)
- Symptomatic arrhythmias are infrequent
- Cyanosis, impaired systemic O\textsubscript{2} delivery
- Myocardial dysfunction

**Worry About**
- Obstructed form typically presents in neonatal period with cyanosis, tachypnea, heart failure, and alveolar edema 2° to pulm venous Htn
- May be assoc with other cardiac anomalies

**Overview**
- All pulm veins drain abn to right atrium (RA), directly or indirectly, via remnants of cardinal or umbilical venous system

- Classification based on anatomic site of abn connection
  - Type 1: supracardiac (49%), most commonly enters left innominate vein
  - Type 2: cardiac (25%), connection to coronary sinus
  - Type 3: infracardiac (18%), distal site of connection usually below diaphragm, connecting with vessel of portal system
  - Type 4: mixed (8%), two or more sites of abn pulm venous connections

- Because venous return to RA is mixture of oxygenated (pulm veins) and deoxygenated (IVC, SVC) blood, R → L atrial shunt causes systemic desaturation; under most circumstances, this is not severe enough to cause significant hypoxemia or end-organ dysfunction

- Pulm venous obstruction results in pulm Htn, diminished pulm blood flow, and significant R → L shunting, with severe hypoxemia and heart failure in the newborn period

**ASSESSMENT POINTS**

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CHF</td>
<td>Tachycardia, cyanosis</td>
<td>Flow murmur</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>PV obstruction</td>
<td>Tachypnea</td>
<td>Pulmonary edema</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency (prerenal)</td>
<td>↓ UO, acidosis</td>
<td></td>
<td>BUN, Cr</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Anesthetic Technique**
- Opioid, NMB, controlled ventilation

**Monitoring**
- IV, arterial line, CVP, or transthoracic atrial/PA line placed by surgeon intraop
- TEE helpful (limited by pt size, potential for airway compression)
- Placement of temporary epicardial pacing wires for potential postop use

**Airway**
- No unique concerns

**Induction and Maintenance**
- IV induction with high-dose opioid, NMB, ± benzodiazepine

**Extubation**
- Usually deferred until after immediate postop period

**Surgical Stages**
- Goals: Direction of pulm venous drainage to left atrium and ligation of abn connection to systemic venous system

- Pulm veins normally converge to a confluence posterior to LA; correction involves creation of unobstructed pathway from pulm veins to this confluence, and ultimately to LA. Anomalous ascending or descending venous connection is ligated.

- Surgery is performed with CPB, occasionally with deep hypothermic circulatory arrest

**Anticipated Problems/Concerns**

- If pt has obstructed pulm veins, at higher risk for periop morbidity/mortality from pulm Htn and/or ventricular dysfunction

- Pulm Htn can result from volatile PVR, kinking at anastomotic site, impaired LV function.

- RV dysfunction resulting directly from pulm Htn can lead to decreased pulm blood flow, diminished LA-LV filling, and dramatic reduction in systemic blood flow

- Etiology of LV dysfunction incl inadequate myocardial protection or inadequate LV preconditioning due to predominant L → R atrial shunt

- Arrhythmias are infrequent, but loss of AV synchrony worsens poor ventricular function. Sequential pacing via temporary epicardial leads and/or pharmacologic management of supraventricular tachycardia is indicated.
Total Hip Arthroplasty

**Risk**
- Incidence in USA: >200,000/y; worldwide about 700,000
- Racial predilection: None
- Very successful at restoring function

**Perioperative Risks**
- Risks: 0.25–2% 30-day mortality (0.7% in large VA study). Risk down 67% over 10 y
- CV collapse ↓ 2° to palp emboli, embolization of fat, marrow, bone, or cement
- Thromboembolism (peripheral venous thromboembolitis in 60% of pts w/o prophylaxis—50% ↓ with prophylaxis)—palp emboli in 2–4% of pts who do not receive prophylaxis

**Assessment Points**

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<tbody>
<tr>
<td>HEENT</td>
<td>Arthritis can involve airway joints</td>
<td>Snoring</td>
<td>Airway</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Impairment due to age; if Fx caused by CV problem—dysrhythmia, etc. Hypovolemia if Fx, bleed into leg</td>
<td>CV status</td>
<td>CV exam</td>
<td>ECG Orthostatic BP by tilt table test</td>
</tr>
<tr>
<td>RESP</td>
<td>Arthritis can be systemic restrictive pulm disease/fat emboli with Fx or with replacement of hip</td>
<td>SOB</td>
<td>Chest exam</td>
<td>O₂ sat</td>
</tr>
<tr>
<td>HEME</td>
<td>Blood loss 2° to Fx</td>
<td>Orthostatic dizziness</td>
<td>Leg; tilt table BP</td>
<td>Hct</td>
</tr>
<tr>
<td>RENAL/CNS</td>
<td>Impairment 2° to age; if Fx, investigate cause: CNS Hx if cause of Fx is CVA, dysrhythmia, etc.</td>
<td>CNS Hx</td>
<td>CNS exam</td>
<td>BUN/Gr</td>
</tr>
</tbody>
</table>


**Monitoring**
- Volume status monitoring of blood loss a major concern—blood loss ↓ with regional anesthesia or intentional hypotension
- Consider CVP or TEE (if GA chosen), several large-bore IVs.
- Check availability of preoperative autologous and/or homologous blood; irradiate directed donor blood; erythropoietin use controversial but common to bring Hgb from below 13 (with or without autologous donation) to over 13 g/dL
- UO, HR, BP not reliable signs of intravascular volume during anesthesia or with postop epidural pain relief
- Myocardial ischemia with ECG, ST segments, even PA cath or TEE if general anesthesia
- Patient’s Sx of angina or CHF may be used but may be distorted if high regional anesthesia (need T6 level for regional)

**Airway**
- Side operated on is usually up, so securing airway after positioning may be difficult (three approaches: posterolateral is most common, but posterior and anterior are used; minimally invasive (4 in.—[10 cm]) or less incisions versus prior 6 to 12 in. incisions (15–30 cm) common. Resurfacing, rather than replacing, now used for under 55-y-old active males with osteoarthrits and minimal underlying hip deformity after approval by FDA with metal-on-metal devices with a polished acetabular head and mated metal head ball which caps the femoral head. Less blood

**Worry About**
- Causes of Fx or arthritis or joint deterioration (CV disease, syncope, inflam joint diseases such as RA, psoriatic CT disease, etc., that are assoc with systemic conditions)
- Periop fluid deficiency
- Hypoxia and/or hypotension on cementing
- Pulm emboli periop

**Overview**
- Diseased articular surfaces are replaced with synthetic materials, thus relieving pain and improving joint kinematics and function
- Assoc with high immediate morbidity, mortality 2° to volume shifts, embolization
- 1–6-d mortality assoc with pulm emboli
- Age, co-morbidities of pts dictate monitoring strategies

**Indications and Usual Treatment**
- Replacement of hip socket (acetabulum) and/or femur with alloys of metals, plastic porcelain for
  - Chronic osteoarthritis
  - Hip Fx (large amounts of blood can be sequestered around Fx site). Investigate cause.
  - Avascular necrosis 2° to steroids, infarction (e.g., sickle cell disease)
  - Usual medical Rx incl NSAIDs, aspirin, chondroitin and glucosamine, exercise, calcium, vita

**Surgical Stages**

**Induction**
- CV instability 2° to volume status
- If regional, consider slow titration

**Skin Incision**
- Large incision over operated hip, leg
- Can observe wound for excessive bleeding

**Dissection** (see diff approaches in Airway section above)
- During dissection down to femur, acetabulum, major blood vessel can be accidentally injured, dif
cult to control—esp femoral, iliac vessels
- Reaming of acetabulum, then femur
- Major blood loss 2° to bone dissection
- High-pressure lavage occasionally used to prep
surface: Embolization of fat possible
- Use of heparin during reaming of femur ↓ risk of thromboembolism

**Definitive Surgery**
- Some are cemented—methacrylate produces hypotension (within 30 min)
- Increased pulm embolization (?fat, air, methy
-methacrylate monomer) that results in increased ventilation-perfusion mismatch or shunt, ↓ O₂ sat, right heart dysfunction
- Decreased myocardial contractility 2° to above
- Can anticipate, hydrate for 10–20 min prior to cementing, ↑ FIO₂, have ephedrine ready
- Duration: ~5 hr
- Fluid shift can be sizable
- Decrease emboli by cath removal of pressure on cement insertion

**Closure/Postoperative Considerations**
- Blood loss of 1–2 units continues—if auto
transfusion used, may have reaction to non-washed cells immediately and may not get as great a long-term boost in Hct as planned (Hcts of 30 desirable if CV disease)
- Keep warm to reduce complications and blood Tx
- Pain score: 6–9
- Pain relief via NSAIDs, PCA, or epidural (start epidural narcotics 1 hr prior to end of surgery)
- Early ambulation reduces thromboemboli

**Anticipated Problems/Concerns**
- Blood volume status and rapid changes due to position, onset of regional and/or general anesthesia
- Extreme age may make less able to tolerate resp insult and CV instability after cementing
- Thromboembolism and infection prophylaxis desirable
- Pain with early ambulation may be considerable
**Total Knee Arthroplasty**

**Risk**
- Incidence in USA: >500,000 cases
- >60 y with osteoarthritis
- Rheumatoid arthritis, trauma, infection, hemophilia, hemochromatosis, pigmented villonodular synovitis, avascular necrosis, obesity

**Preoperative Preparation**
- Consider arterial catheter for dramatic swings in BP with tourniquet inflation, deflation
- Large-bore IV or CV catheter
- Postop bone-cement implantation syndrome (fat embolism syndrome, FES) more common with SBTKR
- Bleeding (major ~1%)
- Infection
- Sciatic and peroneal nerve palsies; more common with valgus deformity
- Pain

**Worry About**
- CV collapse with tourniquet deflation
- Postop bone-cement implantation syndrome (FES)
- Pulm emboli periop
- Intraop bleeding (popliteal artery); postop bone
- Overview
- Replacement of both tibial, femoral components of knee joint, often with methylmethacylate cemented implants.
- Increased risk of FES with intramedullary components

**Postoperative Considerations**
- Significant blood loss over the first 24 hr from surgical drain; if possible, consider autologous blood donation, erythropoetin preop controversi-
  al; cell scavenge and re-infusion
- Unicompartimental knee arthroplasty may have lower periop risks

**ICD-9-CM Codes:**
- 715.26 (Osteoarthritis, knee)
- 714.0 (Rheumatoid arthritis)

**Indications and Usual Treatment**
- Pain (OA, RA)
- Avascular necrosis
- Seronegative spondyloarthropathies
- Post-traumatic arthritis
- Hemophilia arthropathy of knee

**Treatment**
- NSAIDs and COX-2 inhibitors
- Glucosamine and chondroitin sulfate
- Calcium, magnesium, and vitamin D
- Physical therapy and build up of shock absorbing muscles above and below
- Alignment of joints with shoe prostheses
- Surgical wash out of loose fragments, experimental replacement of cartilage with stem cell rebuilding, etc., are all alternatives.

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**Total Knee Arthroplasty**

**Perioperative Management**

**Preoperative Preparation**
- Treat hemophilia if present
- Pre-treatment with LMWH for pts at higher risk for DVT/PE
- Encourage smoking cessation, preop walking, and portion control
- Continue aspirin (81 or preferably 162 mg) pts with stents, Hx CVA, AFib
- Beta-blockers and statins in pts at risk for ischemic events

**Anesthetic Technique**
- Although no clear advantage of regional over GA, regional eliminates manipulation of air-
  way, provides vehicle for postop pain Rx; epidural anesthesia may be extremely difficult with severe arthritis, scoliosis, or ankylosing spondyli-
  tis. Consider paramedian approach.
- Alternatively femoral / sciatic nerve blocks
- Consider GA if on LMWH

**Monitoring**
- Consider arterial catheter for dramatic swings in BP with tourniquet inflation, deflation
- Large-bore IV or CV catheter

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Tracheal Resection

Alex Chen
Edward A. Ochroch

Risk
• Annual incidence of 1,200,000 adults
• 2.7 new cases of malignant tracheal mass per million per year

Perioperative Risks
• Periop mortality ~2%

Worry About
• Difficult ventilation and/or impending airway collapse
• Ventilation strategy changes in different phases of the operation

Overview
• Variable surgical/airway course that is dependent on the lesion’s extent, percent obstruction and location with respect to the carina and vocal cords. Surgery and anesthesia must work in congruence and share the airway
• Resection with end-to-end anastomosis depending on how many rings are involved

ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE | Test
--- | --- | --- | --- | ---
HEENT | Stridor | Resp distress | Auscultation of the trachea, palpitation of the trachea, neck mobility | Bronchoscopy
CARDIO | CV decompensation, pulm Htn | Cyanosis, resp distress, orthopnea | Murmur, hepatomegaly, edema | ECHO, stess, cath (as indicated)
Resp | Obstructive physiology | Exercise intolerance, inspiratory stridor, expiratory wheezing (frequently misdiagnosed as asthma) | Accessory muscle use, tachypnea | CXR, CT 3D recon, PFTs


Perioperative Implications

Preoperative Preparation
• A clear Hx of any previous airway management needs to be reviewed
• Determine which position the pt breaths best—OR rescue
• Presurgery steroid weaning and ventilator weaning and pulm optimization
• Consent for femoral vessel cannulization for CPB in case of complete obstruction
• Set up for immediate rigid bronchoscopy should airway compromise occur
• Readily available small ETTs, fiberoptic cart, jet ventilator, thin injector and/or exchange catheters and surgeons standing by to perform a tracheostomy if necessary
• Premedication: Sedatives reserved for very anxious pts, ± an anticholinergic

Anesthesia Technique
• GA (TIVA due to open airway manipulation)

Monitoring
• Two IVs: TIVA + bolus meds, significant blood loss is rare
• Standard monitors
• Arterial line in the left arm (the innominate artery may be operatively manipulated or compressed)
• Prolonged jet ventilation will necessitate arterial CO₂ sampling
• Minimal fluid shifts: Rare need for CVP, pulmonary artery pressure/CCO Swan (TEE will impinge posterior membranous wall)

Airway
• Preoxygenation and/or denitrogenation vital prior to starting the procedure. It can take considerably longer than usual: small tidal volumes.

• >90% good long-term results may be expected with resection and end-to-end anastomosis
• PFTS (spirometry, flow volume loops: Erect and supine); characteristics of an extra-thoracic tracheal lesion: a greater decrease in inspiratory flow than in expiratory flow; a delay in reaching peak expiratory flow, a reduced peak expiratory and peak inspiratory flow, and/or an abrupt drop of expiratory flow at the end of expiration; with a fixed upper airway obstruction such, there is flattening of both the inspiratory and the expiratory phase

• Neoplasm: Adenoid cystic CA, squamous cell CA, neurofibroma, chordoma, chondroblastoma, hemangiom, pleomorphic adenoma
• Inflammatory lesions: SLE, sarcoidosis, amyloidosis, Wegener's
• Trauma: Mostly related to ET intubation, or from tracheostomy-induced formation of granulation tissue

• Other: Tracheal webs, tracheal agenesis/atries, tracheomalacia, idiopathic laryngotracheal stenosis

Treatment
• Irrigation: Squamous cell CA and adenoid cystic CA: Edema may critically narrow airway
• Bronchoscopy: Dilatation used in emergency situations, final therapy (injection of steroids or mitomycin C may retard the recurrence) or bridge to surgery
• Laser: Great precision with less periop edema; choice of laser determines depth of penetration
• Stents: Palliation, temporizing measure or as an adjunct to surgery to reinforce the new anastomosis
• Tracheal reconstruction: Resection and reconstruction with 1° anastomosis

ICD-9-CM Code: 519.19 (Tracheal stenosis)
Tracheoesophageal Fistula Repair

Risk
- 1/3000 live births
- Dx confirmed by the inability to pass a soft suction cath into stomach

Perioperative Risks
- Periop mortality low in full-term, healthy newborns; almost 100% survival
- Periop mortality approaches 15–60% in infants less than 1800 g
- Tracheomalacia
- Esophageal stricture

Worry About
- Difficult ventilation and/or hypoxemia
- Prematurity: Up to 3% assoc with TEF; consider possibility of retinopathy or prematurity
- VATER syndrome: Vertebral anomalies, anal atresia, tracheoesophageal fistula, esophageal atresia, radial dysplasia

Overview
- Primary repair incl fistula ligation, esophageal anastomosis
- Four types
- Staged repair incl placement of gastrostomy tube under local anesthesia, subsequent ligation of fistula, esophageal repair when more stable

ICD-9-CM Code: 750.3 (Congenital)

ASSESSMENT POINTS

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<thead>
<tr>
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<th>PE</th>
<th>Test (If Indicated)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CV decompensation, cyanosis, CHF</td>
<td>Cyanosis, tachypnea, resp distress</td>
<td>Murmur, cyanosis, enlarged liver, hypotension, bounding pulses</td>
<td>CXR, ECG, ECHO, cardiac cath</td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumonia, subglottic stenosis</td>
<td>Resp distress, tachypnea, stridor</td>
<td>↓ Breath sounds, tachypnea, cyanosis</td>
<td>CXR, ABGs (if indicated), flexible fiberoptic bronchoscopy</td>
</tr>
<tr>
<td>GI</td>
<td>Gastric distention, assoc anal atresia or bowel obstruction</td>
<td>Enlarged abd Resp distress</td>
<td>Tympanic abd, enlarged abd</td>
<td>KUB series</td>
</tr>
<tr>
<td>RENAL</td>
<td>Dysplastic/dysfunction</td>
<td>Anuria</td>
<td>Palpation for kidneys</td>
<td>Créde, cath, or collect urine by bag appliance BUN/Cr</td>
</tr>
</tbody>
</table>


Indications and Usual Treatment
- Carefully define other congenital defects
- Unstable pts: Consider staged procedure
- Premature infants: Consider staged procedure
- Gastrostomy tube placement when gastric distention compromises ventilatory status
- Passage of Fogarty cath through gastrostomy into esophagus can occlude esophagus/fistula, thus promoting ventilation of lungs

Perioperative Considerations
- Vigorous infants may be extubated at conclusion of surgery this preferred for maintenance of repair
- Premature infants and those with significant pulm disease may require continued mechanical ventilation.
- Suction caths marked to point at which they will contact repair

Postoperative Complications
- Pulm aspiration, tracheomalacia, vocal cord paralysis
- At later date, pts at risk for intubation of tracheal diverticulum that may develop at site of fistula closure
- Esophageal stricture, esophageal foreign body entrapment relatively common following repair
- Regional anesthesia may be used as a supplement to general anesthesia and may improve management of postop pain. A single-dose caudal block may be administered. Alternatively, a caudal catheter may be placed and advanced to a thoracic level for intermittent dosing.

Anticipated Problems/Concerns
- Pulm disease
- Difficulty sustaining airway, avoiding hypoxemia and/or hypercarbia
- CV compromise and/or CHD
- Hypovolemia and/or blood loss
- Hypothermia
- Prematurity

Anesthetic Technique
- GA for complete repair
- Local anesthesia for gastrostomy tube placement in staged repair

Monitoring
- Large, well-functioning IV for blood loss
- Consider art line if resp or CV problems
- Urinary catheter

Airway
- Awake intubation and/or careful rapid-sequence
- ETT positioned just above carina to avoid ventilating fistula and to ensure ventilation of both lungs: intentional right main stem intubation with subsequent slow withdrawal of ETT until breath sounds first heard on left usually ensures that ETT optimally placed
- Consider facing the bevel posteriorly during intubation to avoid direct intubation of fistula
- Monitor for kinking and/or obstruction of trachea and/or ETT by surgical traction during dissection, repair
- Monitor for complete obstruction of ETT by blood and secretions, necessitating suctioning and/or replacement
- Precordial stethoscope on left chest to monitor breath sounds intraop; accidental advancement of ETT into right mainstem bronchus may then be detected
- Subglottic stenosis may necessitate placement of smaller diameter ETT than usual

- CHD to 25% assoc with TEF (VSD, ASD, PDA, tetralogy of Fallot), causing CV instability
- CV collapse and/or hypotension due to gastric distention or surgical compression
- Hypothermia, consequent acidosis/metabolic dysfunction

Dissection
- Blood loss usually minimal, although large blood vessels may be transected
- Recurrent laryngeal nerve damage may occur

Definitive Surgery
- Hypercarbia and/or hypoxemia possible from these causes: Compression or retraction of right lung, kinking of trachea and/or ETT from surgical traction, plugging of the ETT, its migration into right mainstem bronchus or fistula, preferential ventilation of fistula
- Hypotension may result from cardiac compression, hypovolemia, or blood loss
- Hypothermia may result from administration of cold IV fluids, cool ambient room, anhydrous gas administration, heating pad malfunction. Metabolic acidosis may result from hypothermia.
- Blood loss can be steady; apparently small losses can be clinically significant in newborn.

Postoperative Considerations
- Vigorous infants may be extubated at conclusion of surgery this preferred for maintenance of repair
- Premature infants and those with significant pulm disease may require continued mechanical ventilation.
- Suction caths marked to point at which they will contact repair

Postoperative Complications
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Anticipated Problems/Concerns
- Pulm disease
- Difficulty sustaining airway, avoiding hypoxemia and/or hypercarbia
- CV compromise and/or CHD
- Hypovolemia and/or blood loss
- Hypothermia
- Prematurity
Tracheotomy/Tracheostomy and Cricothyroidotomy

Overview
- The term tracheotomy is used for temporary and tracheostomy is used for permanent procedure
- May be performed as emergency or elective procedure, open or percutaneous technique
- Advantages of percutaneous tracheostomy: Smaller skin incision, less trauma, bedside procedure, less incidence of wound infection
- Goals of general anesthesia should be to avoid airway fires, optimize oxygenation and ventilation, use the lowest safe inspired O₂ concentration, position the existing ETT such that the damage to its cuff is minimized, control all bleeding points, meticulous suction

Indications and Usual Treatment
- Airway obstruction: Congenital, trauma, infection, neoplasm, foreign body
- Long-term ventilator support: For resp failure in critically care unit
- Provide tracheobronchopulmonary hygiene: facilitate frequent suctioning for secretions in pts with NM diseases, prevention of aspiration
- Planned prophylactic tracheostomy: During extensive head and neck procedures

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
</table>
| HEENT      | Stridor Change in voice drooling| Trauma, tumor, infection | Type of stridor in relation to breathing phase | Inspiratory – supraglottic obstruction
|            |                                 |                   |                             | Expiratory – subglottic obstruction |
|            |                                 |                   |                             | Biphasic – supra and subglottic obstruction |
|            |                                 |                   |                             | Fiberoptic endoscopy |
| CARDIO     | Hypotension Critically ill on ICU| Invasive monitoring | Ionotrope dependence | ECG, ECHO |
| RESP       | Dyspnea, long-term ventilation Cough | Ventilator support, increased O₂ requirements | Decreased breath sounds | ABG, CXR, CT scan/MRI, Fiberoptic endoscopy |
| RENAL      | Renal insufficiency Long term ICU stay | Depending on cause | Serum creatinine, BUN, electrolytes |
| CNS        | Cerebral effects of hypoxia and hypercarbia | Trauma, compromised breathing | Restlessness loss of consciousness | ABG |


Perioperative Management

Preoperative Preparation
- Assess the severity of airway compromise
- Beware while transporting a critically ill pt from the ICU to the OR

Monitoring
- Standard monitoring: ECG, BP, pulse oximetry and capnography
- Arterial line in critically ill pts

Anesthetic Technique/Induction
- General anesthesia for optimal surgical condition
- Local anesthesia in pts with severe airway compromise
- Position: Supine with extended head; may be sitting semi-upright in severe airway obstruction

Surgical Stages

Dissection: Tracheotomy
- Landmark: Midway between the cricoid cartilage and suprasternal notch, site overlying the 2–3 tracheal rings, skin infiltration with lidocaine with epinephrine may help reduce bleeding
- For open tracheostomy, skin incision may be vertical or horizontal, electrocautery used to remove subcutaneous fat, thymic isthmus may be divided, tracheal incision may be T-shaped, U-shaped or involve removal of a small anterior part of the trachea for a permanent stoma
- Before incising the trachea, be sure that the cuff is not in the way of the tracheal incision. Once trachea is opened stop ventilation, deflate the ETT cuff, withdraw the ETT carefully under direct vision until the tip is just above the tracheal stoma.
- Do not remove the ETT until the tracheostomy tube placement is confirmed.
- Confirm placement with chest rise, auscultation for breath sounds and ETO₂
- Fiberoptic endoscopy is useful to assist percutaneous tracheostomy

Cricothyroidotomy
- May be performed with the open, percutaneous or needle cricothyroidotomy techniques
- Open technique: Horizontal stab incision with 20-g scalpel, 1cm horizontal incision above the superior border of the cricoid cartilage, insert the handle of the scalpel to widen the incision, then insert the tracheostomy tube, may be performed in 30 sec and used up to 72 hr
- Percutaneous cricothyroidotomy using Seldinger technique
- Needle cricothyroidotomy may be performed using a cannula and transtracheal jet ventilation (55 psi) commenced in case of emergency situation (cannot intubate cannot ventilate)
- Always confirm ventilation with chest rise, chest auscultation, and capnography

Postoperative Considerations
- Ventilation, critical care management
- Postoperative CXR

Anticipated Problems/Concerns
- Immediate complications: Bleeding from the thyroid gland, pneumothorax (0–4%), pneumomedastinum, injury to recurrent laryngeal nerve, esophagus, and large blood vessels
- Early complications: Increased bleeding around the tracheostomy site on emergence from anesthesia, bloody secretions, tracheitis, mucous plugs, skin infection at site
- Delayed complications: Hemorrhage (1–6 wk after procedure) may be due to tracheoinnominate artery fistula (0.6%), tracheal stenosis, tracheomalacia, trachea-esophageal or tracheocutaneous fistula, scarring, granulation tissue formation
Transjugular Intrahepatic Portosystemic Shunt (TIPS)

**Risks**
- Incidence in USA: Cirrhosis of the liver occurs in 1–4/1000 adults
- 26,000 deaths per year in the USA are related to cirrhosis
- >60% with cirrhosis have portal Htn and 33–98% with cirrhosis have GI varices
- Variceal bleeding occurs in 25–40%
- Portal vein thrombosis occurs 24–32% in cirrhosis and 5–10% in pts referred for TIPS

**Perioperative Risks**
- Technical success rate of the TIPS procedure ranges from 75% to greater than 90%
- Overall direct procedure mortality rate is <2%
- 30-d mortality rate ranges from 4 to 45%
- Rebleeding rate is 10–26% and usually assoc with shunt stenosis or thrombosis
- Postprocedural encephalopathy: 5–55%
- Fever has been reported in 10% of pts
- Child-Pugh or MELD score has been used to predict the overall survival rate
- TIPS in children and post-liver transplant pts is feasible, but with potential difficulties

**Worry About**
- Hypotension 2° to bleeding caused by severe coagulopathy, vein rupture or perforation of liver capsule
- CHF, cardiac arrhythmias: HB, VF, AF
- O₂ desaturation 2° to excessive sedation
- Aspiration
- Tension pneumothorax
- Mental status changes: ↑ encephalopathy postop

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEURO</td>
<td>Hepatic encephalopathy (from confusion to coma)</td>
<td>Disturbances of awareness and mentation, personality change</td>
<td>Asterixis (liver flap), rigidity, hyperreflexia</td>
<td>EEG, ammonia level</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Cardiomyopathy, CAD, arrhythmias (QT prolongation, Tourniquets, etc)</td>
<td>ETOH abuse, smoking Hx</td>
<td>Tachycardia, S₁, edema</td>
<td>ECG, ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Atelectasis, pulm shunting, hypoxemia, hyperventilation, pulm effusion, pulm Htn</td>
<td>SOB, poor exercise tolerance</td>
<td></td>
<td></td>
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<tr>
<td>GI</td>
<td>Ascites, aspiration, variceal bleeding, gastritis, ulcer</td>
<td>↑ Abd girth, hematemesis</td>
<td>Fluid wave, bulging flanks, postural tachycardia</td>
<td>US, paracentesis, endoscopy</td>
</tr>
<tr>
<td>HEPAT</td>
<td>Drug metabolism change</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Hepatorenal syndrome</td>
<td></td>
<td>BUN/creatinine, electrolytes</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypoglycemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia, coagulopathy, incl factors and plt deficiency, Resistant to activated protein C</td>
<td>GI bleed, recurrent TIPS stent stenosis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Indications and Usual Treatment**

<table>
<thead>
<tr>
<th>Accepted Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute variceal bleeding not successfully controlled with medical Rx</td>
</tr>
<tr>
<td>Recurrent variceal bleeding refractory or intolerant to conventional medical Rx</td>
</tr>
<tr>
<td>Particularly helpful when bleeding occurs from inaccessible intestinal or gastric varices or is the result of severe portal hypertensive gastropathy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Probable Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refractory ascites, refractory hepatic hydrothorax, hepatorenal syndrome, Budd-Chiari syndrome, and veno-occlusive disease</td>
</tr>
<tr>
<td>Acute variceal bleed while awaiting Tx</td>
</tr>
</tbody>
</table>

**Usual Treatments**
- Pharmacologic Rx to ↓ portal pressure
- Endoscopic band ligation or sclerotherapy of the varices
- Balloon tamponade, then devascularization
- Surgical portosystemic shunting

**Absolute Contraindications**
- Right-sided heart failure
- Polycystic liver disease
- Severe hepatic failure, unless active variceal bleeding or fulminant Budd-Chiari syndrome
- 1° pulm Htn
- Cavernous portal vein thrombosis

**Relative Contraindications**
- Biliary obstruction, systemic infection, and severe hepatic encephalopathy


**Monitoring**
- Standard monitors; in selected cases, arterial line and central venous catheters can be added

**Induction/Maintenance**
- Rapid-sequence induction common if encephalopathy, abd distention, or recent variceal bleed
- Broad-spectrum antibiotic for gram-negative organisms (e.g., 1 g ceftizoxime) at start
- May be sensitive to all agents

**Emergence**
- Extubation after pts are awake and their protective laryngeal reflexes present
**Surgical Stages**

- **US** is performed to assess the size and patency of portal and hepatic venous systems
- Jugular and hepatic vein access and pressure measurement—right internal jugular vein approach is preferred because a straight path into the infrahepatic IVC. An angiographic catheter is advanced to the infrahepatic IVC and pressures measured. Sheath is advanced into the right hepatic vein, and both free hepatic vein pressure and wedged hepatic vein pressure are measured.
- Identification of portal vein—wedged hepatic venogram is performed with either iiodinated contrast or CO₂ for portal vein localization
- Dilation and stent placement—a puncture needle is advanced to access the portal vein. The needle is removed while a guidewire is advanced to the superior mesenteric vein or splenic vein. Portal pressures are measured and a portal venogram is obtained. An angioplasty balloon is advanced to dilate the transhepatic tract. A bridging expandable stent is deployed and then dilated to 8–12 mm. Ideally, the pressure gradient following shunting should be 6–12 mmHg.
  - Ultrasound within 24 hr to assess patency of the stent
  - EBL: 0–3000 mL

**Postoperative Considerations**

- Monitor in an ICU or step-down unit for 24–48 hr due to potential for portal vein thrombus, worsening encephalopathy, sepsis, bleeding, pulm edema, and fluid and lyte disturbance
- Pain score is 7–8 in first few hours. Usually, opioid is needed only for the first few hours.

**Anticipated Problems/Concerns**

- Portal vein rupture and perforation of liver capsule leads to intra-abdominal hemorrhage
- Cardiopulmonary failure from sudden hemodynamic changes
- Pts with pre-existing LBBB may need pacemaker pre-TIPS due to risk of RBBB
- Encephalopathy may worsen due to ↓ hepatic portal blood flow
Transposition of the Great Arteries (TGA)

Risk
- Incidence: 0.02-0.05% of live births; 7-8% of all congenital cardiac defects, second only to VSD
- Most common cyanotic CHD presenting in infancy
- M:F ratio: 2:3:1.1

Perioperative Risks
- Assoc cardiac anomalies: VSD (40-45%), LV outflow tract (subpulmonic) obstruction (LVOTO) 25%, secundum ASD, aortic arch obstruction
- Systemic or pulm ventricular failure
- Pulm Hm and/or pulm vascular occlusive disease (PVOD) may develop early esp. in presence of aortopulmonary collateral vessels, large VSD
- Cyanotic pts: Polythemia, coagulopathy
- Rhythm disturbances affecting CO
- Use of PGE, to maintain ductal patency may result in aspne, fever, vasodilation, and edema

Worry About
- Neonates with CHF, cyanosis should be evaluated for TGA.
- With intact ventricular septum (IVS), PGE infusion is utilized to maintain ductal patency, blood mixing until balloon atrial septostomy (BAS) performed
- PVR and SVR need to be balanced to maintain optimal ratio of systemic to pulm blood flow

Overview
- d-TGA: Concordance of atrioventricular connections and discordance of ventriculoarterial connections exists creating two parallel circulations.
- Communication between circulations must exist (PDA, ASD, VSD) at one or more levels to allow mixing and survival until surgical intervention
- Echocardiography gold standard of diagnosis
- Without intervention, 30% mortality in first wk, 45% first mo, 90% first y; anoxia, CHF 1st causes of death
- Type of surgical intervention depends on assoc presence of VSD or LVOTO
- Balloon atrial septostomy (BAS): Rashkind procedure, early corrective surgery have improved long-term outcome

Etiology
- Assoc risk: Possible maternal diabetes

Usual Treatment
- PGE, infusion utilized to maintain ductal patency
- Bas frequently performed at bedside or in cath lab to promote mixing preop
- Successful BAS may allow D/C of PGE, preop
- Palliative surgery
- If LVOTO and VSD are present: systemic to PA shunt
- If VSD and advanced PVOD are present, atrial switch (Mustard procedure) may be performed without VSD closure.
- Repair
- Physiologic: Intra-atrial repair (Mustard or Senning procedure) to connect systemic, pulm circuits at atrial level with RV remaining as systemic ventricle
- Anatomic: Arterial switch (Jatene) with coronary artery reimplantation to anatomically correct circulation by anastomosing aorta to systemic ventricle and PA to the pulm ventricle. If severe LVOT obstruction and VSD exist, VSD is closed via intracardiac baffle redirecting LV blood to aorta and RV-PA conduit is placed (Rastelli operation).
- Timing of repair: Pts with IVS: first 1-2 wk of life to prevent reduction in LV mass. If LV regression has occurred PA band may be done before repair to prepare LV. Pts with VSD: first 1-2 mo to prevent development of CHF or PVOD.

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<tbody>
<tr>
<td>CARDIO</td>
<td>CHF</td>
<td>Resp distress</td>
<td>Roles, S, hypotension</td>
<td>ECHO</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm vascular occlusive disease</td>
<td>Dyspnea</td>
<td>Clubbing, cyanosis</td>
<td>CXR, cath</td>
</tr>
<tr>
<td>HEME</td>
<td>Polythemia (if &gt; 6-9 mo)</td>
<td>Bleeding</td>
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<tr>
<td></td>
<td>Coagulopathy, bleeding Thrombocytopenia</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>CVA</td>
<td>Assoc with polythemia</td>
<td>Focal deficit</td>
<td>CT or MRI</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Maintain CO with adequate HR, contractility, preload
- Maintain ductal patency with PGE, (0.01–0.05 μg/min/min) in ductal dependent pts
- Premedication rarely required; consider sedative premedication for infants >6 mo of age

Monitoring
- Arterial line may be placed after induction
- Central line or right atrial line may be used for drug infusion, pressure monitoring

Airway
- Pt may or may not be intubated preop
- Nasal ETT recommended to minimize interference with potential use of TEE

Preinduction/Induction
- Avoid ↑ PVR, can ↓ PBF, intercirculatory mixing
- PVOD present, use ventilatory interventions to ↓ PVR: ↑ FIO2, ↓ PaCO2
- If LVOTO present, ↑ ventilation can ↓ PVR, ↑ pulm blood flow and intercirculatory mixing
- Maintain SVR relative to PVR to maintain effective pulm blood flow and SpO2

If CHF present with VSD, ventilatory manipulations may be deleterious, due to pre-existing ↑ pulm blood flow, difficulty of maintaining systemic blood flow with failing heart
- Anesthetic induction may be accomplished with opioid or pentothal if IV line in place, otherwise inhalation induction may be used.

Maintenance
- Opioids preferred drugs for maintenance anesthesia (fentanyl 10–20 μg/kg); affords hemodynamic stability; does not depress myocardium; blunts reactive pulm Hm. May be used in concert with low doses of volatile agent.
- In infants with TGA and IVS, O2 delivery can be tenuous
- In infants with TGA and VSD, volume overload possible
- Avoid hypercarbia and acidosis
- Pancuronium usually relaxant of choice due to vagolytic properties

Post CPB
- Maintain age-appropriate HR; keep BP low-normal range to avoid excessive bleeding
- Monitor for ischemia, dysrhythmias
- Initiate inotropic and/or vasodilator therapy for ventricular dysfunction
- TEE useful to assess for air, ventricular function, assess coronary arteries, and VSD closure

Extubation
- In ICU when pulm and hemodynamic stability present, postop bleeding controlled, pt awake

Anticipated Problems/Concerns
- Atrial switch
- Interatrial baffle obstruction (systemic or pulm) may occur with resulting low CO or SVC syndrome; pulm venous obstruction may result in low CO, pulm edema
- Baffle leak may allow atrial level shunting
- Dysrhythmias ultimately seen in >60% of pts: Sinus bradycardia may require atrial pacing; junctional rhythm may require AV sequential pacing; rapid AF may require cardioversion
- RV (systemic) dysfunction may occur if right ventriculotomy used
- Arterial switch
- Bleeding from suture lines
- Myocardial ischemia due to coronary reimplantation (air or kinking)
- Inadequate LV function due to insufficient mass, ischemia, or inadequate preservation during CPB—inotropic support
- Supravalvar pulm artery stenosis
- Rastelli operation
- Left ventricular dysfunction or failure
- Arhythias, sudden death
- Conduit stenosis
Transposition of the Great Arteries, L Form (L-TGA)

Risk
- Incidence 0.5% of pts with CHD
- Male slightly greater than female incidence

Perioperative Risks
- 1–10% may have no assoc lesions and may be unrecognized until right ventricular failure occurs
- 25% of pts with no assoc defects develop CHF by age 45 y
- Assoc defects: VSD (60–80%); pulm stenosis (left ventricular outflow obstruction) 30–50%; tricuspid (left AV valve) valve abs incl Ebstein-like malformation (50–80%).
- Increasing risk of AV block, RV dysfunction, worsening TR with increasing age

Worry About
- Pts can present with CHF 2° to large VSD or severe tricuspid (left AV valve) regurgitation
- Significant cyanosis can occur in pts with LVOTO and VSD
- Increased incidence of complete heart block (2% cumulative increase/y)
- Late complications in unoperated pts: Right ventricular dysfunction, CHF
- Assoc defects such as tricuspid (left-sided) regurgitation and AV conduction abn often progress and are risk factors for mortality.

Overview
- L-TGA: Discordant atroventricular connections and discordant ventriculoarterial connections are both present, resulting in a series circulation
- Atrial position is normal, but ventricles are inverted and aorta is anterior and lies to the left of the pulm artery.
- Dextrocardia or mesocardia exists in 25% of pts.
- In the absence of other cardiac lesions pts are physiologically corrected.

Ecology
- No known genetic predisposition

Perioperative Implications

Preoperative Preparation
- Preop medication useful for most pts to allay anxiety and facilitate separation
- Note preop issues with cardiac rate or rhythm
- Arterial line may be placed after anesthetic induction
- Central line or right atrial line may be used for drug infusions and pressure monitoring
- Pacemaker capability should be readily available
- Airway
  - Nasal ETT recommended to minimize interference with potential use of TEE
  - Late complications in unoperated pts: Right ventricular dysfunction, CHF
  - Assoc defects such as tricuspid (left-sided) regurgitation and AV conduction abn often progress and are risk factors for mortality.

Preinduction/Induction
- IV or inhalation induction may be utilized.

Maintenance
- Opioids (fentanyl 10–20 μg/kg) affords hemodynamic stability and avoids myocardial depression.
- May be used in concert with volatile agent.
- Pancuronium usually relaxant of choice due to vagolytic properties

Post CPB
- Monitor for ischemia, dysrhythmias
- Initiate inotropc and/or vasodilator therapy for ventricular dysfunction.
- TEE useful to assess for air, ventricular function, assess coronary arteries and VSD closure.

Usual Treatment
- Digoxin, diuretics, afterload reducing agents utilized in pts with CHF
- Palliative surgery
- Pulm artery banding may occasionally be performed in pts with large VSDs
- Pulm artery banding may be performed esp. in older pts prior to “double switch” (atrial and arterial) procedure to prepare the LV to accept systemic workload.
- Blalock-Taussig shunt (BTS) may be necessary in cyanotic pts with LVOTO and VSD
- Definitive surgery: Goal is to make LV the systemic pumping chamber.
- For pts with no RVOTO: “Double switch” Senning or Mustard procedure/arterial switch procedure: Combined atrial and arterial switch performed to allow left ventricle to become the systemic ventricle
- For pts with VSD and LVOTO: “Double switch” Senning or Mustard procedure/Rastelli procedure: Combined atrial switch and intra-ventricular Rastelli type VSD closure and RV-PA conduit placement

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<tr>
<td>CARDIO</td>
<td>CHF</td>
<td>Poor wt gain, FTT</td>
<td>Jugular venous distention</td>
<td>ECHO</td>
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<tr>
<td></td>
<td></td>
<td>Resp distress</td>
<td>Hepatomegaly</td>
<td>CXR</td>
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<tr>
<td></td>
<td></td>
<td>Poor perfusion</td>
<td>S1 gallop</td>
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<td>Rales</td>
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<td>Edema</td>
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<td></td>
<td>Cardiac murmur</td>
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<td></td>
<td></td>
<td></td>
<td>Clubbing</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>↓ Pulm blood flow</td>
<td>Cyanosis</td>
<td>Pulse oximetry</td>
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<td></td>
<td>AV</td>
<td>SOB</td>
<td>Cardiac murmur</td>
<td>ABG</td>
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<td></td>
<td>block</td>
<td>Exercise intolerance</td>
<td>Slow heart rate</td>
<td>ECHO</td>
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<td></td>
<td></td>
<td>Dizziness/syncope</td>
<td>CHF</td>
<td>EKG</td>
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<tr>
<td>RESP</td>
<td>Congestion/pulm edema</td>
<td>Frequent URLs</td>
<td>Decreased perfusion</td>
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<tr>
<td>HEME</td>
<td>Polycythemia</td>
<td>Bleeding</td>
<td>Plethora</td>
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<td></td>
<td>Coagulopathy</td>
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<td>Coagulation studies</td>
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<tr>
<td></td>
<td>Thrombocytopenia</td>
<td></td>
<td></td>
<td>TEG</td>
</tr>
</tbody>
</table>

Risk
- Incidence of pituitary adenoma: 2.9/100,000/y
- Prevalence: 16.7% of population

Perioperative Risks
- Mortality <1%
- Morbidity 3–5% (diabetes insipidus [DI], CSF leak, carotid artery injury, visual loss, meningitis, hemorrhage)

Worry About
- Endocrine abn (panhypopituitarism, Addison, Cushing’s, thyroid dysfunction)
- DI: increased UO, hypernatremia, dehydration
- Acromegaly (potential difficult mask airway and intubation, increased risk of sleep apnea)
- Increased ICP

Anticipate difficult airway if acromegaly present (may require awake FOB)

• Intracranial hemorrhage 2° to invasion into cavernous sinus

Overview
- Resection performed through nasal, sublabial incisions with aid of microscope
- Newer techniques often use fluoroscopic or MRI guidance, endoscopic approaches, intraop hormonal assays
- Tumors may secrete hormones (GH, ACTH, TSH, prolactin) or be nonfunctional
- Tumors may compress optic chiasm, causing visual field deficits, or may abut or invade cavernous sinus

ICD-9-CM Code: 227.3 (Benign pituitary adenoma)

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<tr>
<td>HEENT</td>
<td>Overgrowth of chin, tongue,</td>
<td>Snoring, sleep apnea</td>
<td>Airway exam</td>
<td>Lateral neck films FOB</td>
</tr>
<tr>
<td></td>
<td>vocal cord paralysis,</td>
<td>hoarseness</td>
<td>Stridor, facial</td>
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</tr>
<tr>
<td></td>
<td>subglottic stenosis</td>
<td></td>
<td>features</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(acromegaly)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>CARDIO</td>
<td>Htn, DM, CHF</td>
<td>Chest pain, dyspnea</td>
<td>CV exam</td>
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</tr>
<tr>
<td></td>
<td>Obesity, ischemia</td>
<td>Exercise tolerance</td>
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<tr>
<td>ENDO</td>
<td>Panhypopituitarism</td>
<td>Cold intolerance</td>
<td>Hemodynamic</td>
<td>GH, TSH, glucose</td>
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<td>Acromegaly (TGH)</td>
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<td>instability</td>
<td>Cortisol level</td>
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<td>Nervousness</td>
<td>CV collapse</td>
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<td>suppression test</td>
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<td>Insulin resistance</td>
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</tr>
<tr>
<td>RENAL/</td>
<td>↑ Aldosterone (ACTH) → ↑ Na*</td>
<td>Oliguria</td>
<td>Pulm/peripheral</td>
<td>ABGs, lytes</td>
</tr>
<tr>
<td>LYTES</td>
<td>↓ K*, metab alkalosis</td>
<td>Thirst, polyuria</td>
<td>edema</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ ADH → DI</td>
<td></td>
<td>Orthostasis,</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>hypotension</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>UO</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Assess and document neurologic deficits, evaluate endocrine function
- Assess for possible difficult intubation and sleep apnea (acromegaly, Cushing’s)
- Avoid sedation, esp. if concerns about increased ICP

Anesthetic Technique
- GA with controlled ventilation

Monitoring
- Routine monitoring plus Foley catheter to follow UO
- Consider arterial line to watch BP during local infiltration and to follow electrolytes periop
- Consider precordial Doppler, or TEE to detect VAE if head elevated >15°

Airway
- Anticipate difficult airway if acromegaly present (may require awake FOB)

• Routine oral intubation if normal airway, consider oral RAE ETT to facilitate surgery
• Packs placed in oropharynx intraop to prevent aspiration of blood and irrigating solution

Induction/Maintenance
- Similar to any craniotomy: balanced vapor, narcotic and relaxant
- Stress dose steroids, antibiotics to cover noso/oropharyngeal flora
- Minimal postop pain; avoid narcotics overdose

Surgical Stages

Incision
- Infiltration of nose and mouth with local anesthetic, cocaine may trigger Htn, tachycardia, arrhythmias

Definitive Surgery
- Watch for DI, hemorrhage from carotid artery or cavernous sinus
- Watch for venous air embolism; D/C NO if suspected

Postoperative Considerations
- Confirm oropharyngeal packing removed, potential for aspiration of blood from nasopharynx, extubate awake
- May need postop steroid replacement
- EBL variable, may be hard to quantitate
- Potential complications: CSF leak, epistaxis, sinusitis or meningitis, intracranial hemorrhage, pneumocephalus, nasal septum perforation

Anticipated Problems/Concerns
- DI 2° to lack of ADH: Increased UO, hypernatremia, increased osmolality, dehydration; treat with IVF, DDAVP as needed
- Hemorrhage from ICA or cavernous sinus can lead to herniation, CN dysfunction, death
- Watch closely for postop neurologic changes
- Addisonian crisis: Hemodynamic instability, CV collapse; treat with steroids
Transurethral Resection of Bladder Tumor

Risk
- Incidence in USA: 69,000 new cases in 2008.
- Fourth most common cancer in men.
- The ninth most common cancer in women.
- Race: Whites to African-Americans 2:1, but African-Americans have mortality rates twice that of whites; whites to Hispanic 2:1, but Hispanic have lower mortality rate than whites.
- Bladder cancer accounts for 3% of all cancer deaths in men and 1.5% in women.
- Gender: M:F ratio: 3:1, but women have 30% mortality higher than men.
- Risk factors: Smoking, age, chemical exposure, chronic accidents abuse, artificial sweeteners, chronic cystitis (calcium indwelling catheters, infection) pelvic irradiation.

Perioperative Risks
- Periop mortality low (<1%).
- Shedding of the tumor cells.
- Ureteral obstruction from tumor or tumor resection.
- Less risk of absorption syndromes than during TURP.
- Clinical bladder perforation and bowel injury.

Worry About
- Bladder perforation.
- Uncontrolled hematura.
- Peroneal and sciatic nerve neuropathy from lithotomy position.
- Obturator nerve stimulation that may lead to bladder perforation.

Overview
- TURBT is indicated as diagnostic and therapeutic procedures in a suspicious of bladder cancer.
- TURBT is relatively simple and quick procedures (30–60 min).
- Bladder cancer can occur at any given age, but the median age is 69 in men and 71 in women and the incidence of bladder cancer increase directly with age.
- Assoc with multiple co-morbidity due to late median age.

ICD-9-CM Code: 57.49 (Other transurethral excision or destruction of lesion or tissue of bladder)

Indications and Usual Treatment
- Most common presenting symptoms: Painless hematuria 85% and irritative symptoms (frequency, urgency, and dysuria).
- Dx tests incl urinary microscopic cytopathology (low sensitivity for low-grade tumors but with 80% sensitivity with high-grade tumors), CT with or without contrast, excretory urography only if CT is not available, cystoscopy.
- Procedure: TURBT is diagnostic by stage resection and therapeutic by resection and fulguration of all grossly visible tumors.
- Repetitive TURBT is common with recurrence or second opinion for muscle invasion.
- Other adjuvant Rx incl periop Mitomycin C (MMC) intravesical to prevent tumor cell implant.
- Combined intravesical chemotherapy with MMC and BCG, intravesical immunotherapy, photodiation therapy, laser therapy, cystectomy.

Perioperative Management

Preoperative Preparation
- Within normal complete blood count, serum electrolyte and coagulation parameters.
- Documented negative UA and culture.
- Intravenous prophylactic antibiotics.

Monitoring
- Standard ASA monitoring.
- Invasive monitoring as CVP depending on the co-morbidity and inability to measure the UOP.

Anesthetic Technique/Induction
- GA: Different induction agents based on medical status, muscle paralysis not required if the tumors on the bladder floor or dome but if the tumors at lateral bladder wall muscle paralysis is required because using electrocautery for resection may lead to stimulation of obturator nerve and subsequent bladder perforation from sudden leg contraction.
- Spinal anesthesia: T10 sensory level sufficient, higher level masks. Xs of perforation of bladder. Spinal anesthesia will not prevent obturator nerve stimulation. Obturator nerve block below pubic ramus if the resection at lateral wall of the bladder.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CAD, PV, CHF</td>
<td>Chest pain, SOB, palpitation, claudication, lower extremities edema</td>
<td>Cardiac exam, JVD, LE edema</td>
<td>ECG, ECHO, stress test</td>
</tr>
<tr>
<td>RESP</td>
<td>COPD</td>
<td>SOB, coughing</td>
<td>Prolonged expiratory phase, wheezing, rales</td>
<td>CXR, PFTs</td>
</tr>
<tr>
<td>RENAL</td>
<td>CKD Chronic UTI</td>
<td>Oliguria, hematuria, disuria, frequency, urgency</td>
<td>Bimanual examination of the bladder and pelvis</td>
<td>UA, BUN/Cr, GFR</td>
</tr>
<tr>
<td>CNS</td>
<td>Cerebrovascular disease, stroke</td>
<td>Kirkali I, Chan T.</td>
<td>exam</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Considerations
- Dorsal lithotomy position.
- Bimanual exam of the bladder is performed under anesthesia.
- Actual resection time usually <30 min.
- Pan-endoscopy to identify and locate the tumors as well the location of ureteric orifices.
- Water used as a bladder irritant during the procedures because of its cytotoly effects.
- IV absorption of irrigating solution rare because of minimal open veins, but lengthy one may lead to absorption of water cause electrolyte imbalances, volume overload and intravascular hemoysis (hemolysis can be avoided by using mannitol, sorbitol, or glycine all are more isotonic).
- Bipolar electroresection is reported to allow transurethral resection in saline (TURIS) as well decrease the risk of obturator reflex.
- Extra or intraperitoneal bladder wall perforation is rare, small perforation of no clinical significance can be treated with bladder drainage and antibiotic postop, but a symptomatic perforation or concern of bowel injury a laparoscopic or open exploration for repair is recommended.
- Avoid bladder overdistension to decrease the incidence of bladder perforation.

Surgical Stages
- Low incidence of tumor seeding after bladder perforation.
- Usually minimal blood loss.

Postoperative Considerations
- Pain and bladder irritation.
- Occult bleeding and clot formation in the bladder.
- Signs and symptoms of peritoneal irritation raise the flag of bladder perforation.
- Peroneal and sciatic nerve injury from lithotomy position.
- Observe for ureteral obstruction if resection near ureteral orifice.

Anticipated Problems/Concerns
- Disease of elderly (CAD, COPD, CKD).
- Sudden hypotension and loss of return of irri-gant suggested perforation.
- Lengthy surgery with change of mental status suggest electrolytes abn and volume overload.
- Occult blood loss (but bleeding and clot can be performed and may require evacuation).
- Be aware that obturator nerve stimulation occurs suddenly if NMB is gone.
Transurethral Resection of Prostate (TURP)

**Risk**
- Histologic evidence of BPH: 50% of men by age 50; 75% by age 80
- BPH clinically significant in 40–50% of these pts
- Approx 400,000/y performed
- Mortality 0.2%–6% (increases with age, co-morbidity)

**Perioperative Risks**
- Age-assoc co-morbidities (cardiac, pulm, cerebrovascular)
- TURP syndrome
- Bladder perforation, hypothermia, coagulopathy and/or DIC, sepsis
- Toxicity of irrigating fluid components

**Worry About**
- TURP SX: Spectrum of clinical, physiologic conditions resulting from absorption of irrigating fluid through exposed venous sinuses of surgical capsule
- Hypervolemia: 20 mL/min absorbed; average 45–60 min
- Neuraxial versus general anesthesia

**Overview**
- TURP SX: Related to surgeon’s experience, aggressiveness with electrocautery loop, size of gland, amount of tissue removed and irrigation used; manifestations from circulatory fluid overload, water intoxication, hyponatremia, pressure and volume of irrigant, duration of resection

**Indications and Usual Treatment**
- TURP appropriate for prostate gland volumes <40–50 mL; alternative approach if >80 mL
- Less invasive, morbidity, expensive c/w open prostatectomy
- Transurethral laser coagulation, microwave thermotherapy are new techniques that eliminate complication of hyponatremia; bipolar electrovaporization allows use of saline irrigation

**ICD-9-CM Code:** 600 (Benign prostatic hypertrophy)

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypervolemia/Htm, dysrhythmia, hypotension/CHF</td>
<td>Angina, palpitations, SOB</td>
<td>BP, HR, JVD, pedal edema, crackles, murmurs</td>
<td>EKG, ECHO, stress test, electrolytes</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>SOB</td>
<td>O, sat, crackles, dyspnea</td>
<td>CXR, ABG</td>
</tr>
<tr>
<td>GU</td>
<td>Hyponatremia, hypo-osmolality, ARF</td>
<td>CRF, DM, Htm</td>
<td>Pedal edema</td>
<td>[Na], osmolarity, Cr, BUN</td>
</tr>
<tr>
<td>CNS</td>
<td>Agitation, seizure, coma, death, transient blindness (glycine)</td>
<td>Mental status, burning smell</td>
<td>Mental status</td>
<td>Neuro exam</td>
</tr>
</tbody>
</table>


**Preoperative Management**

**Preoperative Preparation**
- Careful preop assessment of cardiac, pulm, renal dysfunction
- If neuraxial, R/O coagulopathy, metastasis to spine
- Preop Abx may reduce bacteremia, sepsis; prostate often colonized with bacteria, may enter blood by gland manipulation, opening of venous sinuses

**Monitoring**
- Standard monitors; O, sat may indicate fluid overload; EKG changes (up to 18% pts); assess temp to avoid hypothermia
- Amount of irrigating fluid, hydrostatic pressure, time
- Labs: Na+, osmolarity

**Anesthetic Technique/Induction**
- Neuraxial block (T10 sensory level) allows for neuro exam (HA, restlessness, AMS); no difference in EBL, postop cognitive function, mortality, c/w GA

**Surgical Stages**
- Specialized cystoscope (rectoscope) passed through urethra
- Surgical field visualized by continuous irrigation through resectoscope; distends bladder, washes out blood and tissue from field

**Dissection**
- Moveable electrocautery, cutting wire, loop passed through resectoscope
- Bladder perforation: Resectoscope, overdistention with irrigation; sudden hypotension, generalized abd pain; increased airway PIP

**Postoperative Considerations**
- Tx postop shivering; can dislodge clots, coagulopathy
- Perforations are extraperitoneal, signaled by poor return of irrigating fluids
- DIC can result from thromboplastins released from prostate into circulation; up to 6% of pts may have subclinical DIC; dilutional thrombocytopenia can develop
- TURP Sx can occur within 15 min and up to 24 hr postop

**Anticipated Problems/Concerns**
- Tx of TURP Sx—avoidance, early recognition key; absorbed water must be eliminated; Tx of mild hyponatremia with fluid restriction, loop diuretic; if severe, <115, consider hypertonic saline (cautious use, can result in additional fluid overload, complicating management; central pontine myelinolysis)
- Seizures with benzodiazepine, thiopental; consider ETT if AMS.
Risk
- Leading cause of death among young (1–45 y of age; >60% of all deaths)
- Incidence in USA: 1:10 (7% violent); 60/100,000 for fatal trauma (30% violent)
- Increased risk with concurrent alcohol or drug use
- 50% of trauma deaths are caused by hemorrhagic shock

Perioperative Risks
- Hemorrhagic shock, hypotension, side effects
- Massive transfusion
- Hypoxemia, hyper- or hypocarbia esp. in brain trauma pts
- Lethal triad of trauma: Acidosis, coagulopathy, and hypothermia
- High incidence (40–100%) of systemic inflammatory response syndrome (SIRS) in serious trauma
- Mortality after major trauma 10–25%

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Direct trauma to airway; airway obstruction; maxillofacial fractures</td>
<td>Often clearly visible</td>
<td>Airway Stridor; blood or vomitus in airway; limited ability to open mouth; raccoon eyes or CSF rhinorrhea</td>
<td>Inspection Auscultation X-ray; CT</td>
</tr>
<tr>
<td>RESP</td>
<td>Chest trauma; (tension) pneumothorax; serial rib fractures; flail chest</td>
<td>High degree of suspicion in all major trauma</td>
<td>Breathing Dyspnea, distended neck veins; reduced breath sounds; SQ emphysema; cyanosis (often absent in hemorrhagic shock)</td>
<td>CXR, ABG, CT</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Acute hemorrhage; shock; vascular injury; cardiac tamponade; cardiac contusion</td>
<td>Observed blood loss</td>
<td>Circulation Hypotension, tachycardia, absent pulses, diaphoresis, cold extremities, distended neck veins, UO</td>
<td>ECG, TTE, TEE, FAST, ABG, lactate, CT, CT angio,</td>
</tr>
<tr>
<td>RENAL</td>
<td>Kidney trauma; acute renal failure</td>
<td>Location; crush injury</td>
<td>Hematuria; myoglobinuria</td>
<td>UA; FAST</td>
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<tr>
<td>CNS</td>
<td>Head trauma, neck and spine injury</td>
<td>Unconsciousness</td>
<td>Disability GCS; paraplegia, pain</td>
<td>Head and neck CT</td>
</tr>
</tbody>
</table>


Perioperative Management

**Preoperative Preparation**
- Have emergency release blood set up (for major trauma: 10 PRBC, 10 FFP).
- Adequate preoxygenation is paramount before induction (low O2 reserve)
- Large-bore IV access (often a combination of central and peripheral lines) critical
- Have rapid infusion device and fluid warmer ready.
- Have emergency drugs ready (epinephrine, vasopressin, phenylephrine, norepinephrine).

**Monitoring**
- Standard ASA monitors plus Foley
- Invasive hemodynamic monitoring essential (arterial line); serial ABGs to assess resp and metabolic status
- Consider central venous access; CVP monitoring is of questionable relevance.
- Extended hemodynamic monitoring such as continuous cardiac output, SvO2, and Swan-Ganz catheter may be beneficial for select group of pts. Consider TEE.
- Consider advanced coagulation monitoring (TEG, ROTEM).

**Anesthetic Technique/Induction**
- Have difficult airway equipment, incl a primary surgical airway, ready as well as a functional suction device.
- Airway management is usually by orotracheal intubation and manual in-line stabilization of the C-spine.
- Rapid sequence induction is standard; induction agents that maintain hemodynamic stability are preferred (ketamine, etomidate).

**Fluid Resuscitation**
- Consider permissive hypotension until hemorrhage control has been achieved.
- After initial resuscitation with plasma isotonic crystalloids, switch to PRBC and FFP early.
- All fluids should be warmed.
- Fluid therapy can be guided by TEE or blood lactate values, which are highly indicative for hypovolemia and inadequate tissue oxygenation.
- When coagulopathy becomes clinically relevant, consider fibrinogen concentrate and plts as needed; monitor and treat hyperfibrinolysis when needed.
- Use of buffering agents (bicarbonate) to treat metabolic acidosis is controversial.

**Maintenance**
- Low-dose inhalational agent may be used if hemodynamically tolerated and when brain trauma and intracerebral pressure is not a concern.
- IV anesthestia may consist of opioids and benzodiazepines.

- Pts with multiple trauma are more likely to die from hemorrhagic shock and metabolic failure than from a failure to complete operative repairs.
- Principles of damage control surgery are control of hemorrhage, prevention of contamination and protection from further injury.

**Indications and Usual Treatment**
- Immediate identification of hemorrhage employing US (FAST sonography) and whole body CT scan is crucial.
- Life-saving interventions such as chest tubimg, emergent thoracotomy or laparotomy may be indicated in critical pts even without further workup or imaging.
- In penetrating or violent trauma pts, large vessel injury is a major concern.
Tubal Ligation

Overview
• Tubal ligation is a permanent form of female sterilization, in which the fallopian tubes are severed and sealed or occluded to prevent future fertilization.

Oclusion methods incl:
• Partial salpingectomy: Suture ligation of a small loop of fallopian tube and then removing the interval segment of the loop
• Clips: Clamp the fallopian tubes, thus inhibiting blood flow and causing a small amount of scarring or fibrosis
• Tubal rings: Similar to clips, mechanically blocking the fallopian tubes
• Electrocoagulation/cauterization: Coagulation/burning a small portion of each fallopian tube

• Minilaparotomy: Advantages incl lower rate of serious complication (bowel laceration, vascular injury), less technical surgical expertise required, and hospital stay in postpartum women is not extended
• Laparoscopy: Advantages incl decreased operative time, less postop pain, shorter hospital stay, and more rapid return to normal functional activities

Anesthetic Technique
• Can be performed under local, regional, or GA

Monitoring
• Routine ASA monitors

Airway
• Postpartum pts may have unexpected upper airway edema esp. after a lengthy second stage of labor

Induction
• Local anesthetic technique under IV sedation has been described by skilled surgeons with injection of local anesthetic directly into the wound, then bathing parietal and visceral peritoneum with small volumes of lipid-soluble local anesthetic, and finally injecting the mesosalpinx before fallopian tube ligation.
• In the postpartum period, regional anesthesia (epidural or spinal) is preferred to reduce the risk of failed airway and/or aspiration
• General anesthesia (GA) with controlled ventilation avoids hypercarbia from the mechanical and chemical effects of peritoneal CO₂ insufflation during laparoscopy
• Laparoscopy may be contraindicated if the patient has a history of abdominal surgery and known, extensive intra-abdominal adhesions.

ICD-9-CM Codes: V25.2 (Sterilization); 659.40 (Grand multiparity with current pregnancy unspecified as to episode of care)

Indications and Usual Treatment
• Multiparous pt desiring sterilization
• Medical contraindication to pregnancy (appropriate advance consent must be obtained with re-affirmation at the time of the procedure)

Risk
• 700,000 performed annually in USA as either postpartum mini-laparotomy or interval laparoscopy
• Mortality in USA is 4/100,000 tubal ligations

Perioperative Risks
• Minor risks: Infection, bleeding, bruising, abn or painful scar formation, allergic skin reaction to tape, dressings and/or latex, delayed return of bowel and/or bladder function
• Major risks: Serious bleeding, serious infection, damage to organs (uterus, fallopian tubes, ovaries, bladder, and/or ureters), damage to the intestines (perforation and/or burn injury), nerve injury, blood vessel injury, blood clots, pulm embolus, myocardial infarction, adverse reaction to medications or anesthesia

Worry About
• Postpartum: Airway: aspiration risk (administration of opioid increases the likelihood of delayed gastric emptying in the early postpartum period); hypovolemia (postpartum hemorrhage, uterine atony)
• Interval: Complications of laparoscopy (bowel laceration, vascular injury, hypercarbia, gas embolism, adverse hemodynamics, pneumoperitoneum, Trendelenburg’s position, conversion to laparotomy)
• Peripheral nerve injury due to positioning on the OR table

Monitoring
• Can be performed under local, regional, or GA
• Expected failure rate to achieve an adequate surgical level of up to 25% predisposes practitioners to remove the epidural catheter immediately after delivery and then later induce spinal anesthesia (with 10–12 mg of bupivacaine with or without spinal opioid) in pts desiring regional anesthesia. Other clinicians choose to leave an epidural catheter placed during labor in place.
• If the epidural catheter used for labor analgesia is left in situ: Inspect the catheter site, administer a test dose to confirm nonintraoperative and non-subarachnoid position of the catheter tip, and then extend the sensory level by injecting incremental doses (cumulative 18–24 mL) of local anesthetic with a concentration suitable for surgical anesthesia (2% lidocaine with added epinephrine 1/200,000, 3% 2-chloroprocaine, 0.5% ropivacaine)

Perioperative Management

Preoperative Preparation
• Assess intravascular volume status and hydrate as necessary.
• NPO status: A pt planning to have an elective postpartum tubal ligation should have no oral intake of solid foods within 8 hr of surgery
• Postpartum pts are often given acid-aspiration chemoprophylaxis, incl nonparticulate antacid, H₂-receptor antagonist, and/or metoclopramide
• Pts with pregnancy-induced Htn and/or preeclampsia may safely receive regional anesthesia for postpartum tubal ligation provided that there is no evidence of pulm edema, oliguria, uncontrolled Htn, or thromboembolism; however, the clinical team should carefully weigh the risk of worsening maternal condition in a pt undergoing elective surgery

ASSESSMENT POINTS*

<table>
<thead>
<tr>
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<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT (AIRWAY)</td>
<td>Airway edema</td>
<td>Difficult airway</td>
<td>Airway exam</td>
<td>Hct, pltS</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Hemorrhage</td>
<td>HR, BP (orthostatics)</td>
<td>Hct, pltS</td>
</tr>
<tr>
<td>GI</td>
<td>Gastric volume</td>
<td>Heartburn</td>
<td>Opioids during labor</td>
<td>Wt, BMI</td>
</tr>
<tr>
<td></td>
<td>↓ Gastric pH</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Lower esophageal sphincter tone</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Uterine atony, chorioamnionitis, endometritis, UTI, Pre-eclampsia</td>
<td>Postpartum hemorrhage, fever, diaphoresis</td>
<td>HR, BP (orthostatics), foul lochia, BP, proteinuria, oliguria</td>
<td>Hct, pltS, ↑ WBC, temp</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Headache, visual disturbances, epigastric pain, edema</td>
<td>thrombocytopenia, hemolysis, edema</td>
<td>UO, Hct, pltS</td>
</tr>
</tbody>
</table>
• GA for postpartum pts: Most practitioners still use a rapid sequence induction technique with cricoid pressure and maintain anesthesia with controlled ventilation and IV anesthetic adjuvant drugs, avoiding high-inspired concentrations of volatile halogenated agent for fear of inducing uterine atony/postpartum hemorrhage

• Laparoscopic technique: Deflate the stomach before trocar insertion, obtain large-bore IV access, hyperventilate to maintain normocarbia, reassess ventilation after insufflation (peak airway pressure, minute ventilation, ETCO₂)

Surgical Stages
• Postpartum minilaparotomy: Small infraumbilical incision
• Interval laparoscopy: Introduction of trocars to peritoneal cavity, insufflation of CO₂, and insertion of laparoscope and instruments
• Mini-laparotomy and laparoscopy: Fallopian tubes identified and severed or occluded
• EBL: Minimal (10 mL)
• Duration of surgery: Ideally <30 min

Postoperative Considerations
• Pain: Moderate and of short duration, typically treated with parenteral opioids and oral analgesics
• Local anesthetic infiltration of the mesosalpinx or topical application of a local anesthetic to the fallopian tubes by the surgeon significantly decreases postop opioid requirements
Ureteral Reimplantation

**ASSESSMENT POINTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT/RESP</td>
<td>Generally uninvolved</td>
<td>Snoring</td>
<td>Exercise tolerance</td>
<td>Routine airway exam and</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>lung auscultation</td>
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<tr>
<td>CARDIO</td>
<td>Htm</td>
<td></td>
<td></td>
<td>Measure BP</td>
</tr>
<tr>
<td>IMMUNE</td>
<td>Possible latex allergy in pts</td>
<td>Hx of rash, hives, or anaphylaxis</td>
<td></td>
<td>Electrolytes, BUN/Cr</td>
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<tr>
<td></td>
<td>with spinal dysraphism or</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>extrophy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Possible renal insufficiency</td>
<td>CNS Hx, incl Hx of bladder or bowel dysfunction</td>
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<td>MRI of spine</td>
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<tr>
<td></td>
<td>or RTA</td>
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<tr>
<td>CNS</td>
<td>Weakness or paralysis with</td>
<td>CNS exam</td>
<td>Sacral dimple or hair</td>
<td></td>
</tr>
<tr>
<td></td>
<td>spinal dysraphism</td>
<td></td>
<td>tuft</td>
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</tbody>
</table>


**Perioperative Implications**

**Preoperative Preparation**
- Appropriate fasting interval and premedication (e.g., rectal, oral, IM, or IV midazolam) given pt age and level of preop anxiety
- Routine ECG, noninvasive BP monitoring, pulse oximetry, capnography, temp

**Airway**
- General endotracheal anesthesia for open procedures, while general anesthesia via laryngeal mask airway is generally sufficient when endoscopic surgery is performed

**Induction**
- Mask or IV induction depending on pt age, preference, and risk factors (e.g., GERD)

**Maintenance**
- General endotracheal anesthesia alone or a combined technique (general anesthesia with a single shot caudal or caudal and/or lumbar epidural catheter inserted following induction) are both effective intraop.

**Overview**
- Ureteral reimplantation is usually performed to treat high-grade (Grade III-V) vesicoureteral reflux (VUR), where reflux is assoc with calyceal blunting and, at times, ureteral dilatation
- Untreated VUR can result in chronic and/or recurrent UTIs, renal scarring, renal insufficiency, Htm, and impaired somatic growth; renal failure is uncommon (estimated risk <1%)

**Etiology**
- VUR produces retrograde flow of urine from the bladder through the ureter and, at times, into the kidney
- Dx of VUR is generally made by renal US and voiding cystourethrogram
- 1° VUR is a congenital anomaly resulting in development of an inadequate valvular mechanism at the ureterovesical junction
- 2° VUR is caused by anatomic (e.g., posterior urethral valves or ureteroceles) or functional (e.g., neuropathic bladder or bladder instability) bladder outlet obstruction

**Usual Treatment**
- Most pts are initially managed medically (low-dose prophylactic antibiotics), as 70–90% of low-grade reflux will resolve spontaneously as pt grows
- Indications for surgery incl breakthrough UTIs while on antibiotics, noncompliance, incomplete, increasing or severe reflux (Grades IV and V), deteriorating renal function, persistent reflux in females approaching puberty, and assoc congenital anomalies of the ureterovesical junction

**Postoperative Period**
- Postop length of stay ranges between 1 and 6 d for open ureteroneocystostomy
- Minimally invasive surgical techniques may shorten length of stay, diminish acute postop pain, and decrease the likelihood of inducing urinary retention
- Some minimally invasive extravascular repairs are being performed on an outpatient basis, as are endoscopic Deflux® injections

**Perioperative Risks**
- Mortality extremely rare
- Minimal periop blood loss
- Postop ureteral obstruction (caused by edema, bleeding or blood clots, bladder spasms, or ureteral ischemia) can occur; usually asymptomatic and resolves spontaneously
- Symptomatic obstruction can present with abd pain, N/V, but is usually diagnosed on postop follow-up
- Disruption of bladder innervation possible, esp. with extravesical repair

**Worry About**
- Most pts are healthy children (ASA I-II), but some pts requiring procedure will have significant congenital anomalies (e.g., spinal dysraphism) or GU anomalies (e.g., UPJ obstruction, ureteral duplication, bladder diverticula, posterior urethral valves, bladder or cloacal exstrophy)

**Risk**
- Incidence: 1–3% of healthy children; 30–50% of children with symptomatic UTIs
- Gender predominance: Female
- Racial predilection: Caucasian
- Age: Dx antenatal or in childhood, with majority of surgical procedures performed by 5–6 y of age
- Genetic factors: Unknown but suggested by 30–35% incidence in siblings, and 67% incidence in offspring of affected individuals

**PROCEDURES**

**Regional anesthesia alone is not generally advocated given pt age**
- Combined techniques are not employed in pts with assoc spinal dysraphism
- Local infiltration of the skin incision can be performed before skin closure if a regional technique is not incl in intraop management

**Extubation**
- Generally attempted at the conclusion of surgical procedure

**Adjuvants**
- Ketorolac has been shown to provide analgesia and decrease the frequency and severity of bladder spasms following ureteroneocystostomy

**Postoperative Period**
- Postop length of stay ranges between 1 and 6 d for open ureteroneocystostomy
- Minimally invasive surgical techniques may shorten length of stay, diminish acute postop pain, and decrease the likelihood of inducing urinary retention
- Some minimally invasive extravasacular repairs are being performed on an outpatient basis, as are endoscopic Deflux® injections

**Postoperative Pain Management**
- Postop pain can be incisional or related to bladder spasms, and may require treatment with oral or IV opioids, anticholinergics and bladder smooth muscle relaxants (e.g., oxybutinin and dazepam), NSAIDs, and/or epidural local anesthetic/opioid infusions
- When outpatient surgery is contemplated, pts must meet specific criteria prior to discharge, incl adequate pain control, tolerance of a regular diet, ability to void postop (or comfort with the care of an indwelling urethral catheter), and the ability to ambulate without difficulty
- Complications of postop pain management can incl ileus, resp depression, sedation, emesis, and urinary retention
- Postop pain/bladder spasms may persist after discharge and require treatment with oral opioids, acetaminophen, NSAIDs, oxybutynin and/or dazepam

**Indications**
- Significant reflux (VUR), where reflux is assoc with calyceal blunting and, at times, ureteral dilatation
- Untreated VUR can result in chronic and/or recurrent UTIs, renal scarring, renal insufficiency, Htm, and impaired somatic growth; renal failure is uncommon (estimated risk <1%)

**Extravesical procedures (e.g., modified Lich-Gregoir ureteral reimplantation) have a similar success rate to the intravesical approach.** They leave the bladder intact, lessening the risk of urinary contamination, bladder spasms, and hematuria, but concerns exist about disrupting bladder innervation and causing urinary retention with bilateral reimplantation

**Intravesical and extravasricular repairs through small suprapubic incisions (2–4 cm) can be attempted if minimally invasive surgical techniques are utilized.**

**Risk**
- Pts with spinal dysraphism or GU anomalies (e.g., UPJ obstruction, ureteral duplication, bladder diverticula, posterior urethral valves, bladder or cloacal exstrophy)

**Key Reference:** Constance L. Monitto
Ureteral Stent Placement

Risk
- Incidence of unilateral ureteral obstruction: acute 1:1,000; chronic 5:1,000
- Incidence of bilateral ureteral obstruction: acute 5:10,000; chronic 1:1,000
- M:F ratio: 1:1
- Performed on pts of all ages

Perioperative Risks
- Extremely low mortality (<0.1%)
- Serious complications: 4% of pts
  - Perforation of ureter or adjacent visceral structures
  - Renal hemorrhage requiring transfusion
  - Minor complications: 10% of pts
  - UTI from instrumentation of the urinary tract
  - Irritative bladder symptoms
  - Microscopic hematuria
  - Flank or loin pain from vesicoureteral reflux or stent coiling

Perioperative Management

Preoperative Preparation
- Ensure NPO status, evaluate for volume and electrolyte disturbances
- Establish IV access, consider IVF if signs/symptoms of IV are present
- Anxiolysis and pain control for pt comfort; important for pts with stones

Monitoring
- Standard noninvasive ASA monitors in most cases
- Consider arterial cannulation in critically ill or paraplegic or quadriplegic pts at risk for autonomic hyperreflexia
- Rarely CVP monitoring in pts with acute volume overload or dialysis dependence

Anesthetic Technique/Induction
- General anesthesia most common. Routine induction with inhalational or IV maintenance, consider NMB given that ureters are thin and prone to injury if the pt moves or coughs during the procedure. ETT for secure airway but may consider LMA for short procedures
- Spinal anesthesia: Levels around T8 are desirable
- Epidural anesthesia: May require supplemental IV sedation

Surgical Stages
- Cystoscope insertion: Potential for pt discomfort and/or sympathetic stimulation
- Guidewire introduction into ureter with threading of stent. Fluoroscopic guidance is occasionally required; important to prevent pt movement to avoid perforation of bladder or ureter by ureteroscope
- Majority of stenting procedures are short, typically 15–45 min in duration

Postoperative Considerations
- Pain management: Usually mild discomfort, small doses of IV opioids titrated to effect, ketorolac or prazosin for relief of bladder spasm
- N/V: Not a high risk procedure, consider individual pt risk factors

Anticipated Problems/Concerns
- Lithotomy position: Risk of peroneal nerve injury
- Occasional need for percutaneous placement if endourological approach fails

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Hx of N/V, orthostasis, oliguria</td>
<td>Orthostatic BP, mucous membranes</td>
<td>Electrolytes, H/H</td>
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<tr>
<td>RESP</td>
<td>Pulm edema</td>
<td>Dypsnea, anuria</td>
<td>Crackles, rales, peripheral edema</td>
<td>CXR</td>
</tr>
<tr>
<td>RENAL</td>
<td>Chronic renal insufficiency, acute renal failure</td>
<td>Renal colic pain, oliguria or anuria, recurrent UTI, hematuria or proteinuria</td>
<td>Flank pain, peripheral edema</td>
<td>Urinalysis, BUN, Cr, plasma and urine electrolytes, renal U/S, IVP</td>
</tr>
<tr>
<td>CNS</td>
<td>Uremia, electrolyte disturbances</td>
<td>Obtundation, coma, seizures</td>
<td>Weakness, asterixis, hyperreflexia, tetany</td>
<td>Serum electrolytes, BUN/Cr</td>
</tr>
</tbody>
</table>

Vaginal Delivery, Normal

**Risk**
- Incidence in USA: ~4 million live births per year

**Peripartum Risks**
- Maternal mortality ↓: 12.1 deaths/100,000 live births in 2003 compared with 607.9/100,000 in 1915
- Perinatal mortality rate also ↓: 6.23/1000 live births in 2003
- Thromboembolism, hemorrhage, hypertensive disorders, infection remain common causes of maternal mortality and morbidity
- Decline in anesthesia-related causes noted (UK data)

**Worry About**
- Supine hypotension syndrome
- Difficult airway

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</thead>
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<tr>
<td>CARDIO</td>
<td>↑ CO, ↓ SVR</td>
<td>Previous GA</td>
<td>BP, HR</td>
<td>ECHO, ECG</td>
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<tr>
<td>RESP</td>
<td>Edema, ↑ soft tissue</td>
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<td>Airway exam</td>
<td>None</td>
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<tr>
<td>HEME</td>
<td>↑ Plasma vol &gt; ↑ RBC mass</td>
<td>Sx of easy fatigue with</td>
<td>None specific</td>
<td>CBC: Occasional ↓ plt in</td>
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<tr>
<td></td>
<td></td>
<td>significant anemia</td>
<td></td>
<td>normal preg</td>
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<tr>
<td>HEPAT/RENAL</td>
<td>Significant changes if pregnancy</td>
<td>Epi gastric pain, N/V,</td>
<td>Epigastric</td>
<td>BUN, Cr, LFTIs, UA</td>
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<tr>
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<td>complicated by PH</td>
<td>headache</td>
<td>tenderness,</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>hyperreflexia</td>
<td></td>
</tr>
</tbody>
</table>

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**Indications and Usual Treatment**
- Labor analgesia
- Lumbar epidural
- Spinal
- Combined spinal/epidural
- Parenteral opioids
- Others: Psychoprophylaxis, TENS, hypnotics, inhalational analgesia
- Operative delivery
- Spinal anesthesia
- Epidural anesthesia
- Continuous spinal analgesia
- General anesthesia
- Local anesthesia
- Bilateral pudendal nerve block
- Spinal/epidural anesthesia
- Neonatal resuscitation, esp. in situations of non reassuring fetal heart tracings, meconium-stained amniotic fluid

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**Intrapartum Management**
- Parturients in active labor should consider brief interview with anesthesiologist; emphasis on airway exam, previous anesthetic experience, co-morbid conditions
- Establish fetal status by cardiotocography, relevant prenatal test
- Antacid prophylaxis before any anesthetic intervention
- Establish IV access; consider preload before regional procedure; maintain left uterine displacement at all times

**Monitoring**
- Baseline pulse, BP, temp
- Following regional technique, monitor hemodynamics aggressively for the 1st 30 min; then at 30-60 min intervals
- Equal attention to fetal status at induction, during maintenance of regional analgesia for labor

**Labor Analgesia**

**Lumbar Epidural**
- Local anesthetics, opioids alone or in combination; low-dose, ultra-low dose (0.04% bupivacaine) solutions; latter allow for consideration of ambulation during labor as incidence of motor blockade is low
- Complications
  - Hypotension
  - Inadequate analgesia
  - Dural puncture headache
  - Subarachnoid block
  - Subdural block
  - Nerve damage (rare)
- Contraindications
  - Coagulopathy
  - Infection
  - Pt refusal

**Spinal**
- Intrathecal drugs
- Opioids: Sufentanil, fentanyl, morphine commonly used; addition of bupivacaine 2.5 mg may improve quality, duration of analgesia but increase incidence of motor weakness
- Much ↓ incidence of spinal headache since introduction of pencil-point needles

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**Parenteral Opioids**
- Maternal N/V, sedation
- Decreased beat-to-beat variability in FHR
- Risk of neonatal depression
- Despite low efficacy, remain most common form of labor analgesia

**Anticipated Problems/Concerns**
- Airway: ↑ Incidence of difficult and/or failed intubation with resultant hypoxemia, aspiration
- Water deliveries
- Aortocaval compression
- Peripartum hemorrhage
- Effect of interventions on fetus
- Postpartum neuropathy
- 20–25% of all planned normal spontaneous vaginal deliveries go to C-section
Venous Air Embolism

**Risk**
- Pts with operative field right heart gradient of >5 cm
- Probe patent foramen ovale in 25% of adult population
- Pts for laparoscopic surgery
- Incidence of VAE in children same as adults

**Perioperative Risks**
- Periop mortality <1%, but depends on early detection
- VAE: 40–80% in sitting, 10% in prone, 15% in supine, and 8% in lateral position
- Paradoxical air embolism (as high as 12%)
- Children suffer greater hemodynamic derangements

**Worry About**
- Pulm venous outflow obstruction
- CV collapse
- Paradoxical air embolism

### Overview
- Lethal volume of venous air in adult: 100–300 mL (two cardiac stroke volume)
- Entrained venous air can cause
  - Right ventricle outflow obstruction
  - Paradoxical arterial embolism (coronary embolism, stroke)
- Multiorifice catheter is preferable; the tip must be placed 2 cm below the SVC-atrial junction
- 75% N₂O can increase air bubble size about 3-fold
- Sensitivity for detection of VAE: TEE > precordial Doppler > PAP and ETCO₂ > CVP >BP > ECG
- Mill-wheel murmurs late, catastrophic sign
- PA catheter may provide prognostic information

**ICD-9-CM Codes**: 958.0; 673.0 (Obstetrical)

### Indications and Usual Treatment
- Operative site elevated to gain better exposure, blood drainage
- When VAE occurs
  - Notify surgeon of episode
  - Turn off N₂O
  - Gently apply bilateral jugular vein compression
  - Inflate MAST
  - Aspirate air from central catheter
  - CV support
  - Left decubitus position (Durant’s maneuver)

### Intraoperative Management
- Depends on pathology
- When VAE occurs
  - Notify surgeon of episode
  - Turn off N₂O
  - Gently apply bilateral jugular vein compression
  - Inflate MAST
  - Aspirate air from central catheter
  - CV support
  - Left decubitus position (Durant’s maneuver)

### Postoperative Considerations
- Possible hypoxemia from pulm infarction
- Possible stroke from paradoxical air embolism
- Possible cardiac arrest from massive venous air or paradoxical air embolism

### Anticipated Problems/Concerns
- Hypoxemia, ARDS (late sequelae of massive air embolism and resuscitation)
- Stroke
- MI

### Key Reference:
Ventricular Septal Defect, Repair of

**Overview**
- Spontaneous closure commonly occurs in muscular defects before age 5. Size may predict closure rate: Defects up to 5 mm rarely require surgery whereas defects 6.5 mm or larger almost always require surgery.
- Pulm Htn can be seen by age 1 y if large VSD, multiple VSDs, or PDA exists; Eisenmenger’s syndrome seen in second decade of life
- Goal of surgical therapy is to prevent pulm vascular obstructive disease and to treat intractable CHF assoc with FTT
- Factors determining time of repair incl
  - Degree of L→R shunting
  - CHF unresponsive to medical therapy
  - Increased pulm vascular resistance

**ICD-9-CM Code:** 745.4

**ICD-10-CM Code:**
- **Type I:** 745.40
- **Type II:** 745.41
- **Type III:** 745.42
- **Type IV:** 745.43

**Indications and Usual Treatment**
- Infant and child: CHF and FTT. Infants with trisomy 21 are at particular risk for pulm Htn and should have their VSDs closed early.
- Adults: Unrepaired small defect without evidence of left ventricular volume overload require endocarditis prophylaxis and periodic follow-up. Those with left ventricular volume overload or progressive aortic valve disease usually require closure.
- Other indications for surgical closure incl infundibular defects, chamber enlargement, increasing PVR (>6 wood units/m² despite administration of a pulm vasodilator considered inoperable), and multiple “Swiss cheese” defects refractory to medical management.
- Medical management of symptomatic CHF incl digoxin, diuretics, and after-load reduction agents.
- Surgical closure is performed using cardiopulmonary bypass. Surgical approach depends on defect location but is usually via a right atrial approach.
- Pulm artery banding via a partial sternotomy or right thoracotomy may be used for small infants who are symptomatic, to allow for growth and easier technical repair later. Banding may also be used when there are multiple muscular VSDs and in pts at high risk for cardiopulmonary bypass.
- Transcatheter closure of VSDs has been performed as an alternative to surgery as well as intraop for difficult defects such as multiple muscular VSDs and postop for residual VSDs. Major limitation to this technique is the size of the sheath necessary for device delivery, which precludes its use in infancy.
- Heart and lung transplantation is the only option for end-stage Eisenmenger’s syndrome.

**System**

- **CARDIO**
- Small defect L→R shunt
  - Trivial L → R shunting
  - Incidental murmur found by pediatrician
  - Left parasternal holosystolic murmur
  - Smaller defects are loudest and may have a thrill.
  - Hash pansystolic murmur.
  - Larger defects may have murmurs of constant quality that vary little throughout the cardiac cycle.

- **Large defect L→R shunt**
  - Significant L → R shunting, ↑ PAP
  - FTT, dyspnea, feeding difficulties, recurrent pulm infections

- **Large defect R→L shunt or no shunt**
  - Varying degrees of R→L shunting 2° to pulm vascular obstructive disease
  - Eisenmenger syndrome often cyanotic with clubbing
  - May not have a murmur but may have a loud pulm component of the second heart sound

**Assessment by Hx**
- **CXR normal**
- **ECG normal or may show mild LVH**
- **TTE, TEE, 3D ECHO, MRI cardiac catheterization**

- **CXR: Cardiomegaly, increased pulm vascularitiy**
- **ECG: Biventricular hypertrophy, notched P waves**
- **TTE, TEE, 3D ECHO, MRI cardiac catheterization**

**PE**
- **CXR normal**
- **ECG normal or may show mild LVH**
- **TTE, TEE, 3D ECHO, MRI cardiac catheterization**

**Test**
- **CXR: Right chamber enlargement, dilated main pulm artery, loss of pulm vascularitiy**
- **ECG right axis deviation, RVH TTE, TEE, 3D ECHO, MRI, cardiac catheterization**


**Perioperative Implications**

**Preoperative Preparation**
- Optimal control of CHF
- Premedication
- IV induction or inhalational induction in children
- Airway
  - ETT. PPV may limit degree of L → R shunting

**Maintenance**
- Medium to high-dose opioid with NMB and an inhalational agent
- Avoid maneuvers that excessively lower PVR (hyperventilation, anemia)
- Avoid myocardial depression

**Exubation**
- VSD repair: Extubate in ICU when hemodynamically stable (weaned from inotropes, free of arrhythmias, normothermic, etc.)
• If pt with unrepaired VSD is undergoing non-cardiac surgery, extubate at end of procedure if overall condition good. Avoid worsening L→R shunting (hypoxia, pain, shivering) or worsening CHF (excess fluid administration).

**Adjuvants**

• If PVR is increased, a phosphodiesterase inhibitor (milrinone) or NO may be used to decrease PVR on separation from bypass.

• O₂, furosemide, digoxin for continued CHF
• Inotropic support often necessary
• Temporary pacemaker for heart block

**Postoperative Period**

• Pain management critical
• Pts with increased PVR may require continuation of pulm vasodilators and aggressive diuresis for 48–72 hr.

• Complete heart block that does not resolve by 7–10 d is treated with a permanent pacemaker.

**Anticipated Problems/Concerns**

• Air embolism, shunt reversal, RV failure 2° to pulm Htn
• Pts with large VSDs, CHF, or FTT, are at greatest risk and difficult to wean from bypass.
Ventriculoperitoneal Shunt

Risk
- Elevated ICP
  - Congenital (e.g., intraventricular hemorrhage, meningoencephalocoele, Chiari malformations)
  - Aqueductal stenosis, brain tumors, infection and trauma
  - Shunt malfunction requiring revision (can be as high as 40% of shunt procedures)
  - Assoc with Chiari malformations
  - Overproduction of CSF
  - Normal ICP
  - Assoc dementia, gait disorders in elderly
  - Gender predominance: None

Worry About
- Prevent further elevations in ICP, which can lead to herniation syndromes
- Ventricular dysrhythmias assoc with rapid removal of CSF
- Assoc pathology

Overview
- Procedure to divert CSF from ventricles to peritoneum
- Many pts present with a previously placed external ventricular drainage devise allowing for intracranial pressure (ICP) monitoring if needed
- Proximal catheter passed into lateral ventricle through burr hole, preferably on the right to reduce risk of dominant hemisphere injury
- Distal catheter tunneled SQ; multiorificed tip placed in peritoneum

Indications and Usual Treatment
- Clinical and radiographic evidence of elevated ICP and/or shunt malfunction
- Hx of previous shunts
- Pseudotumor cerebri
- Normal-pressure hydrocephalus with demonstrated improvement in Sx with large-volume lumbar puncture
- If multiple failed ventriculoperitoneal shunts, ventriculojugular, atrial, or pleural shunt may be placed
- Hx of subarachnoid hemorrhage

ICD-9-CM Code: 331.4 (Hydrocephalus: acquired)

Perioperative Risks
- Periop mortality rare
- Intracranial bleeding may occur with placement of proximal tubing

Perioperative Implications
Anesthetic Technique
- GA usual
Monitoring
- Routine
- Consider arterial line in cases of uncontrolled ICP, hemodynamic instability
Airway/Induction
- Normal ICP: IV or mask induction adequate
- Elevated ICP: Atropine (in children), preoxygenate, eriocid pressure, thiobarbiturate, narcotic, lidocaine, rapid-acting nondepolarizing muscle relaxant followed by hyperventilation

Surgical Stages
Positioning
- Table turned 90°, head to surgeon
- Head turned 30° from neutral, bump placed under shoulder ipsilateral to shunt
Dissection
- Small flap turned in parietal region with subsequent burr hole
- Small abd incision, enters peritoneum. Laparoscopic assistance by general surgery is becoming more common in placing the distal portion of the catheter.
- SQ tunnel tracked to pull distal catheter through; can be stimulating, assoc with ↑ anesthetic needs

Definitive Surgery
- EBL: Minimal
- Rapid decompression can be assoc with tachydysrhythmias, hypotension
- Ventriculoatrial shunts can be complicated by air embolism

Postoperative Considerations
- Pt remains flat to avoid over drainage of CSF
- Assoc with minimal pain (pain score: 2)

Anticipated Problems/Concerns
- Elevated ICP with assoc hemodynamic changes
- Shunt misplacement with continued elevated ICP and assoc hemodynamic and neurologic changes

Whipple Procedure (Pancreatico Duodenectomy)

Overview
- Most commonly performed cancer-directed operation for pancreatic cancer
- Distal stomach, gallbladder, common bile duct, head of pancreas, proximal jejunum, duodenum, regional lymphatics removed
- Requires pancreaticojejunosomony, choledochojejunostomy, gastrojejunostomy
- Risk factors incl diabetes, cigarette smoking, alcohol ingestion, familial chronic pancreatitis
- Age, co-morbidities of pt will dictate periop monitoring strategies
- Significant blood loss and fluid shifts with extended Whipple resections (more extensive soft tissue, lymphatic dissections, resection of superior mesenteric vessels, portal vein if necessary)
- ~70% of tumors of head of pancreas unresectable at time of exploratory laparotomy
- Adoption of laparoscopic approach slow due to anatomic complexity of pancreatic surgery

ICD-9-CM Code: 157.0 (Cancer of pancreas)

Indications and Usual Treatment
- Following conditions should be met
  * All evidence of gross tumor can be resected
  * No evidence of distant metastatic disease or extensive vascular or retroperitoneal involvement by work-up
  * Good general health
- Palliative operations directed to relief of obstructive jaundice (cholecytostomyjejunostomy, cholecdochojejunostomy), gastric outlet obstruction (gastrojejunostomy), pain (celiac plexus injection with ethanol)
- 5–y survival ~20% after curative resection
- Combined radiotherapy and chemotherapy have been shown to 5 survival in pts with resectable, unresectable disease

ASSESSMENT POINTS

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<tbody>
<tr>
<td>GI</td>
<td>Gastric outlet obstruction</td>
<td>Vomiting</td>
<td>Abd mass</td>
<td>CT scan</td>
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<tr>
<td>HEPAT</td>
<td>Liver metastases</td>
<td>Pruritus</td>
<td>Hepatomegaly</td>
<td>CT scan</td>
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<td>Bile duct obstruction</td>
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<td>Jaundice, hepatomegaly, palpable nontender gallbladder</td>
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<tr>
<td>NUTRITION</td>
<td>Tumor Malnutrition</td>
<td>Wt loss</td>
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<tr>
<td>ENDO</td>
<td>Tumor</td>
<td></td>
<td>Blood glucose</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Preparation
- Bowel prep routine, requiring rehydration
- Consider referral to high-volume center

Anesthetic Technique
- General
- Combined
  * General/lumbar epidural (narcotics)
  * General/low-thoracic epidural (local anesthesia/narcotics)
  * General/intrathecal narcotics
- Technique needs to allow for unresectability (open, close)

Monitoring
- Large-bore IV access for fluid requirements, blood loss
- Consider central venous, arterial pressure monitoring

Airway
- None

Induction
- Cricoid pressure if gastric outlet obstruction suspected
- Combined technique with low-thoracic epidural requires only moderate volume of local anesthesia (6–8 mL)

Surgical Stages

Dissection
- Small-to-moderate amount of blood loss
- Moderate ongoing fluid requirements
- Resectability determined by absence of distant metastases, extent of major vascular involvement

Definitive Surgery (Pancreatico Duodenectomy)
- Significant volume requirements; consider colloid
- Arterial anomalies can increase operative complexity.
- Isolated venous involvement requiring resection and reconstruction is well accepted.
- Blood loss can be massive with portal vein or vena cava injury.
- Restoring gastrointestinal continuity
- Moderate ongoing volume requirements; consider colloid

Postoperative Considerations
- Pain score: 5–9
- Usual hospital stay: 8–12 d
- Combined technique with neuraxial narcotics
- May develop significant fluid shifts
- May require postop ventilation and/or extended ICU stay
- Glycemic control

Anticipated Problems/Concerns
- Risk of significant fluid shifts, blood loss
- Malnutrition
- Delayed gastric emptying and/or ileus
- Leak or fistula from pancreatic anastomosis

Surgical Stages

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- May require postop ventilation and/or extended ICU stay
- Glycemic control

Anticipated Problems/Concerns
- Risk of significant fluid shifts, blood loss
- Malnutrition
- Delayed gastric emptying and/or ileus
- Leak or fistula from pancreatic anastomosis
ACE Inhibitors

Uses
• Treatment of essential Htn, CHF, and mitral regurgitation
• Numerous studies show that ACE-I improves symptoms and quality of life as well as reduces mortality rate in elderly with heart failure and decreased LVEF.
• Decreases mortality after myocardial infarction
• Safe and effective treatment of Htn in diabetics. Strong evidence ACE-I delays the progression of diabetic renal disease.

Perioperative Risks
• Severe and prolonged hypotension in pts undergoing general anesthesia
• May increase insulin sensitivity and hyperglycemia in diabetics

Worry About
• Decrease glomerular filtration rate and not recommended in pts with renal artery stenosis
• Life-threatening angioedema involves the swelling of head, neck, and tongue
• Hyperkalemia because of decreased production of aldosterone
• Fetal anomalies and fetal and neonatal death

Overview/Pharmacology/Dose
• Captopril is available in oral dose and very effective in treating Htn
• Enalapril has to be converted by esterase in liver to the active metabolite enalaprilat
• Both captopril and enalapril are renally excreted and should be reduced in pt with renal dysfunction
• Lisinopril is absorbed as the active form and offered as once-a-day dosing

Drug Class
• Affects the renin-angiotensin system by blocking the conversion of angiotensin I to the active angiotensin II and delaying bradykinin breakdown and assoc prostaglandins

ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE | Test
--- | --- | --- | --- | ---
HEENT | Angioedema | Swelling of face, neck, tongue | Difficulty speaking, swallowing | Airway exam
 | Bronchospasm | Dyspnea | Wheezing | |
CARDIO | Hypotension | Assess CV response to Rx | |
GU | Renal failure | Orthopnea, dyspnea | Edema | BUN, Cr,lytes
 | Hyperkalemia | |
HEME | Leukopenia, agranulocytosis | Fever | |


Drug Interactions

Preoperative Period
• Assess for evidence of renal insufficiency
• Monitor for hyperkalemia
• ACE-I can be continued until the day of surgery because of the potential benefits in reducing mortality and morbidity
• Consider reducing the ACE-I dose so that hypotension can be avoided

Induction/Maintenance
• Severe and refractory hypotension that can be resistant to vasopressors such as phenylephrine, ephedrine, and norepinephrine
• Vasopressin and analogs can be useful to restore BP
• Use of succinylcholine with elevated K+ may be assoc with cardiac arrhythmia.

Adjuvant/Regional Anesthesia/Reversal
• Hypotensive episodes may be assoc with spinal and epidural anesthesia.

Postoperative Period
• Monitor for hypotension
Acetaminophen

Uses
• Minor analgesic for acute, chronic, and periop pain
• First line agent in WHO analgesia treatment ladder
• First line agent in treatment of pain in pregnancy (class B), compatible with breastfeeding
• Combination therapy formulations (e.g., with opioids) as adjuvant
• Antipyretic

Perioperative Risk
• In normal therapeutic doses, well-tolerated
• Overdose assoc with hepatotoxicity, nephrotoxicity, metabolic acidosis, and hypoglycemia
• Can precipitate asthma exacerbation
• Little peripheral antplatelet effects, although case reports of pts with acetaminophen-induced coagulopathy, randomized controlled studies demonstrated no risk
• Little anti-inflammatory effects

Pharmacokinetics/Pharmacodynamics
• Mechanism of action unclear
• Postulated mechanisms include: Inhibition of prostaglandin synthase, inhibition of cyclooxygenase isoensymes (COX-3), interaction with descending endogenous opioid pathways, interaction with L-arginine-NO-pathway (inhibits substance P-mediated hyperalgesia), augmentation of descending serotoninergic pathway, and increase in cannaboid and/or vanilloid tone.
• Selectively inhibits COX activity with low oxidant environments (i.e., endothelial cells) vs. those with high oxidant environments (pfts).
• Antipyretic activity postulated to be 2° to the blockade of prostaglandin (PG) production and antagonism of prostaglandin endoperoxide H2 synthase (PGHS) and COX centrally (hypothalamus)
• Questionable opioid-sparing effect
• It is rapidly absorbed in GI tract, mostly in the small intestine.
• Half-life (t1/2) 1.25–3 hr, peak plasma concentration 30–60 min
• Serum therapeutic levels 10–30 μg/mL
• Analgesic effect approx 6 hr
• 25% of dose undergoes first pass effect in the liver
• Approx the vast majority (90%) is metabolized by conjugation in the liver via glucuronidation and sulfate conjugation, forming non-toxic metabolites (saturated at doses greater than 150 mg/kg and renally excreted (90–100% recovered in urine within 24 hr)

ASSESSMENT POINTS
System  Effect  Assessment By Hx  Test
CNS  Encephalopathy, antipyretic effects, analgesia  Coma  GCS, CT/MRI (cerebral edema)
PULM  Bronchoconstriction  Labored breathing, wheeze  Increased peak /plateau airway pressure, auscultation
GI  Hepatic dysfunction  N/V, anorexia, sequelia of liver failure  Transaminases, INR, bilirubin, hypoglycemia
RENAL  Renal dysfunction, acute tubular necrosis  Oliguria  BMP, Cr, UA
METABOLIC  Metabolic acidosis  Lactate, ABG


Suspected Toxicity and Treatment
• Overdose accounts for approx 40% of acute liver failure in USA and UK
• Half of the hospitalizations for acetaminophen overdose (150 mg/kg) were unintentional
• Mean dose ingested causing hepatic failure was 24 g.
• Nephrotoxicity occurred in 1–2% of pts with acetaminophen overdose
• If suspected toxicity, don’t delay giving NAC (N-acetylcysteine). Proposed mechanisms of action for antidote incl increasing glutathione stores and conjugation to NAPQI, antioxidant effects, anti-inflammatory effects, increases NO resultant microvascular perfusion
• Serum acetaminophen concentration plotted on Rumack-Matthew nomogram

Symptoms
• Phase I: (0–24 hr): Asymptomatic, anorexia, N/V, malaise, subclinical rise in serum transaminases
• Phase II: (18–72 hr): Right upper quadrant abd pain, anorexia, N/V, increased transaminases levels
• Phase III (72–96 hr): Centrilobular hepatic necrosis, jaundice, coagulopathy, hepatic encephalopathy, renal failure, fulminant hepatitis, death

• Phase IV (96 hr–3 wk): Complete resolution of symptoms and organ failure

Treatment (Pediatric and Adult Populations)
• Gastric decontamination: Within 4 hr of ingestion (charcoal 1g/kg PO)
• NAC administration within 8 hr of ingestion either IV (150 mg/kg over 15 min then 50 mg/kg over 4 hr, then 100 mg/kg over 16 hr) or PO (140 mg/kg, then 70 mg/kg Q4 × 17 doses)
• Supportive measures

Perioperative Implications
• Increased periop morbidity with pts with increased liver function tests
• Pediatric population younger than 5 y have better prognosis than acetaminophen toxicity, potentially related to increased capacity
• Asthma may be co-morbid in pts with chronic acetaminophen use.

Avoidance of anesthesia techniques that impair hepatic/renal blood flow (i.e., halothane)
• Delay elective surgery, follow hepatic function tests and renal function tests to optimize timing of surgical intervention
• Auto-regulatory curve for hemodynamic organ perfusion homeostasis may be altered by women taking acetaminophen chronically 2° to Htn
• Potential coagulopathic augmentation of anti-coagulants (e.g., warafin, NSAIDS)

• Roughly 10% undergo oxidative metabolism via CYP2E, CYP1A2, CYP2A6, and CYP3A4 to form the potentially hepatotoxic and nephrotoxic metabolite N-acetyl-p-benzoquinoneimine (NAPQI)
• Increase in production of NAPQI attributed to P450 system induction or low glutathione levels (which normally detoxify metabolite to mercaptur-ric acid a cysteine conjugates)
• NAPQI can covalently bind to cysteinyln-sulfhydryl groups, causing hepatocellular (zone III) necrosis (oxidative damage, mitochondrial dysfunction)

Drug Class/ Mechanism of Action/Usual Dose
• Available in oral and rectal formulations in the USA, parenteral in Europe
• In USA: 325 mg and 500 mg immediate release tablets, 650 extended-release tablets and rectal suppositories
• Maximum dose 4 g/24 hr, (although American Liver Foundation 3g/24 hr)
• Adjust dose for CrCl: if 10–50 mL/min interval q6h, if <10 mL/min interval q8h

Drug Interactions
• CYP inducers: Barbiturates, bupropion, caffeine, carbamazepine, charcoal-broiled food, cruciferous vegetables, dihydralazine, isoniazid, phenytoin, primidone, rifampin, ritonavir, sulfonpyrazone, ethanol, isoniazid
• Questionable inhibition of glucoronidation with ranitidine, propranolol, ciasipride
• Questionable potentiation of glucuronidation by estrogen-containing contraceptives
• Warfarin, NSAIDs coagulopathic effects may be potentiated by acetaminophen

Anticipated Problems/Concerns
• Pts with salicylate hypersensitivity (urti-caria) have approx 11% of cross-reactivity with acetaminophen
• Long-term use has been linked to Htn in prospective studies in females, no association with males (likely related to imbalance of vasconstrictive and vasodilatory effects of prostaglandins).
• Increased risk of toxicity with baseline hepatic dysfunction (low glutathione levels: HCV, cirrhosis, malnutrition/fasting, HIV) or CYP enzyme induction
• Asthma was found to be 63% higher among acetaminophen users than nonusers in multivari-ate analyses
Alkylating Agents

Uses

- Bone marrow transplants
- Breast and bladder cancers
- Lymphomas and leukemias
- Lung, pancreas and brain cancers
- Ovarian and testicular cancers
- Multiple myeloma
- Sarcomas and melanomas

Worry About

- Extravasation if given by IV infusion
- Prolonged bleeding (thrombocytopenia)
- Aspiration during intubation

Overview/Pharmacology

- First chemotherapy agents (1940s)
- Structurally diverse compounds
- Generate reactive, electron-deficient intermediates
- Covalently bind to DNA bases, esp. guanine, often during mitosis
- Disrupt DNA replication and transcription
- High incidence of cytotoxicity to normal, rapidly dividing cells

Perioperative Risks

- Increased risk of infection
- Aspiration (subsequent to N/V)
- Prolonged succinylcholine action (CTX)
- Fluid retention (HN$_2$)

Worry About

- Extravasation if given by IV infusion
- Prolonged bleeding (thrombocytopenia)
- Aspiration during intubation

Overview/Pharmacology

- First chemotherapy agents (1940s)
- Structurally diverse compounds
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- High incidence of cytotoxicity to normal, rapidly dividing cells

Perioperative Implications

Preoperative Preparation

- Full-stomach precautions
- Risk of infection (leukopenia)
- Adequate hydration (bladder toxicity)
- Check plt count (thrombocytopenia)
- PFT (busulfan, cyclophosphamide)
- MUGA (cyclophosphamide)

Intraoperative

- Risk of aspiration during induction
- Prolonged bleeding
- Plan for RBC transfusion (anemia)
- Maintain UO
- Reduced dose of succinylcholine (CTX, thiotepa)

Postoperative Concerns

- Risk of N/V (most agents)
- Continued fluid hydration
- Monitor cardiac/pulm dysfunction (CTX, busulfan)
- Monitor renal and hepatic function

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**Alpha-2 Adrenergic Agonists**

**Overview/Pharmacology**
- Dexmedetomidine is a high specificity alpha-2A selective (1620:1 alpha-2/alpha-1 activity) imidazole derivative with wide-ranging effects. It binds to postsynaptic alpha-2A receptors on inhibitory neurons in the CNS (predominantly in the locus ceruleus) resulting in its sedative properties (simulating natural sleep and with characteristic preservation of respiration). These effects may also be in part due to activation of central imidazoline receptors in several brainstem nuclei. Dopaminergic modulation in the CNS via alpha-2C receptors probably plays less of a role. The sedation is non-GABAergic and thus is less prone to agitation and/or disinhibition phenomena seen with other agents such as benzodiazepines. Other central locations of alpha-2A postsynaptic agonism lead to inhibition of peripheral sympathetic outflow and are responsible for the vasodepressor and negative chronotropic/inotrope effects via stimulation of inhibitory neurons in the medullary vasomotor centers. This is added to by presynaptic alpha-2A inhibition of norepinephrine release in sympathetic nerve terminals (autoreceptor modulation) and alpha-2C autoreceptor inhibition in the adrenal medulla with net reductions in arterial tone, venomotor tone, stroke volume, and heart rate.
- An initial phase of increased SVR may occur due to postsynaptic alpha-2B agonism in sympathetic nerve terminals with enhanced norepinephrine release.
- Signal transduction is via coupling to G-protein effector systems. Activation of Gβ leads to decreases in adenylyl cyclase activity (with resultant reductions in protein kinase activity), as well as increases in hyperpolarizing K+ currents. Decreases in N-type and L-type Ca2+ currents are also seen and may in part be coupled to activation of Go.
- Both amnestic (not reliably seen) and analgesic properties are also ascribed to dexmedetomidine. Analgesia may occur at multiple sites. Direct presynaptic and postsynaptic alpha-2 agonism in the substantia gelatinosa may diminish substance P and glutamate release (presynaptic heteroreceptor agonism) and directly inhibit second order neurons (postsynaptic agonism); thus ascending nociceptive afferent flow is reduced (in a manner with minimal cross tolerance with opioids). Supraspinal modulation of ascending input may also occur in the CNS itself.
- Clonidine is also an imidazole derivative with similar properties (220:1 alpha-2 and/or alpha-1 activity). In general its effects on the vascular system are more pronounced than those of dexmedetomidine, while its sedative effects are less significant. It has also nonetheless been used to reduce anesthetic requirements in people undergoing general anesthesia, and has been successful as an additive in central neuraxial and peripheral nerve blockade in both extending the duration, and enhancing the quality of sensory neural blockade and avoiding side effects seen with neuraxial opioids used for the same purpose.
- Adjunct agent in general anesthesia (dexmedetomidine, clonidine)
- Sedation in mechanically-ventilated pts (dexmedetomidine)
- Management of withdrawal symptoms (reduction in cardio sympathetic stimulation during naloxone therapy, and abatement of alcohol, nicotine, benzodiazepine, or cocaine withdrawal symptoms) (clonidine)
- Reductions in intraocular pressure via reduced aqueous humor secretion (brimonidine, apraclonidine)
- Reductions in postop shivering (clonidine, dexmedetomidine)
- Additive in central neuraxial and perhaps (question if systemic effect) in peripheral nerve blockade (clonidine)

**Overview/Pharmacology**

**Perioperative Risks**
- Initial phase of acute Htn mediated by postsynaptic alpha-2B vasoconstriction
- Hypotension and bradycardia mediated by postsynaptic central alpha-2A decreases in peripheral sympathetic outflow and presynaptic peripheral alpha-2A/2C inhibition of NE/EPI release
- CV collapse in hypovolemic states or other pts dependent on avoiding reductions in sympathetic tone or SVR to allow maintenance of BP (e.g., trauma pts, aortic stenosis)

**Worry About**
- Rebound Htn (esp. if dexmedetomidine infusions of greater than 24 hr) or any interruptions of clonidine (esp. after 18 hr or in pts taking >1.2 mg daily)
- Assoc xerostomia (which may be beneficial in awake intubation)
- Increases in half time (“context sensitivity”) with prolonged infusions of dexmedetomidine. For example half time of 4 min after 10 min infusion grows to 250 min after 8 hr infusion.

**Drug Class/ Mechanism of Action/Usual Dose**
- Dexmedetomidine: Imidazole derivative given by IV infusion. A loading dose of 1 mcg/kg is given on an infusion pump (200 mcg ampu diluted in 48 mL saline to a final 3 mL volume with resulting concentration 4 mcg/mL) over 10–15 min, followed by an infusion of 0.2–0.7 mcg/kg/hr. Loading doses may be given over slightly longer times (20–30 min) in pts undergoing awake FOI so that response and airway patency may be continually evaluated, and peak sedation may be made to match the end of an assoc topicalization of the airway. Rates may need to be reduced in infusion over 24 hr as half life increases markedly with prolonged infusion. Elimination half life 2–3 hr. May be reversed with atipamezole.
- Clonidine: Imidazoline derivative given in dosages of 100–300 mcg orally 1–4 times daily or via transdermal patch. Elimination half life of 6–10 hr utility as sedative
- Guanfacine and guanabenz: Phenylguanidine derivatives with relatively long half lives (12–24 hr and 4–6 hr respectively). Functional antihypertensives rarely utilized currently.
- Alpha-methylidopa: 1–2 g daily in divided doses. Acts via its central alpha-2 agonist metabolite alpha-methylnorepinephrine. May cause positive Coombs’ test or hemolytic anemia. Safe historic record for use as antihypertensive in pregnancy.
- Bromionidine and apraclonidine: Ophthalmologic agents for use topically in glaucoma
- Tizanidine: Antispasmodic used in treatment of cerebral and spinal spasticity.

**Uses (off-label uses included)**
- Treatment of hypertensive states (clonidine, guanfacine, guanabenz, alpha-methylidopa)
- Sedation in mechanically-ventilated pts (dexmedetomidine)
- Adjunct agent in general anesthesia (dexmedetomidine, clonidine)
- Sedation for awake intubation (dexmedetomidine)
- Management of withdrawal symptoms (reduction in cardio sympathetic stimulation during naloxone therapy, and abatement of alcohol, nicotine, benzodiazepine, or cocaine withdrawal symptoms) (clonidine)
- Reductions in intraocular pressure via reduced aqueous humor secretion (brimonidine, apraclonidine)
- Reductions in postop shivering (clonidine, dexmedetomidine)
- Additive in central neuraxial and perhaps (question if systemic effect) in peripheral nerve blockade (clonidine)

**Table: Drug Effects**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Increased SVR</td>
<td>Headache, palpitations, dizziness, diaphoresis, abd pain</td>
<td>Pulse, BR skin temp and turgor</td>
<td>ECG, PA Cath, TEE</td>
</tr>
<tr>
<td>RESP</td>
<td>Usually minimally reduced</td>
<td>Hypopnea, apnea, cyanosis</td>
<td>Spirometry</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Minute ventilation and preserved CO2</td>
<td></td>
<td>Pulse, Oximetry</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Presynapt</td>
<td>Resp</td>
<td></td>
<td>Capnogram</td>
</tr>
<tr>
<td>CNS</td>
<td>Sedation</td>
<td>Ramsay sedation scale</td>
<td>Somnolence</td>
<td>BIS, Blood, Glucose</td>
</tr>
<tr>
<td>OTHER</td>
<td>Amnesia</td>
<td>Recall</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Analgesia</td>
<td>Pain</td>
<td></td>
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<tr>
<td></td>
<td>Reduced CBF</td>
<td>Dry mouth/nasal decongestion</td>
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<tr>
<td></td>
<td>Xerostomia/antisialogogue</td>
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<tr>
<td></td>
<td>Pt aggregation</td>
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<tr>
<td></td>
<td>Lipolysis inhibition</td>
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<tr>
<td></td>
<td>Insulin secretion inhibition</td>
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</tbody>
</table>

Perioperative Implications

Preoperative Concerns
- Rebound Htn common in pts on clonidine who do not take their dose on the morning of surgery. Pts on clonidine have often been placed on it for refractory Htn and labile BP should be anticipated. Baroreceptor sensitivity is generally preserved.
- Clonidine has some evidence for myocardial protection in CV surgery and can be considered in pts who would benefit from but have contraindication to periop beta blockade.
- Consider adding low dose (30–70 mcg/kg) midazolam to sedation plan for awake FOI if definitive amnesia is strongly desired.

Induction/Maintenance
- Slow controlled induction is preferable when not contraindicated as adjunct decreases in SVR and inotropic state may lead to severe hypotension.
- Treatment of dysrhythmias such as symptomatic bradycardia and AV block may become necessary. Use caution in treating hypotensive bradycardia with anticholinergics alone (esp. in those in whom coronary perfusion is SVR dependent). Increases in HR in a pt with catastrophically low SVR can precipitate severe myocardial ischemia (altered supply/demand ratio).
- Reducions in MAC are common in pts on clonidine and dexmedetomidine (30%–50% in most studies but wide range seen). Titration to hemodynamic variables or BIS may be useful.

Postoperative Period
- Continue to maintain clonidine use postop to reduce risk of rebound Htn.
- Rebound Htn unlikely with dexmedetomidine infusions less than 24 hr duration

Anticipated Problems/Concerns
- Postop hypothermia may be accentuated.
Aminophylline

**Uses**
- Acute and chronic Rx for asthma, COPD
- Rx for neonatal apnea
- As 2nd agent in CPR settings
- Potential for life-threatening CNS, cardiac toxicity
- Administered IV, PO, or rectally

**Perioperative Risks**
- Toxic levels from overaggressive use or coadministration of cimetidine/propranolol
- Increased arrhythmogenicity with pancuronium
- Increased CNS toxicity (lower seizure threshold) with ketamine or in children

**Worry About**
- Prolonged clearance in presence of cimetidine, erythromycin, propranolol, or in pts receiving influenza vaccines
- Enhanced clearance in smokers and pts taking dilantin, barbiturates
- Narrow therapeutic/toxic ratio
- Decreases sedative effect of propofol (but does not alter desflurane MAC)
- May alter bispectral index measurements

**Overview/Pharmacology**
- Methylated xanthine
- Bronchodilatory and anti-inflammatory effects
- Onset of effect within 1 hr from IV dose
- Biotransformed by demethylation in liver; renally excreted
- 7–15% excreted unchanged in urine
- Crosses placenta, found in breast milk

**Drug Class/Mechanism of Action/Usual Dose**
- Methylated xanthine
- Proposed mechanisms of action incl inhibiting phosphodiesterase, antagonizing the effect of adenosine, causing catecholamine release, inhibiting cellular immune function
- Usual dosage: 4 mg/kg q 8–12 hr PO; 5–6 mg/kg IV load followed by 0.2–0.75 mg/kg/hr IV

**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Inotropy and chronotropy; ↓ in SVR, PCWP, BP</td>
<td>Predisposes to ventricular arrhythmia</td>
<td>Auscultation of heart sounds</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchodilation, suppression of cellular immune response</td>
<td>Relief of dyspnea, improvement of bronchospastic symptoms</td>
<td>Auscultation of chest</td>
<td>Peak flow, PFTs</td>
</tr>
<tr>
<td>CNS</td>
<td>Nonspecific CNS stimulation; stimulates central resp drive; alters BIS readings</td>
<td>N/V, irritability, insomnia, delirium, convulsions, stupor, coma</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Concerns**
- Toxic preop blood level 2nd to over-administration or coadministration of drugs that affect clearance (cimetidine, erythromycin, propranolol, verapamil, phenytoin [Dilantin])
- Potential for seizures or malignant arrhythmias if toxic levels
- Presence of underlying bronchospastic disease
- Administration via peripheral vein to avoid cardiotoxicity

**Induction/Maintenance**
- Can interact with halothane or pancuronium to cause ventricular arrhythmias
- Can interact with ketamine to lower seizure threshold
- Continue infusion if carefully monitoring for toxicity
- No proven effectiveness in treating intraop bronchospasm in humans
- Reduction of NMB
- Increases BIS readings

**Postoperative Period**
- Check plasma levels before restarting infusion if toxicity is suspected
- May be used as central resp stimulant in neonates recovering from general anesthesia

**Anticipated Problems/Concerns**
- Most common problems are from narrow therapeutic and/or toxic window, potential for severe CNS, cardiac toxicity.
- Pts taking aminophylline often have severe bronchospastic disease.
- Dialysis or charcoal hemoperfusion can acutely lower blood levels.
Amphetamines

Uses
- Narcolepsy and attention deficit hyperactivity disorder (ADHD)
- Also used to treat enuresis and incontinence
- Not recommended for fatigue or wt loss
- Administered orally and effects can last for several hr

Perioperative Risks
- Chronic use can lead to decreased MAC
- Acute use can increase MAC
- Risk of Htn, tremors, hyperactive reflexes, anxiety, and delirium
- When used with thyroid hormone, diuretics, laxatives it can lead to arrhythmias or cardiac arrest.

Worry About
- Severe Htn, palpitations, confusion, dizziness, and vasomotor disturbances esp. in pts with ischemic heart disease, Htn, rhabdomyolysis, and hyperthyroidism

Overview/Pharmacology
- Causes release of catecholamine resulting in sympathomimetic toxidrome
- Schedule II drug
- Acute use can cause seizures, intracranial hemorrhage, stroke, increased cardiac output, increased heart rate, increased peripheral vascular resistance, angina pain, renal failure, and cardiac arrhythmias.
- Chronic use can cause increased tolerance, dependence, and a depletion of body stores of catecholamine. Also, wt loss and psychotic effects can occur.
- May have a small analgesic effect and can enhance the analgesia produced by opiates
- Metabolized by the liver

Overview/Pharmacology
- Overdose is treated by supportive management, IV hydration for possible rhabdomyolysis, sedation, acidification of urine to enhance elimination, and sodium nitroprusside for severe Htn.
- Be sure to check core temp, obtain an EKG, and evaluate renal function.
- Benzodiazepines, often in high doses, are useful for control of agitation.

Drug Class/Mechanism of Action/Usual Dose
- Phenethylamine derivative
- Amphetamines act indirectly to release biogenic amine neurotransmitters from nerve terminals centrally and peripherally.
- Usual oral dosages
  - Narcolepsy: 5–10 mg bid
  - ADHD: 5–10 mg bid

DRUG EFFECTS

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<tbody>
<tr>
<td>CARDIO</td>
<td>Increases BP, CO, HR, SVR, arrhythmias</td>
<td>Hx of recent/chronic use</td>
<td>Vitals</td>
<td>EKG</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp stimulation</td>
<td>Hx of recent/chronic use</td>
<td>Pulm exam</td>
<td>ABG</td>
</tr>
<tr>
<td>CNS</td>
<td>Increased alertness, electrical activity</td>
<td>Hx of recent/chronic use</td>
<td>CNS exam</td>
<td>EEG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Overdose: Anxiety, psychoses, seizures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Renal failure, lactic acidosis</td>
<td>Hx of recent/chronic use</td>
<td>Vital signs, PE</td>
<td>ABG, electrolytes</td>
</tr>
<tr>
<td></td>
<td>Dehydration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>Mydriasis, diaphoresis, hyperthermia, decreased GI motility</td>
<td>Hx of recent/chronic use</td>
<td>Vitals, PE</td>
<td></td>
</tr>
</tbody>
</table>

Perioperative Implications

Preoperative Concerns
- Recent, acute ingestion
- Concomitant use of MAOI
- Hx of polysubstance abuse
- Monitor BP for Htn, HR, and preop EKG for ischemia

Induction/Maintenance
- With severe Htn, may need to use alpha or beta blocker and/or vasodilators
- Chronic use will decrease anesthetic requirements
- May increase analgesic effect of opioids

- Diminished response to vasopressors from chronic abuse
  * Adjuvant /Reversal
  * Drug interactions noted are the major concerns

Postoperative Period
- Continue to monitor CV, CNS hyperactivity, UO, body temp
- Withdrawal symptoms are non-life threatening

Anticipated Problems/Concerns
- Ask about Hx of substance abuse. Worry about concomitant use of other drugs as well.
- Ask about most recent usage due to changes in anesthesia requirements and need for further labs and/or tests.
- Possible drug interactions
Angiotensin II Receptor Blocking Drugs

Uses
• AT1-receptor antagonists or sartans, are a group of pharmaceuticals which modulate the renin-angiotensin-aldosterone system. Their main use is in Htn, diabetic nephropathy, and CHF.

Perioperative Risks
• ARBs do not inhibit ACE, they do not cause an increase in bradykinin, which contributes to the vasodilation produced by ACE inhibitors and also some of the side effects of ACE inhibitors (cough and angioedema).
• Dementia: pts who were already suffering from Alzheimer’s disease or dementia, and found those subjects had up to a 50% lower chance of being admitted to nursing homes or dying if they were taking ARBs.

Worry About
• Rebound Htn if drug acutely withdrawn esp. with longer-acting agents
• Refractory hypotension in pts undergoing general anesthesia. BP responds to vasopressin agonists.
• Questionable increased risk of MI with ARBs

Pharmacology Overview
• Renin-angiotensin cascade begins with the cleavage of angiotensin by rennin, angiotensin I converted by ACE to angiotensin II, angiotensin II receptors activated by binding of angiotensin
• Clinical effects of angiotensin II (vasoconstriction, sodium/water retention, renin suppression, etc.) are mediated by the angiotensin receptor 1 (AT1)
• Blockade of AT1 receptors directly cause vasodilation, reduces secretion of vasopressin, reduces production and secretion of aldosterone, among other actions—the combined effect of which is reduction of BP.
• Three important PD/PK factors: Pressor inhibition, AT1 affinity, biological half life (example Losartan 100 mg 25–40%, 1000-fold, 6 hr or valsartan 80 mg 30%, 20,000-fold, 6 hr).
• Contraindicated in pregnancy

Mechanism of Action/Usual Dose
• The activated receptor in turn couples to Gq/11 and thus activates phospholipase C and increases the cytosolic Ca++ concentrations, which in turn triggers cellular responses such as stimulation of protein kinase C. Activated receptor also inhibits adenylate cyclase and activates various tyrosine kinases.
• Available in once-a-day dosing
  • Candesartan (Atacand) 4–32 mg
  • Irbesartan (Avapro) 150–300 mg
  • Losartan (Cozaar) 50–100 mg
  • Telmisartan (Micardis) 40–80 mg
  • Valsartan (Diovan) 80–320 mg

DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Lowers BP</td>
<td>Assess response to Rx</td>
<td>BP</td>
<td>Monitor BP, can also have tachycardia and bradycardia with lowering BP, careful when D/C</td>
</tr>
<tr>
<td>GI</td>
<td>↑ in LFTs</td>
<td>Rare reversible hepatotoxicity reported</td>
<td></td>
<td>Watch for rebound Htn, LFTs</td>
</tr>
<tr>
<td>METAB</td>
<td>Hyperkalemia</td>
<td></td>
<td>K+</td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Angioedema reported</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Microcytic anemia</td>
<td></td>
<td>CBC</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Can cause ARF if pts with renal artery stenosis or diffuse infrarenal stenosis</td>
<td></td>
<td>BUN/Cr</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Rare headache, dizziness, fatigue insomnia</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Risks
• Reduced responsiveness to vasopressor, potential risk of rebound Htn in withdrawal (potential risks in general/neuron-vascular surgery).
• Potential risk of ARF in major bleeding and kidney ischemia

Induction/Maintenance
• Watch for refractory hypotension, which needs treatment with vasopressin agonist

Adjuvants/Regional Anesthesia/Reversal
• No known interactions

Postoperative Concerns
• Resumption of preop drugs for BP control if no ARF
• Only available in PO forms

Anticipated Concerns
• Recommended preop withdrawal for 12–24 hr.
Aspirin (Acetylsalicylic Acid)

**Overview/Pharmacology**
- **Cyclooxygenase inhibitor**
- Chronically taken for
  - MS pain (e.g., arthritis, neuralgia)
  - Prevention of CV events
  - Claudication
  - Acutely taken for
  - Acute, mild to moderate pain (e.g., headache, myalgia)
  - Fever
  - Dysmenorrhea
- Usual dose, 325–1000 mg q3–4h for acute illnesses and pain
- 62.5–325 mg for plt inhibitor effects
- Alternatives: Acetaminophen, other NSAIDs (ibuprofen, naproxen), steroids, opioids, gold, ticlopidine, dipyridamole, pentoxifylline

**Uses**
- People in USA consume 10,000–20,000 tons annually
- Rx for mild and/or moderate pain, fever, arthritis, prevention of myocardial infarction

**Perioperative Risks**
- Peptic ulcer disease
- Plt dysfunction
- Hemorrhage
- Stroke
- Interstitial nephritis
- Reye's syndrome

**Worry About**
- Displacement of protein-bound drugs: e.g., warfarin, sulfonlureas, thiopental, methotrexate
- Potentiation of anticoagulants

**Perioperative Implications**

**Preoperative Concerns**
- D/C 1 wk prior to surgery for full reversal of plt inhibition (need only ½ of normally functioning plts, so if no dilution effect expected, need only 48 hr off low-dose ASA); May see hyperthrombotic state around 7–10 d, particularly in pts with coronary stents
- Continue aspirin in pts with coronary stents unless contraindicated.
- May potentiate the effects of protein-bound drugs

**Induction/Maintenance**
- Possible mildly exaggerated effects of thiopental

**DRUG EFFECTS**

<table>
<thead>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Hyperventilation, resp alkalosis</td>
<td>Tachypnea</td>
<td></td>
<td>ABGs</td>
</tr>
<tr>
<td>GI</td>
<td>Gastritis</td>
<td>Dyspepsia</td>
<td></td>
<td>Endoscopy</td>
</tr>
<tr>
<td></td>
<td>PUD</td>
<td>N/V, hematemesis, melena</td>
<td></td>
<td>Upper GI, x-rays, stool heme, Hgb</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperglycemia, corticosteroid release</td>
<td>Glucose</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Plt dysfunction</td>
<td>Bleeding, bruising</td>
<td>Nematoma, petechiae</td>
<td>Bleeding time</td>
</tr>
<tr>
<td>HEPAT</td>
<td>Hepatocellular damage</td>
<td>Nausea, anorexia</td>
<td>Hepatomegaly, jaundice</td>
<td>SGOT, SGPT, alk phos</td>
</tr>
</tbody>
</table>

**TOXICITY**

| CARDIO | Vasomotor paralysis | Hypotension |    |      |
| RESP   | Hypoventilation, resp acidosis | Hypopnea |    | ABGs |
| DERM   | Eruptions | Pruritus | Aeneiform, erythematous, pruritic, eczematoid, or desquamative lesions |    |      |
| RENAL  | Renal failure due to analgesic nephropathy | Oliguria, anuria | Edema, rales | BUN/Cr, UA, CXR |
| CNS    | Headache, tinnitus, drowsiness, dizziness, diminished vision and hearing | Sweating, confusion, convulsions, coma |    |      |
| ACID-BASE | Metabolic acidosis | ABGs |    |      |

**Drug Class/Mechanism of Action/Usual Dose**
- NSAID
- Acetaminophen, other NSAIDs (ibuprofen, naproxen), steroids, opioids, gold, ticlopidine, dipyridamole, pentoxifylline

Asthma Drugs, New

Michael Bishop
Yulia Ivashkov

Uses
- Asthma therapy is based on the recognition that it is primarily an inflammatory disease.
- There are no major changes in the last 5y in treatment of acute asthma.
- Treatment of chronic asthma incl:
  - Inhaled corticosteroids are a first-line therapy. Introduction of a selective inhaled corticosteroid ciclesonide may further minimize the adverse effects of inhaled corticosteroids (specifically, oral thrush).
  - Use of inhaled anti-inflammatory drugs such as nedocromil (mast-cell stabilizer) and cromolyn for mild persistent asthma.
  - Oral anti-leukotriene drugs zileuton, montelukast and zafirlukast
  - Recombinant humanized monoclonal anti-IgE antibody, omalizumab, for treatment of allergic asthma
- Drugs for chronic treatment have no place in the face of an acute asthma attack for which beta adrenergic agonists remain first-line therapy.

Risks
- Oral antileukotriene drugs are recommended for use in mild to moderate persistent asthma, and are less effective in asthma exacerbations, than inhaled corticosteroids. They have no direct bronchodilatory effects. They do not prevent an asthma attack in the face of airway stimulation.
- Zafirlukast and zileuton may cause drug interactions due to cytochrome P450 isoenzyme involvement (inhibition of warfarin and theophylline metabolism)
- Zileuton can cause hepatotoxicity, and requires monitoring of liver function.
- Potential safety concerns for omalizumab incl risk of development of anaphylaxis. Side effects may also incl diarrhea, vomiting, menorrhagia, and increased hematoma formation. It is given as a SQ injection once in 2 to 4 wk.

Overview
- Leukotrienes are potent bronchoconstrictors. They also cause chemotaxis, mucus secretion, edema, and eosinophilia. The anti-leukotriene modifiers prevent formation of leukotrienes from arachidonic acid via the lipoxygenase pathway.
- Zileuton inhibits the first step of the pathway: Conversion of arachidonic acid to leukotriene A4 by the enzyme 5-lipoxygenase.
- Zafirlukast and montelukast act as receptor antagonists for the cysteinyl leukotrienes, leukotrienes C4, D4 and E4.
- Clinical effects of anti-leukotriene drugs are modest. The most beneficial effect was shown in pts with exercise-induced asthma and asthma assoc with allergic rhinitis. They may be used as a monotherapy in persistent mild asthma.
- Symptoms of atopic asthma may be mediated through a number of IgE-dependent mechanisms. Omalizumab is a recombinant humanized monoclonal anti-IgE antibody that markedly reduces free circulating IgE. It is used as an add-on therapy in severe persistent asthma not responding to a traditional therapy.

Perioperative Implications
- Other than the above mentioned drug interactions with warfarin and theophylline, there are relatively few implications of the drugs themselves.
- It is critical to recognize that pts on these drugs are asthmatic and that these drugs will not necessarily prevent bronchoconstriction to an acute stimulus.
- Prophylactic beta adrenergic agonists prior to surgery are indicated in pts on these drugs and are the first line of acute therapy.
- These drugs are sometimes used in an effort to lower doses of oral steroids. Pts with recent steroid tapers may be at risk during the periop period due to adrenal suppression.
- Anesthesia providers should recognize that omalizumab can be a cause of anaphylaxis.
Atorvastatin (Lipitor)

**Uses**
- Synthetic lipid-lowering agent
- Selective, competitive HMG CoA reductase inhibitor (the rate-limiting enzyme of LDL cholesterol synthesis)
- Lowers LDL and triglycerides and raises HDL (the latter at high doses, e.g., 40 mg/d and over to the average 70 kg pt)
- Augments elimination of LDL and VLDL by upregulating LDL receptors on surface of hepatocytes
- Statins also modify endothelial function, decreasing inflammatory responses, and thrombogenicity, while increasing plaque stability.
- Evidence widely shows statins reduce the rates of MI, stroke, and coronary and all cause mortality in the 1° and 2° prevention settings.
- Three studies indicate it reduces incidence of age-related cognitive dysfunction.
- Also been shown to ↓ risk of first major coronary event in patients with known risk factors for CAD and normal LDL cholesterol levels

**Perioperative Risks**
- Clinically important side effects of all statins incl myositis (may be modified by creatine or CoQ10 administration) and liver dysfunction.
- Incidence of ↑ transaminase levels of more than 3× normal is <2%.
- LFT elevations completely reversible and resolve within a few weeks on D/C of the drug
- Severe hepatic dysfunction or cirrhosis not reported
- Assoc rarely with rhabdomyolysis in the periop period of major surgeries
- Myositis rarely seen with monotherapy. Increases significantly (to nearly 30%) when used in combination with immunosuppressives (cyclosporine, tacrolimus), azole antifungal agents (ketocanazole, itraconazole), fibrinic acid derivatives (gemfibrozil), niacin, erythromycin, clarithromycin, and fluvoxamine. Greater risk with those on higher doses of statins, > age 65 y; with baseline renal insufficiency and uncontrolled hypothyroidism.

**Pharmacokinetics/Pharmacodynamics**
- Antilipemic drug: Interferes with production and enhances uptake of cholesterol and its lipoprotein complexes
- Orally administered and rapidly absorbed
- Hepatic first-pass metabolism causes a low systemic bioavailability.
- Undergoes extensive metabolism to active metabolites
- Elimination of parent drug and metabolites occurs primarily in bile after hepatic and/or extrahepatic metabolism
- <2% of a dose recovered in urine

**Induction/Maintenance**
- No reported cases of atorvastatin assoc intraop/periop events, although other statins have been assoc with adverse events such as rhabdomyolysis and concomitant myoglobinuria and renal failure
- If surgery involves significant muscle damage in the setting of a statin, consider placement of urinary catheter for early detection of myoglobinuria and avoidance of succinylcholine.

**Adjuvants/Regional Anesthesia/Reversal**
- No interactions known
- Avoid grapefruit premedication if MAC proposed as grapefruit within 2 hr decreases metabolism of this statin.
- Mean plasma elimination T ½ 14 hr, but T ½ of HMG CoA reductase inhibitory activity is 20–30 hr due to the active metabolites. As a result, drug has greater efficacy than other statins.
- Second most efficacious LDL lowering statin (26–60%)
- Unique structure, long half-life and hepatic selectivity possible explanations for increased potency
- Unlike other statins, increased dosages do not necessarily result in increased ability to raise HDL, but seems to require higher dosage to have an HDL raising effect

**Drug Class/Mechanism of Action/Usual Dose**
- Cholesterol lowering, statin
- Inhibition of HMG CoA reductase results in decreased synthesis of hepatic cholesterol
- Compensatory ↑ in hepatic LDL receptor production then results in an ↑ in uptake of LDL cholesterol from the circulation
- Reduces elevated total cholesterol, LDL-C, apo B, triglyceride levels and ↑ HDL-C
- Usual initial dosage 10 mg once daily. Maintenance dosage 10–80 mg once daily.
- No dosage modifications needed for renal insufficiency

**Drug Interactions**
- Risk of myopathy ↑ with concurrent administration of cyclosporine and erythromycin
- Antacids ↓ plasma concentrations by 35%
- Digoxin levels can ↑ by 20%

**Anticipated Problems/Concerns**
- Assess for CAD

**Perioperative Implications**

**Preoperative Concerns**
- Assess for CAD and other assoc conditions such as Htn, diabetes, atherosclerotic CVD
- When possible, HMG CoA reductase inhibitors should be continued periop but with hydration and observation to avoid significant rare skeletal muscle damage.
- Based on a T ½ of 30 hr for its active metabolites, the drug should be D/C 3–5 d preop if such D/C is desired
- Should also be D/C in pts with symptoms suggestive of a myopathy or with other risk factors predisposing to the development of renal failure and/or rhabdomyolysis such as hypotension, trauma, and severe acute infection

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- Antacids ↓ plasma concentrations by 35%
- Digoxin levels can ↑ by 20%

**Anticipated Problems/Concerns**
- Assess for CAD
Atropine

**Uses**
- Primary indication: Treatment for reflex-mediated bradycardia
- Preop medications to reduce oral and tracheobronchial secretions
- Production of mydriasis and cycloplegia
- Biliary and ureteral smooth muscle relaxation
- Treatment of organophosphate poisoning
- Bronchodilation esp. administered as aerosol
- Treatment for organophosphate poisoning; this poisoning may cause side effects known as SLUDGE: Salvation, Lacrimation, Urination, Diaphoresis, Gastrointestinal motility, and Emesis

**Pharmacology**
- Structurally resembles cocaine and has weak analgesic properties
- Competitively antagonize the effects of the neurotransmitter acetylcholine at the postganglionic sites known as muscarinic receptors.
- Muscarinic cholinergic receptors are found in salivary glands, smooth muscles of the GI and GU tract, and the heart.
- At high doses, the drug produces partial block of autonomic ganglia (nicotinic receptors)
- Parasympatholytic effects of anticholinergic drug decreasing the actions of rest and digest
- Lipid-soluble, easily pass the blood-brain barrier
- Plasma T ½ 2.3 hr
- Metabolism by hydrolysis in the liver, excreted by kidneys

**Dosages (70 kg adult):**
- 0.4–0.5 mg IV for intraop bradycardia
- 1–2 mg IV before reversal of muscle relaxants
- 1–2 mg IV before intrinsic sinus node dysfunction or organophosphate poisoning repeated prn
- 0.4–0.5 mg SQ, IM for control of secretions
- 0.01–0.02 mg/kg in infants and children (minimum 0.1mg)

**Special Considerations**
- 2–4 mg diluted with NS may be given in the ETT in absence of IV access
- For cardiopulmonary resuscitation larger doses may be necessary.

**Central Anticholinergic Syndrome**
- Atropine can enter the CNS and produce symptoms ranging from restlessness, hallucinations, somnolence, depressed ventilation to loss of consciousness. This syndrome can be mistaken for delayed recovery from anesthesia. Physostigmine is the specific treatment.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Mechanism of Action/Effects</th>
<th>PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Decreased secretions Block sphincteric and ciliary muscles</td>
<td>Dry mouth/upper airways Mydriasis (Dilated pupils)</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Block vagal effects of M, receptors on SA node</td>
<td>Tachycardia (Palpitations)</td>
</tr>
<tr>
<td>RESP</td>
<td>Dry airways</td>
<td>Thick secretions</td>
</tr>
<tr>
<td>GI</td>
<td>Decreased gastric acid production</td>
<td>Decreased peristalsis</td>
</tr>
<tr>
<td>GU</td>
<td>Block sphincteric muscles</td>
<td>Pain in lower abd (Enlarged bladder)</td>
</tr>
<tr>
<td>CNS</td>
<td>Toxic doses</td>
<td>Restlessness, disorientation, delirium</td>
</tr>
<tr>
<td>DERM</td>
<td>Flushing</td>
<td>Red body, dry skin (Possible increase of body temp)</td>
</tr>
</tbody>
</table>

**Key References:**
Benzodiazepines (Midazolam, Lorazepam, Diazepam)

**Uses**
- Prescribed for the treatment of anxiety
- Used for conscious sedation and premedication

**Perioperative Risks**
- High levels assoc with hypnosis, unconsciousness, resp depression, apnea

**Worry About**
- Combination with opioids or other CNS depressants may result in severe resp depression, apnea, hypotension

**Overview/Pharmacology**
- Anxiolysis, sedation, hypnosis, muscle relaxation, anterograde amnesia, anticonvulsant
- Midazolam: Short-elimination T½ (2.5 hr)
- Lorazepam: Intermediate-elimination T½ (15 hr)
- Diazepam: long-elimination T½ (30 hr)
- Metabolized by hepatic microsomal oxidation and glucuronide conjugation
- Midazolam
  - IV: Peak effect in 2–4 min
- Diazepam
  - IV: Peak effect in 1–2 min, painful injection, thombophlebitis
  - IM: Painful, unpredictable absorption, do not use
- Midazolam
  - IV: Peak effect in 2–4 min

**Drug Class/Mechanism of Action/Usual Dose**
- Anxiolytic, sedative, hypnotic
- Potentiation of gamma-aminobutyric acid-mediated neural inhibition
- Safe use involves careful titration to the desired effect
- Usual dosage for premedication and conscious sedation:
  - Midazolam
    - IV: 0.5–1 mg, repeated; maintenance infusion: 0.04–0.10 mg/kg/h
    - IM: 0.04–0.10 mg/kg/h
    - Oral: 15 mg (0.5 mg/kg in children up to 20 kg)
  - Lorazepam
    - IV: 0.25 mg, repeated
    - IM: 0.05 mg/kg, max 4 mg
    - Oral: 0.5–4 mg
  - Diazepam
    - IV: 1–2 mg, repeated
    - Oral: 5–10 mg

**Drug Effects**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Decreased systemic vascular resistance and cardiac output</td>
<td>Arterial BP</td>
<td></td>
</tr>
</tbody>
</table>
| RESP   | Central resp depression | Resp rate | Tidal volume
  | Apnea | | Minute volume, capnography, oximetry |
| CNS    | Anxiolysis | Slurred speech, drowsiness, ataxia |
|        | Sedation | Unresponsiveness |
|        | Hypnosis | |
|        | Amnesia | |
|        | Anticonvulsant | |
|        | ↓ Cerebral metabolic rate and cerebral blood flow | |

**Perioperative Implications/Possible Drug Interactions**
- Elderly: Reduce dose up to 5-fold (5–10%/decade reduction)
- Cimetidine, ranitidine (microsomal cytochrome P450 inhibitors), and liver cirrhosis ↓ clearance, enhanced effect may be seen
- Smoking and enzyme-inducing drugs increase diazepam clearance.
- Renal failure increases diazepam T½
- Monitor ventilation
- Induction/maintenance
- Synergistic interaction with anesthesia induction agents, and opioids

**Regional Anesthesia**
- Possibly exacerbates resp depression during spinal anesthesia (mechanism unknown)
- Combination with opioids or other CNS depressants may result in severe resp depression, apnea, hypotension
- Large doses result in prolonged drowsiness and resp depression, esp. in the elderly; reversal with flumazenil
- Undesirable degree of amnesia

**Preoperative Concerns**
- Elderly: Reduce dose up to 5-fold (5–10%/decade reduction)
- Cimetidine, ranitidine (microsomal cytochrome P450 inhibitors), and liver cirrhosis ↓ clearance, enhanced effect may be seen
- Smoking and enzyme-inducing drugs increase diazepam clearance.

**Key References:**
- Riker RR, Fraser GL. Adverse events associated with sedatives, analgesics, and other drugs that provide patient comfort in the intensive care unit. Pharmacotherapy. 2005;25:88S–18S.
- Maxa JL; Ogu CC; Adeeko MA; Swane TG. Continuous-infusion flumazenil in the management of chlordiazepoxide toxicity. Pharmacotherapy. 2003;23:1513–1516.

**Anticipated Problems/Concerns**
- Combination with opioids or other CNS depressants may result in severe resp depression, apnea, hypotension
- Large doses result in prolonged drowsiness and resp depression, esp. in the elderly; reversal with flumazenil
- Undesirable degree of amnesia
Beta-Adrenergic Receptor Antagonists (Blockers)

Uses
- Precise role in periop period (risks and benefits) still controversial
- Used in management of essential Htn
- Effective in decreasing infarct size
- Used to ↓ HR
- Available as oral and IV preparations
- Used to suppress cardiac dysrhythmias
- Value in prevention of excess SNS activity
- Must be continued periop in pts who have been taking them (Class I AHA indication)
- Reduced CV morbidity and mortality in pts with symptomatic ischemic heart disease for major surgery with periop use of β-blockers (major vascular surgery only)
- Whether one should give β-blockers periop (prophylactically) to pts with cardiac risk factors alone remains unclear

Perioperative Risks
- Nonselective blocker may precipitate bronchospasm
- May worsen or precipitate CHF in pts with ↓ LV function
- May cause hypotension, bradycardia

Worry About
- Decreased ventricular performance esp. with underlying cardiac dysfunction
- Can worsen lung disease, esp. with nonspecific blockers and Hx of COPD or bronchospasm

Overview/Pharmacology
- All β-blockers are derivatives of isoproterenol
- β-adrenergic receptor agonists classified as partial or pure agonists on basis or absence of intrinsic sympathomimetic activity
- Partial agonists often better tolerated than pure antagonists in pts with ↓ LV function
- β-blocker may produce varying degrees of membrane stabilization in heart (detectable only at extremely high plasma concentration)
- Effective in both acute, chronic management

Perioperative Risks
- β-blocker should be continued in periop period (in pts previously on β-blockers)
- May have value in pts with symptomatic cardiac disease preventing for major vascular surgery (Class I indication-AHA guidelines)
- Acute D/C can result in excess SNS activity that manifests in 24–48 hr

Induction/Maintenance
- Myocardial depression observed with inhaled or injected anesthetic is worsened with addition of β-blocker
- Esmolol has been assoc with profound brady-cardia in presence of inhaled anesthetics

Drug Class/Mechanism of Action/Usual Dose
- All β-blockers bind selectively to β-receptors
- β-blockers interfere with ability of other drugs/substances with sympathomimetic activity to activate β-receptors
- Action of β-blockers negates effect of catecholamines, other sympathomimetics on heart and smooth muscle of airways, blood vessels
- Bind to β-receptor by competitive inhibition
- Exhibit selective affinity for β-adrenergic receptors
- Binding of agonists to the β-receptor is reversible
- Chronic administration is assoc with ↑ in number of β-adrenergic receptors
- Principal method of clearance hepatic, renal, or plasma hydrolysis (esmolol)
- Elimination T½ specific to individual agents, depends on dose, protein binding, route of administration (oral/IV)


DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>↓ HR</td>
<td>Relief of angina</td>
<td>HR</td>
<td>ECG; stress ECG</td>
</tr>
<tr>
<td></td>
<td>↓ CO</td>
<td></td>
<td>BP</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ LV function</td>
<td></td>
<td>↓ HR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Coronary vascular resistance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Myocardial O2 consumption</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>↑ Airway resistance (esp. nonselective agents)</td>
<td>↑ Wheezing</td>
<td>↑ Bronchospasm</td>
<td>FEV1, ↑ Peak airway pressure</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperglycemia</td>
<td></td>
<td></td>
<td>Laboratory measurements of K¹ and glucose</td>
</tr>
<tr>
<td></td>
<td>Hypokalemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Fatigue, lethargy, peripheral paresthesia, withdrawal hypersensitivity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OB</td>
<td>All cross placenta, fetal effect: Bradycardia, hypotension, hypoglycemia</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Anticipated Problems/Concerns
- When β-blockers are administered in presence of anesthetic drugs they may unmask direct negative inotropic effects of concomitantly administered anesthetic; this effect results in profound ↓ in BP, CO

Adjuvants/Regional Anesthesia/Reversal
- Bradycardic effects often can be reversed by atropine
- Isoproterenol most effective at reversing negative cardiac (both dromotropic and inotropic) effects; but need to administer 1 dose of isoproterenol (2–25 µg/min) to reverse negative cardiac effect
- CaCl² (250–1000 mg) or glucagon (1–5 mg) administered IV (adult) effectively reverses myocardial depression
- Life-threatening bradycardia may require insertion of transvenous pacemaker
**Bicarbonate Sodium**

**Uses**
- Management of hyperkalemia
- Promote alkaline diuresis ( hastens secretion of salicylates, TCA; renal protective in rhabdomyolysis, transfusion reaction)
- Possible protection against contrast-induced nephropathy (CIN) in pts with compromised renal function.
- Alkalize local anesthetics (weak bases); uncharged form can cross bilipid cell membrane to bind to Na^+ receptor (site of action) intracellularly. Decreases time to onset, minimum concentration required. Can reduce pain with SQ injection.
- Controversial efficacy in metabolic acidosis. Usually reserved for situations when pH <7.1.

**Perioperative Risks and Implications**
- Hypernatremia; Na^+ retaining states (CHF, pulm edema, cirrhosis) can worsen
- Hyperosmolality
- Hypercapnia

**Drug Class/Mechanism of Action/Usual Dose**
- Electrolyte and alkalizer, dosing is specific to abn being treated.
- Hyperkalemia: 1 ampule of NaHCO_3 (50mEq/50 mL) over 3 min
- Metabolic acidosis: [0.2 × weight (kg) × (desired HCO_3-,measured HCO_3)] = bicarbonate dose in mEq; half of calculated dose is administered initially over 30 min; reassessment of ABG: pH, HCO_3, BE.
- Alkalizes urine, which prevents reabsorption of TCA, salicylates, Hg, and myoglobin into renal vasculature, aiding in excretion.
- Protection against CIN (unlabeled use): 154 mEq/L of NaHCO_3 in D5W, with an initial infusion of 3 mL/kg/hr in a 1 hr prior contrast bolus, 1mL/kg/hr for 6 hr post procedure.
- Local anesthetics: Typically 1 cc NaHCO_3; 10 cc lidocaine.

**DRUG EFFECTS**

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Acidosis can reduce inotropy, responsiveness to catecholamines. NaHCO_3 can exacerbate/cause CHF, edema from Na^+ load</td>
<td>BP, cardiac output, TEE</td>
</tr>
<tr>
<td>RESP</td>
<td>Pulm edema; hypercapnia, requiring adjustment in minute ventilation</td>
<td>X-ray, ABG</td>
</tr>
<tr>
<td>CNS</td>
<td>AMS/ increased ICP from hypercapnea, coma from hyperosmolality</td>
<td>Neurologic exam</td>
</tr>
<tr>
<td>Renal</td>
<td>Alkalizes urine, aiding in diuresis of toxic compounds; impaired renal function may be incapable of handling sodium load; protection against contrast nephropathy</td>
<td>Urine pH &gt;7, Cr, BUN</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Hypernatremia can reduce renin/aldosterone; hyperosmolality can increase ADH</td>
<td>ABG, HCO_3 levels, BMP, BNP, serum osm</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Concerns**
- Perform ABG, [K^+] prior to initiation of NaHCO_3 therapy, if possible.
- Plasma-expanding properties may make it useful in pts with HD shock.
- Consider controlled ventilation over spontaneous (LMA). If ETT in situ, evaluate if minute ventilation can be augmented to handle increased CO2 loads.
- Digoxin: Hypokalemia ca,n exacerbate effects.

**Induction/Maintenance**
- If excess NaHCO_3 creates alkalosis, basic drugs (opioids, local anesthetics) have increased activity due to higher non-ionized fraction crossing lipid membranes.
- Hypokalemia
- Other physiologic effects: Left shift in oxy-Hg curve, accelerated lactate production, CSF acidosis

**Special Considerations**
- Treatment for hyperkalemia is temporizing; does not reduce total body K^+.
- Necessitates adequate ventilation to excrete CO2 (produced when neutralizes H^+); otherwise, resp acids can result.
- Metabolic acidosis: Dx and Tx of underlying cause (hypovolemia, anemia, hypoxia, uremia, sepsis) should be undertaken; NaHCO_3 should be viewed only as temporizing measure.

**Overview/Pharmacology**
- H^+/K^+ ion membrane exchanger shifts K^+ intracellularly, reducing intravascular K^+.
- Excess HCO_3- ions are excreted renally, alkalinizing the urine.
- Na^+ + HCO_3- + H^+ ←→ H_2O + CO_2 via carbonic anhydrase.
- Rapid bolus can cause transient fall in MAP, rise in ICP; can be alleviated by slow IV infusion.
- 100 mEq of NaHCO_3 produces 2.24 L of CO2; equivalent to 10 min of CO2 production.

**Postoperative Period**
- Therapy should be guided by ABG analysis of pH, HCO_3, BE.

**Anticipated Problems/Concerns**
- No convincing evidence exists that NaHCO_3 confers any survival benefit in cardiac arrest/VF. Should not be used routinely in CPR. Consider when prolonged VF, severe acidosis, hyperkalemia, medication overdose (TCA, salicylate).
- No convincing evidence that improves mortality when used to treat metabolic acidosis, even at pH <7.1; additionally, use is not benign; can cause hypernatremia, hyperosmolality, hypervolemia, alkalosis, volume overload, hypokalemia, left shift oxyhemoglobin curve, CSF acidosis.
- Alternatives for buffering acidic states are limited and have no proven advantage over NaHCO_3.
- Carbicarb (equimolar sodium carbonate and NaHCO_3) may cause very alkaline pH, local tissue injury, dysrhythmias; vasodilator effect can reduce coronary perfusion pressure.
- THAM (tris(hydroxymethyl)aminomethane) acts intracellularly (vs NaHCO_3); can cause hypero.
- Tribonate (NaHCO_3+THAM+phosphate+acetate) may be more effective treating intracellula.

Bleomycin

**Uses**
- Treatment of squamous cell carcinoma
- Treatment of melanomas and sarcomas
- Treatment of testicular carcinoma
- Treatment of Hodgkin’s and non-Hodgkin’s lymphoma
- Sclerosing agent for malignant pleural effusion

**Perioperative Risks**
- Greater than 10%
  - Acute febrile reactions (25–50%)
- Dermatologic
  - 50% of pts can develop erythema, rash, striae, induration, hyperkeratosis, vesiculation, and peeling of the skin. This is predominantly seen on the palmar and plantar surfaces of the hands and feet.
  - Hyperpigmentation (50%), alopecia, and nail bed changes.
  - These effects are usually dose related and reversible with D/C.
- Gastrointestinal: Stomatitis and mucositis (30%), anorexia, weight loss
  - Between 1–10%
- Respiratory: Tachypnea, dyspnea, interstitial pneumonitis, and pulmonary fibrosis (5–10%)
  - Idiosyncratic reaction: A severe reaction similar to anaphylaxis has been reported in 1% of lymphoma pts treated with bleomycin. These reactions typically occur after the first or second dose.
  - Pulmonary fibrosis: The most severe toxicity of bleomycin, with risk increasing in elderly pts, those receiving >400 units total lifetime dose and possibly smokers and pts receiving concurrent O2 therapy
  - Experienced physician: Should be administered under the supervision of a physician experienced in delivering chemotherapy

**Contraindications**
- Hypersensitivity of bleomycin or any component of the formulation
- Severe pulm disease
- Pregnancy

**Worry About**
- Sustained O2 conc >30%
- Liberal use of maintenance fluids

**Overview/Pharmacology**
- 1 U bleomycin = 1 mg activity of bleomycin
- \( T_{1/2} = 2 \text{ hr} \)
- If Cr <35 mL/min exponentially ↑ \( T_{1/2} \)
- 70% is recovered in urine as active bleomycin

**Drug Class/Mechanism of Action**
- Mixture of cytotoxic antibiotics isolated from Streptomyces verticillus
- Cytotoxic action caused by inhibition of DNA synthesis
- Usual dose: 0.25–0.5 U/kg (10–20 U/m2) to 400 U (total dose)

**ASSESSMENT POINTS**

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Raynaud’s (rare)</td>
<td>Color changes in fingers</td>
<td>Observation</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Interstitial pneumonitis (10%)</td>
<td>Dose (&gt;250 U), age (&gt;65 y)</td>
<td>Dyspnea, fine rales and cough, fever</td>
<td>PFTs (↓ TLC, ↓ VC)</td>
</tr>
<tr>
<td></td>
<td>Pulmonary fibrosis (1%)</td>
<td>Previous lung disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>N/V</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>(Not assoc with pancytopenia)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Mucocutaneous toxicity (50%)</td>
<td>1–3 wk after start of Rx (dose 150–200 U)</td>
<td>Urticaria, hyperpigmentation, hyperkeratosis, alopecia</td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Period**
- Assess bleomycin cumulative dose (>250 U)
- Assess age (>65 y)
- Assess previous lung disease Hx
- Ask about previous radiation to thorax
- Obtain PFTs, CXR, ABGs

**Postoperative Period**
- Limit delivered O2 to <30% if adequate for O2 sat >89%.
- Limit fluids and avoid fluid overload.

**Key Reference:** McEvoy GK, ed. AHFS 95 Drug Information. Bethesda, MD: 600–602.

**Anticipated Problems/Concerns**
- Cyclophosphamide, radiation Rx (thorax) potentiates pulm toxicity.
- Cisplatin potentiates renal insufficiency.
- Vinca alkaloids (vincristine, vinblastine, VP-16) potentiates Raynaud’s phenomenon.
- Mitomycin C exhibits similar properties to those of bleomycin but with milder effects.
Calcium-Channel Blockers

Uses
- Prescribed to treat Htn, angina, supraventricular arrhythmias, cerebral vasospasm, and HCM

Perioperative Profile/Risks
- A significant proportion of the surgical population come to surgery chronically taking calcium channel blockers. Many pts receive Ca²⁺-channel blockers because of the systemic Htn, CAD or supraventricular arrhythmias. A combination of ACE inhibitors and CCB are indicated in diabetic pts with Htn. This class of drug effectively decreases myocardial O₂ demand through its effects on AV conduction, inotropy, and vasodilation of systemic and coronary vasculature. There is also theoretic evidence of an effect on plt function but clinically this has not been demonstrated.

Worry About
- Hypotension, meta-analysis of both cardiac and noncardiac RCTs shows a 50% increase in the incidences of unplanned periop hypotension.
- Neither RCTs or nonrandomized trials have demonstrated increased incidence of CHF; or the need for inotropic support
- AV nodal block or asystole has not been demonstrated, however, there is an increased utilization of temporary cardiac pacing after cardiac surgery.

Bradyarrhythmia, requiring treatment, has been demonstrated in a frequency similar to beta blockers.
- In both cardiac and noncardiac surgery, beneficial effects have been demonstrated; acute withdrawal may precipitate acute coronary ischemia.
- Pft function is a theoretic risk, however, increased bleeding or increased transfusion requirements have not been demonstrated in meta-analysis or nonrandomized trials.

Overview and Pharmacology
- Ca²⁺ channels: Functional pores in cardiac and smooth muscle cell membranes—allow calcium to flow down an electrochemical gradient. Channels are also present in sarcoplasmic reticulum and mitochondria. Calcium is a primary generator of the cardiac action potential and intracellular events regulating muscular contraction.
- Calcium enters through voltage-dependent or receptor-operated channels. Most of the effects of calcium channel blockers is regulated by components of the L (long lasting) type receptor.
- Amlodipine is the most widely prescribed calcium channel blocker; a half life of 30–50 hr; a bioavailability, PO onset of action 2–3 hr, peak effect 8 hr, 85% eliminated by first-pass hepatic metabolism with elimination T½ of 3–7 hr. The IV effects are almost immediate.
- Diltiazem: 89–90% PO absorption, 40–70% bioavailability, PO onset of action <15 min, peak effect 30 min, 60% metabolized by liver, remainder excreted by kidneys, T½ 3.5–6.0 hr
- Bepridil: >90% absorption, >80% bioavailability, PO onset of action 2–3 hr, peak effect 8 hr, hepatic elimination with T½ 26–64 hr
- Hepatic disease may necessitate decreased dosing of verapamil and other Ca²⁺-channel blockers

Drug Class/Mechanism of Action
- Four different classes of Ca blockers:
  - 1,4 dihydropyridine (e.g., amlodipine, nifedipine, nicardipine),
  - Phenylalkylamines (e.g., verapamil)
  - Benzothiazepines (e.g., diltiazem)
  - Diarylaminopropionolamine ether (e.g., bepridil)
- Mechanisms of action: Amlodipine—blockade of voltage-dependent L-type inactive Ca²⁺-channel receptor that has recently undergone activation and cannot open; the other 3 classes bind to specific receptors within the L-type channels,
- The dose periop use of calcium channel blockers (nicardipine, diltazen, and verapamil) should be titrated to effect.

ASSESSMENT POINTS

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Ischemic protection, myocardial depression,</td>
<td>Short-acting nifedipine</td>
<td>Hypotension,</td>
<td>BP measurement, ECG, ECHO for ventricular</td>
</tr>
<tr>
<td></td>
<td>vasodilation, AV conduction slowing</td>
<td>should be avoided, due to</td>
<td>bradycardia</td>
<td>contractility</td>
</tr>
<tr>
<td>CEREBRAL</td>
<td>Cerebral vasodilation and ↓vasospasm, there is no indication of increased stroke in clinical studies</td>
<td>Ongoing assessment of neurologic status in pts at risk for vasospasm</td>
<td>Changes in neurologic assessment</td>
<td>Cranial Doppler or angiogram</td>
</tr>
<tr>
<td>NEURO</td>
<td>Potentiation of NMBs</td>
<td>Increased risk of aspiration if extubated with residual block</td>
<td>Prolonged block</td>
<td>Use of NMB monitor</td>
</tr>
<tr>
<td>ENDO</td>
<td>Nifedipine delays insulin release and ↓serum glucose in DM; diltiazem has no effect on insulin, glucagon, growth hormone, cortisol levels</td>
<td>Better glucose control in DM pts on nifedipine</td>
<td>Blood glucose</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Concerns
- Continue chronic Ca²⁺-channel blockers throughout the periop period to minimize ↑BP, or ischemic syndromes
- Careful assessment of baseline hemodynamic variables
- Drug interactions: Verapamil ↑ digoxin levels; cimetidine and ranitidine ↑ serum levels of Ca²⁺-channel blockers through ↓hepatic blood flow

Monitoring
- Routine
- Pacing capability if assoc AV block, or CHF
- Arterial line if BP instability likely

Airway
- No special concerns

Preinduction/Induction
- Assess hemodynamics and ECG before induction

Maintenance
- Goal-directed fluid therapy may decrease incidence of hypotension
- Volatile anesthetics may potentiate vasoactive effects
- Effects of Ca²⁺-channel blockers can be antagonized by administration of calcium or by administration of other pressor agents.

Extubation
- Check TOF if using NMBs; potentiation of succinylcholine, pancuronium, p-tubocurarine, atracurium, vecuronium have been described.

Potential for incomplete reversal of NMBs owing to interaction with Ca²⁺-channel blockers on the postsynaptic membrane and blockade of Ca²⁺-channels in skeletal muscle.

Anticipated Problems/Concerns
- Hypotension
- Bradycardia
- AV nodal block and the increased use of temporary pacemakers
- Paradoxical aggravation of myocardial ischemia with the use of short-acting nifedipine due reflex sympathetic stimulation and tachycardia
**Capsaicin**

**Uses**
- Topical analgesic if repeatedly used in diabetic neuropathy, postherpetic neuralgia, osteoarthritis, rheumatoid arthritis, and other painful disorders
- Spray used as less-lethal force by law enforcement
- May cause apoptosis in breast and prostate cancer cells

**Perioperative Risks**
- None reported
- After exposure to aerosolized capsaicin, e.g., in a trauma pt restrained by police, airway irritability, coughing, and bronchospasm may occur.

**Pharmacokinetics/Pharmacodynamics**
- Topical effects only since very poorly absorbed from the skin.

**Drug Class/Mechanism of Action/Usual Dose**
- Analgesic; 8-methyl-N-vanillyl-noneamide, an agonist of temp-sensitive TRPV1 receptor.
- Selective binding to afferent C fibers in the skin causes neuronal excitation and release of substance P.
- Repetitive application causes depletion of substance P and calcitonin gene-related peptide from C fibers and eventually reduced sensitivity to pain.

- Reversible degeneration of small nerve fibers in the epidermis occurs with long-term use.
- Various creams and patches available over-the-counter and by prescription in a wide range of strengths as low as 0.025%.
- For best results, repeated application of topical capsaicin is necessary several times a day for several weeks.
- An 8% patch has been recently approved by the FDA and an injectable capsaicin product (Qutenza) is in clinical trials at the time of this writing.

**DRUG EFFECTS**

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<tbody>
<tr>
<td>DERM (topical)</td>
<td>Burning pain at site, possibly redness</td>
<td>Hx of chronic pain</td>
<td>Thermal allodynia at site, or hypaesthesia at site of application</td>
<td></td>
</tr>
<tr>
<td>EENT (topical or inhaled)</td>
<td>Lacrimation, blepharospasm</td>
<td>Accidental exposure of topical agent to the eye; exposure to aerosol in confrontation with law enforcement.</td>
<td>Eye exam</td>
<td></td>
</tr>
<tr>
<td>RESP (inhaled)</td>
<td>Extreme airway irritability, coughing and bronchospasm, and possibly death with heavy exposure.</td>
<td>Exposure to aerosol in confrontation with law enforcement or industrial accident.</td>
<td>Auscultation, pulse oximetry</td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**
- None known

**Drug Interactions**
- None known

**Anticipated Problems/Concerns**
- Only moderate to poor efficacy in chronic pain syndromes, which have an anatomically superficial and localized pain generator.
- Compliance is poor because of burning sensation with application.
Carbamazepine

**Uses**
- FDA-approved (since 1976) for
  - Epilepsy: Complex partial seizures; generalized tonic-clonic seizures; and mixed seizure pattern of the above mentioned, with the exception of petit mal seizures
  - Bipolar disorder with manic or mixed episodes
  - Trigeminal and glossopharyngeal neuralgia
- Off-label use for
  - Pain syndromes, e.g., phantom limb pain, complex pain syndromes, diabetic neuropathy
  - Psychiatric disorders, e.g., PTSD, ADHD, mania, schizophrenia, bipolar disorder in remission, dementia, alcohol withdrawal
  - Other disorders, e.g., prevention of seizures, paroxysmal choreoathetosis, RLS, diabetes insipidus
- Oxcarbazepine, a keto-analogue of CBZ
- FDA-approved (since 2000)
- Indicated for partial seizures

**Perioperative Risks/Worry About**
- PHARM: Clinically significant drug interactions
- CNS: Increased sedation, dizziness, and ataxia
- CVS: Aggravation of HTN, hypotension, CAD, arrhythmias, and AV block (rare, but potentially life-threatening)
- LAB: Higher incidence of hyponatremia, aplastic anemia, agranulocytosis, thrombo- and leukopenia as well as elevated LFTs and hypothyroidism

**Overview/Pharmacology**
- Pharmacokinetics/pharmacodynamics
  - Rapid oral absorption
  - Peak plasma concentrations: Tablet 2–6 hr after ingestion, suspension 1.5 hr, extended release up to 26 hr
  - Plasma protein binding: 70–80%, epoxide metabolite 50%
  - Volume of distribution Vd: Children: 1.9 L/kg; adults: 0.59–2 L/kg
  - Initial half-life values: Range from 25–65 hr, ↑ to 12–17 hr on repeated doses
  - Metabolization: To 98% in the liver:
    - Cytochrome P450 3A4 as the major isomorph responsible for formation of the principle metabolite carbamazepine-10,11-epoxide (antiseizure effects), accounting for many of the dose-limiting side effects
  - Induction of its own metabolism, half-life therefore variable (autoinduction completed after 3–5 wk of a fixed dosing regimen); dose adjustment necessary 2–4 wk after initiation of therapy
  - Excreted (72% in urine, 28% in feces), largely in form of hydroxylated and conjugated metabolites, only 3% unchanged, indicating that renal insufficiency can potentially increase CBZ toxicity
  - Faster metabolism in children <15 yr
  - Esp. in geriatric pts neuropsychiatric symptoms with agitation, confusion, psychosis possibly due to anticholinergic and tricyclic effects of CBZ and pre-existing renal insufficiency
  - No known substantial gender influence on pharmacokinetics
  - Race not studied in pharmacokinetics, but differences in HLA-B genes account for the frequency of side effects like Stevens-Johnson syndrome
  - Drug-drug interactions relevant for anesthesia
- Increased CBZ plasma levels with potential toxicity due to CYP3A4 inhibitors: macrolide antibiotics such as clindamycin and clarithromycin, azoles like fluconazole, metronidazole, dihydroartemisinin, verapamil, cimetine, propranolol, valproate, possibly levetraacetam, acetazolamide, some antidepressants (esp SSRIs and also other triyclics), oxfibutynin, grapefruit juice
- Decreased CBZ plasma levels with potentially ↑ risk for seizures due to CYP3A4 inducers CBZ (autoinduction): theophylline, phenytoin, phenobarbital, and other barbiturates
- Increased levels of substances and risk for their toxicity due to CBZ: phenytoin, lithium (potentially fatal neurotoxic reactions), acetaminophen, theophylline (also ↓), alcohol
- Decreased levels of medications and therefore less effectiveness due to CBZ: hormonal contraceptives (frequent—potential of pregnancy), anticoagulants (e.g., warfarin), caspofungin, corticosteroids, other antiepileptics like valproic acid with the exception of topiramate, benzodiazepines (e.g., midazolam, phenobarbital) and other barbiturates, tramadol, methadone and theoretically all opioids, muscle relaxants (diazepam, rocuronium, pancuronium, succinylcholine, vecuronium, and, to a much lesser extent also, (cis-)atracurium and questionably mivacurium), aprepitant, statins, felodipine, neuroleptics like clozapine and, to a lesser extent, haloperidol and others, tricyclics, theophylline (also ↑)

**Side effects**
- CNS
  - Most frequent and already within upper normal range of plasma level
  - Dizziness, drowsiness, fatigue, confusion, ataxia, impaired cognition, transient diplopia, oculomotor disturbances, nystagmus, blurred vision
  - Rare
  - Speech disturbances, dystonia, peripheral neuritis/neuropathy and paresthesias, depression/agitation, suicidal ideation, visual hallucinations, headache, hyperacusis, tinnitus, myalgia, isolated cases of malignant neuroleptic syndrome, activation of latent psychosis
- RESP
  - Less frequent
  - Pulm hypersensitivity with fever, dyspnea, or pneumonitis
- CVS
  - Rare (but potentially fatal) above upper range of plasma level
  - CHF, edema, aggravation of HTN, hypotension syncope and collapse, aggravation of CAD/myocardial infarction, arrhythmias (esp. bradycardia) and AV block, thrombophlebitis, thromboembolism, or lymphadenopathy, edema
- HEM
  - Rare
  - Aplastic anemia, agranulocytosis, pancytopenia, thrombocytopenia, leukopenia, leukocytoysis, acute neutrophilic purpura
- DERM
  - Less frequent
  - Toxic epidermal necrolysis and Stevens-Johnson syndrome, erythematous rashes, urticaria, photosensitivity reactions, exfoliative dermatitis

**Contraindications**
- Hypersensitivity to any drug with tricyclic compounds (e.g., amitriptyline, desipramine, etc.) and MAO inhibitors, higher in pts with hypersensitivity to phenytoin and phenobarbital
- Complete AV block (risk of asystole)
- Bone marrow depression (aggravation)
- Porphyria (increases porphyrin precursors)
- Pregnancy (teratogenic, malformations, pregnancy category D)
- Co-administration with nefazodone (hepatoxicity, insufficient plasma level of nefazodone)

**Acute Toxicity**
- Lowest known lethal dose in a 24-year-old adult: 3.2 g, and in a 3-year-old girl 1.6 g
- First signs and symptoms appear after 1–3 hr, primarily with NM dysfunctions like seizures, coma, and resp depression; CV disturbances with arterial hypotension milder and only with very high doses (>60 g)

**Drug Class/MEchanism of Action/Usual Dose**
- Derivative of iminostilbene (5H-Dibenzo[b,f]azepine) with a structure of a tricyclic 2° amine similar to other tricyclic compounds
- Reduces polysynaptic responses and blocks post-tetanic potentiation through stabilization of voltage-gated sodium channels protein type V subunit alpha similar to phenytoin
- Potentiation of GABA(A) receptors
- Mild anticholinergic, central antidiuretic, anti-arrhythmic, antidepressant, sedative, and NMB activity
- Very potent inducer of CYP 450 3A4 and as such significant drug-drug interactions possible with a variety of drugs used in anesthesia
- Available only oral as normal, chewable, and extended release tablets and suspension
- Usual dose range between 200 and 1600 mg/day in adults (max. 2000 mg/d), 10 mg/kg – 35 mg/kg in pediatrics
- Monitored and guided by blood level (4–12 mcg/mL)
**Oxcarbazepine**

- Pro-drug, activated to elicarbazepine in the liver, converted into active metabolite 10-hydroxy CBZ

### DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Heart failure</td>
<td>SOB, palpitations</td>
<td>Auscultation</td>
<td>X-ray, ECHO</td>
</tr>
<tr>
<td>CNS</td>
<td>Sedation</td>
<td>Clinical assessment</td>
<td>Clinical assessment</td>
<td></td>
</tr>
<tr>
<td>METAB</td>
<td>Hyponatremia</td>
<td>Edema, drowsiness</td>
<td>Oliguria</td>
<td>Electrolytes</td>
</tr>
<tr>
<td>HEM</td>
<td>Anemia/aplasia</td>
<td>Palor, Petechias</td>
<td>Clinical assessment</td>
<td>CBC</td>
</tr>
<tr>
<td>RENAL</td>
<td>Renal insufficiency</td>
<td>Azotemia, Acute oliguria</td>
<td>Cr, BUN, ABG</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumonitis</td>
<td>Pulm hypersensitivity</td>
<td>Clinical assessment</td>
<td></td>
</tr>
</tbody>
</table>

**Preoperative Concerns**

- Continue CBZ medication
- Check blood level to assess potential risk of toxicity, esp. in old pts with polypharmacy.
- Check for pathologic laboratory tests in mostly affected organ systems:
  - HEM: Electrolytes, leukocytes, erythrocytes, plt
  - HEP: Liver enzymes
- REN: Kidney function with urinalysis and BUN
- METAB: Thyroid function (TSH)
- Assess CVS: Physical capacity and ECG (arrhythmias, AV-block incl second- and third-degree block, ischemic changes, QT-prolongation)
- Assess resp in regards to pulm hypersensitivity/pneumonitis

**Induction/Maintenance**

- Take potential for drug-drug interactions seriously.
- Increased dosages of anesthetics very likely, concerning esp IV sedatives like thiopentone (additive effects of CBZ with propofol and ketamine, but antagonistic effects with etomidate in animal studies), midazolam and muscle relaxants (incl cis-jatracurium) as well as opioids since their primary metabolism occurs via CYP 450 3A4

**Perioperative Implications**

**Anticipated Problems/Concerns**

- Toxic plasma levels of CBZ possible with co-medication (e.g. macrolide antibiotics and levetiracetam) which possibly result in delayed wake-up, drowsiness, and tendency to syncope postop
- Nerve stimulator recommended to dose and titrate muscle relaxants
- Be attentive for arrhythmias, esp. bradycardia, aggravation or appearance of AV-block and cardiac ischemia as an additive effect of concomitant use of cardiodopressive drugs that are used during anesthesia
- Reckon with aggravated arterial hypo- or Htn
- Consider increased tendency to hyponatremia esp. in certain surgeries and co-administration of drugs (e.g. in craniotomies due to the administration of manitol)
- Adjust anesthetic medications (e.g., dosage of opioids) and plan (e.g., potentially extended monitoring) according to possible pre-existing cardiac, hepatic, and renal damage
- Reckon with possibly increased risk for laryngospasm and bronchospasm during emergence (although rare)
- Develop anesthetic plan to facilitate early mobilization to avoid thromboembolic events

**Postoperative Period**

- Continue with CBZ medication; bear in mind however that change from tablets to suspension might cause higher plasma peak levels with potentially increased side effects.

- Monitor esp. geriatric pts for increased confusion/agitation, AV-block, bradycardia or SIADH
- Inform pts who take oral hormonal contraceptives about decreased safety and the need of additional safety precautions for anticonception.
- Monitor for serious and fatal dermatologic reactions, incl toxic epidermal necrolysis (TEN) and Stevens-Johnson syndrome (SJS) esp. in the presence of an inherited variant of the HLA-B gene, HLA-B*1502
- Often in pts originating from certain Asian countries (15% of the population in Hong Kong, Thailand, Malaysia, and parts of the Philippines, 10% of Taiwan and 4% in North China, but only 2–4% of South Asians incl Indians, and only <1% of people of Japan and Korea)
- HLA-B*1502 practically absent in people who are not of Asian origin (e.g. Caucasians, Native Americans, African–Amerincans and Hispanics)

Uses

- One of the treatment modalities available to prevent cancer cells from multiplying, invading, metastasizing and killing the host; also inhibit multiplication of normal cells, therefore have a high incidence of debilitating systemic side effects.

<table>
<thead>
<tr>
<th>DRUG CLASS</th>
<th>Agents</th>
<th>Used to Treat</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alkylating Agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alkyl sulfonate</td>
<td>Busulfan (Myleran)</td>
<td>Chronic granulocytic leukemia, acute leukemia, lymphoma</td>
</tr>
<tr>
<td>Ethylenimine derivative</td>
<td>Thiotepa</td>
<td>Testis, ovary, cervix, bladder, endometrial, lung, soft tissue, bone</td>
</tr>
<tr>
<td>Metal salt</td>
<td>Carboblatin, cisplatin</td>
<td>Solid tumors, lymphoma</td>
</tr>
<tr>
<td>Nitrogen mustard (HN₂)</td>
<td>Chlorambucil (Leukeran), cyclophosphamide (Cytoxan), Ifosamide, Melphanal (Alkeran), Mechlorethamine, estramustine</td>
<td>Chronic lymphocytic leukemia, Non-Hodgkin’s, Waldenstrom’s macroglobulinemia, Trophoblastic tumors</td>
</tr>
<tr>
<td>Nitrosourea</td>
<td>Carmustine (BCNU), lomustine, streptozocin</td>
<td>Brain tumors, lymphomas</td>
</tr>
<tr>
<td>Triazene</td>
<td>Dacarbazine, temozolamide</td>
<td>Melanomas, brain tumors</td>
</tr>
<tr>
<td><strong>Antimetabolites</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antifolate</td>
<td>Methotrexate, pemetrexed</td>
<td>Solid tumors, leukemia</td>
</tr>
<tr>
<td>Purine analogs</td>
<td>Cladribine, clofarabine, fludarabine mercaptopurine, thioguine</td>
<td>Leukemias, lymphomas, osteosarcoma, ALL</td>
</tr>
<tr>
<td>Pyrimidine analogs</td>
<td>Azacitidine, cytarabine, decitabine, fluorouracil, decitabine, flouxuridine, gemcitabine</td>
<td>Myelodysplastic syndromes</td>
</tr>
<tr>
<td><strong>Natural Products</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotics</td>
<td>Bleomycin, danuro hicin, doxorubicin (Adriamycin), Mitomycin, valrubicin</td>
<td>Lymphomas, head and neck, testis, vulva, anus, skin cancer</td>
</tr>
<tr>
<td>Enzyme</td>
<td>Asparaginase</td>
<td>Acute leukemias, ovary, breast</td>
</tr>
<tr>
<td>Mitotic inhibitor</td>
<td>Vinblastine, vincristine, vindesine, vinorelbine</td>
<td>Rhodomyosarcoma, anal carcinoma, Wilms tumor, breast, lung, ALL</td>
</tr>
<tr>
<td>Topoisomerase inhibitors</td>
<td>Irinotecan, etoposide, teniposide, topotecan</td>
<td>Lung, brain, lymphoma</td>
</tr>
<tr>
<td>Microtubule polymer stabilizer</td>
<td>Docetaxel, paclitaxel</td>
<td>Neuroblastomas</td>
</tr>
<tr>
<td><strong>Moleculally Targeted Agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monoclonal antibody</td>
<td>Alemtuzumab, cetuximab, rituximab, bevacizumab</td>
<td>B and T cell leukemia, head and neck (cetuximab), colon (cetuximab, panutumab), renal cell carcinoma, lung (bevacizumab), breast (trastuzumab, bevacizumab)</td>
</tr>
<tr>
<td>Gene expression modulators</td>
<td>Retinooids, renoids</td>
<td>Multiple myeloma</td>
</tr>
<tr>
<td>Interleukin 2 receptor toxin</td>
<td>Deneleukin, difitox</td>
<td>Graft vs. host disease, APMIL</td>
</tr>
<tr>
<td>Receptor tyrosine kinase inhibitor</td>
<td>Dastinib, sorafenib, semaxanib, sunitinib, gefitinib</td>
<td>GI, breast, lung cancers, Non small cell lung cancer (gefitinib/erlotinib), pancreas (erlotinib), renal cell carcinoma (sorafenib, sunitinib, axitinib), hepatocellular carcinoma (sorafenib)</td>
</tr>
<tr>
<td><strong>Biological Response Modifiers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interferons</td>
<td>Interferon α-2a, interferon α-2b</td>
<td>Melanoma, renal cell carcinoma, multiple myeloma, kaposis, CML, mycosis fungoides, non-Hodgkins</td>
</tr>
<tr>
<td>Interleukins</td>
<td>Aldesleukin (interleukin 2), hang R.O, oprelvekin, difitox</td>
<td>Myelodysplastic syndrome, multiple myeloma</td>
</tr>
<tr>
<td>Nonspecific immune modulation</td>
<td>Thalidomide, lenalidomide</td>
<td>Cytopenia</td>
</tr>
<tr>
<td>Myeloid and erythroid stimulating factor</td>
<td>Epoitin, filgrasin</td>
<td></td>
</tr>
<tr>
<td><strong>Miscellaneous Agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bisphosphonates</td>
<td>Pamlidronate, zoledronic acid</td>
<td>Breast cancer, cancer with bone metastasis</td>
</tr>
<tr>
<td>Methylhydralazine derivative</td>
<td>Procarbazine</td>
<td>Hodgkin’s, lymphosarcoma, reticulosarcoma</td>
</tr>
<tr>
<td>Photosensitizing agent</td>
<td>Profimer</td>
<td>Lung, cholangiocarcinoma, bladder</td>
</tr>
<tr>
<td>Platelet reducing salt</td>
<td>Anagrelide</td>
<td>Chronic granulocytic leukemia, polycythemia rubra vera</td>
</tr>
<tr>
<td>Substituted urea</td>
<td>Hydroxyurea</td>
<td>Head and neck cancer, acute leukemia</td>
</tr>
</tbody>
</table>

(Continued)
Mechanism of Action

- Alkylating agents: Alkylate the DNA double strands with resultant inhibition or inaccurate replication of DNA.
- Antimetabolites: Inhibit mitosis by blocking or altering the substrate and inhibit DNA synthesis.
- Natural products: Inhibit microtubular proteins with resultant metaphase mitotic arrest, some cause DNA strand breakage and arrest cell multiplication.
- Antibiotics: Antitumor antibiotics, affect the synthesis of nucleic acids.
- Enzymes: Deprive cancer cells of essential amino acids by a chemical reaction with the essential substrate, (e.g., asparaginase catalyses the reaction of asparagine to aspartic acid and ammonia)
- Molecularly targeted agents (monoclonal antibodies, tyrosine kinase inhibitors etc.): Directed to inhibit/antagonize vascular endothelial growth factors or their receptors.
- Adjuvants: Interleukins (IL-2): Enhance killer cell activity, lymphocyte mitogenesis and cytotoxicity.
- Interferon α 2a, 2b: Direct inhibition of tumor cell growth and modulation of the immune response, activation of NK cells, modulation of antibody production and induction of histocompatibility antigens.
- Hormones and hormone antagonists: Inhibit tumor growth by exploiting hormone sensitive tumor receptors.

Perioperative Risks

- Cardiac: Caused by antibiotic chemotherapeutic agents. Acute toxicity: acute fulminant cardiomyopathy; chronic toxicity: cardiomyophathy and biventricular failure. Adriamycin and daunorubicin can cause acute fulminant or chronic recurrent cardiomyopathy. Biventricular failure is dose related, increased risk when dose >550 mg/m² and with advancing age. Echocardiography prior to treatment with adriamycin and post if symptoms develop will help in early DX and further progression of heart failure by altering treatment regimens. High dose cyclophosphamide and busulfan can cause cardiotoxicity too. Interleukins can cause sepsis like syndrome with vasodilatation and hypotension.
- Pulmonary: Interstitial lung disease bronchiolitis obliterans and organizing pneumonia (BOOP), and predisposition to O₂ toxicity. Bleomycin, busulfan and the monoclonal antibodies (Rituximab, cetuximab, bevacizumab, alemtuzumab) can predispose to interstitial lung disease. Gemcitabine can cause pneumonitis and fibrosis. Serial estimation of DLCO can help diagnose onset of interstitial lung disease at an early stage. Vincristine can cause severe bronchospasm during its administration. Carmustine and lomustine can cause delayed pulm fibrosis. Interleukins can cause pulm edema from leaky capillaries.
- Hematology: All chemotherapeutic agents cause cytopenia. Severe bone marrow depression is more frequent with alkylating agents, antimitabolites, plant alkaloids, antibiotics and nitrosoureas. Aplastic anemia, anemia, thrombocytopenia and neutropenia predispose to increased risk of infection and hemorrhage.
- Nervous system: Acute cerebellar syndrome with 5FU. Methotrexate can cause leukoencephalopathy. Vincristine can cause autonomic and PNS toxicity. Cyclophosphamide and ifosfamide can cause water retention with resultant hyponatremia and coma. High dose cis-platinum can cause ocular toxicity.
- Renal: Hemorrhagic cystitis with ifosfamide and cyclophosphamide. Methotrexate can cause renal tubular necrosis. Streptozocin can be nephrotoxic.
- Hepatobiliary: 6-Mercaptopurine, thioguanine, nitrhamycin and L-asparaginase can be hepatoxic.
- Immune suppression: All chemotherapeutic agents cause immune suppression from neutropenia, but the incidence is higher with alkylating agents, antimitabolites and plant alkaloids.
- Fluid and electrolytes: Interleukins cause capillary leak syndrome similar to systemic sepsis, resulting in fluid retention causing airway and pulm edema and circulatory instability. Treatment cycles of acute leukemias, lymphomas, particularly Burkett lymphoma, can be complicated by tumor necrosis syndrome due to rapid cell destruction (high sensitivity to chemotherapy).
- Endocrine: Use of steroids with chemotherapeutic agents impairs blood sugar control. Steroids can cause wt gain, truncal obesity and moon facies.

Anesthetic Implications

- Drug interactions: Prolongation of succinylcholine by inhibition of pseudocholinesterase (cytoxan)
- Vincristine causes muscle wasting; therefore, risk of hyperkalemia with succinylcholine. Thiopeta can further prolong pancuronium. NSAIDs can elevate m ethotrexate levels. Procarbazine can exaggerate CNS depression of sedatives and analgesics, therefore consider careful titration of analgesics and CNS depressants.
- Airway: Cytopenias, particularly thrombocytopenia, can result in easy bruising and airway hemorrhage/bleeding can complicate laryngoscopy and intubation. High dose steroid regimen can result in moon facies and airway edema. Stomatitis and mucosal dryness accompanies many chemotherapy regimens predisposing to easy mucosal bruising even with gentle airway manipulation.
- Venous access: Chemotherapeutic agents cause venous sclerosis, and IV access can be difficult. Will often require a semipermanent central venous access (portacath/PICC lines).
- Pulmonary: High inspired O₂ concentration can cause pulm toxicity in pts treated with bleomycin. Chemotherapy induced pulm toxicity can present as restrictive lung disease.
- Renal: Chemotherapy induced impaired renal function can alter drug excretion of some active metabolites. Periop NSAIDs can worsen pre-existing renal disease. Risk of postop renal failure is high when renal function is already impaired.
- Neuropathy: Care with positioning; careful documentation of pre-existing neuropathy prior to performing regional techniques.
- Hematology: Thrombocytopenia can increase the risk of hemotoma; low plt count may contraindicate neuraxial anesthesia. May require transfusions due to anemia/aplastic anemia.
- Photodynamic therapy (profimer): Pts are extremely photosensitive, will need to be in a location without bright lights.

Class and Type | Agents | Used to Treat
--- | --- | ---
Hormones and Hormone Antagonists | Fluoxymesterone | Prostate cancer
Androgen, Androgen antagonist | Bicalutamide | Breast cancer
Estrogen, estrogen antagonist | Diethylstilbestrol | Breast
Thyroid hormones | Tamoxifen | Thyroid
Levothyroxine | | |

Chloramphenicol (Chloromycetin)

Uses
- Infections such as typhoid fever, meningitis (Haemophilus influenzae, Neisseria meningitidis, Streptococcus pneumoniae), plague (Yersinia pestis), and rickettsiosis not treatable with other antibiotics
- Infections in pts with hypersensitivity to penicillin

Risks
- Anemia (dose dependent), aplastic anemia (dose independent)
- P450 (CYP) inhibition

- Grey Baby Syndrome (in premature infants because of the lack of liver UDP-glucuronyltransferase)

Worry About
- Increased T½ of dicumarol, warfarin, chlorpropamide, phenytoin, tolbutamide

Overview/Pharmacology
- Inhibition of protein synthesis by interfering with the incorporation of amino acids into ribosomes (inhibits 50S peptidyltransferase, bacteriostatic)
- Active against gram-positive and gram-negative bacteria incl Salmonella typhi, Proteus, and Rickettsia

- Decreases P450 (CYP—multiple isoforms) activity thus changing T½ of P450-dependent drugs such as warfarin (see above)

Drug Class/Usual Dose
- Antibiotic for otherwise intractable gram-negative infections (e.g., salmonellosis, Haemophilus influenzae, meningitis)
- Usual dosage: 50 mg/kg/d IV in divided doses

Drug Effects
- Newborns who cannot glucuronide-conjugate chloramphenicol may develop abd distention, cyanosis, vascular collapse, and death (rare) known as Grey Baby Syndrome

Key References:
Cimetidine

Michael F. Roizen

Uses
- Incidence in USA: >1,000,000 plus
- Rx for ulcers, gastric reflux, gastric hypersecretion
- High levels assoc with confusional states in elderly

Perioperative Risks
- Drug interactions esp. with local anesthetics (↑ toxicity), aldomet, clonidine (CNS toxicity)

Worry About
- Decreased hepatic P450 clearance of drugs, ↓ hepatic blood flow, ↑ fentanyl, phenothiazine, β-blocker drug, lidocaine, with ↑ potential for toxicity

Overview/Pharmacology
- H₂ antagonist
- Cleared by renal excretion; ↓ dosage intervals to 12 hr with Cr clearance of 0–20 mL/min/1.73 m²
- Decreased hepatic metab of drugs requiring specific cytochrome P450 (β-blocking agents, Ca²⁺-channel blockers, theophylline, phenothiazines) or drugs requiring liver for first-pass metab (by ↓ hepatic blood flow—lidocaine, β-blocking agents)

Drug Class/Mechanism of Action/Usual Dose
- H₂ antagonist
- Chronically taken for ulcer Rx, prophylaxis or to raise gastric pH for prophylaxis or Rx of gastric reflux
- Acutely taken for prophylaxis against pulm aspiration and part of prophylaxis against immune or nonimmune CV effects from immune or nonimmune release of H₂
- Usual dose: 100–300 mg bid
- Alternatives
  * Other H₂ antagonists; longer-acting agents have displaced much of use; now available OTC
  * Antibiotics to ↓ Helicobacter pylori (tetracycline + metronidazole + bismuth)


D RUG E FFECTS

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<tr>
<th>System</th>
<th>Effect</th>
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</tr>
</thead>
<tbody>
<tr>
<td>HEPATIC</td>
<td>↓ Hepatic drug metab</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>↓ Gastric acid secretion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Weak antiandrogenic effect; gynecomastia (men)</td>
<td>Gynecomastia</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Renal</td>
<td>BUN, Cr</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Placenta—crosses placental barrier, excreted in milk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Poor penetration to CNS; with high doses in pts, esp. with impaired renal function, assoc with disorientation to coma</td>
<td>CNS exam</td>
<td></td>
</tr>
</tbody>
</table>

Perioperative Implications/Possible Drug Interactions

Preoperative Concerns
- Cimetidine + clonidine or aldomet assoc with CNS dysfunction
- Decreased clearance of phenothiazines, phenytoin, theophylline

Induction/Maintenance
- Fentanyl T₁/₂ may be prolonged 2° to direct or indirect ↓ in hepatic BF by cimetidine

Adjuncts/Regional Anesthesia/Reversal
- Increased biologic availability of lidocaine and other local anesthetics and thus toxicity
- Increased NMB agent requirements anecdotally reported (mechanism unknown)

Anticipated Problems/Concerns
- Decreased hepatic P450 clearance of drugs, ↓ hepatic blood flow, ↑ fentanyl, phenothiazine, β-blocking drug, lidocaine potential for toxicity
- CNS dysfunction by itself (esp. in aged and those with ↓ renal function) or with clonidine and aldomet
Cisplatin

Uses (See also Chemotherapeutic Agents)
• Pts undergoing chemotherapy for testicular, ovarian, or bladder cancer

Perioperative Risks
• End-organ damage, esp. renal and neurotoxicity
• Renal toxicity prominent, seen in 28–36% of pts after 1 dose: Effect cumulative, minimized by aggressive hydration, allowing renal function to return to baseline between treatments
• Decrease in renal tubular function is dose-related, typically occurs during 2nd wk of administration
• Hyperuricemia, hyponaglemesia, hypocalcemia, hypotremesia, hypokeramia, hypophosphatemis has been reported and are related to renal tubular damage. Allopurinol Rx reduces uric acid levels.
• Neurotoxicity happens to 85% of pts at total dose over 300 mg/m². Peripheral sensory neuropathy, hearing loss, autonomic neuropathy, Lhermitte’s sign (electrical sensation running down the back and into the arms), seizures, and encephalopathy predominates.

DRUG EFFECTS

<table>
<thead>
<tr>
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<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Ototoxicity (31% of pts) manifested as tinnitus or loss of hearing; more pronounced in children</td>
<td>Total exposure</td>
<td>Audiometry</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Anaphylactic-like reactions with edema, bronchospasm reported</td>
<td>SOB after administration, palpitations, CV exam</td>
<td>ECG</td>
<td></td>
</tr>
<tr>
<td>HEPAT</td>
<td>Transient elevations in liver enzymes reported with use of cis-DDP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>N/V severe, triggered by action at chemoreceptor trigger zone of medulla</td>
<td>N/V within 1–4 hr up to 24 hr</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Mild–moderate myelosuppression (25–30%)</td>
<td></td>
<td>CBC</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Renal toxicity – typically nonoliguric with increased loss of water, electrolytes, and hypomagnesemia</td>
<td>BUN, Cr, electrolytes, Mg²⁺</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Hearing loss, autonomic neuropathy, and encephalopathy. Seizures with high acute doses.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Peripheral neuropathies in a stocking/glove distribution with prolonged Rx of 4–7 mo</td>
<td>Total exposure (&gt;300 mg/m²)</td>
<td>Neuro exam</td>
<td>Pinprick vibration</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Concerns
• Determine the total exposure of the pt as most toxicities are related to total dose
• Estimate GFR from serum creatinine or post-chemotherapy studies of renal function

Adjuvants/Regional Anesthesia/Reversal
• One case report of a 14-y female pt who experienced profound nerve injury after an interscalene block with bupivacaine and epinephrine and after having had previous extensive chemotherapy which incl cisplatin (840 mg/m²) (Hebl et al. Anesthesia & Analgesia (2001), 92: (1): p. 249).

Pharmacokinetics/Pharmacodynamics
• Reaches site of action by diffusion
• High concentrations in kidneys, liver, prostate, intestines, testes; low CNS penetration
• T½ 20–30 min following bolus administration or infusion of 50 or 100 mg/m²; clearance is 15–16 L/hr/m²; vol of distribution, 11–12 L/m²
• Highly protein-bound, poorly dialyzable
• Cleared renally at rate greater than that of Cr; 13–17% of parent compound excreted within 1 hr after administration

Drug Class/Mechanism of Action/Usual Dose
• Inorganic platinum-containing compound (cis-diamminedichloroplatinum [cis-DDP])
• Disrupts DNA helix, preventing duplication
• In chemotherapy of metastatic ovarian and testicular CA and advanced bladder CA, often used in combination with other drugs, particularly cyclophosphamide (Cytoxan)

• Contraindicated (relatively) in pts with pre-existing renal disease, hearing loss, myelosuppression; use of other nephrotoxic or ototoxic agents (e.g., aminoglycosides) may increase toxicity
• Must be administered IV (See the current oncology literature for dosage, administration guidelines, and protocols. Has been used in doses of 20 mg/m² for 5 d for testicular cancer, 75–100 mg/m² once every 4 wk for ovarian tumors in combination with other agents, 50–70 mg/m² for advanced bladder CA.)
• Pretreatment hydration of 1–2 L over 12 h before administration and infusion of cis-DDP in a dilute vol with mannitol recommended. Repeat courses usually not given until renal function returns to baseline, circulating blood elements are at acceptable levels, and audiometric and hepatic function monitoring have been completed.

Drug Interactions
• Plasma levels of anticonvulsants may become subtherapeutic with the use of cisplatin.
• Clearance of renally excreted drugs in proportion to previous damage
• Avoid aminoglycosides (increased toxicity)
• Should not be administered through needles or IV sets containing aluminum, which reacts with cisplatin, causing precipitation
• cis-DDP and equipment used for administration should be handled as potentially carcinogenic
• May be irritating to the skin; if extravasated may cause local soft tissue toxicity
Uses

- Indicated for reduction of atherosclerotic events in pts with known atherosclerosis confirmed by recent stroke, TIA, MI, or established arterial disease; after PCI with or without stent placement

Perioperative Risks

- Increased risk of bleeding if not D/C 5 d before surgery
- Risk of coronary events increase post stent placement if full course of post procedure antiplatelet therapy is not completed. Risk in drug-eluting stent (DES) may persist for years if clopidogrel D/C

Worry About

- Hypersensitivity reactions (rare): Bronchospasms, angioedema, and anaphylactoid reactions
- Increased bleeding intra- and postop
- Elective surgery undertaken <4 to 6 wk from bare metal coronary stent (BMS) placement demonstrated a high occurrence of stent thrombosis. Elective surgery <365 d after DES demonstrated high rate of stent thrombosis. It is recommended to postpone elective noncardiac surgery until the course of antiplatelet treatment is completed, thus reducing the risk of bleeding complications and stent thrombosis. 5-7 d delay recommended after D/C.
- Black Box Warning: Variant alleles for the cytochrome P450 enzymes, specifically CYP2C19, have been shown to decrease the conversion of clopidogrel to its active metabolite. Poor metabolizers (mainly Caucasians/Asians) may not receive full drug benefit. Pharmacogenomic testing is available.

Overview/Pharmacology

- Inhibitor of plt aggregation. Active metabolite irreversibly modifies the plt ADP receptor, affecting the plt for its entire lifespan after exposure.
- Plt inhibition can be seen within 2 hr of loading dose (300–600 mg) of clopidogrel bisulfate with steady-state of inhibition reached between d 3 and 7 (maintenance dose 75–150 mg)
- Bleeding time and plt aggregation returned to baseline in 5 d after D/C.
- Metabolized: Liver
- Excretion: urine, feces, and breast milk

Drug Class/Usual Dose

- Antiplatelet
- Normal metabolizers: 75 mg PO daily (no effect whether taken with meals or not)
- Slow metabolizers: 150 mg PO daily

Contraindications

- Hypersensitivity to the drug or components of it
- Active pathologic bleeding such as intracranial bleeding or peptic ulcers
- CYP2C19 poor metabolizers
- PPIs diminish the antiplatelet effect of clopidogrel's active metabolite

Drug Interactions/Perioperative Implications

- Markedly ↑ risk of surgical bleeding if D/C <5 d. Decision to continue or D/C drug depends on risk of surgical bleeding versus risk of coronary thrombosis. Discussion between cardiologist/surgeon/anesthesiologist critical.
- Avoid use with CYP2C19 inhibitors (e.g., Prilosec)
- Use with NSAIDs increases GI bleeding risk
- Concomitant use with warfarin increases bleeding risk

Rhabdomyolysis, ARF, ESRD
Thrombocytopenia, enhanced plt
Oliguria, anuria
Exposure

Cocaine

Overview/Pharmacology
- Cocaine is an ester local anesthetic and sodium-channel blocking drug, classified as a Class I antiarrhythmic agent
- Blocks presynaptic reuptake of norepinephrine, dopamine, and serotonin, resulting in activation of SNS
- May produce negative inotropic, chronotropic effects on heart muscle
- Impairs reuptake in brain of dopamine, serotonin, tryptophan
- Accumulation of dopamine in synaptic cleft may lead to acute euphoria, increased alertness
- Cocaine 4% topical solution is FDA-approved as a local anesthetic used on mucous membranes. Cocaine is useful for ENT surgery and for an awake fiberoptic intubation (not to exceed 3 mg/kg; 1 mg/kg is recommended)

ICD-9-CM Codes: 305.6 (Nondependent); 364.2 (Dependent)

Etiology
- Cocaine abuse
- OD during ENT surgery; ER use (part of tetracaine, epinephrine, cocaine mix)

Usual Treatment
- Supportive
- Myocardial ischemia induced by cocaine should be treated initially with O2, sublingual aspirin, and benzodiazepines. If there is ongoing ischemia, use of nitroglycerine, verapamil, or phentolamine to reverse cocaine-induced coronary vasoconstriction may be necessary.
- β-Blockers may worsen coronary vasoconstriction and should be used with great caution if pt presents with signs of ischemia or acute cocaine toxicity.
- In management of short-lived arrhythmias, drug treatment should be avoided if possible, as antiarrhythmic agents and cocaine may have a synergistic depression of contractile function.
- For sustained hemodynamically tolerated SVT assoc with AV nodal re-entry, adenosine is safe and free of major side effects. If adenosine is unsuccessful, administration of an α-antagonist and β-blocker in combination is likely to be both safe and effective. No reliable information on the safety and efficacy of other antiarrhythmic drugs.
- Supraventricular or ventricular tachyarrhythmias assoc with hemodynamic compromise require urgent DC cardioversion.

### Drug Effects

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hematopoiesis, enhanced plt aggregation promoting thrombus formation</td>
<td>Bleeding problems, vasoconstriction</td>
<td>Plt</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Pneumomediastinum, bronchoconstriction, pulm edema</td>
<td>Exposure</td>
<td>Wheezing</td>
<td>CXR</td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenia, enhanced plt aggregation promoting thrombus formation</td>
<td>Headache, N/V</td>
<td>Neuro exam</td>
<td>CT scan</td>
</tr>
<tr>
<td>OB</td>
<td>Preterm labor, premature rupture of membranes, abruptio placenta, spontaneous abortion, meconium-stained amniotic fluid</td>
<td>Exposure</td>
<td>Oliguria, anuria</td>
<td>K+, Cr, CK, urine myoglobin</td>
</tr>
<tr>
<td>GU</td>
<td>Rhabdomyolysis, ARF, ESRD</td>
<td>Exposure</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


### Preoperative Implications

Preoperative Concerns
- Self-reporting of drug abuse unreliable, 35–55% deny cocaine use but have at least 1 positive urine assay
- Hx of smoking, alcohol use, positive syphilis serology, and use of other illicit drugs should alert to possibility of cocaine abuse.
- Difficult IV access due to sclerosis of peripheral veins
- Consider urine screen (reliable for only 14–60 hr after use).

Monitoring
- Routine
- Consider arterial line if Hx of acute intoxication, recent exposure

Airway
- Intranasal cocaine use may cause perforation of nasal septum, oropharyngeal ulcers, or chronic sinusitis
- Temp rise, sympathomimetic effects assoc with cocaine can mimic malignant hyperthermia

Exubation
- No special issues

Adjutants
- Ester local anesthetics and succinylcholine, which undergo metabolism by plasma ChE, may compete with cocaine, resulting in decreased metabolism of both
- Cocaine decreases seizure threshold, enhances convulsant effect of other local anesthetics.

Postoperative Period
- Myocardial ischemia
- Pain medication requirements in chronic abusers are same as for nonabusers.

### Adverse Effects

- Perioperative Risks
- Hemodynamic instability, sympathetic discharge
- Myocardial ischemia
  - Increased myocardial O2 demand (↑ HR, ↑ BP, ↑ LV contractility)
  - Decreased myocardial O2 supply (↓ endotherm, ↓ NO resulting in coronary vasoconstriction)

- Worry About
  - CV: Htn, tachycardia, dysrhythmias, MI, cardiomyopathy, premature coronary atherosclerosis, sudden cardiac death
  - Neurologic: Intracerebral bleed, seizures
  - Pulmonary: Pneumomediastinum, cocaine-induced asthma, hypersensitivity pneumonitis, chronic cough, pulm edema, diffusing capacity alv-veins
  - OB: Placenta previa, abruptio placentae, premature labor, fetal distress

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Cromolyn Sodium

**Uses**
- Approved by the FDA in 1973 as the first prophylactic nonsteroidal drug available for treatment of chronic asthma
- Alternative initial maintenance therapy for mild persistent and moderate persistent asthma
- Preventative only. Not effective during acute episodes of bronchospasm
- Most beneficial for allergic-component and exercise-induced asthma
- May be beneficial in allergic rhinitis and atopic ocular diseases
- Oral formulations for the management of mastocytosis, ulcerative colitis, and food allergies

**Overview/Pharmacology**
- Inhibits antigen-induced degranulation of pulm mast cells, eosinophils, neutrophils, monocytes, and lymphocytes
- Prevents release of histamine, leukotrienes, and other autacoids
- Reverses and suppresses leukocyte activation
- Inhibits cough reflex
- Does not directly relax bronchial smooth muscle
- No apparent steroid-sparing effect; inferior to inhaled corticosteroids on measures of lung function and morbidity in 2006 Cochrane Review.
- Administered by inhalation route to treat asthma
- 8–10% of inhaled dose reaches lung parenchyma and is readily absorbed
- $T_{1/2} = 80–90$ min; peak plasma concentration within 15 min
- Active drug excreted unchanged in urine (50%) and bile (50%)
- Can be taken prophylactically 15–20 min before exercise or exposure to known allergen to prevent bronchospasm

**Drug Class/Mechanism of Action**
- Cromolyn sodium (disodium cromoglycate) is a derivative of 2-chromone-carboxylic acid
- Direct mechanism of action in asthma is poorly defined
- One proposed explanation is ↓ in accumulation of intracellular $Ca^{2+}$ in sensitized mast cells
- Another possible mechanism is Cl− channel blockade in antigen-sensitized pulm C-fibers
- Effective in preventing degranulation of mast cells only if given prior to antigenic challenge

**Usual Dose**
- Cromolyn sodium for inhalation (Intal) via special nebulizer (20 mg/2 ml) or metered spray (2 puffs [1 mg/puff] 3–4 times daily for asthma)
- 4% liquid nasal spray (Nasalcrom) given as 1 spray to each nostril 3–6 times daily for allergic rhinitis
- 4% ophthalmic solution (Opticrom) given as 1–2 drops to each eye 4–6 times daily for atopic eye conditions

**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>System</th>
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</thead>
<tbody>
<tr>
<td>RESP</td>
<td>Inhibition of pulm mast cell degranulation; ↓ release of histamine and leukotrienes; reverse or suppress leukocyte activation</td>
<td>↓ Episodes of exercise- or antigen-induced bronchospasm after chronic use</td>
<td>↓ Bronchial hyperactivity as measured by histamine or methacholine challenge</td>
</tr>
</tbody>
</table>

**Perioperative Implications/Possible Drug Interactions**
- Continue administration periop. Do not D/C abruptly.
- Cromolyn sodium is of no benefit in treating an acute exacerbation of asthma.
- Adverse effects are infrequent
  - Unpleasant taste (most common)
  - Direct irritation (e.g., wheezing, coughing)
  - Dizziness, nausea, rash
  - Urticaria, anaphylaxis (extremely rare)
- No significant drug-drug interactions with cromolyn sodium are known.
- Compatible in nebulized solution with albuterol, levalbuterol, ipratropium, and budesonide
- Pregnancy Category B. No known evidence of teratogenicity.

Dexmedetomidine (Precedex)

Ori Gottlieb

**Uses**
- Alpha-2-adrenergic agonist
- Sedation of intubated pts in an intensive care setting
- Sedation of non-intubated pts prior to and/or during surgical and other procedures
- Anxiolytic for use during awake fiberoptic intubations and cases where monitored anesthesia care is appropriate
- May continue infusion during extubation as does not cause resp depression
- Useful in awake craniotomy which requires intact cooperative mental status
- Pediatric sedation in the NICU/PICU and for specific procedures (e.g., MRI)

**Perioperative Risks**
- Initially ↑ BP and ↓ HR (esp. when bolus used)

**Worry About**
- CV effects if bolus used (↑ BP and ↓ HR)
- Recall: No amnestic component to its sedation
- Consider reducing dose in hepatic failure. Titrate to effect.
- Dosing in renal failure unchanged as no known active metabolites
- Education is vital as the pt will likely be more aware and/or cooperative than with other sedatives. This is esp. important in the ICU where nurses and/or physicians are used to a less cooperative mental status from sedated pts.

**Overview/Pharmacology**
- Imidazoline derivative with direct effect at the presynaptic alpha-2 receptors induces a drop in cAMP production by inhibitory G proteins.

**Drug Class/Mechanism of Action/Usual Dose**
- Imidazoline derive; highly selective alpha-2 agonist
- Alpha-2 receptors in brainstem (locus ceruleus) induce less alertness or sedation
- ICU sedation: 0.2–0.7 mcg/kg/hr
- Awake FOI/MAC sedation: 0.2–1 mcg/kg/hr
- Bolus in PI labeled at 1 mcg/kg over 10 min. Beware CV effects.
- Allow 15–20 min of infusion for effect if dosing without bolus.

**Perioperative Implications**

**Preoperative Concerns**
- May be used as an anxiolytic, esp. where resp depression is detrimental

**Induction/Maintenance**
- Bolus achieves faster sedation at the risk of ↑ BP and ↓ HR

- After the delay assoc with the alpha-2 negative feedback loop, BP may drop due to a decrease in plasma norepinephrine levels

**Drug Effects**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Initial ↑ BP followed by ↓ BP Bradycardia</td>
<td>None</td>
<td>SpO₂/ETCO₂</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>None</td>
<td>None</td>
<td>Anxiolysis</td>
<td>VAS</td>
</tr>
<tr>
<td>CNS</td>
<td>Cooperative sedation Reduces MAC requirements of inhaled agents Mental status/responsiveness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>Antisialagogue Analgesic adjunct Xerostomia Patient feedback</td>
<td>Dry mouth</td>
<td>VAS</td>
<td></td>
</tr>
</tbody>
</table>


**Postoperative Period**
- Useful in the postop period to reduce narcotic requirements in those at risk for pulm complications (e.g., morbidly obese, obstructive sleep apnea)
- Unlike with clonidine, rapid withdrawal is not problematic.
**Digitalis (Digoxin)**

**Uses**
- A glycoside extracted from leaves of the foxglove (digitalis lanata), available in oral and IV preparations
- Treatment of CHF, atrial fibrillation and flutter
- Avoid in pts with ventricular extrasystole or VT, as it may precipitate VF due to increased cardiac excitability
- Prevention of supraventricular arrhythmias following thoracotomy
- Narrow therapeutic range —2 mcg/L
- Cardiac side effects: Arrhythmias and conduc- tion disturbances

**DOSING/PHARMACOKINETICS**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Onset</th>
<th>Peak</th>
<th>( T_{1/2} )</th>
<th>Dose</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digoxin: IV</td>
<td>5–30 min</td>
<td>1–3 hr</td>
<td>34 hr</td>
<td>0.5–1.0 mg</td>
<td>0.25 mg/d</td>
</tr>
<tr>
<td>PO</td>
<td>1–3 hr</td>
<td>4–6 hr</td>
<td>34 hr</td>
<td>0.75–1.2 mg</td>
<td>0.125–0.5 mg/d</td>
</tr>
<tr>
<td>Digitoxin: PO</td>
<td>3–6 hr</td>
<td>6–12 hr</td>
<td>7 d</td>
<td>0.8–1.2 mg</td>
<td>0.05–0.3 mg/d</td>
</tr>
</tbody>
</table>

**Excretion**
- Digoxin: Renal, mostly unchanged; ↓ dose for ↑ Cr
- Digitoxin: Hepatic degradation

**Drug Interactions**
- Diuretics: ↓ serum K⁺, ↑ toxicity
- Plasma levels increased by quinidine, amio-
darone, verapamil, captopril, erythromycin
- Plasma levels decreased by antacids, phenytoin, metoclopramide, and cholestyramine

**Treatment for Toxicity**
- Due to Na⁺/K⁺+ATPase inhibition, hyper-
kalemia may be a feature and should be corrected
- Hypokalemia exacerbates toxicity and should be corrected.
- Severe bradycardia: Atropine or pacing pre-
ferred over catecholamines
- Ventricular arrhythmias: Treat with lidocaine or
phenytoin
- Digoxin specific Fab: Indicated for digoxin lev-
els >10 mcg/L, life-threatening arrhythmias or uncontrolled hyperkalemia

**Drug Class/Mechanism of Action**
- Direct action: Inhibition of Na⁺,K⁺+ATPase, produ-
cing ↑ intracellular Na⁺ ↓ K⁺, resulting in increased intracellular Ca²⁺, leading to positive inotropic effect.
- Decrease K⁺ intracellular: slowing of AV con-
duction and of the pacemaker cell
- Indirect effect: Enhances release of acetylcho-
line at the cardiac muscarinic receptors - This slows conduction and prolongs the refractory period in AV node and bundle of his.

**Perioperative Implications**

**Preoperative Concerns**
- Do not D/C digitalis preop. Withdrawal in heart failure pts may lead to recurrence of failure symptoms
- When changing from oral to IV therapy, dos-
age should be reduced by 20% to 25%.
- Correct and maintain serum K⁺.
- Decreasing dose with increasing serum Cr

**Possible Drug Interactions**
- Decrease AV block with β-adrenergic, amio-
darone and Ca²⁺-channel blocking drugs
- Decrease dose with concurrent quinidine

**Anticipated Problems/Concerns**
- Ventricular rate with AFib and/or AFLT is a rough bioassay for digoxin level; fast ventricular rate with AFib indicates inadequate serum level of digoxin.

**Drug Worry About**
- Hyperventilation can cause alkalosis leading to relative hypokalemia-toxicity.
- Renal insufficiency (decreased digoxin clear-
ance and need for dose alteration, not appreciably removed by dialysis)

**Overview/Pharmacology**
- General pharmacologic effect: Positive inotropic and slowing of ventricular response

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**Key References:**
Diuretics

Indications
- Prescribed for pts with Htn, CHF, elevated ICP, edema, hemoglobinuria, low intraop UO
- Mannitol may function as a renal preservative by free-radical scavenging and toxin dilution
- Fenoldopam is a selective dopamine-1 agonist. As a vasodilator, it lowers BP and augments renal blood flow which improves UO and GFR. It may serve as a renal protectant. Usual dose begins at 0.03 μg/kg/min titrated to effect.

Perioperative Risks
- Hypokalemia
- Hypovolemia
- Low intraop UO
- Hyperkalemia with aldosterone antagonists
- Hypomagnesemia

Worry About
- Hypokalemia, hypovolemia
- Low intraop UO if hold usual diuretic preop
- Hypokalemia provoking and/or aggravating digitalis toxicity
- Deafness with ECA (ethacrynic acid)
- Nephrotoxicity of cephaloridine is enhanced by furosemide
- End result of diuretic use is ↑ UO with net loss of H₂O and solute esp K⁺, Mg²⁺
- Onset of diuresis is within 10 min after IV administration
- With exception of aldosterone antagonist and K⁺-sparking diuretics, all others cause K⁺ loss
- Serum K⁺ <3.5 mEq/L in 15% of pts, <3.0 mEq/L in up to 10% of diuretic-treated pts
- Chronic diuretic-induced hypokalemia is less arrhythmogenic than acute, but serum K⁺ <3.0 mEq/L assoc with 2-fold greater incidence of ventricular arrhythmias than K⁺ >3.0 mEq/L.
- Site-specific action assoc with additional effect if diuretics from 2 classes used

Drug Class/Mechanism of Action/Usual Dose
- Diuretics belong to osmotic, carbonic anhydrase inhibition, benzoazide, high-ceiling (loop), K⁺-sparking, or aldosterone antagonist class of drugs, based on mechanism of action
- Only osmotic and loop diuretics used intraop
- Osmotic diuretic: Mannitol—ascending loop, limits H₂O reabsorption; onset of action 5–15 min after IV dose; renal clearance
  - Usual dose: Mannitol 0.25–2.0 g/kg (give as drip and not bolus to avoid hypotension)
- Loop diuretics—ascending loop, limit NaCl reabsorption; onset of action 5 min after IV dose; T½ 1–2 hr; duration of action 3–6 hr; renal clearance
  - Usual IV dose for 70 kg person: Furosemide: 5–40 mg (0.1–1.0 mg/kg); ECA: 25-50 mg (0.5–1.0 mg/kg); bumetanide: 0.5–1.0 mg q 2–3 h; max 10 mg/d

Perioperative Implications/Possible Drug Interactions

Preoperative Concerns
- In chronic hypertensive pts treated with diuretics, a significant intravascular volume contraction may exist, making them more prone to hypotension following induction of anesthesia and any acute blood loss.
- Hypokalemia: Check serum K⁺, consider enhanced digitalis toxicity
- Hypomagnesemia is common in pts treated with loop or thiazide diuretics and predisposes to ventricular arrhythmias, and should be suspected when hypokalemia is noted
- Enhanced oto- and nephrotoxicity of loop diuretics are assoc with rapid administration of large IV doses and concurrent use of another nephrotoxic drug, e.g., aminoglycoside antibiotic, another loop diuretic, and some cephalosporins, esp., cephaloridine
- Probably best to continue chronic dose through the periop period, incl day of surgery. (UO will decline if diuretic not given on day of surgery.)

Induction/Maintenance
- Intraop loop diuretic use may significantly decrease serum K⁺ level with diuresis

Advantages
- Enhanced renal clearance of other drugs, e.g., NMB agents, provoked by diuresis is not clinically problematic

DRUG EFFECTS

<table>
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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Transient (&lt;24 hr) deafness or vertigo may follow IV rapid bolus ECA; less common after furosemide or bumetanide; rarely permanent</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Transient ↑ in venous capacitance causes hypotension with rapid IV loop diuretic administration; acute transient ↑ in intravascular volume precedes diuresis with mannitol; vasodilation with fenoldopam</td>
</tr>
<tr>
<td>GI</td>
<td>Diarrhea may follow ECA use</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypokalemia, metabolic alkalosis</td>
</tr>
<tr>
<td>GU</td>
<td>Diuresis</td>
</tr>
<tr>
<td>CNS</td>
<td>Mannitol ↓ ICP following transient increase. The latter may be mitigated by coadministration of furosemide</td>
</tr>
</tbody>
</table>


Anticipated Problems/Concerns
- Pts receiving diuretics preop should be considered volume-contracted until proven otherwise.
- Hypokalemia assoc with diuresis will be aggrivated by hyperventilation, which further lowers serum K⁺ by an additional 0.5 mEq/L for each 10 mmHg decrease in PaCO₂.
- Catecholamine β effect (endogenous and/or exogenous); also lowers serum K⁺.
- Low intraop UO in a euvoletic pt if ADH/ stress mediated will, in authors’ experience, respond to very low dose (e.g., 2–5 mg furosemide) with increased UO

Christopher Giordano
Nikolaus Gravenstein
Dobutamine

**Indications**
- Administered to pts with low cardiac output (CO) 2° to decreased right or left ventricular function assoc with HF, MI, or cardiac surgery
- Used for treatment of pulm Htn with right ventricular dysfunction
- Provocative test for Dx of CAD (e.g., dobutamine stress-echocardiography)

**Perioperative Risks**
- Risk of tachyarrhythmias

**Worry About**
- Tachycardia and tachyarrhythmias, worse at high doses
- Ventricular ectopy
- Rarely, hypotension or Htn may be observed
- Hypokalemia may occur

**Overview/Pharmacology**
- Inotrope used intravenously for increasing CO simultaneous with a decrease in systemic and pulm vascular resistance
- Was considered to have relatively greater inotropic than chronotropic actions, but recent investigation does not support this contention
- $\beta_1$-agonist with lesser effect at $\beta_1$-receptors and minimal effects at $\alpha$-receptors
- Increases intracellular Ca$^{2+}$ by elevating cAMP through effects on $\beta_1$-receptors
- Increases SA-node automaticity and AV nodal and intraventricular conduction
- May cause systemic and pulm vasodilation through $\beta_2$-receptor stimulation

**Drug Class/Mechanism of Action/Usual Dose**
- Synthetic catecholamine
- $\beta$-adrenergic action increases adenyl cyclase activity
- Increases CO by increasing SV and HR and decreasing SVR
- Usual dosage: 1–10 $\mu$g/kg/min IV
- Combined use with other agents to increase cardiac output via different mechanisms (e.g., milrinone, sodium nitroprusside)
- Concurrent use of dobutamine with epinephrine may reduce efficacy of epinephrine

**Adjuvant/Regional Anesthesia/Reversal**
- Combining therapy with inotropes that are not $\beta_1$-agonists such as milrinone may provide greater than additive effects
- Improvement in cardiac output may also be achieved by adding sodium nitroprusside if SVR is high
- Excessive effect can be reversed with $\beta$-adrenergic antagonists such as esmolol
- Consider using digoxin prior to dobutamine in pts with AFIB and rapid ventricular response
- May be ineffective or larger doses required in pts receiving $\beta$-blockers

**Anticipated Problems/Concerns**
- Sinus tachycardia can occur, and in pts with AFib, the ventricular rate may increase 2° to enhanced AV conduction.
- Pulm V/Q mismatch 2° to pulm vasodilation and loss of hypoxic pulm vasoconstriction may lead to a decrease in $P_{aO_2}$
- Initiation or exacerbation of ventricular arrhythmias if myocardial ischemia present
- Contraindicated in IHSS
- Prolonged use assoc with $\beta$-receptor downregulation and theoretically reduced effectiveness

**Drug Effects**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>↑ HR</td>
<td>Relief of dyspnea</td>
<td>Capillary perfusion, JVD, UO, raies</td>
<td>Mixed venous $O_2$ sat, CO, PVR, SVR data</td>
</tr>
</tbody>
</table>

**Preoperative Concerns**
- Assess systemic perfusion
- Monitor BP, CO, PCWP
- PA catheter essential for adequate drug titration

**Induction/Maintenance**
- Despite adequate CO and BP before induction, there may be a decrease in these values during induction of anesthesia.
- Therapy should be guided by measures of adequacy of systemic perfusion such as CO, mixed venous $O_2$ sat, and ABG tensions.

**Postoperative Period**
- Duration of treatment determined by assessment of cardiac function with PA catheter

Dopamine

**Indications**
- A flexible molecule that fits into many receptors to cause direct β1- and β2-receptor stimulation, as well as some α-stimulation, and fits into dopaminergic receptors as well.
- Hypotension
- Cardiac failure/shock with low-to-normal SVR
- Oliguria or periods of renal stress such as vascular surgery, sepsis, cardiopulmonary bypass, and concurrent use of other vasopressors. The concept of therapeutic renal dose is now outdated.
- Bradycardia

**Perioperative Risks**
- Tachycardia, angina, arrhythmias
- N/V
- Vasodeconstriction and Htn (possible gangrene of extremities)
- Skin sloughing and necrosis if infiltrated in SQ tissue
- Depression T-lymphocyte function (hypoproteinemia)
- Oliguria or periods of renal stress such as vascular surgery, sepsis, cardiopulmonary bypass, and concurrent use of other vasopressors. The concept of therapeutic renal dose is now outdated.

**Pharmacology**
- Preparations: 200-, 400-, 800-mg ampules (must be diluted before IV administration). It must not be diluted in alkaline solutions.
- Endogenous central and peripheral neurotransmitter

**Overview/Mechanism of Action**
- Mixed indirect and direct sympathomimetic effects, by activating dopamine (DA1 and DA2). β1- and α1-adrenergic receptors in dose-dependent fashion
- Presynaptic DA1 receptors (0.2–0.4 µg/kg/min) inhibit endogenous norepinephrine and prolactin release.
- Postsynaptic DA2 receptors (0.5–3.0 µg/kg/min) produce vasodilation in renal, mesenteric, coronary, cerebral arteries.
- β1-Adrenergic receptors (0.4–10 µg/kg/min) activates adenylyl cyclase and ↑ myocardial cAMP concentration, ↑ myocardial contractility, inotropy.
- α1-Adrenergic receptors (>10–20 µg/kg/min) produce progressive vasocostriction
- Metabolism: Substrate for both MAO and COMT

**DRUG EFFECTS**

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>↑ Cardiac inotropy; vasoconstrictor activity</td>
<td>Improved mental status, perfusion</td>
<td>Pulses, BP Capillary refill</td>
<td>↑ Cardiac output; UO</td>
</tr>
<tr>
<td>RESP</td>
<td>↓ Hypoxic drive</td>
<td>Hypoventilation</td>
<td>Resp rate, depth</td>
<td>ABGs</td>
</tr>
<tr>
<td>RENAL</td>
<td>↑ Renal blood flow; ↓ renal Na+, water reabsorption</td>
<td>UO</td>
<td>Urine volume</td>
<td>Urine volume, electrolytes Cr, CrCl</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Dopamine Infusion Issues**
- Ensure adequate intravascular volume
- Consider invasive monitoring
- Continuous arterial and PAP catheters; central venous and pulm artery occlusion pressure (“filling pressures”); thermodilution cardiac output
- UO and Cr clearance

**Adjuvants**
- Additional inotropes—other β-agonists (dobutamine, epinephrine, etc.) or phosphodiesterase inhibitors (milrinone or amrinone) may be needed to ↑ cardiac contractility.

**Cautions**
- Watch for arrhythmias
- May increase heart rate and LV wall stress excessively (dopamine >10 µg/kg/min frequently causes progressive tachycardia and ↑ diastolic ventricular filling pressure)

- Diminished response in pts with chronic CHF or active sepsis
- In cardiogenic shock, myocardial lactate may ↑
- Long-term infusions may suppress immune (T-cell) function with ↓ prolactin
- May exacerbate glaucoma
- If the pt has recently taken a monoamine oxidase inhibitor, the rate of dopamine
- Metabolism by the tissue will fail and the dose should be cut to one-tenth the usual

**Related Agent: Dopexamine**
- Dopexamine, a synthetic analogue of dopamine, lacks any direct α1-adrenergic agonist activity, expressing only β1-adrenergic and dopaminergic (DA2) agonist action
- DA, and β2, arterial vasosilation reduces cardiac afterload while simultaneously increasing blood flow to the kidneys, intestines, liver, spleen
- Dopexamine (doses between 1 and 4 µg/kg/min) significantly ↑ cardiac index while decreasing systemic and pulm vascular resistances after cardiac surgery

- HR ↑, but not SV index thus, dopexamine combines positive inotropic, chronotropic, vasodilatory, diuretic, natriuretic properties
- Fenoldopam mesylate (Corlopam) is a pure DA1-receptor agonist inducing selective coronary, renal, mesenteric, and peripheral arterial vasodilation. It causes a linear, dose-dependent reduction in systolic and diastolic BP (pharmacokinetic T½ = 5 min); produces potent renal vasodilation and natriuretic actions (similar to dopamine), and ↑ UO even in the setting of ↓ BP. May replace renal dose dopamine. No known drug interactions with β-blockers, α-blockers, Ca2+-channel blockers, or ACE inhibitors. Has the disadvantage of causing reflex tachycardia. Can cause asymptomatic T wave flattening on the ECG.
- Infusions have proven ineffective in prevention of contrast-induced nephropathy. Use with caution in pts with glaucoma. Contains sulfites.
Doxorubicin (Adriamycin)
Daunorubicin (Cerubidine)

**Toxicity**
- Two phases of toxicity, acute and chronic
  - Acute toxicity: Cardiac (may be from direct effects of histamine)
    - ECG changes and conduction disturbances: ↓ QRS voltage; nonspecific ST changes; T wave flattening
      - Rhythm disturbances: Supraventricular tachyarrhythmias; PVCs
        - Decreased EF
      - N/V, alopecia, diarrhea, mucositis
      - Bone marrow suppression (may limit dose acutely; counts lowest about 2 wk after beginning therapy
      - Infiltrated drug with IV delivery may cause extensive tissue necrosis, requiring wide debridement
  - Late toxicity: Cardiac
    - Most acute toxic cardiac effects (except ↓ QRS voltage) diminish with time
    - Late toxicity occurs weeks to months after administration; reports of onset up to 5 y after dosing
    - Effects are permanent (some indication that children may recover with time)
      - CHF unresponsive to inotropic drugs
      - Increased risk of CHF with higher doses but heart failure can occur after 1st dose
        - Risk 0.1–7% up to 550 mg/m²
        - Risk rises sharply after 550 mg/m² to 50% at 1000 mg/m²
        - Risk increased by radiation of LV, other cardiotoxic drugs, prior LV dysfunction
        - Risk greater in young children
        - Risk of CHF ↓ by divided dosing (e.g., weekly)
        - Myocardial function evaluation requires measurement of EF by ECHO or MUGA (serial CXR, ECGs, systolic time intervals, and other clinical signs not reliable)
        - Myocardial biopsy but not myocardial function shows characteristic changes

**Perioperative Risks**
- Acute: Anemia, thrombocytopenia, cardiac arrhythmias, and conduction disturbances
- Late: Cardiac contractile dysfunction (variable; may be severe)

**DRUG EFFECTS**

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<tr>
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<th>Test</th>
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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Conduction</td>
<td>Exercise tolerance</td>
<td>Unreliable CHF signs; orthopnea, DOE, etc.</td>
<td>ECG, MUGA</td>
</tr>
<tr>
<td></td>
<td>Contractile force</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Mucositis, diarrhea</td>
<td>Volume indicators</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Marrow suppression</td>
<td>Bleeding</td>
<td>Unreliable</td>
<td>CBC with plt count</td>
</tr>
</tbody>
</table>


**Related Drugs**
- Epirubicin (Ellence) is a newer derivative of doxorubicin, with similar anesthetic-related features. It may be found as part of breast cancer chemotherapy. It may be slightly less cardiotoxic on a milligram basis.
- Idarubicin (Idamycin, Idamycin PFS) is a newer derivative of daunorubicin, with similar anesthetic-related features. It may be found as part of acute myelogenous leukemia chemotherapy. An oral form of idarubicin has been tested as part of long-term chemotherapy regimens.

**Anticipated Problems/Concerns**
- LV dysfunction periop with pulm edema
- Risk of infection in acute toxicity
Ephedrine

**Uses**
- Treatment of hypotension resulting from sympathetic blockade, vasodilatory and/or cardiopressive effects of IV and/or inhalational anesthesia agents
- Acute hypotension due to undetermined shock, complete heart block (Stokes-Adams attack)
- As a pressor following overdose with ganglionic-blocking and antiadrenergic agents
- Orally, is used for allergic disorders, asthma, nasopharyngeal congestion, coryza, hay fever
- Topically, for treatment of nasal congestion, coryza, vasomotor rhinitis, potential sinusitis

**Perioperative Risks**
- May trigger arrhythmias, incl life threatening ventricular arrhythmias, if myocardium more sensitive to catecholamines (e.g., due to inhalational agents, particularly halothane and enflurane)
- May cause acute Htn resulting in intracranial hemorrhage and CV collapse
- Hypertensive crisis may occur if used in pts taking MAOIs and TCA antidepressants

**Possible Drug Interactions**
- Increased response in pts receiving β-blockers
- Ephedra preparations (e.g., ma-huang, mormon tea), guarana and caffeine may lead to additive effects
- Additive effects and risk of toxicity is assoc with urinary alkalinizers

**Induction/Maintenance**
- As in Perioperative Concerns

**Anticipated Problems/Concerns**
- Myocardium sensitization to catecholamine by some inhalational agents (e.g., halothane)
- Tachyphylaxis with repeated doses
- Possibility of interactions with other drugs that affect ANS, incl herbas and cocaine
- Precipitates ischemia in susceptible pts
- Maternal tachycardia/other arrhythmias, potential fetal acidosis

**Possible Drug Interactions**

<table>
<thead>
<tr>
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<th>Test</th>
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</thead>
<tbody>
<tr>
<td>RECEPTORS</td>
<td>α₁: +</td>
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<tr>
<td></td>
<td>β₁: +</td>
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<tr>
<td></td>
<td>β₂: +</td>
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<tr>
<td>CARDO</td>
<td>HR: +</td>
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<td></td>
<td>Contractility: +</td>
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<td></td>
<td>Automaticity: +</td>
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<td></td>
<td>Peripheral resistance: +</td>
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<td></td>
<td>CO₂: +</td>
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<td>Mean BP: +</td>
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<td>PAP: +</td>
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<tr>
<td>RESP</td>
<td>Airway resistance: −</td>
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<td></td>
<td>Resp stimulant: +</td>
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<tr>
<td>VASC. BED FLOW</td>
<td>Skin/viscera: −</td>
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<td></td>
<td>Muscle: +</td>
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<td>Kidney: −</td>
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<td></td>
<td>Coronary: +</td>
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<tr>
<td></td>
<td>Cerebral: +</td>
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<tr>
<td>ENDO</td>
<td>O₂ consumption: +</td>
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<td></td>
<td>Blood glucose: +</td>
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<td></td>
<td>Blood lactic acid: NC</td>
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<tr>
<td>GU</td>
<td>Uterine relaxation; restores uterine BF in hypotension from epidural/spinal</td>
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<tr>
<td>CNS</td>
<td>Mild stimulant</td>
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<tr>
<td></td>
<td>Mild mydriasis</td>
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</tbody>
</table>

Epinephrine

**Uses**
- Intraop: Systolic dysfunction—weaning from CPB, in critical care for CV collapse from many causes: cardiogenic (incl RV failure), distributive (incl anaphylaxis)—stabilizes mast cells, obstructive shock
- Addition to local anesthesia to prolong action (1:200,000) and for hemostasis (field block)
- Nebulized racemic epinephrine for airway edema: Postop stridor or laryngotracheobronchitis in children, not usually for anionotropic edema
- Inhalational forms for mild asthma
- Topical solutions for vasoconstriction (nasal, ophthalmic)
- Large repeated doses in cardiac arrest

**Perioperative Risks**
- Increased risk of dysrhythmias (limit to 1 µg/kg with halothane, 2–3 µg/kg with isoflurane, enfurane)
- May precipitate myocardial ischemia
- Severe Htn/stroke if dosed incorrectly
- Large doses may precipitate pulm edema
- Avoid topical application where reduced perfusion could lead to ischemic tissue damage

**Overview/Pharmacology**
- Potent β₁, β₂, α₁ and α₂ stimulant. More potent at both β₁ and β₂-receptors than norepinephrine.
- Water soluble—no blood-brain barrier penetration
- ↑ Pulse pressure at low doses because of β₂ effect
- β Stimulation causes increased intracellular cAMP
- α Stimulation causes increased intracellular Ca²⁺ by G protein interaction as well as increased turnover of phosphoinositol
- α Stimulates inhibits adenylyl cyclase
- Metabolized by MAO, COMT; conjugated and excreted in urine
- Biologic activity terminated principally by uptake in postganglionic sympathetic nerve terminals

**Drug Class/Usual Dose**
- Naturally occurring sympathomimetic
- Dosage: Depends on route and clinical situation—low, moderate, high doses
- IV: Mix 1 mg in 250 mL (4 µg/mL); adult bolus doses for ↓ BP from anaphylaxis: 10–20 µg as starting dose, ↑ as needed
- Infusion, mainly β at 0.01–0.03 µg/kg/min, increasing α at 0.03–0.15 µg/kg/min, predominant α at 0.15–0.3 µg/kg/min. Cardiac arrest dose: 0.5–1 mg q5 min.
- Subcutaneous: 10 µg/kg for mild to moderate allergic reactions, severe asthma

**Drug Effects**

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<tbody>
<tr>
<td><strong>Receptors</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>α: + + (dose-dependent)</td>
<td></td>
<td>Palpitations</td>
<td></td>
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<tr>
<td>β₁: + +</td>
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<tr>
<td>β₂: + +</td>
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<tr>
<td><strong>Cardio</strong></td>
<td>HR: + +</td>
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<tr>
<td>Contractility: + +</td>
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<tr>
<td>Automaticity: + +</td>
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<tr>
<td>SVR + (dose-dependent)</td>
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<tr>
<td>CO: + +</td>
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<tr>
<td>Mean BP: + (dose-dependent)</td>
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<tr>
<td>PAP: +</td>
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<tr>
<td><strong>Resp</strong></td>
<td>Airway resistance: –</td>
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<tr>
<td>Resp stimulant: +</td>
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<tr>
<td><strong>Vasc bed flow</strong></td>
<td>Skin/viscera: – –</td>
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<tr>
<td>Kidney: – –</td>
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<tr>
<td>Coronary: +</td>
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<tr>
<td>Cerebral: +</td>
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<tr>
<td><strong>Endo</strong></td>
<td>O₂ consumption: + +</td>
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<tr>
<td>Blood glucose: + +</td>
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<tr>
<td>Blood lactic acid: + + (with infusion)</td>
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<tr>
<td>Hypokalemia</td>
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<tr>
<td>Serum FFA: + +</td>
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<tr>
<td><strong>Gu</strong></td>
<td>Relaxation of uterus</td>
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<tr>
<td><strong>Cns</strong></td>
<td>Mild stimulant</td>
<td>Anxiety, agitation,</td>
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<tr>
<td>Mild mydriasis</td>
<td></td>
<td>headache</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Mydriasis, arousal</td>
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</tbody>
</table>

* Minimal increase; + + moderate increase; + + + marked increase; – minimal decrease; – – moderate decrease

Epsilon-Aminocaproic Acid (EACA) (Amicar)

Uses
- EACA is a hemostatic agent used in the treatment of excessive bleeding assoc hyperfibrinolysis
- Indications: Hemorrhage caused by systemic hyperfibrinolysis*, surgical or non-surgical hematuria*, surgical bleeding assoc with CPB, hemorrhagic reaction to fibrinolytic drugs, bleeding in pts with hereditary blood coagulation disorders, treatment and prophylaxis in hemophiliacs undergoing dental surgical procedures, 2nd prophylaxis against 2nd hemorrhage following intraocular bleeding, recurrence of subarachnoid hemorrhage, hemorrhage in pts with thrombocytopenia
- Methods of administration: Oral solution, IV solution, topical, intravesical

*FDA approved

Perioperative Risks
- Increased risk of developing thrombosis in hemophilic pts, who are concurrently treated with Factor IX concentrate or anti-inhibitor coagulant complex

Worry About
- EACA should not be used when there is evidence of an active intravascular clotting process. When there is uncertainty as to whether the cause of bleeding is primary fibrinolysis or disseminated intravascular coagulation (DIC), this distinction almost certainly must be made before administering EACA. EACA has too great a hazard to benefit ratio to be used in the presence of DIC without concomitant heparin.

Overview and Pharmacology
- EACA prevents formation of excessive plasmin, thereby inhibiting fibrinolysis
- EACA enhances hemostasis when fibrinolysis contributes to bleeding

Drug Class/Mechanism of Action/Usual Dose
- EACA is an antifibrinolytic agent of the lysine analogue class.
- EACA binds competitively to lysine-binding sites within the plasminogen/plasmin molecule, which interferes with the ability of plasmin to lyse fibrin clots.
- The optimal dosage in the setting of CPB is undefined, but the following is a commonly used regimen in adults: Initial loading dose is 5 g IV over 1 hr, followed by a continuous infusion at 1 g/hr, maximum recommended daily dosage is 30 g
- Plasma concentrations are increased in pts with severe renal dysfunction, but no quantitative recommendations for dosing adjustments are available

Perioperative Implications/Possible Drug Interactions
Preoperative Concerns
- In the presence of hematuria originating in the upper urinary tract, EACA can cause intrarenal obstruction due to clot retention

Drug Interaction
- EACA is contraindicated in hemophilic pts who are treated with Factor IX concentrates or anti-inhibitor coagulant complex, unless the risk of thrombosis is outweighed by the potential benefit of EACA

Induction/Maintenance
- Close hemodynamic monitoring of cardiac pts because of the risk of hypotension and sinus bradycardia, particularly with rapid IV administration and in hypovolemia
- Monitor renal function in pts with renal dysfunction and consider dosage adjustments depending on clinical response and degree of renal function impairment.
- Consider transfusion of pts, FFP, and cryoprecipitate in the presence of bleeding not caused by hyperfibrinolysis

Postoperative Period
- Continue assessment of bleeding and monitoring of coagulation profiles after D/C of EACA therapy

Anticipated Problems/Concerns
- EACA should not be administered without a definite diagnosis and/or laboratory finding indicative of hyperfibrinolysis (hyperplasminemia) because of the potential for thrombotic complications in pts with DIC and underlying hypercoagulable states

DRUG EFFECTS

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Dizziness, confusion, delirium, headache, seizure</td>
<td>Neuro exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardio</td>
<td>Hypotension, bradycardia</td>
<td>Vital signs</td>
<td>ECG</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>N/V/D</td>
<td>Oliguria</td>
<td>BUN/Crea</td>
<td></td>
</tr>
<tr>
<td>Renal</td>
<td>Renal failure, urinary tract obstruction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heme</td>
<td>Thrombosis</td>
<td>Potential causes for DIC</td>
<td>Evidence for paradox of simultaneous thrombosis and bleeding</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Myopathy, rhabdomyolysis</td>
<td>Myalgia, malaise, fatigue</td>
<td>Muscle weakness</td>
<td>CPK</td>
</tr>
</tbody>
</table>

Fluoxetine (Prozac)

**Uses**
- Incidence in USA: Taken by approx 5 million
- Rx for depression, obsessive-compulsive disorder, bulimia nervosa

**Perioperative Risks**
- May be assoc with periop anxiety
- Drug interactions with β-blockers, phenytoin, benzodiazepines, antipsychotics (may increase levels by inhibition of metabolism)

**Worry About**
- Suicidal behavior, psychotic or extrapyramidal reactions (rare)
- Serotonin syndrome with concomitant administration of MAO inhibitors, tricyclic antidepressants, antipsychotics, or meperidine
- Increased risk of abn bleeding

**Overview/Pharmacology**
- Selective inhibitor of serotonin reuptake
- Administered as racemic mixture of R-and S-enantiomers
- S-enantiomer more potent than R-enantiomer
- Active metabolites, R- and S-norfluoxetine, formed by demethylation
- Eliminated mainly through oxidative metabolism and conjugation
- Long elimination T½: 1–10 d for fluoxetine; 3–20 d for norfluoxetine
- Fluoxetine inhibits (and probably metabolized by) liver cytochrome P450 enzymes CYP2D6 and possibly CYP3A4: May inhibit metabolism, ↑ levels of β-blockers, benzodiazepines, antipsychotics
- Difficult to establish relationship between plasma conc of fluoxetine and effect, probably because these are four active compounds (R- and S-fluoxetine and R- and S-norfluoxetine) that require separate measurements

**Drug Class/Mechanism of Action/Usual Dose**
- Selective inhibitor of serotonin reuptake chronically taken for depression, obsessive-compulsive disorder, bulimia nervosa
- Not useful for acute administration, since full antidepressant effect may be delayed until 4 wk of treatment or longer
- Initial PO dose, 20 mg/d
- Maximal dose, 80 mg/d
- Alternatives: Other antidepressant medications

**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Bradycardia, dysrhythmia</td>
<td>(rare)</td>
<td>Pulse</td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>Slight BP increase</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Extrapyramidal symptoms (rare), mania (rare), serotonin syndrome (rare)</td>
<td>Headache, anxiety, tremor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>SIADH secretion (rare)</td>
<td></td>
<td>Urine specific gravity</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Nausea, wt loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Serotonin syndrome (rare)</td>
<td>Arthritic complaints (infrequent), muscle rigidity</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Implications/Possible Drug Interactions**
- Headache, anxiety, nausea are common symptoms
- May inhibit cytochrome P450 enzymes and ↑ the serum concentrations of other drugs (β-blockers, phenytoin, benzodiazepines, antipsychotics) and potentiate their effects
- Do not give to pregnant pts without assessing risk-benefit ratio

**Anticipated Problems/Concerns**
- Approx 7% of Caucasians lack the cytochrome P450 (CYP2D6) that probably metabolizes fluoxetine; these individuals may develop higher serum concentrations of fluoxetine and be more prone to side effects.
- Serotonin syndrome, characterized by agitation, confusion, diaphoresis, and muscle rigidity, may develop in pts who receive a combination of fluoxetine and MAO inhibitors

Uses
- Prevention of folic acid deficiency
- Treat megaloblastic anemia
- Experimental treatment for major depressive disorder
- Treat folic acid deficiency caused by anorexia, chronic oral contraceptive use, chronic use of some anti-epileptic drugs, alcoholism, malabsorption diseases (e.g., sprue), bowel resection, and diverticulosis
- Reduces incidence of NTD (spina bifida) and congenital heart defects in developing fetus
- Reduces homocysteine; may have CV benefits (no evidence of such from randomized trials, but lots of anecdotal evidence)

Perioperative Risks
- Overdosage chronically increase cancer proliferation—demonstrated in epidemiologic studies and in-vitro studies with breast cancer
- Exposure to NO disrupts folic acid metabolism; repeated exposure can cause deficiency
- Supraphysiological doses (>15 mg/d), may decrease seizure threshold in pts on some anti-epileptic medications

Worry About
- Allergic reactions (rare); most to the parenteral form
- Loss of appetite, nausea, lethargy, stomach pain, insomnia
- Supraphysiological doses (>15 mg/d) increase in all symptoms listed above
- May cause seizures (>15 mg/d); higher risk in epileptics

Overview/Pharmacology
- Vitamin with close synergistic relationships with vitamin B12, ascorbate, and zinc
- Very little found as folic acid in nature; converted to tetrahydrofolate (THF)
- Absorption most efficient in the jejunum
- Loss from the body is prevented by efficient enterohepatic recirculation
- Some fecal excretion; very little excreted in the urine
- Alcohol decreases blood levels by interfering with enterohepatic recirculation
- THF accepts and denotes one carbon group in amino acid degradation and metabolism reactions

Drug Class/Mechanism of Action/Usual Dose
- Vitamin
- Accepts and denotes one carbon group in amino acid degradation and metabolism reactions (i.e., in the synthesis of glycine from serine)
- Critical for cell division because required for purine and thymidine synthesis
- Oral and parenteral forms
- RDA 400 μg/d for healthy individuals; 600 μg/d for pregnant women
- Higher requirements (anemia, antifolate drug therapy, etc); 1 mg 1-3X/d (PO, IM, IV)
- Given as a multivitamin containing vitamin B12, because can mask vitamin B12 deficiency and accompanying neurologic damage

Perioperative Implications

Preoperative Concerns
- Deficiency may cause anemia
- Consider general nutritional status (i.e., if evidence of poor diet, folic acid deficiency likely)
- Consider specific underlying conditions (i.e., anorexia, alcoholism, malabsorption disorders)
- Continue periop supplementation as needed

Induction/Maintenance
- Same as Preoperative Concerns
- Avoid repeated use of N2O

Adjuvants/Regional Anesthesia/Reversal
- Same as Preoperative Concerns

Postoperative Period
- Same as Preoperative Concerns

Anticipated Problems/Concerns
- Rare allergic reactions
- Generally none in otherwise healthy pts
- May cause seizures (>15 mg/d) in seizure pts already on anti-epileptics

DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Improves O2 delivery</td>
<td>Better exercise tolerance</td>
<td>Hgb</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Improves cell division</td>
<td>Less diarrhea</td>
<td>Better hydration/absorption</td>
<td></td>
</tr>
<tr>
<td>ENDO/METAB</td>
<td>Improves nucleic acid/protein synthesis</td>
<td>Wt gain</td>
<td>Folate level</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Improves RBC synthesis</td>
<td>Better exercise tolerance</td>
<td>Hgb</td>
<td></td>
</tr>
</tbody>
</table>

Gold (Auranofin, Aurothioglucose, Aurothiomalate)

**Uses**
- Treatment for rheumatoid arthritis pts who do not respond to NSAIDs, steroids, or other disease-modifying antirheumatic drugs (DMARDs)
- Gold Rx as monotherapy dramatically decreasing 2° to preferred use of other DMARDs: e.g., methotrexate and use of biologic TNF inhibitors (infliximab and etanercept)

**Risks**
- Cutaneous reactions from erythema to exfoliative dermatitis (up to 30% of pts)
- Mucous membrane lesions: Stomatitis, pharyngitis, gastritis, colitis (20% of pts)
- Diarrhea is common in patients taking Auranofin (oral formulation)
- Gold deposition in dermis in dose-dependent manner, also renal tubule deposition
- Chrysiasis (gray-to-blue pigmentation of skin) possible with large cumulative doses; effect of transcutaneous Hgb saturation measurement unknown
- Proteinuria is common w/IM gold treatment (5–40%), usually resolves with cessation of treatment
- Cholestatic hepatic toxicity possible—resolves with cessation of therapy

**Overview/Pharmacology**
- Aurothioglucose, aurothiomalate administered IM, require close follow-up for side effects
- Auranofin administered orally, has differing pharmacokinetics, leading to potent immunosuppression and higher risk of rare side effects (thrombocytopenia, aplastic anemia)
- Oral preparation has lower incidence of common side effects, but slower elimination

**Key Reference:** Kean WF, Kean IR. Clinical pharmacology of gold. Inflammopharmacol. 2008;16:112–125.

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Glossitis, pharyngitis, (cricoarytenoid arthritis often present in RA pts)</td>
<td>ROM, decreased cervical range of motion and oral mouth opening</td>
<td>Lateral neck radiograph, neck CT, MRI</td>
</tr>
<tr>
<td>MSK</td>
<td>RA pts w/c-spine involvement (atlantoaxial subluxation) and sometimes TMJ involvement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Tracheitis, pneumonitis, pulm fibrosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Hepatitis</td>
<td>LFTs</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Proteinuria, hematuria, membranous glomerulonephritis (contraindicated during pregnancy, breastfeeding)</td>
<td>Renal function, pregnancy</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Encephalitis, peripheral neuritis</td>
<td>CNS exam</td>
<td></td>
</tr>
</tbody>
</table>

**Drug Interactions**
- None during anesthesia, but chrysiasis may interfere with pulse oximeter function

**Drug Interactions**
- Lesions of skin, mucous membranes may make these tissues friable
- RA may require fiberoptic or nasal endotracheal intubation
- Hematologic problems (thrombocytopenia, leukopenia) may manifest as bleeding or postop infection

**Perioperative Implications/Possible Drug Interactions**
- Severe RA; difficulty positioning on operating table
- Airway: Laryngeal arthritis, cervical instability, and pulm fibrosis; other problems of arthritis may be encountered
- Stomatitis, pharyngitis, tracheitis may make mucous membranes fragile

**Drug Interactions**
- None during anesthesia, but chrysiasis may interfere with pulse oximeter function

**Anticipated Problems/Concerns**
- Assess cervical instability and TMJ involvement in all severe RA pts (80% have upper C-spine involvement)
- Beware of renal, hepatic, and pulm dysfunction
**Haloperidol (Haldol)**

**Uses**
- Rx for
- Psychotic disorders in ambulatory population (PO)
  - Agitation caused by delirium in ICU pts (IV or IM)

**Perioperative Risks**
- Laryngospasm
- Extrapyramidal symptoms
- Neuroleptic malignant syndrome
- Cardiac arrest at high doses

**Worry About**
- May exacerbate symptoms in pts with Parkinson's disease
- Potential concern for neurotoxic metabolites
- Extrapyramidal symptoms less common with IV than PO doses

**Overview/Pharmacology**
- Dopaminergic antagonist
- Precise mechanism of action unknown
- Onset time: 5–20 min for IV; 30–60 min for PO
- Long (and variable) serum T½ (13–60 hr)
- 90–94% bound to serum proteins
- Therapeutic plasma concentration in range of 4–40 µg/L, but large variability among pts
- Clearance by hepatic metabolism
- Metabolized to reduced haloperidol, which has ~ 10% of activity of parent drug; reduced haloperidol may be oxidized and reconverted to haloperidol
- Renal excretion of parent drug is negligible

**Drug Class/Mechanism of Action/Usual Dose**
- Dopaminergic antagonist

**Overview/Pharmacology**
- Chronic administration
  - Management of psychotic disorders
  - Control of tics and vocal utterances of Tourette's disorder
  - Acutely taken to control agitation caused by delirium
- Usual PO dose 1–6 mg/d
- Usual IV or IM dose
  - 0.5–2 mg for mild agitation
  - 5 mg for moderate agitation
  - 10 mg for severe agitation (+10 mg/hr infusion)
- Alternatives
  - Other antipsychotic medications
  - Other antidelirium medications (e.g., physostigmine)
- Usually for agitation caused by delirium; agitation caused by anxiety/pain can be treated with benzodiazepines/narcotics

**System** | **Effect** | **Test**
--- | --- | ---
HEENT | Laryngospasm (infrequent side effect) | 
CARDIO | Hypotension or Htn, cardiac arrest (high doses) | 
HEPAT | ↓ Metabolism and ↑ serum concentration with hepatic disease | Monitoring of haloperidol concentrations is indicated only in pts with poor response at high doses or with hepatic disease
GI | Nausea | 
ENDO | Gynecomastia | 
GU | Urinary retention | 
CNS | Extrapyramidal symptoms (akathisia, dystonia, tardive dystkinesia) | 
MS | Neuroleptic malignant syndrome (NMS) | 


- Encephalopathic syndrome with combined use of lithium and haloperidol
- May potentiate effects of general anesthetics and narcotics

**Anticipated Problems/Concerns**
- Laryngospasm infrequent but life-threatening
- Cardiac arrests reported with high (~10 mg) doses
- IV haloperidol is not approved by the FDA for routine use
- NMS may develop 1–3 d after haloperidol administration and is characterized by muscle rigidity, hyperthermia, tachycardia, altered consciousness, and elevated serum creatine kinase concentrations. A mild form of NMS may occur in as many as 1% of pts given haloperidol.
Hormone Replacement Therapy (HRT)

Uses
  • To treat the physiologic and physical manifestations of hypogonadism due to hypogonadism or primary ovarian failure
  • To prevent or alleviate the signs and symptoms associated with surgical or age-related menopause, including vaginal dryness, uterine atrophy, irritability, depressed mood, osteoporosis, hyperlipidemia, CV disease, and vasomotor symptoms such as hot flashes.
  • A generic term that encompasses the use of unopposed estrogen therapy and combinations of estrogens and progestins.

Perioperative Risks
  • Increased risk for coagulopathy due to changes in coagulation and fibrinolytic pathways.
  • Within the first year of use, coagulopathy may cause a slight increase in coagulation factors II, VII, IX, X, and XII, while decreasing the anticoagulation factors Protein C, Protein S, and antithrombin III, enhancing clot formation.
  • When used in combination with a progestin, may result in decreased levels of plasmogen-activator inhibitor protein-1 (PAI-1) resulting in increased fibrinolysis and prolonged bleeding.

Worry About
  • Increased risk of thrombosis including DVT, pulmonary embolism, stroke, and myocardial infarction with estrogen replacement therapy if used without concomitant aspirin therapy (can be continued throughout the day of surgery for all but plastic, eye, and some neurologic operations).
  • Increased risk of fibrinolysis and prolonged bleeding with combination estrogen-progestin regimens.
  • Alterations in drug metabolism due to induction of various cytochrome P450 CYP isozymes.
  • Changes in drug distribution as a result of increased hepatic production of serum binding proteins.

Overview/Pharmacology
  • HRT provides pharmacologic effects of one or more estrogens, often in combination with progestrone or a chemical analogue, called a progestin.
  • Conjugated estrogens and synthetic progestins have been most commonly used in HRT.
  • Estrogens: A group of 18-carbon steroid compounds that occur naturally in three major forms: estrone, estradiol, and estriol. All steroids contain 4 condensed rings, designated A-D. The phenolic A ring is the principal structural feature that is responsible for selective, high-affinity binding to the estrogen receptors. As with most steroid hormones, estrogens can diffuse readily across cell membranes. Once within the cell, they bind and activate estrogen receptors that in turn up-regulate gene expression. Estrogen receptors are abundant throughout the body and can be found in the female reproductive tract, mammary glands, hypothalamus, endothelial cells, vascular smooth muscles, lung, brain, and bone.
  • Progestins: A family of 21-carbon steroids that are synthetic derivatives of the 19-nortestosterone structure. Designed to have progestinic effects similar to progesterone, progestins work by binding to an intracellular progesterone receptor resulting in transcriptional activation. Physiologic actions include endometrial proliferation, suppression of uterine contractility, mammary gland development, and thickening of endocervical gland secretions.

Drug Class/Mechanism of Action/Usual Dose
  • Classified as steroid hormones, estrogens and progestins bind to specific receptors and have widespread effects on many tissues in the body.
  • Various formulations are available for oral, parenteral, transdermal, or topical administration.
  • To reduce the risks of HRT, lower-dose estrogen therapy regimens are preferred over high-dose therapy. The typical daily oral dose of conjugated estrogen is 0.625 mg; however, initial treatment should start at a dose of 0.3 mg/d and dose adjustments made based on clinical response. Additional available dosages are 0.9, 1.25, and 2.5 mg.
  • The most commonly prescribed progestin is medroxyprogesterone acetate. It is typically given in a cyclic regimen (5–10 mg/d) or continuous regimen (2.5mg/d). Better choice is a micronized progesterin (Prometrium, for example) which doesn’t oppose the effect of estradiol on arterial function.

DRUG EFFECTS

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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Retinal vascular thrombosis, ↑ corneal curvature, ↑ lacrimal secretion</td>
<td>Changes or loss of vision, contact lens intolerance</td>
<td>Pale retina with cherry red macula, retinal hemorrhages</td>
<td>Ophthalmologic exam</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Fluid retention, Htn improved lipoprotein profiles: ↑ HDL, ↓ LDL</td>
<td>Swelling and wt gain, Htn</td>
<td>Edema</td>
<td>Physical exam, BP, lipid profile</td>
</tr>
<tr>
<td>GI</td>
<td>Pancreatitis, N/V, gallstone formation</td>
<td>Abd pain, intolerance to fatty foods</td>
<td>RUQ or epigastic pain</td>
<td>Amylase, lipase, alk phos, RUQ U/S, bilirubin</td>
</tr>
<tr>
<td>HEPAT</td>
<td>Adenoma enlargement cholesteatis</td>
<td>Abd pain, yellowing of skin</td>
<td>Hepatomegaly, jaundice</td>
<td>RUQ US, LFTs, bilirubin</td>
</tr>
<tr>
<td>GU</td>
<td>Abn uterine bleeding, changes in cervical secretions, increase in fibroid size, vaginal candidiasis</td>
<td>Vaginal bleeding, vaginal discharge, vaginal itching/burning</td>
<td>Enlarged lobulated uterus, vaginal discharge</td>
<td>Gynecologic exam, GYN US, KOH prep</td>
</tr>
<tr>
<td>HEME</td>
<td>Increased coagulation if not given with concomitant aspirin increased fibrinolysis</td>
<td>DVT, PE, MI, CVA Prolonged bleeding</td>
<td>LE swelling, SOB, CP, neuro deficits</td>
<td>PT/PTT, D-dimer, duplex US, CT angiography, fibrinogen, antithrombin III, Protein C</td>
</tr>
<tr>
<td>DERM</td>
<td>Chloasma, melasma, rashes, alopecia, hirsutism</td>
<td>Skin and hair changes</td>
<td>Hyperpigmentation, erythema, papules, nodules, hair changes</td>
<td>Dermatologic exam</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Concerns
  • Changes in angiotensin-aldosterone system may result in elevated BP and/or renal failure.
  • Increased risk for thrombosis if not given with concomitant aspirin. Its undergoing procedures associated with moderate to high risk for venous thromboembolism should stop hormone therapy at least 4 to 6 wk prior to surgery. Rigorous prophylaxis for DVT must be observed in the perioperative period.
  • The risks associated with temporary D/C of hormone therapy include withdrawal bleeding, hot flashes, and other menopausal symptoms.

Induction/Maintenance
  • Alterations in the activity of various cytochrome P450 CYP isozymes may require dose adjustment of heptically cleared drugs.
  • HRT may induce metabolism of drugs which are glucuronidated, incl some benzodiazepines and analogues.
  • Progestin metabolite, allopregnanolone, may affect the excitability of neurons through direct modulation of the GABA-A receptors exerting hypnotic/sedative, anxiolytic, and anesthetic.

Postoperative Period
  • Increased risk for thrombosis extends into the postoperative period. A heightened suspicion for postoperative DVT, pulmonary embolism, stroke, and myocardial infarction must be maintained. Restart or continue aspirin which should be always given concomitantly unless contraindicated.
  • Activation of fibrinolytic pathways in pts using combined estrogen-progesterin replacement therapy may result in postoperative bleeding.

Anticipated Problems/Concerns
  • Coagulopathy, esp. increased risk for thromboembolism, remains a top concern for women using HRT.
Insulin Receptor Modifiers

### Uses
- Oral anti-diabetic agents for Type 2 diabetes.

### Perioperative Risks
- **Hypoglycemia:** Rare on monotherapy as the action is glucose mediated; caution is needed when used in combination with insulin or sulphonylureas (SUs).
- **Hepatotoxicity:** Though less likely with the currently available thiazolidinediones (TZDs), it is important to note that an earlier TZD (troglitazone) was withdrawn from market due to hepatotoxicity. Recommended monitoring would be liver function tests postop.
- **CV risk:** Increased sodium and water retention. Worsens heart failure esp. in NYHA class III and IV. Risk is higher with concomitant use of insulin.

### Worry About
- **Precipitation of heart failure:** Volume expansion and sodium retention
- **Hypoglycemia:** When used in conjunction with insulin and SUs

### Key References:

### DRUG EFFECTS

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<tr>
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<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>↑ Body wt (ave. of 3–5 kg)</td>
<td>Pre-drug body wt</td>
<td>↑ SQ fat and ↓ visceral fat</td>
<td>Improved waist-hop ratio</td>
</tr>
<tr>
<td>CARDIO</td>
<td>↑ Fluid retention Vasodilatation Worsening heart failure, esp. in NYHA III &amp; IV</td>
<td>↑ Ankle swelling</td>
<td>Edema</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>↑ Anemia</td>
<td>Easily fatigued</td>
<td>Pallor</td>
<td>Hb – ↓ of up to 2–4 g</td>
</tr>
<tr>
<td>SKEL</td>
<td>↑ of fractures</td>
<td>Spontaneous fractures; low impact fractures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENT</td>
<td>Sinusitis, pharyngitis, URI</td>
<td>Coryza, headache, rhinorrhea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Hepatotoxicity (1:100,000)</td>
<td>Loss of appetite, abd pain Jaundice, dark urine LFTs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypoglycemia (in combination with insulin and SUs)</td>
<td>Sweating, tremors, blurring of vision, palpitation Tachycardia, altered consciousness</td>
<td>Glucose</td>
<td></td>
</tr>
<tr>
<td>EYES</td>
<td>↑ Macular edema (rare)</td>
<td>Blurring of vision ↓ visual acuity</td>
<td>Fundus exam</td>
<td></td>
</tr>
<tr>
<td>REPRO</td>
<td>↑ Ovulation (↑ chances of pregnancy in PCOS women)</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

### Anticipated Problems/Concerns
- Resistant hyperglycemia needing large doses of insulin, esp. in obese individuals who have responded well to TZDs
- Use of beta-blockers may mask signs of hypoglycemia
- In doubt, stop or delay the resumption of TZDs.

### Latest Development
- Recently, the FDA has introduced a stringent guideline for using Rosiglitazone because of the potential increased risk of myocardial infarction (MI). The European equivalent (EMEA) has suspended the drug from the market across Europe. Extreme caution is therefore required for patients on Rosiglitazone undergoing surgery.
Isoproterenol (Isuprel, Medihaler-ISO)

Uses
- Primarily used in the treatment of bradycardia with heart block.
- Temporary control of bradycardia in denervated heart transplant pts unresponsive to atropine.
- Adjunct therapy in the treatment of hypovolemic, cardiogenic, and septic shock.
- Treatment of Adams-Stokes attacks (except when caused by ventricular tachycardia or fibrillation).
- May be used as an inhaled aerosol to treat bronchospasm during anesthesia, asthma exacerbation, chronic bronchitis, and emphysema.
- Unlabeled use as pharmacologic overdrive pacing for refractory tachycardia.
- Provokes vasovagal syncope during tilt table testing.
- Unlabeled use in mesotherapy for body contouring by promoting release of stored fat.

Perioperative Risks
- Isoproterenol may have a deleterious effect on an injured or failing heart by increasing myocardial O2 demand, and decreasing effective coronary perfusion.
- May lead to myocardial ischemia, may transiently increase blood glucose levels and may induce thyroid storm in susceptible individuals.
- Avoid with halothane and desflurane to reduce the potential for sensitization of the myocardium to effects of sympathomimetic amines.
- Avoid with MAOIs to prevent synergistic effects with released catecholamines.
- Administration may cause N/V.

Perioperative Implications
- Use is contraindicated in pts with myocardial ischemia, pre-existing tachyarrhythmia, angina pectoris, heart block caused by digitalis toxicity and cardiac glycoside intoxication.
- Caution is necessary when administering isoproterenol to pts with CAD, diabetes, hyperthyroidism, and sensitivity to sympathomimetic amines.
- Contains sodium metabisulfite which may cause allergic reaction and asthma exacerbation in susceptible individuals.
- Paradoxical airway resistance with repeated, excessive use of inhalation preparations.
- Animal reproduction studies have not been conducted. It is not known whether isoproterenol can cause fetal harm when administered to a pregnant woman. Excretion in breast milk is unknown. Should be given if clearly indicated.

Dosage and Administration
- Usual route of administration is IV infusion or bolus IV injection. Can also be given IM or SQ as well. In dire emergencies, the drug can be administered by intracardiac injection.
- Should be started at the lowest recommended dose and gradually increased to limit risk of ventricular arrhythmias.
- Bronchospasm during anesthesia (adults): IV 0.01 to 0.02 mg. Repeat as necessary. INH 1–2 doses 0.08 mg/Inh.
- Shock and hypoperfusion (adults): IV 0.5 mcg to 5 mcg/min.
- Heart block, Adams-Stokes attacks, and cardiac arrest (adults): Bolus IV 0.02 mg to 0.06 mg as initial dose with subsequent dose range of 0.01 mg to 0.2 mg; IV infusion 2–10 mcg/min, with titration to pt response; IM 0.2 mg initial dose with a subsequent dose range of 0.02 mg to 1 mg; SQ 0.2 mg initial dose of 0.2 mg with subsequent dose range of 0.15 mg to 0.2 mg; intracardiac 0.02 mg initial dose, with subsequent dosage and administration method dependent upon ventricular rate and rapidity with which cardiac pacemaker can take over when drug is withdrawn.

DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Tachycardia, increased contractility, increased CO, decreased SVR</td>
<td>Palpitations, angina</td>
<td>Pallor, flushing, hyper-/ hypotension</td>
<td>ECG, pulse arterial blood pressure, CVP</td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchodilation</td>
<td>Dyspnea</td>
<td>Pulm edema</td>
<td>Respirations, peak airway pressure</td>
</tr>
<tr>
<td>CNS</td>
<td>Stimulant</td>
<td>Headache, anxiety, weakness, insomnia</td>
<td>Nervousness, restlessness, tremor</td>
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<tr>
<td>GI</td>
<td>Vasodilation of mesenteric beds</td>
<td>N/V</td>
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</tr>
<tr>
<td>ENDO</td>
<td>Hypokalemia</td>
<td>Increased serum glucose</td>
<td>Basic metabolic panel</td>
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</tbody>
</table>

Lithium Carbonate (Lithobid)

**Uses**
- For treatment of manic episodes for bipolar and some schizoaffective disorders
- Approved for maintenance therapy to help prevent episodes of mania or depression
- As an augmenting agent for antidepressants. Has also been used to treat aggression, PTSD, and conduct disorder in children
- For neuropenia associated with chemotherapy, HIV therapy, and other medications (acute exposure to lithium can cause leukocytosis; chronic exposure may cause aplastic anemia)
- For hyperthyroidism, e.g., Graves disease (may eventually lead to hypothyroidism)
- May be used to treat syndrome of inappropriate antidiuretic hormone secretion (causes nephrogenic DI)

**Perioperative Risks**
- Extremely narrow therapeutic level with desired serum levels of 0.6–1.2 mEq/L
- Interaction with depolarizing and nondepolarizing muscle relaxants causes a prolonged response, specifically with pancuronium and succinylcholine.

**Pharmacokinetics/Pharmacodynamics**
- At cellular level, acts as imperfect substitute for Na+, intracellular accumulation of lithium decreases phosphatidylinositolby interfering with hydrolysis of myoinositol-1-phosphate in the brain. Specific mechanism of action unknown.
- Decreases availability of norepinephrine at central adrenergic synaptic cleft because increased reabsorption into storage granules. Also interferes with calcium depolarization-mediated release of norepinephrine and dopamine centrally.
- May also inhibit ability of some hormones to activate adenylyl cyclase
- Apparent volume of distribution 0.6–1 L/kg
- Almost complete absorption from GI tract; peak levels 2–4 hr after oral dose
- Initial distribution in extracellular fluid, subsequent accumulation in tissues
- No plasma protein binding
- Eliminated exclusively by renal excretion with a half-life of 20–27 hr after a single dose. One third to ½ acute dose excreted in 6–12 hr; 80% reabsorbed in the proximal convoluted tubules.
- Reabsorption is related to sodium balance. Na+ depletion causes retention of lithium; increase lithium levels from thiazide diuretics, ECA, furosemide; Na+ loading causes increased excretion of lithium.
- Lithium clearance is 20% of creatinine clearance
- Low therapeutic index: 0.8–1.25 mEq/L, toxic at levels >1.5 mEq/L

**Drug Class/Usual Dose**
- Lithium salt
- Daily dose is individualized; requires regular monitoring of lithium levels. Usual adult dose varies: 900–2400 mg/d in 3–4 divided doses or 900–1800 mg/d in two divided doses of sustained release.

**Pharmacokinetics**
- Serum levels cause benign ST interval/T wave changes
- Toxicity: Malignant arrhythmias, heart block, hypotension
- Enlarged tender thyroid; hypothyroidism rare
- Nephrogenic DI
- Tremor, drowsiness, coma, convulsions
- Toxicity: May cause drowsiness, EEG slowing
- Therapeutic levels cause benign ST interval/T wave changes
- Dose, intercurrent illness, drugs precipitating toxicity
- Neck pain, hypothyroid symptoms
- Polyuria, polydipsia
- Dose, concomitant therapy, illnesses
- CNS Exam
- Lithium level

**DRUG EFFECTS**

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</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Therapeutic levels cause benign ST interval/T wave changes</td>
<td>Dose, intercurrent illness, drugs precipitating toxicity</td>
<td>CVS exam</td>
<td>ECG</td>
</tr>
<tr>
<td>ENDO</td>
<td>Enlarged tender thyroid; hypothyroidism rare</td>
<td>Neck pain, hypothyroid symptoms</td>
<td>Thyroid</td>
<td>FT, E/TSH</td>
</tr>
<tr>
<td>GU</td>
<td>Nephrogenic DI</td>
<td>Polyuria, polydipsia</td>
<td>Urine/serum electrolytes/osmolality</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Toxicity: Tremor, drowsiness, coma, convulsions</td>
<td>Dose, concomitant therapy, illnesses</td>
<td>CNS exam</td>
<td>Lithium level</td>
</tr>
</tbody>
</table>


**Possible Drug Interactions**
- Drugs that affect GFR or promote renal sodium wasting leads to increased lithium levels and risk of toxicity: Thiazide diuretics, ECA, furosemide, ACE inhibitors, and carbamazepine
- Increased risk of neurotoxicity: Verapamil, dilantin, metronidazole
- Increased risk of serotonin syndrome with fluoxetine

**Induction/Maintenance**
- Must be aware of signs of lithium toxicity
- May have reduced requirement for inhaled and injected anesthesia
- Possibility of prolonged NM blocking effects
- Delayed recovery from barbiturates reported

**Preoperative Concerns**
- May have reduced requirement for inhaled and IV anesthetics (reduced MAC)
- Toxicity expressed by GI symptoms (nausea/vomiting), psychiatric symptoms (anxiety, insomnia, irritability, mood lability), neurologic symptoms (dizziness, headache, parasthesia, tremor), and somatic symptoms (fatigue, myalgias, chills, rhinorrhea)
- May be used to treat syndrome of inappropriate antidiuretic hormone secretion (causes nephrogenic DI)

**Anticipated Problems/Concerns**
- Be aware of signs and symptoms of toxicity. Toxic levels can be decreased with osmotic diuretics (do not use furosemide), administration of saline, or dialysis.
- Renal toxicity is common with chronic lithium therapy. Nephrogenic DI is the most common manifestation. Electrolyte balance is very important.
- Hypothyroidism is the most common endocrine disorder cause by chronic lithium therapy.

**Acute Exposure**
- Acute exposure to lithium can cause leukocytosis; chronic exposure may cause aplastic anemia.
- Severe CV collapse; arrhythmias, heart block possible with toxicity
- Abrupt D/C of lithium does not cause withdrawal affect and can be continued soon after surgery.
- Lithium is contraindicated in pregnancy with increased risk for cardiac anomalies (Epstein’s anomaly). May be excreted in breast milk. Lithium should be avoided in first trimester of pregnancy.
- Affects response of commonly used anesthetic drugs.
Magnesium Sulfate

Uses
- Hypomagnesemia and magnesium deficiency in critically ill pts
- Treatment of tics de points, digoxin toxicity, atrial or ventricular arrhythmias
- Treatment of pre-eclampsia, eclampsia, prevention of seizures due to eclampsia, tocolysis
- Management of conditions with catecholamine excess (tetanus, pheochromocytoma, attenuation of stress response during laryngoscopy)
- Orally as cathartic or laxative
- Acute asthma
- Adjuvant to other agents during general anesthesia to reduce the requirements of analgesics, muscle relaxants, and hypnotics
- Controlled hypotension

Perioperative Risks
- Hypotension
- Potentiation of nondepolarizing NMB and muscle weakness in pts with high levels of serum magnesium (levels greater than 8 mEq/L)
- Inadvertent use in pts with impaired-renal function can lead to a state of hypermagnesemia; however, hypomagnesemia may increase the risk of periop arrhythmias.

Worry About
- Dose adjustment via titration is vital during simultaneous administration with muscle relaxant in pt on magnesium sulfate. One third of normal dose is sufficient for maintaining NMB.
- Magnesium deficiency is very undesirable in periop period and in critical care.
- Administration can cause decreased systemic vascular resistance (SVR) leading to hypotension in septic pts.
- Decreased responsiveness to vasopressors due to effect of magnesium on catecholamine reuptake

Overview/Pharmacology
- Magnesium is an emerging drug in anesthesia (molecule of the century); it is the fourth-most common cation in the body and second-most common intracellular cation after potassium.
- Physiological antagonist of calcium and has a fundamental role as a cofactor in over 300 enzymatic reactions.
- Magnesium is available as an inorganic phosphate.
- CVS: Reduces the systemic vascular resistance in high doses. Prolongs SA node conduction time and reduces the rate of SA node impulse formation. Excess catecholamine induced vasconstriction, arrhythmogenic effects and diastolic dysfunction are attenuated by magnesium.
- Antiepileptic properties and the action on CNS are not very defined. Various postulations for neuroprotection incl cerebral vasodilatation, blood-brain barrier protection, and anticonvulsant actions.

DRUG EFFECTS

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<tbody>
<tr>
<td>CARDIO</td>
<td>Vasodilatation, sympathetic blockade, inhibition of catecholamine release, decreased myocardial contractility after a bolus dose of more than 2.5 g</td>
<td>Light headaeness, flushing or sensation of warmth if given in an awake pt</td>
<td>Bradycardia, low BP, poor peripheral and systemic perfusion due to vasodilatation and low cardiac output</td>
<td>Check levels CO monitoring (non-invasive and invasive)</td>
</tr>
<tr>
<td>RESP</td>
<td>Resp depressant effect due to NMB. Bronchodilator</td>
<td>Resp insufficiency Improvement in asthmatic pts</td>
<td>Hypoxia, hypoventilation, sedation, hypercapnea</td>
<td>Monitor levels, pulse oximetry, ABG</td>
</tr>
<tr>
<td>CNS</td>
<td>Anti epileptic, NMDA receptor blockade, potentiation of NMB</td>
<td>Cessation of convulsions Analgesic adjuvant Prolonged recovery from non-depolarizing blockade, muscle weakness</td>
<td>Postictal phase Decreased deep tendon reflexes Improvement in analgesia</td>
<td>Monitor levels</td>
</tr>
<tr>
<td>MS</td>
<td>Weakness, increased sensitivity to non depolarizing relaxants</td>
<td>Respiratory depression Heightened response to muscle relaxants</td>
<td>Weakness, lethargy, absent or reduced deep tendon reflexes (DTR)</td>
<td>Monitor DTR, twitch monitoring</td>
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<tr>
<td>OB</td>
<td>Tocolytic, Arrests labor, decreased uterine tone</td>
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</tbody>
</table>

Perioperative Implications

Preoperative Concerns
- Exaggerated hemodynamic response to induction agents and central neuroaxial blockade
- Enhanced effects of CNS depressants and potentiation of nondepolarizing blockade
- Monitoring levels is important in pt with renal failure.

Induction/Maintenance
- Dose of induction agent to be titrated as profound drop in BP can occur.
- Use of muscle relaxants to be avoided unless indicated. Use 30–40% of required maintenance dose. Succinylcholine can be safely used as it is not affected by magnesium.

Drug Class/Mechanism of Action/Usual Dose
- Key actions are calcium antagonism via calcium channels, regulation of energy transfer, membrane sealing, or stabilization. Presynaptically inhibits release of acetylcholine at the NM junction.
- Emergency treatment: IV 2–4 grams (8–16 mmol) initially slowly over 20 min, followed by 10 gram (40 mmol) over next 5 hr.
- It can be given by IM route, however, it is very painful.
- Eclampsia: 4 g IV over at least 5 min followed by 1 g hr for 24 hr after the last seizure. Therapeutic levels: 4–8 mEq/L. Clinical signs of toxicity inc loss of reflexes and resp insufficiency. Assoc with development of cerebral palsy.

Subramanian Sathishkumar
Sanjib Adhikary

**Marijuana**

**Uses**
- Many states have passed laws to allow compassionate use of marijuana to alleviate symptoms of debilitating diseases such as pain, nausea and/or vomiting, cachexia, glaucoma, spasms, and seizures.
- Trials are under way for using cannabinoid derivatives as an aid for wt loss; and for preventing relapse of cigarette smoking and opioid abuse.

**Perioperative Risks**
- Bronchospasm, laryngospasm, uvular edema, pharyngitis
- Inability to filter unnecessary information and perform complex mental tasks

**Worry About**
- Multiple drug consumption
- Drug interactions with other intoxicants such as cocaine and ethanol or commonly prescribed medications (antidepressants, protease inhibitors, sildenafil)
- Panic reactions and acute psychosis, which is more common with oral intake
- Fetal effects if pregnant

**Overview/Pharmacology**
- Marijuana is rapidly titrated by inhalation with maximal effects after 2–4 hr. Oral administration within an hour peaks and has prolonged effects lasting approx 5 hr 2° to absorption in the GI tract.
- THC, the main active cannabinoid in marijuana, undergoes first-pass metabolism in the liver into active metabolites.
- The plasma elimination half-life of marijuana is 56 hr (28 hr with chronic use). Its tissue half life is about a week and complete elimination may take 1 mo with chronic use.
- Metabolites are eliminated via urinary excretion or secreted through the biliary tract.
- Cannabinoids are highly lipid soluble and accumulate in breast milk.
- Cannabinoids easily cross both the blood-brain barrier and placenta.

**Drug Class/Mechanism of Action/Usual Dose**
- Cannabinoid receptors are coupled to G proteins, which inhibit adenyl cyclase.
- Cannabinoid receptors are present on many peripheral and central sites, esp. the hippocampus, striatum, basal ganglia, cortex, cerebellum, and spinal cord.
- Inhalational use of marijuana is usually titrated to effect. The dose of dronabinol for appetite stimulation is 2.5–5 mg daily. Nausea 2° to chemotherapy can be as much as 5 mg/m², 6 times a day.

**DRUG EFFECTS**

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypotension</td>
<td>Recent exposure</td>
<td>Vital signs</td>
<td>Urine tox screen</td>
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<tr>
<td></td>
<td>Tachycardia (bradycardia with chronic use)</td>
<td>Duration and amount of use</td>
<td>Injected conjunctiva</td>
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<td></td>
<td>Vasodilatation</td>
<td>Use of other recreational drugs</td>
<td>Reduced oculomotor racking</td>
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<td></td>
<td>Myocardial depression with higher doses</td>
<td>Tobaco/alcohol Hx</td>
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<td></td>
<td>Increased myocardial O₂ demand</td>
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<td></td>
<td>Increased cerebral blood flow (decreased with chronic use)</td>
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<tr>
<td>RESP</td>
<td>Coughing</td>
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<td></td>
<td>Decreased O₂ -carrying capacity 2° to CO intake with inhalation</td>
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<td></td>
<td>Bronchial dilation</td>
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<td></td>
<td>Increased ventilation (decreased with larger doses)</td>
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<td></td>
<td>Bronchitis</td>
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<td></td>
<td>Decreased transport of secretions</td>
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<td></td>
<td>Squamous metaplasia</td>
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<td></td>
<td>Emphysema</td>
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<tr>
<td>CNS</td>
<td>Euphoria/dysphoria</td>
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<td></td>
<td>Lethargy</td>
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<td>Impairment of coordination</td>
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<td>Changes in perception</td>
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<td>Decreased ability to perform complex thoughts or actions</td>
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<td>Decreased nausea</td>
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<td>Dizziness</td>
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<td></td>
<td>Hallucinations</td>
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<td></td>
<td>Panic reactions</td>
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<td></td>
<td>Ataxia/dysarthria</td>
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<td>Confusion</td>
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<td>Amnesia</td>
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<td>Anticonvulsant/proconvulsant</td>
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<td>Schizophreniform symptoms</td>
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<td></td>
<td>Poor judgment</td>
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<td></td>
<td>Increasing cognitive impairment with chronic use</td>
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<tr>
<td></td>
<td>Depression</td>
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<td>VISION</td>
<td>Decreased intraocular pressure</td>
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<td>Possible rebound increase in intraocular pressure with cessation</td>
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<td>Poor oculomotor tracking</td>
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<tr>
<td>Immune</td>
<td>Decreased resistance to infection</td>
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<td>Impairment of macrophages</td>
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<tr>
<td>GU</td>
<td>Urinary retention</td>
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<tr>
<td>Pregnancy</td>
<td>Preterm labor</td>
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<td>IUGR</td>
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<td>VSD in fetus</td>
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<td>Delay in cognitive development</td>
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**Perioperative Implications**

**Preoperative Concerns**
- Chronic use could lead to prolonged intoxication lasting several days to storage in adipose tissue and reuptake of active metabolites in the gut.
- Pts may be sedated or have signs and symptoms of bronchitis and asthma.
- Marijuana may increase opioid effects on ventilation.

**Induction/Maintenance**
- May interact with medications that affect heart rate.
- Reduces the MAC and may cause pronounced myocardial depression with potent inhaled anesthetics.

**Postoperative Period**
- Increased postop agitation and confusion
- Motor function and coordination may be reduced for a longer period than anticipated.
- Some pts may experience withdrawal. Signs include restlessness, irritation, agitation, nausea, and cramping.

**Anticipated Problems/Concerns**
- Anesthesiologists should anticipate interactions with anticholinergics, barbiturates, and depressants.
- Increased risk of having resp complications during anesthesia
- Periop agitation
- Recent use may impair pts' ability to give consent. Chronic use may lead to difficulty following postop instructions.
- Interactions with the effects of chronotropic medications
- Cannabinoids have prolonged action in pts of increasing age and pts with liver disease.
- Anesthesiologists should encourage preop discontinuance of the drug for elective cases and consider delaying elective cases with recent use.
Metformin (Glucophage)

Uses
- Oral antihyperglycemic drug used in the management of type 2 diabetes
- Most popular oral hypoglycemic drug in the United States and one of the most prescribed drugs overall.

Perioperative Risks
- Metformin-assoc lactic acidosis (MALA)
- Hypoglycemia (rare)

• Risk of metformin accumulation and MALA increases when there is renal impairment.

Pharmacokinetics/Pharmacodynamics
- Oral bioavailability 50–60%
- Absorbed from the small intestine
- Binding to plasma proteins is negligible
- Not metabolized
- Excreted unchanged in the urine
- Half-life is approx 6 hr, however antihyperglycemic effects last >24 hr

Drug Class/Mechanism of Action/Usual Dose
- Biguanide oral antihyperglycemic
- Decreases hepatic glucose production
- Decreases intestinal absorption of glucose
- Improves insulin sensitivity by increasing peripheral glucose uptake and utilization
- Usually dosed 500 mg twice a day
- Maximum recommended daily dose is 2550 mg

ASSESSMENT POINTS

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<thead>
<tr>
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<tbody>
<tr>
<td>ENDO</td>
<td>Hypoglycemia</td>
<td>Use of other oral antihyperglycemic, decreased PO intake, alcohol consumption Elderly, debilitated, or malnourished pts, and those with adrenal or pituitary insufficiency more susceptible</td>
<td>Irritability, seizures, bradycardia, hypotension, resp failure</td>
<td>Serum glucose (&lt;50 mg/dL)</td>
</tr>
<tr>
<td>METAB</td>
<td>Lactic acidosis</td>
<td>Presence of predisposing conditions: Disease states that increase production of lactic acid (CHF, hypoxic states, shock, septicemia) or decrease removal of lactic acid (severe liver disease, alcohol)</td>
<td>Non-specific Hypotension and resp failure have been reported</td>
<td>Serum lactate, serum bicarbonate, ABG, metformin levels</td>
</tr>
<tr>
<td>GI</td>
<td>Diarrhea, N/V, flatulence, indigestion, abd discomfort</td>
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<td></td>
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</tr>
<tr>
<td>CNS</td>
<td>Headache</td>
<td></td>
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</tr>
<tr>
<td>Other</td>
<td>Asthenia, megaloblastic anemia</td>
<td></td>
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</tbody>
</table>


Perioperative Implications

Preoperative Concerns
- Metformin should be temporarily D/C preop
- Metformin should be D/C at the time of, or prior to, use of IV contrast dye, withheld for 48 hrs subsequent to the procedure, and reinstituted only after renal function has been reassessed.
- Co-morbidities that may increase the incidence of MALA in pts taking metformin are renal dysfunction, heart failure, resp failure, impaired liver function, sepsis, and severe dehydration.

Induction/Maintenance
- No known drug interactions
- No known contraindications

Adjuvants/Regional Anesthesia/Reversal
- No known contraindications

Postoperative Concerns
- Metformin should be withheld in pts having postop renal complications
- Do not resume metformin until the pt is tolerating a PO diet

Drug Interactions
- Cimetidine competes for the renal tubular secretion of metformin and concomitant use leads to higher metformin serum levels.
- Other cationic drugs such as vancomycin, digoxin, procainamide, and trimethoprim may interact similarly

Anticipated Problems/Concerns
- MALA and risk factors for MALA can be assessed for (hypovolemia, hypotension, hypoxemia)
- Hyperglycemia from withholding metformin in the periop period
Monoamine Oxidase Inhibitors; Reversible Inhibitors of Monoamine Oxidase

Uses
- Monoamine oxidase (MAO) inhibitors are indicated for refractory depression, depression with prominent anxiety, low psychomotor activity, and severe phobias.
- Other indications incl Htn, narcolepsy, and intractable headache.

Perioperative Risks
- Hypertensive crisis
- Serotonin syndrome (central serotoninergic hyperactivity) may be fatal and manifests as Htn, hypotension, resp depression, tachycardia, diaphoresis, hyperthermia, muscle rigidity, seizures, agitation, headache, and coma.

Worry About
- Side effects incl orthostatic Htn, agitation, tremor, seizures, muscle spasms, urinary retention, dysuria, paresthesias, hepatotoxicity, jaundice, sedation, vision changes, hallucinations, dryness of the mouth, and constipation.

Induction/Maintenance
- Consider arterial cannula for close monitoring of BP.
- Phentolamine can prolong the duration of succinylcholine by inhibiting pseudocholinesterase
- Interaction with opioids, particularly meperidine, can lead to the potentially fatal serotonin syndrome.
- Consider regional techniques to avoid opioids; morphine or fentanyl are preferred if necessary
- N.O and volatile agents are acceptable
- Pronounced response to vasopressors and sympathetic stimulation can occur; direct-acting vasopressors of short duration at a reduced dose are preferred (such as phenylephrine at ½ the usual dose)
- Avoid drugs that increase sympathetic activity such as ketamine, pancuronium, cocaine, and epinephrine (in local anesthetics)

Postoperative Period
- Judicious opioid use if needed
- Use adrenergic α- or β-antagonists or direct-acting vasodilators for Htn

Anticipated Problems/Concerns
- Pain control
- Hemodynamic instability
- Timing and dosing of MAO inhibitor resumption

Overview/Pharmacology
- MAO is an endogenous mitochondrial enzyme which inactivates neurotransmitters by deamination.
- MAO inhibitors block oxidative deamination of naturally occurring amines, which permits neurotransmitter accumulation and increased adrenergic receptor activation.
- 2 MAO isoenzymes (types A and B) differ in their substrate selectivities.
- MAO A is selective for serotonin, dopamine, and norepinephrine.
- MAO B is selective for tyramine and phenylethylamine; ineffective as antidepressants
- Nonselective (irreversible MAO inhibitors) agents: Phenelzine (Nardil), isocarboxazid (Marplan), and tranylcypromine (Parnate)
- Nonselective agents may interfere with many other enzymes
- Selective agents (reversible MAO A inhibitors): Moclobemide, brofaromine, lazabemide, toloxetine, and cimoxatone

PERIOpERATIVE IMPLICATIONS

Preoperative Concerns
- Avoid co-administration of MAO inhibitors and SSRIs within 6 wk to avoid serotonin syndrome
- Check LFTs as hepatotoxicity and/or hepatic enzyme inhibition may exaggerate depressant effects of opioids, benzodiazepines, barbiturates, antihistamines, anticholinergics, and tricyclic antidepressants.
- Controversy persists regarding D/C prior to elective surgery.
- Previous recommendations were cessation 2–3 wk prior to surgery, but the decision should take into consideration the pt’s indication and dependence (risk of suicide).
- Effective anxiolysis to avoid sympathetic hyperactivity

Other References

Key Reference:
Nicotine Replacment Therapies (NRTs)**

**Uses**
- NRTs are FDA-approved devices that are effective in helping treat tobacco dependence, acting on nicotinic acetylcholine receptors to mimic or replace the effects of nicotine, the highly addictive chemical from tobacco products.
- NRTs are available OTC (gum, transdermal patch, sublingual lozenge/tablet) and by prescription (nasal spray, inhaler).
- NRTs provide only nicotine; they do not contain the carcinogens and toxic gases that are found in cigarette smoke.

**Perioperative Risks**
- While there is no clear evidence that NRTs increase CV risk in medically stable pts, the safety of NRTs in critically ill pts or in pts undergoing cardiopulmonary bypass surgery, esp. off-pump cardiac surgery, is questionable.
- During MRI procedures, transdermal nicotine patches that have metallic components can cause cutaneous burns if a pt wears them during the scan.
- Nicotine gum or sublingual lozenges/tablets can cause hiccups, nausea, and heartburn; this could potentially increase aspiration risk for pts undergoing general anesthesia.

**Worry About**
- A fatal nicotine dose for adults is around 60 mg. Individual cigarettes contain 1–3 mg of nicotine. While it is difficult to overdose solely by smoking, the use of nicotine patches, nicotine gum, or other NRTs concomitantly with tobacco smoking could place the user at risk. Make sure smoker takes off patch prior to surgery as increased skin blood flow with inhalation agents could increase absorption from skin depot or patch.
- Nicotine poisoning manifests as nausea, salivation, abd cramps, vertigo, mental confusion, difficulty breathing, increased heart rate, skeletal muscle weakness, and seizures.
- Nicotine withdrawal can create a negative emotional state, anxiety and irritability, perception of increased stress, difficulty concentrating, increased appetite, headache, and insomnia.
- Nicotine medication via NRTs is generally considered safer than cigarette smoking since exposure to toxic combustion products is averted; however, concerns exist regarding the safety of long-term nicotine exposure and its effect on CV disease, cancer, wound healing, and fetal development.
- NRTs can cause irritation to the skin or inside of the mouth.

**Overview/Pharmacology**
- Nicotine from NRTs is absorbed from the skin, the resp tract, or buccal mucous membranes. These methods deliver nicotine to the bloodstream more slowly than smoking.
- Nicotine’s half-life is approx 2 hr. It is metabolized primarily by the liver and partly by the lungs. Cotamine, which can be a urinary marker of nicotine exposure, is the principle metabolite with ⅓ the pharmacologic activity of nicotine.
- Nicotine and its metabolites are eliminated by the kidneys and in breast milk.
- Nicotine can cause the induction of liver microsomal enzymes, resulting in faster metabolism of some anesthetics, analgesics, and sedatives.

**Drug Class/Mechanism of Action/Usual Dose**
- Nicotine is a highly addictive alkaloid. It is a sympathomimetic drug that stimulates autonomic ganglia and acts as a central nicotinic cholinergic agonist, thereby facilitating neurotransmitter release (i.e., dopamine, norepinephrine, serotonin, glutamate, GABA).
- A typical pack-per-day smoker absorbs 20–40 mg/day. The dose of NRTs is variable: transdermal patches (5–22 mg/24 hr); gum, lozenges, tablets (1–4 mg each); inhaler (cartridge contains 10 mg); nasal spray (0.5 mg/spray).
- Nicotine can have unpredictable effects, initially acting as a stimulant and then as a depressant.

**Drug Effects**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>↑ HR, BP, cardiac contractility; coronary and peripheral vasoconstriction</td>
<td>Palpitations, chest pain</td>
<td>Cardiac exam, heart sounds</td>
<td>Vital signs</td>
</tr>
<tr>
<td>RESP</td>
<td>↑ Ventilation (stim. of aortic and carotid body chemoreceptors)</td>
<td>↑ Resp rate</td>
<td>Resp exam, breath sounds</td>
<td>O₂ sat, resp rate</td>
</tr>
<tr>
<td>GI</td>
<td>Vomiting, diarrhea, heartburn, initial ↑ salivary secretions</td>
<td>Dyspepsia, nausea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Stimulation</td>
<td>Initially tremor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>↓ Insulin sensitivity, may aggravate or precipitate diabetes</td>
<td></td>
<td>Blood sugar; HbA1c</td>
<td></td>
</tr>
<tr>
<td>IMMUNO</td>
<td>May be a tumor promoter through angiogenesis, ↑ cell proliferation, and ↓ apoptosis</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

**Induction/Maintenance**
- Pts who are smokers or receiving NRTs may experience resistance to some anesthetic or analgesic agents as a result of increased metabolism from induced hepatic enzymes.
- Nicotine is a sympathomimetic agent and also has effects on autonomic ganglia. Smokers receiving nicotine patches preop have been observed to show exaggerated increases in heart rate after tracheal intubation. NRTs may have hemodynamic effects that may need to be addressed in the periop period.
- While NRTs have been deemed safe in medically stable pts, they may be assoc with worsened outcome in critically ill pts or in pts undergoing cardiopulmonary bypass surgery, esp. off-pump cardiac surgery.

**Postoperative Period**
- Smoking contributes to acute physiologic effects such as increased sympathetic tone, lung inflammation, and tissue hypoxia as well as long-term pathophysiologic changes such as atherosclerosis and COPD, placing these pts at higher risk for postop complications.
- Nicotine withdrawal should be considered as a cause of postop agitation or anxiety.

**Anticipated Problems/Concerns**
- NRTs have proven to be both safe and effective in treating tobacco dependence in medically stable pts, even in those with smoking-related diseases. While more studies are needed, current evidence suggests that NRTs can be valuable tools to manage tobacco dependence in the periop period.
- Use of NRTs in the periop period is far preferable to continued smoking per most experts in the field.

**References**
Nitric Oxide (NO), Inhaled

Uses

- Children: Acute or chronic pulmonary hypertension associated with persistent pulmonary hypertension of newborns (PPHN), meconium aspiration, CHD, and postnatal diaphragmatic hernia
- Adults: Acute or persistent pulmonary hypertension associated with ARDS, pulmon embolism

Perioperative Risks

- Methemoglobinemia (esp. breathing >20 ppm NO)
- NO, and peroxynitrite formation

Worry About

- Methemoglobinemia; measure metHb, esp. for infants, within 6 hr and then every 24 hr
- Measure inhaled NO and NO$_2$ levels continuously
- Do not give if high NO$_2$ levels (>2 ppm)

- Do not allow NO to stagnate in ventilator or breathing circuits; it slowly converts to toxic NO$_2$ gas
- High inhaled NO levels may inhibit platelet aggregation
- In severe heart failure, reducing PVR with NO may raise LAP
- Rebound pulmonary hypertension during NO withdrawal

Overview/Pharmacology

- Inhaled NO activates guanylate cyclase in lung vessels, arterioles, and increased cGMP levels, causing selective pulmonary vasodilation
- Very rapid and avid binding with RBC Hgb inactivates NO and thereby prevents systemic vasodilation
- NO is metabolized to nitrate and nitrite and is excreted in urine

- Supplied as stock gas of ≤10,000 ppm by vol NO in nitrogen or other inert gas
- Inhaled NO is mixed with O$_2$-containing gas immediately before breathing via intratracheal catheter, ventilator, mask, or nasal prongs

Drug Class/Mechanism of Action/Usual Dose

- NO is a free radical with short T$_1/2$ in aqueous solutions (∼1 sec)
- It combines with ferrous-heme ring of guanylate cyclase, and thereby stimulates the conversion of GTP to cGMP; cGMP reduces intracellular Ca$^{2+}$, causing smooth muscle relaxation and modulates other cell functions by regulating gene expression; cGMP is broken down by phosphodiesterases
- Usual inhaled NO dose is 0.1 to 20 ppm by vol

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESP</td>
<td>↓ PVR</td>
<td></td>
<td>↓ PAP↑ CO</td>
</tr>
<tr>
<td>↑ Gas exchange</td>
<td>Skin color</td>
<td>↑ Pao, ↑ Sao, ↓ Paco</td>
<td></td>
</tr>
</tbody>
</table>

Postoperative Period

- Slowly wean from NO over hours if possible watching for abrupt worsening of oxygenation or pulmonary hypertension with the D/C of NO (“rebound” effects)

Anticipated Problems/Concerns

- Beware rapid D/C of inhaled NO$_2$ reactive pulmonary hypertension and hypoxemia, RHF may ensue. These effects may not be seen while reducing NO$_2$ doses, but may be dramatic with D/C of iNO therapy, and can even occur after D/C of NO.
- Do not allow NO stock tanks to deplete
- Provide NO freshly mixed in O$_2$-containing gas for manual ventilation even when briefly disconnecting from ventilator for suctioning or moving pt.
- If inhaled NO does not reverse hypoxemia despite mechanical ventilation with PEEP, high-frequency oscillatory ventilation, etc., ECMO may be required.

Perioperative Implications

- Check for heart failure; do not use in severe heart failure (e.g., PCWP >25 mmHg) or with pulmonary venous disease (e.g., pulm vein stenosis, pulm veno-occlusive disease). Use of inhaled NO in these settings can cause severe pulm edema with hypoxemia and decreased lung compliance. Some pts with mild left heart dysfunction (diastolic dysfunction) may also develop worsening pulmonary edema with iNO.

Monitoring

- Must monitor
  - Inhaled NO, NO$_2$ levels
  - metHb levels
- Consider monitoring
  - PA pressure
  - RV ECHO
  - ABG/s, SpO$_2$

Induction/Maintenance

- Inhale 0.1–20 ppm in ARDS (usual dose: 5–15 ppm) Initiate therapy with a higher dose (usually 20 ppm) in the setting of ARDS with moderate or severe pulmonary hypertension, and lower doses (5–10 ppm) to reduce intrapulmonary shunt (e.g., ARDS).
- In persistent pulmonary hypertension of newborns, begin therapy at 20 ppm, and progressively reduce the dose to 5 ppm or less with improved oxygenation (e.g., FIO$_2$ ≥0.60) and PAP by echocardiogram. Inhaled NO therapy should not be initiated without first optimizing lung volume, ventilation, cardiac performance, and systemic BP.
- Ideal doses need better definition but lower doses are most effective for improving oxygenation by matching ventilation and perfusion, and higher doses to treat pulmonary hypertension. Failure to respond in infants with PPHN may reflect underlying lung developmental abnormalities or structural (anatomic) heart disease.
- Give as little NO as possible to reduce oxidative burden of lung.

Adjuncts

- Phosphodiesterase inhibitors (e.g., sildenafil) increase sensitivity and duration of the dilatory effect of inhaled NO, but must be used with caution as they can cause systemic hypotension.

**Nitroglycerin**

**Uses**
- Rx for pts with angina
- CHF
- In MI, ↓ infarct size
- Prinzmetal’s angina
- Can be given as patch, paste, PO, sublingually prn
- Uterine relaxation
- May be beneficial in reducing postop morphine usage for pain management

**Perioperative Risks**
- Development of hypotension
- Drug rash (rare)

**Worry About**
- Severe hypotension, esp. with regional anesthesia

**Overview/Pharmacology**
- Used for both chronic Rx and acute management
- Prophylactic nitroglycerin not shown to ↓ incidence of intraop MI
- Tolerance to drug from prolonged IV infusion or continuous patch can occur.
- Metabolized by reductive hydrolysis in liver
- Rapidity of onset, duration of action directly related to method of administration
  - SL: Onset 1–2 min, duration <1 hr
  - Oral: Peak effect 60–90 min, duration 3–6 hr
  - Paste: Onset 60 min, duration 4–8 hr
  - Patch: Duration up to 24 hr
- Prolonged use can → tolerance (↓ effectiveness)
- Nitroglycerin paste and/or patch may have uneven absorption intraop

**Drug Class/Mechanism of Action/Usual Dose**
- Organic nitrate
- Activates guanylate cyclase; ↑ cGMP levels in smooth muscle, other tissues; increases NO
- Usual dosage:
  - SL: 0.4 mg prn
  - Paste: 1/2″–1″
  - Isordil: 5–30 mg q 6 h
  - IV: 0.5–2.0 μg/kg/min
- Bolus for uterine relaxation (slow 50 μg; may repeat × 1 with caution if has regional anesthesia actively causing sympathectomy)

**Value Points**

<table>
<thead>
<tr>
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<th>Effect</th>
<th>Assessment by Hx</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Vasodilation of veins &gt; arteries</td>
<td>Relief of angina</td>
<td>BP</td>
<td>PCWP</td>
</tr>
<tr>
<td></td>
<td>Redistribution of coronary blood flow</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Decreased pulm vascular resistance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Uterine (smooth muscle) relaxation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Dilation of meningeal arterial vessels</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Concerns**
- Assess volume status
- Consider monitoring:
  - BP (arterial catheter)
  - PA catheter (may give useful information if nitroglycerin infusion used)

**Induction/Maintenance**
- May interact with other induction agents to cause hypotension
- Ideally should be given IV because of uneven absorption intraop (binding sites on tubing)

**Postoperative Period**
- Pts on chronic nitroglycerin may benefit by resumption of agent
- Can give as patch or paste after rewarming of pt

**Anticipated Problems/Concerns**
- Tolerance to nitroglycerin manifests by ↓ hemodynamic effects; a function of dose, frequency of administration
- Many inhalational agents and opiates have some aspect of hemodynamic effects of nitroglycerin—e.g., venodilation, ↓ O₂ demand

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

**Risks**
- Incidence in USA: 100 million NSAID prescriptions are written per year; 17 million Americans using NSAIDs daily
- 2 subclasses: Nonselective (COX1 and 2) inhibitors and selective COX2 inhibitors (coxibs).
- Have analgesic, anti-inflammatory, and anti-pyretic properties.

**Indications**
- NSAIDs are the first step in the WHO analgesic ladder, typically considered drugs of choice for mild to moderate pain.
- Can be taken OTC or prescribed for chronic somatic pain states (e.g., arthritis) and rheumatologic disorders
- Also given IV, IM, and PO postop as part of a multimodal treatment regimen for acute pain

**Worry About**
- Pt dysfunction
- Renal insufficiency
- Drug interactions
- Allergic reactions

**Overview/Pharmacology**
- Most NSAIDs are weak acids (pKa 3–5) of diverse chemical structure and half-lives.
- Well absorbed from the stomach and intestinal mucosa.
- Highly protein bound (>95%), usually to albumin.
- Work by inhibiting cyclooxygenase, which is a key enzyme in the synthesis pathway of prostaglandins
  - Leads to a ↓ in prostaglandin synthesis, therefore decreasing the inflammatory response as well as the sensitizing effect of prostaglandins on nociceptors (both central and peripheral).
  - Two isoforms of the COX enzyme have been identified.
  - COX-1: Expressed constitutively in most cell types and has an essential role in functions such as gastric protection, platelet aggregation, renal function
  - COX-2: Traditionally considered to be induced by tissue injury/inflammation, now known to be constitutively expressed in some tissues (e.g., brain and/or kidney).
- Undergo liver metabolism to inactive metabolites, which are then excreted by the kidney
- Have a low abuse potential but also possess a ceiling analgesic effect

**Perioperative Implications**

**Preoperative**
- Preop nonselective NSAID use has been associated with increased intraop blood loss due to platelet inhibition.
- Unlike aspirin, NSAID platelet inhibition is reversible, common practice is to hold the NSAID for a period of 5 half-lives before surgery (e.g., ibuprofen 1 d, naproxen 5 d).
- Coxibs do not affect platelet function and therefore do not need to be held.
- NSAIDs displace albumin-bound drugs and can potentiate their effects (e.g., warfarin).

**Regional Anesthesia**
- According to ASRA consensus guidelines, NSAIDs do not significantly increase the risk for spinal hematoma in pts undergoing neuraxial anesthesia.
- The use of NSAIDs alone should not interfere with the performance of neuraxial blocks or the timing of neuraxial catheter removal.

**Intraoperative**
- Intraop administration of NSAIDs has been shown to cause a slight increase in the need for reoperation in surgeries at high risk for postop bleeding (e.g., tonsillectomy/CABG).
  - Decision to administer an NSAID should consider the need for improved analgesia, pts ability to achieve hemostasis, and the risk of postop bleeding inherent to the surgery.
  - May exacerbate asthma, esp. in pts with a Hx of NSAID-induced bronchospasm, angioedema, urticaria, or rhinitis.

**Postoperative**
- NSAIDs can be resumed while cautiously monitoring for GI bleeding/renal dysfunction, avoid resumption in seriously ill pts.
- Risk of adverse effect on renal function the same for both non-selective NSAIDs and COX-2 inhibitors.
  - Use caution when initiating therapy in pts with pre-existing heart/kidney disease, use of loop diuretics, or loss of blood volume > 10%.
  - Both the nonselective NSAIDs and coxibs have been implicated in potentially inhibiting bone healing.
  - May be prudent to avoid in cases where bone formation is esp. crucial (e.g., spinal fusion).

**Drug Class**
- Traditional or nonselective NSAIDs are both COX-1 and COX-2 inhibitors
- Have several different subclasses
  - Salicylate (salts, aminosalicylates, and cholins)
  - Propionic (ibuprofen, ketoprofen, naproxen, fenoprofen)
  - Indole (indomethacin, sulindac, tolmetin)
  - Fenamate (mefenamic, meclofenamate)
  - Mixed (piroxicam, ketorolac, diclofenac)
- Coxibs are selective COX-2 inhibitors.
  - Only celecoxib is commercially available.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
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<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARPO</td>
<td>Hm, HF,</td>
<td>Worsening SOB</td>
<td>BP, edema, rales, chest pain</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Nasal polyps, rhinitis, dyspnea, bronchospasm, angioedema</td>
<td>In asthmatics</td>
<td>Wheezing</td>
<td></td>
</tr>
<tr>
<td>HEPAT</td>
<td>Hepatitis</td>
<td>N/V, anorexia</td>
<td>Jaundice</td>
<td>Stool heme, Hgb, upper endoscopy</td>
</tr>
<tr>
<td>GI</td>
<td>Gastropathy (can be asymptomatic), GI bleeding, esophageal disease, pancreatitis</td>
<td>Hx ulcers, heartburn</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>↑ Bleeding</td>
<td>Easy bruising/bleeding</td>
<td>Pallor</td>
<td>Bleeding time, Hgb</td>
</tr>
<tr>
<td>DERM</td>
<td>Urticaria, erythema multiforme, rash</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Renal insufficiency, sodium/fluid retention, papillary necrosis, interstitial nephritis</td>
<td>BP, edema, wt changes</td>
<td>↑ K+/BUN/Cr, ↓ UO, biopsy</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Headache, aseptic meningitis, hearing disturbances</td>
<td>Cognitive dysfunction, somnolence, confusion</td>
<td></td>
<td>CSF</td>
</tr>
</tbody>
</table>

Nutritional Support

Risk
- Up to 40% of pts may be undernourished on admission to hospital and two-thirds of all pts lose wt during hospital course.

Perioperative Risks of Malnutrition
- Decreased resp, cardiac, skeletal muscle mass, strength
- Decreased visceral protein mass, altered GI mucosal barrier
- Altered humoral, cell-mediated immunity
- Altered neutrophil function
- Increased pulmonary, thromboembolic complications
- Pts with protein-calorie malnutrition have ↑ risk for postop cardiac, noncardiac complications
- Increased risk for nosocomial infections and decreased wound healing
- Increased risk for multiple organ failure
- Increased length of hospital stay

Worry About
- Hypo- or hyperglycemia, depending on additives to TPN
- Decreased ability to secrete insulin in malnourished pts
- Increased free fraction of certain protein-bound drugs with low albumin levels
- B-12 and/or folate deficiency leading to anemia
- Higher rates of infection with TPN
- Excess carbohydrate administration via TPN may lead to increased CO₂ production and increased difficulty in weaning from ventilatory support and hepatic steatosis.
- Excess fat administration via TPN may lead to hyperlipidemia, decreased immune function, and reduced reticuloendothelial function.

Overview
- Nutritional risk index (NRI) = 1.519 × serum albumin (g/L) + [0.417 × (current wt/usual wt) × 100]. (Malnutrition defined as NRI <100; severe malnutrition defined as NRI <83.5.)

Assessment Points

<table>
<thead>
<tr>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>&gt;10% loss of body wt over 6 mo</td>
<td>Hx of renal, hepatic dysfunction, Hx of short gut</td>
<td>Muscle wasting, ↓ Triceps and skinfold thickness, ↓ mid-arm circumference</td>
<td>Alb &lt;2.5 g/dL, Total lymphocyte count &lt;1500 cells/mm³</td>
</tr>
</tbody>
</table>


Preoperative Concerns
- Monitoring
  - Weekly monitoring of wt, electrolytes, magnesium.
  - Weekly monitoring of zinc, liver function tests, PT/PTT.
  - Nutritional variable: Prealbumin and transferrin better indicators of nutritional status due to their shorter half life compared to albumin. Failure to improve or maintain adequate levels usually represents inadequate nutritional support, intercurrent systemic inflammatory response, or advanced organ failure.

Induction/Maintenance
- TPN is usually continued intraop
- Monitor glucose
- Malnutrition may predispose a pt to having a higher risk for pseudocholinesterase deficiency, use succinylcholine with caution.

Adjuvants
- For morbidly obese pts use ideal wt for calculation of TPN requirement.
- For severely underweight pts use 1/2 difference between pt’s ideal wt and actual wt.

Anticipated Problems/Concerns
- Caloric and glucose overload can result in hyperglycemia and hepatic dysfunction.
- Fat overload can result in WBC dysfunction, and infectious complication, and increased CO₂ production.

TPN Composition
- Fluid: 30 mL/kg/d, additional losses
- Calories: 25–30 Kcal/kg/d
- Glucose: 3.0–5.0 g/kg/d
- Fat: 1.0–1.5 g/kg/d
- Protein: 1.5–2.0 g/kg/d
- Additives:
  - Multivitamins in the form of balanced formula should be provided daily.
  - IV formula requires addition of vitamin K, 2 mg/d
  - Trace elements should be given daily to pts with GFR >20 mL/d; magnesium: 15–20 mg/d; zinc 15–20 mg/d. (Requirement for replacement is based on serum level.)

Special Formulas
- Modified amino acid formula is more efficient in restoring positive nitrogen balance, ↓ ureagenesis, and ↑ support of protein synthesis.
Oral Contraceptives

Uses

- Prevention of pregnancy
- Treatment of the following
  - Dysmenorrhea
  - Metrorrhagia/Fe deficiency anemia
  - Acne
  - Endometriosis
  - Functional ovarian cyst
  - Hyperandrogenism and/or polycystic ovarian disease
  - Premenstrual syndrome and/or premenstrual dysphoric disorder
  - Perimenopausal vasomotor symptoms
  - Mittleschmerz

Perioperative Risks

- Hypercoagulability increased risk of venous and arterial thrombosis when given without concomitant aspirin, esp in blood Type A+ women

Worry About

- Thromboembolic events—increased relative risk of 2.7 (without aspirin)

- Hyperkalemia (drospirenone and/or ethinyl estradiol)
- Treatment failure and/or pregnancy. “Typical user” failure rates reported as high as 9%. Preop beta-HCG assay may be indicated in sexually active pts.

Overview/Pharmacology

- Oral preparations of synthetic estrogen, progestin generally well absorbed
- Metabolized by the liver and excreted in urine and feces

DRUG CLASS/MECHANISM OF ACTION/USUAL Dose

- Estrogens
  - Mestrol
  - Ethinyl
  - Estradiol
- Progestins
  - Norethindrone
  - Norgestrel
  - Norethindrone acetate
  - Ethynodiol diacetate
  - Levonorgestrel
  - Norgestimate
  - Desogestrel
  - Drospirenone
  - Prometrium
  - Combination estrogen and progestin drugs inhibit ovulation by negative feedback effect on the hypothalamus; altering normal pattern of gonadotropin secretion by the anterior pituitary; cervical mucus thickens, and is unfavorable to sperm even if ovulation occurs. Classified as
    - Monophasic: Same ratio of progestin and estrogen in each pill
    - Biphasic: Two phases of altered progestin and estrogen ratio
    - Triphasic: Progestin and estrogen ratio varied in three phases
  - First and second generation progestin only drugs have a lower risk of thromboembolism. Third generation progestins carry 6-9-fold increase of VTE similar to the risk during pregnancy if given without concomitant aspirin.

Key References:

Induction/Maintenance

- Consider thromboprophylaxis on an individualized basis judged according to additional genetic and acquired risk factors.

Postoperative Period

- Surveillance for DVT and PE. Restart aspirin.
- Early mobilization, resume agents 2 wk after surgery or mobilization.
Oral Hypoglycemics

Jyotsna Rimal

DRUGS

Uses
- Oral hypoglycemics are used for Type II DM or non-insulin dependent diabetes mellitus (NIDDM) not controlled by diet or wt loss where cells are resistant to secreted insulin.
- Main classes of agents:
  - Insulin secretagogues: Sulfonylureas, meglinitides, dipeptidyl peptidase-4 inhibitors (DPP-4)
  - Insulin sensitizers: Biguanides and thiazolidinediones
- Others: Alpha–glucosidase inhibitors
- Diabetes is the leading cause of blindness, kidney failure, and non-traumatic amputations, and a leading cause of arterial diseases such as stroke, memory loss, impotence, and myocardial infarctions.
- Prevalence of diagnosed and undiagnosed diabetes in the USA, all ages, 2007 was 23.6 million; 17.9 million are diagnosed and 5.7 million are undiagnosed. Expected to increase to 60+ million by 2030.
- Total diabetic pts in USA of which 50.6% only on oral hypoglycemics. Estimated diabetes costs in the USA in 2007 total: $174 billion (direct and indirect).

Perioperative Risks
- Sulfonylureas: Hypoglycemia, N/V, cholestatic jaundice, allergic skin reaction, agranulocytosis, aplastic anemia (pt with G6PD deficiency), hemolytic anemia, alcohol-induced flushing with chlorpropamide, SIADH with chlorpropamide and tolbutamide, potentially teratogenic effects
- Meglitinides: Hypoglycemia
- Dipeptidyl peptidase-4 inhibitors (DPP-4): Hypoglycemia
- Biguanides: Metformin: lactic acidosis, nausea and/or vomiting, diarrhea, anorexia, metallic taste
- Thiazolidinediones: Edema, hepatitis
- Alpha-glucosidase inhibitors: Diarrhea, flatulence

Worry About
- Sulfonylureas: may be potentiated by the following agents: NSAIDs, warfarin, salicylates, azole antifungal drugs, MAO blockers, blockers, sulfonamides, phenylbutazone, ethanol, probenecid, chloramphenicol
- Meglitinides: Azole antifungals, clarithromycin, gemfibrozil causes increase in plasma concentration of meglitinides (Repaglinide–Prandin) causing hypoglycemia.
- Metformin: Cimetidine causes an increase in the plasma concentration of metformin, decreased absorption of vitamin B12, and folate.
- Thiazolidinediones: Hepatitis, water retention in pts with NYHA Grade III and IV heart failure.
- Alpha-glucosidase inhibitors: An episode of hypoglycemia should be treated with monosaccharides-glucose tablets, since the drug prevents the absorption of polysaccharides.

Overview/Pharmacology
- Sulfonylureas
  - First generation: Tolbutamide, acetohexamidine, tolazamide, chlorpropamide
  - Second generation: Glibenclamide (glyburide), glipizide
  - Third generation: Glimeperide (amaryl)
  - Clearance: Metabolized by the liver and excreted in the urine
- Meglitinides: Repaglinide (prandin), nateglinide (Starlix)
  - Clearance: Metabolized by the liver and excreted in the urine
- DPP-4 inhibitors: Sitagliptin (Januvia), saxagliptin (Onglyza)
  - Clearance: Metabolized by the liver and excreted in the urine (80%)
- Biguanides: Metformin, glucophage, rionet, fortamet, glumetza
  - Clearance: The drug does not bind to plasma proteins and is excreted unchanged in the urine.
- Thiazolidinediones: Rosiglitazone (Avanda), pioglitazone (Actos)

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Water retention</td>
<td>SOB</td>
<td>Edema</td>
<td>CXR</td>
</tr>
<tr>
<td>Thiazolidinediones</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Lactic acidosis</td>
<td>Tiredness, dysrhythmia</td>
<td>Tachypnea</td>
<td>Lactate</td>
</tr>
<tr>
<td>Metformin</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Hypoglycemia</td>
<td>Altered mental status, convulsion, coma</td>
<td>Sweating, tachycardia</td>
<td>Blood glucose</td>
</tr>
<tr>
<td>Sulfonylureas</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meglitinides</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPP-4 inhibitors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Concerns
- Generally withhold sulfonylureas, meglitinides and dipeptidyl peptidase-4 inhibitors on the day of surgery to avoid hypoglycemia.
- Hold metformin on the day of surgery because complications or alterations in renal function arising intraop may potentiate the risk of development of lactic acidosis (very rare risk).
- Type II diabetics usually not prone to ketoacidosis.

- Blood sugar measurement required before surgery to control hyperglycemia with regular insulin and dextrose solution.
  - Induction/Maintenance
    - Hypoglycemia should be treated with IV dextrose solution.
    - For uncontrolled intraop hyperglycemia use insulin drip with frequent blood sugar measurements.

Postoperative Period
- Avoid hypoglycemia

- Blood sugar measurements required frequently, use regular insulin to control hyperglycemia as needed until pt able to resume oral agents.

Anticipated Problems/Concerns
- Sulfonylureas, meglitinides, and dipeptidyl peptidase may cause significant hypoglycemia if given to NPO status pt.
- Metformin may increase the risk of lactic acidosis.
- Sulfonylureas may be potentiated by various other drugs as listed above.
Penicillins

**Uses**
- Prescribed for pts with infections by sensitive organisms, primarily *Pneumococci* and those in genera *Streplococci*, *Staphylococci*, *Neisseria, Pseudomonas, Proteus, Haemophilus*, etc., used as prophylaxis for subacute bacterial endocarditis (penicillin G benzathine)
- Can be administered PO, IM as regular or slow-release repository form, or IV

**Worry About**
- Hypersensitivity reactions (0.7–4%): rash, fever, bronchospasm, vasculitis, serum sickness, exfoliative dermatitis, Stevens-Johnson syndrome, angioedema, anaphylaxis
- Hyperkalemia when penicillin G potassium is administered IV (1.7 mEq K+/1 × 10⁶ units penicillin G), esp. if administered rapidly
- Plt dysfunction, defective hemostasis after ticarcillin, and penicillin G
- Rare bone marrow depression, granulocytopenia, hepatitis
- Headaches, seizures after 1 dose of 5 MU of penicillin G procaine
- Clearance lower in neonates and infants
- After PO ingestion, nausea and diarrhea, rarely *Clostridium difficile* pseudo-membranous colitis

**Overview/Pharmacology**
- Used to treat wide spectrum of infectious diseases
- Many penicillins are acid-labile (pH2 destroys antibiotic), often not administered orally
- Actively and rapidly excreted by renal tubule
- T₁/₂ of penicillin markedly increased in anuria
- Dosage should be decreased in renal failure
- Other organic acids, e.g., probenecid, can compete at the renal tubule for excretion, prolonging the T₁/₂ of the antibiotic
- High concentration in urine
- Ampicillin and amoxicillin often administered with β-lactamase inhibitors such as clavulanate and sulbactam

**Drug Class/Mechanism of Action/Usual Dose**
- Penicillins are organic acids consisting of a β-lactam ring to which is attached a side chain and a thiazolidine ring; they inhibit bacterial cell wall synthesis primarily by inhibiting the transpeptidase reaction that is essential for bacterial cell wall synthesis
- Dose and route of administration depend on penicillin used and severity of disease treated

### DRUG EFFECTS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Absorption After Oral Dose</th>
<th>Resistance to Penicillinase</th>
<th>Dose IV</th>
<th>Antimicrobial Spectrum</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin G</td>
<td>Poor; −1/3 of dose; take on empty stomach</td>
<td>No</td>
<td>1–10 MU q 4–6 h</td>
<td><em>Streptococcus, Neisseria</em></td>
<td>↑ K⁺ (1.7 mEq K⁺/1 × 10⁶ units Pen G); &gt;20 1 × 10⁶ U/day can cause seizures; inhibits plt aggregation</td>
</tr>
<tr>
<td>Penicillin V</td>
<td>Moderate 2–5; Pen G</td>
<td>No</td>
<td>0.5 gm q 6 h PO</td>
<td>Like Pen G</td>
<td>K⁺ salt</td>
</tr>
<tr>
<td>Dicloxacillin</td>
<td>Good (30–80% of dose on empty stomach)</td>
<td>Yes</td>
<td>0.5–1 gm PO q6h</td>
<td><em>Staphylococcus aureus</em></td>
<td>90–95% bound to albumin; not removed by dialysis</td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>Good; take on empty stomach</td>
<td>No</td>
<td>1–2 g q6h (250–500 mg q 6 h PO)</td>
<td><em>gm + coci, gm neg, H. influenza, E coli, P. mirabilis</em></td>
<td></td>
</tr>
<tr>
<td>Ticarcillin</td>
<td>Poor</td>
<td>No</td>
<td>50–75 mg/kg q6h</td>
<td><em>Pseudomonas, Enterobacter, Proteus (indole +)</em></td>
<td>CHF 2° to Na⁺ overload; 5 mEq Na⁺/g; low K⁺ 2° to obligatory cation excretion with anion, ↓ in plt aggregation</td>
</tr>
<tr>
<td>Piperacillin</td>
<td>Poor</td>
<td>No</td>
<td>2–6 g q8h</td>
<td><em>P. aeruginosa, Enterobacter, some Klebsiella, other gm negatives, gm + coci, L. monocytogenes</em></td>
<td>Same as ticarcillin: 2 mEq Na⁺/g</td>
</tr>
<tr>
<td>Mezlocillin</td>
<td>Poor</td>
<td>No</td>
<td>1.5–4.5 g q6h</td>
<td><em>Pseudomonas, Enterobacter, Klebsiella sp.</em></td>
<td>2 mEq Na⁺/g</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Concerns**
- Is pt allergic to any penicillins? What exactly happens when the drug is taken (rash vs. anaphylaxis)?
- If pt on large doses of penicillin G, ticarcillin, or piperacillin, are serum electrolytes normal?

- Hemostasis, esp. plt aggregation, may be inhibited by the antibiotics.
- If plt has renal insufficiency or failure, dose of antibiotic should be q12h or less frequently.

**Induction/Maintenance/Postoperative Period**
- Penicillins should have no effect on induction or maintenance unless allergic reaction occurs; no known interactions with any anesthetic agents.

**Anticipated Problems/Concerns**
- Relate to administration of large amounts of Na⁺, K⁺, and organic anions (acids). Possible bleeding problems due to plt dysfunction.

Phencyclidine (PCP)

Uses
- DEA Schedule I drug of abuse with no present medical indications
- Street terms for phencyclidine: Angel dust, supergrass, killer weed, embalming fluid, rocket fuel, wack, ozone
- Common routes of administration: Smoking (often laced in marijuana cigarettes), oral ingestion; less common is IV injection.

Risk
- Psychosis, seizures, anticholinergic type syndrome
- Experimentally PCP causes irreversible brain damage, per excitotoxicity, with the typical bull’s eye neuronal cell and vacualization

Perioperative Risks
- Aggressive and/or psychotic behavior, Htn, stroke, hyperthermia, rhabdomyolysis, aspiration
- Kidney failure, aspiration, malignant Htn, prolonged action

Worry About
- With moderate-high doses elevated HR and BP, hypersalivation, sweating, fever, repetitive movements, muscle rigidity on stimulation
- With high doses, anesthesia, stupor, coma, convulsions can occur.

Overview/Pharmacology
- Effects due to parent compound, highly lipid soluble, pK of 8.6, peak effects 15 min when smoked and 2 hr by ingestion, distribution 4 hr, elimination up to 48 hr. Metabolites are active and are described in elimination time up to weeks in chronic users.
- Metabolized in the liver; urinary excretion of metabolites at low doses, excretion of free drug at high doses, only small fraction of the drug excreted unchanged.
- Produces an acute state of intoxication lasting 4–6 hr but may produce a chronic state of psychosis that can last for up to several days. With low-moderate doses, acute intoxication incl staggering gait, slurred speech, nystagmus, numbness of extremities, sweating, catatonic muscular rigidity, blank stare, changes in body image, disorganized thought, drowsiness, apathy, anterograde amnesia, possibly aggressive behavior
- Arylcyclohexylamine
- Acts at the N-methyl-D-aspartate receptor as a noncompetitive antagonist, but weak dopamine, serotonin, and noradrenergic agonist.
- Experimentally PCP-induced desphorylation of ERK ½ and Akt and dephosphorylation of GSK3-beta (activation) that is prevented by lithium.

Davide Cattano

ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE | Test
--- | --- | --- | --- | ---
CARDIO | Tachycardia, Htn | Quantification, chronicity, acuity of drug exposure | Vital signs Diaphoresis | Blood, urine toxicity screens
RESP | Tachypnea vs. depression | Concurrent drug exposure (e.g., alcohol) | Resp rate Sat O₂% | Observation, temp
CNS | Psychosis, coma, convulsions, analgesia | Pupils, speech, reflexes | |
ANS | Hypersalivation vs. dry mouth, hyperthermia | |


Induction/Maintenance
- Ketamine contraindicated (cross-tolerance) unless used cautiously to treat addiction and/or withdrawal symptoms (rare, escalating doses); probably avoid NO and isoflurane.
- Not selective beta blockers with alpha effects (labetalol)
- Clonidine and dexmedetomidine

Postoperative Period
- Psychosis (acute vs. withdrawal), rhabdomyolysis, anticholinergic syndrome
- Precaution with muscle relaxant reversal

Anticipated Problems/Concerns
- Phenothiazines, anticholinergics, acidification of urine
- There is no withdrawal, but addiction tolerance is common.
Phenothiazines

**Uses**
- Phenothiazines share a tricyclic core functional group and have multiple clinical applications.
- Phenothiazine neuroleptics such as chlorpromazine are used in the treatment of schizophrenia and psychosis.
- Phenothiazine antihistamines such as promethazine and prochlorperazine are highly effective antihistametics.
- Chlorpromazine can effectively treat uncontrollable hiccups and acute migraine headaches.

**Perioperative Risks**
- Severe extrapyramidal symptoms may arise from phenothiazines' antidopaminergic activity.
- Tardive dyskinesia may result from long-term use and may be irreversible.
- Contraindicated in Parkinson's disease; may worsen tremor and Parkinsonism.
- Autonomic dysfunction may result from phenothiazines' sympathetic and anti-cholinergic effects.

**Pharmacokinetics/Pharmacodynamics**
- Phenothiazines undergo hepatic metabolism; use caution in pts with hepatic dysfunction.
- Inactive metabolites excreted in bile/urine; pharmacokinetics rarely affected by renal failure.
- Phenothiazines are highly protein-bound (>90%).
- Prochlorperazine is used at doses of 2.5–10 mg IV/IM/PO q4–6h (max 40 mg/d), 25 mg PR q12h

**ASSESSMENT POINTS**

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<tr>
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</thead>
<tbody>
<tr>
<td>ANS</td>
<td>α₁ Adrenoceptor blockade, antimuscarinic action</td>
<td>Orthostatic hypotension, tachycardia</td>
<td>Orthostatic</td>
<td>Orthostatics</td>
</tr>
<tr>
<td>CNS</td>
<td>Extrapyramidal symptoms</td>
<td>Acute or chronic</td>
<td>Tachycardia, bradycardia</td>
<td>EEG</td>
</tr>
<tr>
<td></td>
<td>Neuroleptic malignant syndrome</td>
<td>Muscle cramps, delirium</td>
<td>Rigidity, tachycardia, lethargy, delirium</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sedation</td>
<td>Decreased seizure threshold</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIAC</td>
<td>Conduction defects: Long QT, ventricular and supraventricular arrhythmias</td>
<td>Can cause sudden death</td>
<td></td>
<td>EKG</td>
</tr>
<tr>
<td>PULM</td>
<td>Resp depression</td>
<td>May potentiate opioids</td>
<td>Low resp rate</td>
<td>SpO₂, ABG, ETCO,</td>
</tr>
<tr>
<td>VASC</td>
<td>Tissue necrosis (promethazine)</td>
<td>Arterial injection or tissue extravasation</td>
<td>Gangrene</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Leukopenia, agranulocytosis</td>
<td>Usually in chronic dosing only</td>
<td></td>
<td>CBC</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Concerns**
- Obtain baseline EKG for all pts taking chronic phenothiazines to assess cardiac conduction and QT interval.
- Neuroleptic malignant syndrome may present in pts undergoing chronic treatment, but may be precipitated by other antidopaminergic agents such as metoclopramide.

**Induction/Maintenance**
- Autonomic insufficiency may contribute to profound intraop hypotension.

**Adjuvants/Regional Anesthesia/Reversal**
- Treat extrapyramidal side effects with diphenhydramine, benztropine, or benzdiazepines.
- Neuroleptic malignant syndrome is treated with bromocriptine, dantrolene, and aggressive hydration/monitoring.

**Postoperative Concerns**
- Clinically significant resp depression if given to pts <2 yo or those with pulm disease, esp. if combined with opioids.
- Arrhythmias and prolonged QT.
- Increased risk of sedation and delirium in elderly pts.

**Drug Class/Mechanism of Action/Usual Dose**
- Phenothiazines antagonize many receptors, incl dopamine receptors (primarily D₂), muscarinic receptors, serotonin receptors, α₁ adrenergic receptors and H₁ histamine receptors.
- Phenothiazine neuroleptics mediate their antipsychotic effects by blocking mesolimbic D₂ receptors, but D₁ blockade in striatum causes extra-pyramidal side effects.
- Phenothiazine anti-emetics are potent H₁ histamine antagonists and exert antimuscarinic and anti-D₂ dopaminergic activity at the chemoreceptor trigger zone.
- Promethazine is used at doses of 6.25–25 mg IV/IM q4–6h, IV doses given in diluted form slowy into well-functioning large-vein IV.
- Prochlorperazine is used at doses of 2.5–10 mg IV/IM/PO q4–6h (max 40 mg/d), 25 mg PR q12h.

**Drug Interactions**
- Increased risk of extrapyramidal symptoms if given with other antidopaminergic medications (typically metoclopramide).
- May increase concentration of other hepatically metabolized (CYP2D6) drugs (some beta-blockers and tricyclic antidepressants).

**Anticipated Problems/Concerns**
- Assess mental status, resp status, and CV function after administration, particularly in early postop period.

Phenoxybenzamine

**Uses**
- Incidence in USA: 3,600/y
- Rx for preop pheochromocytoma; occasionally, chronic Rx of pheochromocytoma, sympathetic hyperactivity states, carcinoid syndrome, benign prostatic hypertrophy (BPH)

**Perioperative Risks**
- Drug interactions: Sometimes requires very high doses of \(\alpha\)-adrenergic agents to produce vasoconstriction
- Vasodilation, orthostatic hypotension accentuated in hypovolemic pts

**Overview/Pharmacology**
- \(\alpha\)-Blocker (relatively selective \(\alpha_1 \gg \alpha_2\)) by covalent (irreversible) binding to a receptor; compensatory response calls for production or availability of more (spare) receptors
- Effect develops slowly; peak effect not attained for 2 hr after IV or 4 hr after oral administration
- Absorption from GI tract incomplete
- Renal excretion of 50% in 12 hr, 80% in 24 hr
- \(T_{1/2}\) of effect over 24 hr, effects accumulate for at least 4–6 d
- High lipid solubility at body pH

**Induction/Maintenance**
- Can produce ↑ sedation, ↓ anesthetic requirements by ⅓ (not studied but anecdotally reported)

**Muscle Relaxants**
- No interactions known

**Regional Anesthesia/Reversal**
- No interactions known

**Anticipated Problems/Concerns**
- May need very high doses of vasopressors to ↑ vascular resistance, BP in pt taking large doses
- CNS dysfunction by itself

**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Vasodilation of mucous membranes of nasopharynx, miosis</td>
<td>Nasal congestion</td>
<td>Mouth breathing</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Antihypertensive agent Postural hypotension, reflex tachycardia ↑ CO</td>
<td>Orthostatic dizziness</td>
<td>Orthostatic VS</td>
<td>Hct ECG</td>
</tr>
<tr>
<td>GI</td>
<td>↑ Intestinal motility, causes diarrhea</td>
<td>Orthostatic hypotension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Stimulates insulin release ↑ Presynaptic norepinephrine release (blockade of presynaptic (\alpha_2) receptors inhibiting release of norepinephrine)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>↑ Blood volume, Na(^+) retention inhibits contraction of vas deferens Impairs ejaculation</td>
<td>CNS exam</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Depression, sedation, fatigue Extrapyramidal symptoms rarely N/V; motor excitability rare</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Phenyleylephrine (Neo-Synephrine)

Uses
- Prescribed mainly as nasal decongestant or ophthalmically for mydriasis Rx, capillary decongestion
- Reliable vasopressor in treatment of hypotension
- Prolongs local anesthetic duration in regional anesthesia
- Available as parenteral IM/IV and various ophthalmic and/or nasal preparations

Perioperative Implications

Preoperative Concerns
- Assess LV function and Hx of CAD
- Consider arterial catheter if phenylephrine infusion anticipated (carotid endarterectomy, relative hypovolemia)
- Assess renal function (Cr)
- For nasal intubations, phenylephrine can be used as a nasal vasoconstrictor in a mixture with 3–4% lidocaine

Induction/Maintenance
- Monitor ECG for signs of ischemia due to increased ventricular work or coronary artery spasm
- May ↓ hepatic blood flow due to α-adrenergic-medicated vasoconstriction of portal venous vasculature

Perioperative Risks
- Risk of Htn increases left heart work; may precipitate myocardial ischemia, MI
- Infusions to augment systolic BP ↑ incidence of myocardial ischemia in pts undergoing carotid endarterectomy
- Increased pulm vascular resistance, right heart work
- Bradycardia may occur (usually not severe) related to baroreceptor reflex
- Decreased renal, splanchnic blood flow
- May ↑ uterine artery vascular resistance, ↓ uterine artery blood flow in pregnant pts
- Systemic absorption of topical preparations may cause Htn, headache, tremulousness, myopathy
- α-adrenergic-agonist activity causes systemic and PA vasoconstriction, resulting in ↑ impedance to forward flow, ↑ BP
- Rapidly metabolized by MAO
- IV duration less than 5 min
- May terminate supraventricular tachycardia by vagal reflex from baroreceptor stimulation
- Increased SVR during CPB
- Increased perfusion pressure to vital organs in hypovolemic pts until vol restored, CPR
- May be used in conjunction with nitroglycerin to elevate coronary perfusion pressure in hypotensive pts with myocardial ischemia
- Decreasing right to left shunts in pts with cyanotic spells (tetralogy of Fallot)
- Vasopressor of choice in hypertrophic cardiomyopathy, systolic anterior motion of mitral valve and aortic stenosis, when ↓ inotropy or tachycardia undesirable

Worry About
- Increase preload, afterload may worsen LV failure in pts with LV dysfunction
- Raised PA pressures may worsen RV dysfunction
- May ↓ renal blood flow

Overview/Pharmacology
- Direct α1-agonist activity causes systemic and PA vasoconstriction, resulting in ↑ impedance to forward flow, ↑ BP
- Rapidly metabolized by MAO
- IV infusion of 20–200 μg/min/70 kg
- Ophthalmic solutions: 2.5–10%
- Supraventricular tachycardia dose: 150–800 μg titrated to ↑ BP

Overview/Pharmacology
- IV bolus: 50–100 μg
- IV infusion: 20–200 μg/min/70 kg
- Ophthalmic solutions: 2.5–10%
- Supraventricular tachycardia dose: 150–800 μg titrated to ↑ BP

Adjuvants/Regional Anesthesia/Reversal
- Can be used when severe hypotension presents immediate danger to compromised myocardium or other end organ (e.g., brain)
- With a failing heart, increasing afterload and preload may ↓ left-sided filling pressures enough to precipitate pulm edema
- Advantageous in catecholamine-depleted pts (chronic cocaine or amphetamine abuse), or in pts on tricyclic antidepressants or MAO inhibitors, when indirect vasopressors are unpredictable


ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Mydriasis without cycloplegia</td>
<td>PE</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Production of aqueous humor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Vasoconstriction of veins and arteries</td>
<td>BP</td>
<td>PCWP</td>
</tr>
<tr>
<td></td>
<td>↑ Systolic and diastolic BP</td>
<td>HR</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>↑ PVR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>↓ Renal blood flow</td>
<td>UO</td>
<td>BUN, Cr</td>
</tr>
</tbody>
</table>

**DRUGS**

**Worry About**
- Increase preload, afterload may worsen LV failure in pts with LV dysfunction
- Raised PA pressures may worsen RV dysfunction
- May ↓ renal blood flow

**Overview/Pharmacology**
- Direct α1-agonist activity causes systemic and PA vasoconstriction, resulting in ↑ impedance to forward flow, ↑ BP
- Rapidly metabolized by MAO
- IV infusion of 20–200 μg/min/70 kg
- Ophthalmic solutions: 2.5–10%
- Supraventricular tachycardia dose: 150–800 μg titrated to ↑ BP

Phenytoin

**Indications**

- Management of generalized tonic-clonic (grand mal) and complex partial seizures
- Prophylaxis against seizures after trauma or surgical intervention
- Treatment of ventricular arrhythmias, esp. those assoc with digitalis or tricyclic antidepressant toxicity
- Treatment of prolonged QT interval
- Treatment of epidermolysis bullosa and chronic pain syndromes

**Overview/Pharmacology**

- Drug of choice for status epilepticus
- Treatment for acute and chronic seizures
- Onset of action: 30–60 min
- Protein binding >90% in adults
- Elimination half-life: 22 hr
- 95% hydroxylated and conjugated in liver with glucuronic acid for renal excretion
- Therapeutic range: 10–20 mcg/mL

**Drug Class/Mechanism of Action/Usual Dose**

- Hydantoin derivative
- In the CNS, helps to limit nerve impulse generation thereby limiting seizure focus spread by:
  - Decreasing influx of Na⁺ ions across cell membranes in the motor cortex
  - Decreasing presynaptic Ca²⁺ release
  - Decreasing extracellular K⁺ concentration
- In the heart works to limit re-entrant arrhythmias by:
  - Prolonging the effective refractory period and suppressing ventricular pacemaker automaticity
  - Shortening action potential for status epilepticus (IV and PO dosages are the same)
- Pediatric: Loading dose—15–20 mg/kg in single or divided doses, then 5 mg/kg/d in divided doses
- Adult: Loading dose—10–15 mg/kg, then 5–6 mg/kg/d in three divided doses

**Perioperative Implications**

**Preoperative Concerns**

- Pts with renal, liver disease or decreased nutritional states can increase level of free phenytoin

**Induction/Maintenance**

- Rapid administration of phenytoin may cause hypotension, bradycardia, arrhythmias. Administer at rate of less than 50 mg/kg/min
- Larger doses of nondepolarizing muscle relaxants may be required
- Shorter duration of nondepolarizing muscle relaxants

**Contraindications**

- For treatment of cardiac arrhythmias: 1.5 mg/kg IV every 5 min for maximum dose of 15 mg/kg or 1.5 g

**Perioperative Risks**

- Hypotension, bradycardia, cardiac arrhythmias and/or collapse with rapid IV administration (likely due to propylene glycol vehicle)
- Venous irritation and/or pain
- Decreased efficacy of muscle relaxants

**Concerns**

- Pts with renal failure, jaundice, or other causes of hypoalbuminemia may exhibit phenytoin toxicity
- Increased P450 clearance may cause decreased effectiveness of certain drugs: Antibiotics, oral contraceptives, procainamide, and oral anticoagulants

**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Nystagmus at toxic levels, &gt; 20mg/mL</td>
<td>Gingival Hyperplasia with chronic use</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Hypotension, bradycardia, cardiac arrhythmias with rapid administration</td>
<td>Vital signs, monitoring</td>
<td></td>
</tr>
<tr>
<td>PULM</td>
<td>Resp depression</td>
<td>Saturation, resp rate monitoring</td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Rash; Stevens-Johnsons syndrome (rare)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI/LIVER</td>
<td>Constipation, vomiting, nausea, hepatitis: increased hepatic drug metabolism; toxicity in low albumin states; avoid or limit Ethyl alcohol use</td>
<td>GI irritation if not taken with food</td>
<td>Albumin</td>
</tr>
<tr>
<td>HEME</td>
<td>Folic acid depletion, hyperglycemia, leukopenia, thrombocytopenia, agranulocytosis</td>
<td>CBC with differential</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Toxicity in uremic pts</td>
<td>BUN/Cr</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Ataxia, diplopia, drowsiness, lethargy, coma, nystagmus, mood changes</td>
<td>CNS exam</td>
<td></td>
</tr>
</tbody>
</table>

**Key Reference:** Phenytoin in University of Maryland Medical Center Medical Encyclopedia. 2009-downloadable at [www.umm.edu/ency/article/002632.htm](http://www.umm.edu/ency/article/002632.htm).
Physostigmine, Eserine

Uses
- Central anticholinergia; for diagnosis and treatment. Hx of anticholinergic ingestion and/or exposure: (atropine, scopoline, belladonna, jimson weed, toxic mushrooms, tricyclics, phe-nothiazines, antihistamines, benzodiazepines, opiates, inhalation anesthetics, propofol, GHB)
- “Blind as a bat”: Mydriasis and loss of accommodation
- “Dry as a bone”: Urinary retention and dry mucus membranes
- “Hot as a hare”: Hyperthermia from loss of sweating
- “Red as a beet”: Cutaneous vasodilitation to counteract hyperthermia
- “Mad as a hatter”: Fluctuating consciousness, delirium, disorientation, hallucinations, phantom behaviors, coma, paraesthesia during recovery
- Postop delirium, from agitation to excessive somnolence
- Glaucoma, ciliary muscle contraction = miosis = facilitates outflow of aqueous humor
- Treatment for antimuscarinic xenobiotic toxicity (a chemical compound that is foreign to a living organism, e.g., benzodiazepines, tricyclics, antihistamines, jimson weed)
- NMB reversal (neostigmine better choice as it avoids central CNS effects)
- Hereditary ataxies
- Alzheimer’s disease (may improve STM but not used clinically)
- Analgesia (decreases morphine consumption postop)

Perioperative Risk
- Cholinesterase inhibition = Excess acetylcholine. Can lead to 3 sets of problems: Analogous to organophosphorous compound poisoning, a cholinergic crisis (basically the opposite from anticholinergic syndrome above)
- Muscarinic cholinergic (parasympathetic over-stimulation (DUMBELS)
  D = Defecation, diarrhea, diaphoresis, GI distress
  U = Uremia
  M = Miosis
  B = Bronchorrhea, bronchospasm, stridor
  E = Emesis (nausea)
  S = Salivation
- nicotinic cholinergic excess (continuing depolarization of motor endplate leading to fasciculations at low dose and progressive weakness at high dose)
- CNS (anxiety, confusion, tremors, seizures, resp depression, coma)

Worry About
- Reactive airway disease
- Peripheral vascular disease
- Diabetes
- Bowel or bladder obstruction
- Pre-existing intraventricular conduction delay, long QT
- Pre-existing AV block
- Pregnancy (Class C)
- Sulfite allergy (contains sodium bisulfite preservative)

Overview/Pharmacology
- Physostigmine is a parasympathomimetic carbamate derived from the beans and/or seeds of an aquatic leguminous plant (calabar or ordeal bean). Used in the Old Calabar region of Nigeria as part of the Esere witchcraft ritual (believed to test the guilt or innocence of a person accused of a crime)

Drug Class/Mechanism of Action/Usual Dose
- Physostigmine is a tertiary amine and a competing substrate for cholinesterase enzymes, thus decreasing the breakdown of acetylcholine.
- 1.5 mg given over 60 min = Vd 2.4+/– L/kg; t1/2 16.4+/– 3.2 min; peak plasma conc 3+/–0.5 mg/mL; clearance 0.1 L/min/kg.
- Inhibition of plasma cholinesterase within 2 min of infusion start. Half-life of plasma cholinesterase inhibition = 83.7+/–5.2 min.
- For glaucoma: Not commonly used due to systemic absorption and side effects. Replaced by other agents. Physostigmine ointment placed 1–3 times daily. Physostigmine solution 0.25%, 0.5% used 1–4 times daily while holding pressure over medial canthus/tear duct to minimize absorption.
- For reversal of central anticholinergia: Physostigmine salicylate (antilirium) 1 mg/mL; dose = 0.04 mg/kg or 1–2 mg IV/IM. IV given slowly, no more that 1 mg/min every 20–60 min as effective and necessary, or until side effects develop.

Perioperative Implications

Preoperative Concerns
- Scopolamine, antihistamines, benzodiazepines (esp. in elderly) Can contribute to central anticholinergia.
- Use of jimson weed (belladonna) or hallucinogenic mushrooms
- Interaction with vasopressors (possible Htn tachycardia).
- Long QT or AVB (increases chance for asystole).
- Tricyclic antidepressant use (asystole has occurred in treatment of tricyclic overdose).

Induction/Maintenance
- Not used

Postoperative Period
- Many if not all anesthetics can cause anticholinergic signs and symptoms.
- Differential diagnosis: Metabolic (hyper/hypo-glycemia, electrolyte imbalance, sepsis, MH, NMS); respiratory (hypo, hypercarbia); neuro (CVA, seizures); psychiatric (narcolepsy, psychosis); iatrogenic (residual NMB, bladder distention, prolonged anesthetic effects/sensitivity).

Anticipated Problems/Concerns
- Physostigmine can be very effective in reversing excessive sedation or agitation assoc with anticholinergia. However, anticholinergia is usually self-limited and is a diagnosis of exclusion; although confirmed by a positive response to physostigmine. Also, physostigmine side effects are unpredictable and can be severe (asystole, seizures). In general, they are limited to exaggerated parasympathetic effects: N/V, stomach pain, salivation, urination, defecation, miosis, inability to focus, lacrimation, sweating, bronchospasm, bronchorrhea, dyspnea, bradycardia or tachycardia, hypertension or Htn, irregular pulse, muscular twitching; but weakness, seizures, collapse, coma, pulm edema, death (i.e., cholinergic crisis) can occur.
- Avoid in pts receiving other cholinergic agents (methacholine, bethanol)
- Avoid in pts receiving depolarizing NMBs (succinylcholine)
- Atropine is the antidote for physostigmine overdose and central cholinergic symptoms.
- Glycopyrrolate is the antidote for peripheral cholinergic excess.
Prilocaine (Citanest)

**Indications**
- Infrequently used local anesthetic in USA, still used extensively in Germany
- Administered either as an injection local anesthetic 4% (with or without epinephrine) or topically as EMLA, a eutectic mixture of 2.5% prilocaine and 2.5% lidocaine
- Widely used for anesthesia and analgesia during circumcisions. Newborns at higher risk for toxicity and methemoglobinemia
- Percutaneous anesthesia with EMLA for venipuncture, ulcer debridement, and skin graft harvesting
- Liposuction
- Painless treatment of hydrocele

**Perioperative Risks**
- Toxicity from excessive dose
- Hypersensitivity reaction
- Methemoglobinemia

**Worry About**
- Metabolism to o-toluidine, which causes Hgb to be reduced to methemoglobin

**Overview/Pharmacology**
- 2-Propylamino-o-propionotoluidide
- Pharmacokinetics: \( T_{1/2} \alpha \approx 0.5 \) min; \( T_{\beta} \approx 5 \) min, \( V_d \), 261 L; \( T_{\gamma} \approx 1.5 \) hr, clearance rate 2.84 L/min
- (distributed at rapid rate from blood to tissue)

**Drug Class/Mechanism of Action/Usual Dose**
- Intermediate-acting amide local anesthetic (less readily metabolized than esters); this ↓ in metabolism ↑ risk of adverse reactions

**Drug Effects**
- Addition of epinephrine does not affect block duration, a result of vasodilating action of prilocaine
- Contraindicated in pts with G6PD deficiency
- Its increased volume of distribution reduces its CNS toxicity, making it suitable for IV regional blocks.
- It must not (DO NOT!) be used on mucous membranes or abraded skin, as rapid absorption may result in systemic toxicity.

**Toxicity**
- **Methemoglobinemia**
  - Dose-response relationship exists between amount of prilocaine and methemoglobinemia (occurs with \( \geq 600 \) mg, \( >8 \) mg/kg). Occurrence related to chemical structure: Prilocaine has one less methyl group in benzene ring than lidocaine; metabolism in liver results in formation of o-toluidine, which oxidizes Hgb to methemoglobin.
  - In healthy persons, methemoglobinemia usually is not a problem.
  - Methemoglobinemia is significant when methemoglobin exceeds 10% of total Hgb (shift to left with less release of O\(_2\)). Cyanosis observed; methemoglobinemia of concern if anemic or pregnant (when maternal transfer leads to methemoglobinemia of fetus).
  - Methemoglobinemia is more common in neonates due to decreased resistance of fetal hemoglobin to oxidant stresses and the immaturity of enzymes in the neonate that convert methemoglobin back to the ferrous state.
  - Treatment: If spontaneous reversal does not occur, IV injection with 1–2 mg/kg of 1% methylene blue solution (tetramethyl-thionine chloride)

**Other Toxicity**
- CNS, CV systems; generally 4–7 × the amount producing convulsions → CV collapse
- Toxicity assoc with \( >400 \) mg \( (>8 \) mg/kg)
- Intercostal injection leading to higher blood levels than with epidural

**ASSESSMENT POINTS**

<table>
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<tr>
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<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Toxicity</td>
<td>Metallic taste, tinnitus</td>
<td>↑ PAP</td>
<td>↓ PVR</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Pulm vasoconstriction</td>
<td>Systemic vasodilator</td>
<td>Nervous decrease</td>
<td>↓ SVR</td>
</tr>
<tr>
<td></td>
<td>Systemic vasodilator</td>
<td>Nervous decrease</td>
<td>Twitching, hyperreflexia possible</td>
<td>↓ CO</td>
</tr>
<tr>
<td></td>
<td>Neg inotrope</td>
<td>Neg chronotrope</td>
<td>Resp depression</td>
<td>ECG: ↑ P-R, ↓ QRS</td>
</tr>
<tr>
<td>CNS</td>
<td>Toxicity: More sensitive than CV</td>
<td>Shivering, twitching, tremors in face, extremities, progressing to tonic-clonic seizure</td>
<td>Twitching, hyperreflexia possible</td>
<td>Resp depression</td>
</tr>
<tr>
<td>PNS</td>
<td>Block nerve transmissions</td>
<td>Loss of sensation and motor function</td>
<td>Nerve stimulator:</td>
<td>Twitch height</td>
</tr>
<tr>
<td>MS</td>
<td>IV may augment NM blocker (both depolarizing and nondepolarizing)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Possible Drug Interactions**
- In large doses, blocks NM transmission; in smaller doses, enhances NMB from nondepolarizing and depolarizing NM blockers
- Acidosis, hypercarbia, hypoxia may potentiate nec chronotropic, isotropic actions

**Preoperative Considerations/Induction/Maintenance**
- Routine

**Anticipated Problems/Concerns**
- EMLA cream = eutectic mix of 5% lidocaine + prilocaine base for topical cutaneous anesthesia. EMLA applied under occlusive bandage for 45–60 min to obtain effective cutaneous anesthesia. EMLA cream produces anesthesia to a maximum depth of 5 mm. The efficacy of this combination lies in the fact that the mixture of prilocaine and lidocaine has a melting point less than that of either compound alone; existing at room temp as oil that can penetrate intact skin. Guidelines are available to calculate maximum amount of cream that can be applied and area of skin covered.
- EMLA → methemoglobinemia when large amounts used in children, particularly in newborn
- Methemoglobinemia if \( >600 \) mg given or given to anemic or pregnant pts


**Procainamide (Procan, Procanabid, Pronestyl)**

**Uses**
- Treats recurrent or sustained hemodynamically stable monomorphic VT (MVT) (IIa/C)/(IIa/C)
- Treats focal atrial tachycardia in hemodynamically stable pts (IIa/C)
- Treats recurrent atrial flutter (only in combination with AV-nodal-blocking agent) (IIb/A)
- Treats SVT during pregnancy

**Perioperative Risks**
- Potential for hypotension 2° to ganglionic blockade more likely than myocardial depression
- Nausea in pts on oral procainamide (related to levels of N-acetyl procainamide?)
- Chronic use can cause lupus-like syndrome; 25–50% of pts develop rash, small-joint arthralgias positive ANA. Resolves with cessation or administration of N-acetyl procainamide

**Worry About**
- Ventricular dysrhythmias if plasma concentration of NAPA >30 μg/mL

**Overview/Pharmacology**
- Analog of procaine
- Na⁺ channel blocker (intermediate recovery)
- Decreases automaticity, increases refractory periods, slows phase 4 depolarization
- Highly lipophilic but no relationship between drug properties and volume of distribution
- Major metabolite: N-acetyl procainamide (NAPA) does not block Na⁺ channels but equipotent in prolonging action potentials
- Rapid hepatic conjugation by N-acetyl transferase (t₁/₂ = 3–4 hr) to active metabolite NAPA

**Drug Class/Mechanism of Action/Usual Dose**
- Class IA antiarrhythmic; Na⁺-channel blocker
- Marked slowing of conduction by blocking of sodium channels (SA node and intraventricular conduction)
- IV loading dose: 1 g IV given at 20 mg/min (up to 17 mg/kg); IV maintenance 2–4 mg/min IV
- Narrow therapeutic window: Therapeutic plasma levels: procainamide 4–10 mcg/mL, NAPA 15–25 mcg/mL
- Toxic level: Procainamide >10 mcg/mL

**Induction/Maintenance**
- Caution with drugs that slow cardiac conduction (e.g., other Na⁺ channel blockers, beta-blockers)
- Use of other Na⁺ channel blockers
- LA toxicity with major conduction blocks
- Arrhythmias with high plasma concentration
- Myocardial depressive effect worsened by hyperkalemia
- Potentiates activity of NMBs

**Postoperative Period**
- Toxicity
- Arrhythmias caused by slowed conduction

**Perioperative Implications**

**Preoperative Concerns**
- Hx of arrhythmia, ischemic or structural heart disease
- Ventricular function
- Plasma concentration of procainamide
- May be used to treat contractions in pts with myotonic dystrophies; should be continued periop

**Postoperative Period**
- Evaluate regimen, pt compliance
- Monitor blood levels of procainamide and NAPA
- Neurologic assessment

**Anticipated Problems/Concerns**
- Monitor for clinical signs of toxicity: Torsades, heart block, arrhythmias, confusion, lupus syndrome
- Not well tolerated for long-term control of atrial tachycardias because of dosing regimen, complications
- In renal pts: Concentrations of procainamide and NAPA may rise to toxic levels => reduce dose, monitor levels of both


**Drug Effects**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Slowing of conduction</td>
<td>Assess for clinically symptomatic bradycardia, heart block, CHF</td>
<td>Auscultation of heart sounds, ECG</td>
<td>Continuous ECG monitoring</td>
</tr>
<tr>
<td>RESP</td>
<td>Lupus-related pleuritis or pneumonia</td>
<td>Assess for dyspnea</td>
<td>Auscultation of lung fields</td>
<td>O₂ sat monitoring</td>
</tr>
<tr>
<td>CNS</td>
<td>High plasma concentrations may cause confusion/disorientation and/or seizures; rarely muscle weakness</td>
<td>Evaluate regimen, pt compliance</td>
<td>Monitor blood levels of procainamide and NAPA</td>
<td>Neurologic assessment</td>
</tr>
</tbody>
</table>

*The first number and lower-case letter refer to the ACC/AHA system of classifying guidelines while the upper-case letter refers to the level of evidence.*
Propylthiouracil—Antithyroid Drugs

**Uses**
- Incidence in USA: 5% of pregnant women, plus 480,000/y develop hyperthyroidism
- Rx for hyperthyroidism, goiter associated with hyperthyroidism
- Definitive Rx to control hyperthyroidism in anticipation of spontaneous remission
- Rx for hyperthyroidism in conjunction with $^{131}$I or $^{125}$I to hasten recovery while awaiting effects of radiation therapy
- Rx for hyperthyroidism to control disorder in preparation for surgery

**Perioperative Risks**
- Side effects of drug: Hyperthyroidism (see Hyperthyroidism or Hypothyroidism in Diseases section); liver failure esp. in pts with liver transplants; be careful in pregnancy.

**Worry About**
- Agranulocytosis (less than 0.5% of treated pts develop this side effect)

**Overview/Pharmacology**
- Antithyroid drug: Absorbed within 20–30 min; effect begins to ↓ in 2–3 hr (methimazole $T_{1/2}$ estimated to be 6–13 hr)
- Drug and metabolites cleared by renal excretion
- Antithyroid drugs cross placenta, can be found in breast milk

**Drug Class/Usual Dose**
- Antithyroid drug: Interferes directly with synthesis of thyroid hormones by preventing incorporation of iodine into tyrosyl residual thyroglobulin; inhibits coupling of iodotyrosyl residues to form iodothyronines by inhibiting peroxidase enzyme
- Depletes pre-formed hormone over time; only then do clinical effects become noticeable ($T_{1/2}$ of thyroid hormones is $3$ d in circulation)
- Other useful antithyroid Rx drugs incl those inhibiting conversion of less active $T_{3}$ into more active $T_{4}$, such as propranolol; methimazole, carbimazole do not appear to do so with anti-$\beta$-blocker effect (e.g., propanolol and others); those that inhibit release of pre-formed thyroid hormone (e.g., iodine). Also temporarily inhibits synthesis and ↓ vascularity of thyroid glands.
- A thioureylene

**Perioperative Risks**
- Decreased hyperthyroidism and thyrotoxicosis
- Decreased goiter size in hyperthyroidism

**Acute Rx Uses**
- Relieves symptoms of hyperthyroidism while waiting for $^{131}$I or $^{125}$I to take effect

**DRUG EFFECTS**

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<tr>
<th>System</th>
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</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Goiter shrinkage; occasionally goiter develops if hypothyroidism occurs</td>
<td>Snoring, hoarseness, neck pain</td>
<td>Ask pt to vocalize “e”; examine airway, neck</td>
<td>Check CXR (PA, lat), lat neck films; if needed, CT scan of neck</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Assess CV response to Rx</td>
<td></td>
<td>Rhythm strip or full ECG if CV system is involved by either Hx or PE</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Rare hepatotoxicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Mild anemia, thrombocytopenia; agranulocytosis as toxic reaction to propylthiouracil or methimazole (0.05–0.12% of pts)</td>
<td>Hx of sore throat or fever often heralds agranulocytosis</td>
<td>Skin/mucous membranes for infection/petechiae; purpura if at risk</td>
<td>CBC with plt count; differential leukocyte count</td>
</tr>
<tr>
<td>DERM</td>
<td>Rare depigmentation of hair</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Pain/stiffness in joints (rare side effect)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Placenta—crosses placental barrier and is excreted in breast milk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Headache, paresthesia rare side effects. Shaking, anxiety, emotional instability are signs that hyperthyroidism not yet controlled.</td>
<td>Reflex speed, tremor, nervousness, mental status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Need to assess if euthyroid</td>
<td>Refer to all other systems, esp. reflex speed, tremor, heat intolerance, wt loss, fatigue, weakness, anorexia, ↑ appetite</td>
<td>Reflex speed; HR</td>
<td>Free $T_{4}$ level if unable to assess if euthyroid by Hx, PE</td>
</tr>
</tbody>
</table>


**Possible Drug Interactions**

**Preoperative Period**
- Assess euthyroid state (see table)
- Fairly certain sign that remission may have occurred is ↓ in size of goiter

**Induction/Maintenance**
- No interactions known

**Adjuvants/Regional Anesthesia/Reversal**
- No interactions known

**Postoperative Concerns**
- Resumption not necessary if surgery to correct hyperthyroidism successful
- Be careful in pts with Hx of liver disease, pregnant or breastfeeding

**Anticipated Problems/Concerns**
- Assess for hyperthyroidism, agranulocytosis

**Chronic Rx Uses**
- Decreased hyperthyroidism and thyrotoxicosis
- Decreased goiter size in hyperthyroidism

**Acute Rx Uses**
- Relieves symptoms of hyperthyroidism while waiting for $^{131}$I or $^{125}$I to take effect
**Pyridostigmine Bromide**

**Uses**
- Therapy for myasthenia gravis (MG) which is caused by decreased postsynaptic acetylcholine (ACh) receptors
- Antagonism of nondepolarizing neuromuscular blocking drugs (NMBD)
- Therapy for glaucoma
- Therapy for atony of GI and urinary tracts

**Perioperative Implications**

**Preoperative Concerns**
- In MG pts, skeletal muscle response to repetitive impulses is augmented by increased availability of ACh
- Chronic administration in MG pts may alter effects of NMBD, and some may consider omission or reduction of morning dose on the day of surgery.

**Induction/Maintenance**
- While nicotinic effects are desirable, muscarinic effects should be attenuated by an anticholinergic (typically glycopyrrolate 0.05 mg per 1 mg of pyridostigmine)
- Paralysis may be prolonged by excessive doses which can produce a depolarizing NMB

**Pharmacology**
- Oxydiaphoretic (acid-transferring) inhibitor of acetylcholinesterase (AChE)
- Transfers a carbamate group to AChE and forms a covalent bond at the esteratic site
- Quaternary ammonium ion which is poorly lipid soluble; does not effectively penetrate GI tract or blood-brain barrier (no CNS side effects)
- Onset is 10–15 min (versus 5–10 min for neostigmine); duration is 4 hr (similar to neostigmine)
- 20% as potent as neostigmine

**Postoperative Period**
- Incidence of recurarization in renal pts is not ↑ as clearance of both AChE inhibitors and NMBD is similarly affected.
- MG pts taking >750 mg/d have ↑ potential for resp insufficiency
- Myasthenic and cholinergic crises may occur after periop alterations in AChE inhibitor therapy.

**Drug Class/Mechanism of Action/Usual Dose**
- Reversibly inhibits AChE which increases the concentration of ACh at the motor endplate
- May be administered PO, IV, or IM
- Dose is 0.1–0.4 mg/kg IV

**Anticipated Problems/Concerns**
- If maximal dose of pyridostigmine (0.4 mg/kg, or 20 mg in adults) fails to antagonize the residual blockade, it is not advisable to redose the AChE inhibitor as this may lead to further motor weakness.
- Causes of inadequate antagonism incl profound blockade, resp acidosis, hypokalemia, hypermagnesemia, hypothermia, verapamil, and antibiotics such as aminoglycosides and polypeptides.

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**DRUG EFFECTS**

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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Bradyarrhythmias, hypotension</td>
<td>Presyncope, angina, confusion</td>
<td>HR, BP, orthostasis</td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>↑ Secretions, bronchospasm</td>
<td>Dyspnea, wheezing</td>
<td>Auscultation</td>
<td>PFTs</td>
</tr>
<tr>
<td>GI</td>
<td>↑ Secretions, ↑ motility, spasms</td>
<td>Diarrhea, abd pain</td>
<td>Palpation</td>
<td>Electrolytes</td>
</tr>
</tbody>
</table>

Quinidine

- See Also Procainamide

Uses
- Used in the treatment of ventricular arrhythmias (hemodynamically stable wide complex tachycardia) or supraventricular arrhythmias (i.e., torsades de pointes). Acidosis, hypomagnesemia, and hypokalemia increase risk of adverse events.

Perioperative Risks
- Plasma levels >2 μg/mL results in prolongation of the PR interval, QRS complex, and QT interval which may lead to life-threatening ventricular arrhythmias (i.e., torsades de pointes). Acidosis, hypomagnesemia, and hypokalemia increase risk of adverse events.

Worry About
- High plasma levels assoc with hepatic and renal disease because quinidine is metabolized by the liver and subsequently excreted by the kidney (dose must be reduced and plasma levels followed)
- Thrombocytopenia possible 2° to drug-plt complexes that cause the production of antibodies
- N/V/D occur in approx 1/3 of people
- Decreasing plasma levels in assoc with rifampin, phenytoin, barbiturates
- Increasing plasma levels in assoc with amiodarone, cimetidine
- Causes serum levels of digoxin to increase to toxic levels

DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>↑ QRS duration, vagolytic cardiac/ peripheral α blockade, negative inotropic, ↓ conduction velocity</td>
<td>↑ HR, Dyspnea, Syncope</td>
<td>CXR (3° AV block), Alnormal S, Bradycardia, asystole</td>
</tr>
<tr>
<td>GI</td>
<td>Diarrhea (18%), nausea (18%)</td>
<td>α Blockade</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Thrombocytopenia</td>
<td>Mucosal bleeding</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Headache (13%), dizziness (8%), tinnitus, blurred vision, tremor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MISC</td>
<td>Drug-induced SLE, anaphylactoid reactions, aggravation of asthma (caution)</td>
<td>Rash, Joint pain</td>
<td></td>
</tr>
</tbody>
</table>


Preoperative Implications/Possible Drug Interactions

Preoperative Preparation
- ECG should be closely monitored when initially starting quinidine. Consider serial measurements of QRS duration and QT interval on the ECG to prevent arrhythmias; QRS should be <140 ms or ≤50% increase from baseline
- Quinidine will cause increased plasma levels of warfarin and digoxin; monitor levels closely and ↓ dosage to prevent toxicity.
- Decrease dose in hypoproteinemia, liver failure, and renal failure

Induction/Maintenance
- Possible ↓ in hepatic metabolism of halogenated agents
- ↓ Levels of plasma proteins after CPB augment free fraction of drug

Adjuvants/Regional Anesthesia/Reversal
- Quinidine may ↑ muscle weakness in pts with myasthenia gravis, ↓ effect of anticholinesterases, enhances NMB and may lead to recurrence of skeletal muscle blockade in immediate postop period.
- Quinidine can enhance the effects of vasodilating, negative inotropic, sinus node depressant agents (e.g., β-blockers, verapamil, rauwolfia alkaloids, bretylium) leading to severe hypotension or syncope.
- Concurrent administration of other class IA drugs, amiodarone, or phenothiazines ↑ risk of torsades de pointes
- Quinidine has attenuates effects of anticholinergic drugs

Anticipated Problems/Concerns
- Quinidine contraindicated when ventricular arrhythmias assoc with or caused by QT prolongation (risk of torsades)
- Usually administered orally due to adverse effects assoc with parenteral routes
- IM injection very painful
- IV routes cause vasodilation and myocardial depression, esp. when rapidly infused. D/C if hypotension persists.
- Must weigh risk-benefit ratio in each pt due to proarrhythmic qualities of quinidine even when plasma levels in therapeutic range; may actually have increased mortality.

Overview/Pharmacology
- Dextroisomer of quinine and thus also has antiinflammatory and antipyretic effects
- Quinidine blocks the fast inward sodium channel, which leads to a prolongation of phase 0 of the cardiac action potential and therefore a decreased heart rate
- Well absorbed from GI tract (80% bioavailability) and peak plasma concentrations occur in 60-90 min with effects lasting 6-8 hr (up to 12 hr with extended release formulations)
- Elimination through kidneys, 20% unchanged, 80% after hepatic metabolism through hydroxylation to an inactive metabolite
- 90% bound to plasma proteins
- Serum concentration should be monitored to fit narrow therapeutic range: 2–5 μg/mL
- Interacts with drugs that alter hepatic enzyme function, other highly protein-bound drugs
- Urine alkalinization leads to decreased excretion

Drug Class/Mechanism of Action/Usual Dose
- Class IA antiarrhythmic (use-dependent Na+ channel blocker which causes prolongation of the cardiac action potential and is responsible for effectiveness in tachyarrhythmias)
- Dosage
  - Quinidine gluconate (324 mg tabs): 648–2592 mg divided bid or tid
  - Quinidine gluconate IM form: 400–2400 mg daily
  - Quinidine sulfate (200 mg, 300 mg, 300 mg ER tabs): 400–4000 mg divided bid or tid
- Quinidine is rarely administered IV due to peripheral vasodilation and myocardial depression, which can lead to severe hypotension
- Doses must be titrated to the individual based on plasma levels and EKG findings
- Alternatives: Other class IA drugs (e.g., procaainamide, disopyramide, cibenzoline, pirmenol)
Riboflavin (Vitamin B<sub>2</sub>)

**Other Names**
- Flavin, flavine, lactoflavin, riboflavin, vitamin G.
- The name riboflavin comes from ribose (the sugar that forms part of its structure) and flavin, the ring-moiety that imparts the yellow color to the oxidized molecule.

**Etiology/Sources**
- Organic: Leafy green vegetables, milk, cheese, kidneys, legumes, tomatoes, yeast, mushrooms
- Synthetic: Water-soluble B-complex vitamins and vitamin B<sub>1</sub> preparations.

**Indications/Uses**
- Riboflavin is required for a wide variety of cellular processes in humans and animals.
- Taken orally for the prevention of common deficiency with general nutritional deficiency (malnutrition, starvation, chronic alcoholism) and for preventing migraine headaches.
- Also used in the treatment of alcoholism, atroflavinosis, acne, burning feet syndrome, burns, canker sores, carpal tunnel syndrome, cataracts, congenital methemoglobinemia, eye fatigue, glaucoma, lactic acidosis induced by NRTI (anti-retroviral) drugs, multiple acylcoenzyme A dehydrogenase deficiency, liver disease, memory loss (incl Alzheimer’s disease), RBC aplasia, sickle cell anemia, and ulcers.
- Riboflavin is also used to increase energy levels, boost immune system function and athletic performance, slow aging, promote healthy reproductive function, and to maintain healthy hair, skin, mucous membranes, and nails.

**Perioperative Risks**
- Excessive intake causes increased excretion of unchanged riboflavin in the urine.
- Deficiency causes anemia, neuropathy, and more friable lip tissue and oral/tongue mucosa.
- Use caution with tape and pressure at or near facial, nasal areas if seborrhea is present.

**Anticipated Problems/Concerns**
- Riboflavin is likely safe when taken orally, no toxic effects have been reported.
- During pregnancy, it is likely safe when used at the RDA of 1.4 mg/d.
- When lactating, it is likely safe when used at the RDA of 1.6 mg/day.
- Adequate phosphorus must be given along with riboflavin and other vitamins when refeeding starved pts to prevent depletion of phosphate stores and energy of cells.
- Exposure to light can destroy vitamin B<sub>2</sub>.

**Overview/ Mechanism of Action/Pharmacology**
- Component of the electron transfer chain in mitochondria, oxidative metabolic coenzymes.
- Absorbed from upper GI tract by specific transport mechanism involving phosphorylation of an enzyme to FMN by the enzyme flavokinase.
- Central component of cofactors FAD and FMN and is therefore required by all flavoproteins.
- Like other B vitamins, riboflavin plays an important role in energy metabolism and for the metabolism of fats, ketone bodies, carbohydrates, and proteins.
- Distributed to all tissues, but little stored and the rest is excreted unchanged in the urine.

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect of Deficiency</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEEENT</td>
<td>Sore throat, cheilosis, glossitis, corneal vascularization, cataracts</td>
<td>Burning tongue, soreness in mouth and throat</td>
<td>Red, fissured lips; blue-red tongue with edematous surface—“cobblestone tongue”</td>
<td>Urinary excretion of &lt;30 mcg/24 hr of riboflavin</td>
</tr>
<tr>
<td>HEME</td>
<td>Anemia</td>
<td></td>
<td>Reticulocytopenia, normochromic normocytic anemia</td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Seborrheic dermatitis of face, dermatis of arms and trunk</td>
<td>Burning, itching eyes</td>
<td>Rough, sharkskin appearance of nose</td>
<td></td>
</tr>
<tr>
<td>PNS</td>
<td>Neuropathy</td>
<td>PNS function exam</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Drug Class/Usual Dose**
- Water-Soluble B-Complex Vitamin; RDA For adults is 0.6 mg/1000 kcal/d diet.

**Possible Drug Interactions/Perioperative Implications**
- Aspirin: Concomitant use with riboflavin may cause gastric intolerance.
- Beta-blockers: Theoretically, concomitant use with riboflavin may enhance migraine prevention without increasing adverse effects.
- Nucleoside reverse transcriptase inhibitor (NRTI) drugs: Riboflavin is reported to reverse lactic acidosis caused by these antiretroviral drugs.
- Probenecid: Decreases riboflavin absorption.
- Propantheline: Delays and increases riboflavin absorption.
- Drug influences on nutrient levels and depletion:
  - Antibiotics: Can destroy normal GI flora, which can cause decreased production of B vitamins.
  - Metoclopramide: Concomitant use can decrease riboflavin absorption in the GI tract.
  - Oral contraceptives: By an unknown mechanism the use of oral contraceptives can reduce serum vitamin B<sub>2</sub> levels. The need for supplementation has not been adequately studied.
  - Phenothiazines: Use of phenothiazines can increase urinary vitamin B<sub>2</sub> excretion and reduce serum vitamin B<sub>2</sub> levels.
- Probencid: Inhibits dietary riboflavin absorption.
- Probantheline bromide: Delays and increases supplemental riboflavin absorption.
- Absorption depends on flavokinase activity, which in turn depends on thyroid hormone status and is inhibited by TCAs and chlorpromazine.
- Preop normochromic normocytic anemia in a nutritionally depleted pt responds to riboflavin administration.
- Boric acid poisoning induces riboflavin deficiency.
Neisseria meningitidis

- Secreted in saliva, tears

Assessment by Hx

- Decreased duration of action of narcotic and barbiturates due to P450 (CYP2D6) enzyme induction
- Pts receiving antiarrhythmic therapy, digoxin, theophylline, phenytoin, or glucocorticoid therapy may need increased doses of these drugs due to enzyme induction

Overview/ Pharmacology

- Complex macrocyclic antibiotic
- H2O-soluble at acidic pH; inhibits gram-positive and many gram-negative organisms, incl E. Coli, Pseudomonas, Proteus, Klebsiella, N. meningitidis, H. influenzae, M. tuberculosis
- Increases in vitro activity of streptomycin and isoniazid
- Eliminated by biliary clearance with significant enterohepatic circulation

Perioperative Implications/ Possible Drug Interactions

Preoperative Concerns

- Decrease duration of action of benzodiazepines, narcotics, barbiturates due to hepatic (P450, CYP2D6) enzyme induction
- Adequacy of pre-existing drug regimens should be verified (see Special Considerations)

Induction/ Maintenance

- Decreased narcotic and analgesic efficacy: barbiturates, methadone, diazepam, midazolam; β-blockers have increased clearance, decrease duration of action
- Halothane metabolism increases with increased risk of hepatotoxicity

Adjutants/ Reversal

- Mycobacteria quickly develop resistance when rifampin used alone; administer with isoniazid and/or streptomycin

Special Considerations

- Risk of hepatic dysfunction periop ↑ by pre-existing hepatic disease
- Delays oral absorption of ASA
- Decreases T1⁄ requiring ↑ doses to maintain adequate therapeutic levels: Digoxin, digitoxin, quinidine, propranolol, metoprolol, verapamil, coumadin, theophylline, phenytoin, prednisone, cortisol, cyclosporine, oral hypoglycemic agents, ketoconazole, fluconazole
- Can precipitate opioid withdrawal symptoms in an opioid dependent pt because of enhancing the hepatic enzymatic metabolism of opioids

Drug Class/Mechanism of Action/Dose

- Rifamycin antibiotic family
- Inhibits DNA-dependent RNA polymerase in bacteria and mycobacteria; nuclear eukaryotic RNA polymerase not affected

Perioperative Risks

- Hepatic dysfunction, most likely in presence of pre-existing liver disease and when used in combination with other hepatotoxic agents like isoniazid

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>OVERALL</td>
<td>Secreted in saliva, tears</td>
<td>Orange sputum, tears, conjunctiva, sweat</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEENT</td>
<td>Fatigue, drowsiness, dizziness, ataxia, confusion, weakness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Jaundice</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Elevated transaminases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Interstitial nephritis, ATN, renal failure (with high doses)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


- Rifampin

- T1⁄ of 1.5–5 h, increases with hepatic dysfunction

Worry About

- Induces hepatic microsomal (P450, CYP2D6) activity, decreases T1⁄ of hepatically metabolized drugs
- Theoretical increases risk of halothane hepatitis
- Hemolytic anemia, thrombocytopenia (rare)

DRUGS

Rifampin

- Induction/ Maintenance
- Clin Pharmacol Ther
- Rifampin used alone; administered with isoniazid and/or streptomycin
- Rifampin induces microsomal enzyme activity in liver, results in ↓ efficacy, duration of action of hepatically metabolized drugs

Perioperative Risks

- Administered for chemoprophylaxis of meningococcal infections, with β-lactams for staphylococcus endocarditis, osteomyelitis, for methicillin-resistant S. aureus infections, in conjunction with isoniazid and streptomycin for active TB
- Usual dose: 600 mg qd; pediatric dose 10 mg/kg qd, PO or IV
- Possible interaction of rifampin with 5HT3 and opioid system as well as mediators of itching is proposed. Dose 300 mg bid IV
- Should be administered 1 hr before or 2 hr after meals PO

Anticipated Problems/Concerns

- 10% on therapy may develop hepatitis; pts with pre-existing liver disease are at higher risk
- Rifampin induces microsomal enzyme activity in liver, results in ↓ efficacy, duration of action of hepatically metabolized drugs
Serotonin: Agonists, Antagonists, and Reuptake Inhibitors

**Indications**
- Serotonin (5-hydroxytryptophan, 5-HT) not given as a drug
- Partially selective receptor agonists (used mostly for Rx of acute migraine headaches)
  - Sumatriptan (Imitrex) 5–20 mg nasal, 25–100 mg/d PO mg
  - Naratriptan (Amerge) 2.5 mg/d PO
  - Rizatriptan (Maxalt) 5 mg/d PO
  - Zolmitriptan (Zomig) 5 mg nasal, 2.5 mg/d PO
  - Metoclopramide (Reglan) 5–15 mg qid PO, 2–10 mg IV (Rx for GERD, gastroparesis, N/V)
- Partially selective receptor 5-HT₁ antagonists (used for Rx of N/V)
  - Dolasetron (Anzemet) 12.5 mg IV or 100 mg PO 30–60 min before emergence to prevent postop N/V or before chemoRx
  - Ondansetron (Zofran) 4–8 mg tid PO to prevent N/V due to emergence or emetogenic chemoRx treatment
  - Granisetron (Kytril) 10 µg/kg IV, 1 mg/bid PO, TD patch (Sancuso) for prevention of N/V due to chemoRx; for postop N/V
  - Palonosetron (Aloxi) 0.25 mg IV 30 min before chemoRx
  - Selective serotonin reuptake inhibitors (SSRIs) (all used PO for Rx of major depression and personality disorders)
    - Citalopram (Celexa) 20–40 mg/d PO
    - Escitalopram (Lexapro) 10 mg/d PO
    - Fluoxetine (Prozac) 20–80 mg/d PO
    - Fluvoxamine (Luvox) 25–50 mg/d PO
    - Paroxetine (Paxil) 20–50 mg/d PO
    - Sertraline (Zoloft) 50–200 mg/d PO

**Perioperative Risks**
- Sumatriptan: Not for pts with IHD, angina, Prinzmetal’s angina, severe Htn
- Metoclopramide: Not for pts with pheochromocytoma, on MAOIs; may worsen mental depression; effect antagonized by narcotics
- SSRIs can cause serotonin syndrome (hyperthermia, muscle rigidity, myoclonus, rapid mental change) if given in the presence of MAOIs; may increase coumadin, digitalis effects by reducing plasma protein binding; increased suicide risk <24 y age

**Worry About**
- Sumatriptan and other 5-HT agonists: pts taking these may have exacerbation of anginal Sx
- Ondansetron, granisetron, etc: chemoRx pts may exhibit ↑ N/V during anesthesia
- SSRIs: Serotonin syndrome: concomitant use of MAOIs, displacement of other drugs highly bound to plasma protein (digoxin, antianginals, beta-blockers, tricyclic antidepressants) increased bleeding with coumadin, so monitor prothrombin time

**Overview/Pharmacology**
- Serotonin secreted 90% by enterochromaffin cells of GI tract; released into plasma by unclear mech, neuronal stimuli; some taken up, much is stored in plts; 5-HT receptors on vascular endothelium stimulate release of NO to promote vasodilation, but receptors on vascular smooth muscle promote vasoconstriction. Excess release involved in carcinoid syndrome, due to enterochromaffin cell neoplasm. As an amine neurotransmitter, serotonin also secreted, stored, released by raphe nuclei in brainstem (serotonergic neurons).
- Serotonergic neurons diffusely innervate most regions of CNS; with other neurotransmitters is involved in modulating mood, depression, anxiety, migraine headache, sleep, appetite, temp regulation, perception of pain and itch, regulation of BP
- Abn in secretion or receptor activation likely underlie mental depression, migraine headache, sensitivity to pain, sleep pattern, and central BP control. In CNS, 5-HT receptor activation increases K⁺ conductance to promote membrane hyperpolarization, → mostly inhibitory action. As CNS neurotransmitter, 5-HT modulates effects of other monoamine transmitters—e.g., norepinephrine, dopamine, and other transmitters such as ACh, glycine, GABA. Inhibition of 5-HT reuptake elevates mood, normalizes behavior.

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Htn, IHD (agonists)</td>
<td>MAO drug interaction</td>
<td>BP</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ECG: Longer P-R and QT; intervals (agonists)</td>
<td>Diarrhea, abd pain, asthma, flushing, hyperglycemia, PAT, SVT</td>
<td>BP, CNS</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypotension (SSRIs)</td>
<td>Drug levels</td>
<td>Bleeding</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Serotonin syndrome (5-HT)</td>
<td>5-HT, kallikreins</td>
<td>Bleeding</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Altered drug levels (SSRIs)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Carcinoid syndrome (↑ 5-HT)</td>
<td>Dysrhythmias, bleeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Leukopenia (agonists)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Psychosis, depression, altered mood, Sz</td>
<td>Mental disorder</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**
- Avoid narcotics in pts with carcinoid syndrome (surgery or 5-HT antagonists usual Rx for carcinoid tumor)
- Use caution in giving metoclopramide; pt must not be taking MAOIs—e.g., isocarboxazid (Marplan), phenelzine (Nardil), or tranylcypromine (Parnate)
- Check pt’s drug profile if Hx of migraine; ↑ risk of coronary vasoconstriction with sumatriptan
- Check pt’s drug profile if Hx of schizophrenia; may have low WBC count if taking clozapine
- Check pt’s drug profile if Hx of major depression; if taking coumadin or digitalis, levels may be ↑.
Sildenafil Citrate

Indications
- Treatment of erectile dysfunction (Viagra)
- Sildenafil (Revatio) is used to improve the ability to exercise in people with pulm arterial Htn
- Oral sildenafil is used as part of multimodal management of severe periop pulm Htn and right ventricular dysfunction in clinical settings such as:
  - Heart transplantation
  - Pulm Htn assoc with CHD
  - Pulm Htn assoc with mitral valve disease

Perioperative Risks
- None for elective surgery based on $\frac{T_{1/2}}{T_1}$
- Drug may still be present in emergent surgery

Worry About
- Potentiation of vasodilating agents
- Hx of coronary ischemia or congestive heart failure
- Severe hepatic impairment

Overview/Pharmacology
- Sildenafil citrate was discovered by accident during testing as a treatment for heart disease.
- Terminal $T_{1/2}$ 4–6 hr
- Total protein binding 96%, also distributed in tissues
- Bioavailability 41%
- Metabolized in liver via the cytochrome P450 isoenzymes, 3A4 (major route) and 2C9 (minor route)
- Active N-desmethyl metabolite
- Peak plasma concentration 60 min
- Excreted via feces (80%), kidney (13%), and semen (<0.001% of a dose)
- Metabolism may be delayed after a high-fat meal and in pts with liver disease
- Contraindicated in pts with hypersensitivity to sildenafil products and pts taking nitroglycerin or other organic nitrates
- Precautions: Anatomic deformities of the penis, conditions predisposing pts to priapism, bleeding disorders or active peptic ulceration, retinits pigmentosa or other retinal abn, coronary ischemia or CHF, multidrug antihypertensive regimens
- Excretion in breast milk is unknown

Drug Class/Mechanism of Action
- Potent and selective inhibitor of phosphodiesterase type V (PDE V)
- PDE V isofrom is responsible for breaking down cyclic guanosine monophosphate (cGMP) in corpus cavernosum. cGMP relaxes smooth muscle to cause local vasodilatation and swelling of corpora as they fill with blood.
- With sexual arousal, NO is produced in cavernosal tissue to stimulate the secretion of cGMP.
- Sildenafil inhibits PDE V, causing 35% increase in cGMP levels.
- Sildenafil inhibits PDE V in the lung, thus increasing cGMP levels in the lung to cause pulm vasodilatation and improvement in pulm Htn.

Usual Dose
- Supplied in 100-mg, 50-mg, 25-mg tablets
- May be taken 0.5–4 hr prior to sexual activity
- Dose ranges from 25–100 mg, with a maximum frequency of once-a-day orally
- Dose adjustments required in pts with severe renal and hepatic impairment
- For geriatric pts (>65 y), starting dose should be 25 mg

Perioperative Implications
- Risk primarily related to emergent cases based on $T_1$
- Caution with the concomitant use of hypotensive agents
- Precautions to prevent reflux and regurgitation
- Pts on regular sildenafil for pulm vasodilation will have significant pulm Htn that is often asso with significant underlying lung disease and right ventricular dysfunction.

Drug Interactions
- Concurrent use of nitrates may cause hypotension.
- Drug interactions with cytochrome P450 inhibitors (e.g., ketoconazole, erythromycin, and cimetidine) can be expected, and during concomitant therapy a lower dose is suggested.
**Statins**

**Uses**
- Incidence in USA: Estimated 20 million
- Primary indications include:
  - Hyperlipidemia: Hydroxymethylglutaryl coenzyme-A (HMG CoA) reductase inhibitors (statins) are powerful drugs for lowering low-density lipoprotein (LDL) cholesterol and certain brands—atorvastatin in high doses and rosuvastatin—increase healthy HDL cholesterol.
  - 1st and 2nd prevention of CV disease: CV benefits (reduction in myocardial infarction and stroke) in pts with hypercholesterolemia. Benefits also in normocholesterolemic pts with elevated markers of inflammation (e.g., C-reactive protein (CRP)).
  - 2nd, less robustly proven, benefits: Decreased risk of sepsis, venoembolic disease, osteoporosis, cancer, and dementia; blood pressure reduction; renal function preservation; reduced morbidity in heart failure.

**Perioperative Risks**
- **Myopathy:** Incidence among nonoperative chronic statin users is 2–11% for myalgias, 0.5% for myositis, <0.1% for rhabdomyolysis. Incidence increased in severe renal insufficiency (CrCl <30 mL/min).
- **Hepatic dysfunction:** Incidence of persistent elevations in aminotransferases is 0.5–3%, and 0.1% for 20-fold increase in alanine aminotransferase. Reversible following D/C.
- **Incidence of myopathy and transaminisits increased when cytochrome P-450 3A4 inhibitors, incl cyclosporine, tacrolimus, azole antifungals, fenofibrates, protease inhibitors, and macrodote antibiotics are used concomitant with those statins that are extensively metabolized by cytochrome P-450 3A4 (lovastatin, simvastatin, and to a lesser extent atorvastatin).**
- **Lipophilic statins may be associated with more adverse events than hydrophilic statins**

**Overview/Pharmacology**
- Statins inhibit the reduction of HMG CoA to mevalonate, the rate-limiting step in cholesterol biosynthesis. Statins primarily inhibit hepatocyte cholesterol synthesis and increase LDL receptor transcription and hepatic LDL cholesterol uptake. Consequently, statins reduce systemic concentrations of LDL cholesterol by 25–55%. Plasma HDL cholesterol levels may rise by 8–10% with atorvastatin and rosuvastatin.
- The reduction in intracellular isoprenoid synthesis, which reduces prenylation of small GTPases (e.g., Ras, Rhe), and may mediate the beneficial pleiotropic (non-lipid lowering) effects of statins. These effects include atherosclerotic plaque stabilization, inflammation reduction, reversal of endothelial dysfunction (through eNOS upregulation), decreased thrombogenicity, reduced reactive O2 species generation (through NADPH-oxidase assembly inhibition). Improved survival occurs primarily in pts with elevated serum CRP levels. The statin-induced reduction in serum CRP concentration occurs unrelated to lipid levels at baseline or during therapy.
- Statins are orally administered once daily and peak plasma concentrations achieved in 1–3 hr.
- The hepatic cytochrome P-450 system metabolizes most statins to active and inactive metabolites, and statins are primarily excreted in bile.

### PHARMACOKINETICS

<table>
<thead>
<tr>
<th>Statin</th>
<th>Dose (mg)</th>
<th>Elimination Half-life, hrs</th>
<th>Protein Binding</th>
<th>Solubility</th>
<th>Cytochrome P-450 Isozyme</th>
<th>Active Metabolites</th>
<th>Renal excretion, %</th>
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<tr>
<td>Atorva-</td>
<td>10–80</td>
<td>15–30</td>
<td>80–90</td>
<td>Lipophilic</td>
<td>3A4</td>
<td>Yes</td>
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<td>Fluv-</td>
<td>20–80</td>
<td>0.5–2.3</td>
<td>&gt;99</td>
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<td>2C9</td>
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<td>Lova-</td>
<td>20–80</td>
<td>2.9</td>
<td>&gt;95</td>
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<td>3A4</td>
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<td>Prava-</td>
<td>10–40</td>
<td>1.3–2.8</td>
<td>43–55</td>
<td>Hydrophilic</td>
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<td>Rosuva-</td>
<td>5–40</td>
<td>19</td>
<td>88</td>
<td>Hydrophilic</td>
<td>2C9</td>
<td>No</td>
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<td>Simva-</td>
<td>10–80</td>
<td>2–3</td>
<td>94–98</td>
<td>Lipophilic</td>
<td>3A4, 3A5</td>
<td>Yes</td>
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</table>

### SIDE EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Presentation</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEPAT</td>
<td>Transaminitis</td>
<td>Asymptomatic</td>
<td>None</td>
<td>LFTs</td>
</tr>
<tr>
<td>MS</td>
<td>Myositis</td>
<td>Myalgia, cramps, aches</td>
<td>Muscle tenderness</td>
<td>Creatinine kinase</td>
</tr>
</tbody>
</table>

**Perioperative Implications**
- Pts with coronary disease or a coronary disease risk equivalent (DM, symptomatic carotid artery disease, peripheral arterial disease, ab aortic aneurysm, chronic kidney disease, or multiple risk factors that confer a 10-yr risk of CHD greater than 20%) should receive statin therapy. As such, pts on statin therapy should be examined preop for coronary and peripheral vascular disease.
- Concern for statin accumulation and muscular and hepatic side effects among pts receiving major surgery led the ACC/AHA/NHLBI to recommend short-term periop statin administration cessation in 2002. Periop observational studies, however, have not assoc statin use with an increased risk of myopathy or rhabdomyolysis. In fact, preop cessation of statin therapy was assoc with significant CV harm in pts undergoing cardiac and major vascular surgery.
- Two randomized trials and large observational studies suggest pleiotropic effects of statins improve outcomes in the periop period for major cardiac and vascular surgery.
- In the DECREASE III trial, 497 vascular surgery pts were randomly assigned to either 80 mg of extended release fluvastatin daily or placebo at least 30 d before the procedure and continued for at least 30 d after surgery. The 2nd endpoint of myocardial ischemia, within 30 d of surgery, occurred significantly less often in the fluvastatin group (10.8 vs. 19.0 percent; hazard ratio 0.55, 95% CI 0.34–0.88). The 2nd endpoint of the composite of death from CV causes and myocardial infarction also occurred significantly less often in the fluvastatin group (4.8 versus 10.1 percent; hazard ratio 0.47, 95% CI 0.24–0.94). There was no evidence of an increase in skeletal muscle or hepatic injury in the fluvastatin group.
- Among percutaneous coronary intervention pts, statin therapy administered 12 hr pre-catheterization reduced composite of myocardial ischemic events and death in several placebo controlled RCTs. Another RCT (ARMYDA) reported that 7 d of preop atorvastatin reduced post cardiac surgery atrial fibrillation and hospital length of stay.
- The non-lipid lowering (i.e., pleiotropic) effects of statins originate from improved endothelial function and reduced inflammation. In rodent and cell models, statins increase NO availability, reduce reactive O2 species generation, and reduce circulating markers of inflammation (interleu kin-6, P-selectin) within 6-18 hr.
- Statin therapy is recommended as early as possible before surgery for pts undergoing elective major vascular surgery who have not been receiving a statin. Statin therapy should not be discontinued in the periop period in statin-using pts.
- Physician-scientists hope pleiotropic effects of statin therapy will provide periop protection for heart, brain, and kidney, but as yet, sufficient data is lacking.

Steroids

Uses
- Replacement therapy for pts with structural or functional disorders of the adrenal cortex, pituitary, or hypothalamus
- Adrenocorticosteroids for Addison's disease (primary adrenocortical insufficiency), for 2° or 3° adrenocortical insufficiency, and for congenital adrenal hyperplasia
- Sex hormones for deficiency (as in 1° hypogonadism and postmenopause) and for contraception
- Glucocorticoids are used to treat
  - Inflammation: Crohn's disease, ulcerative colitis, asthma, arthritis, airway and cerebral edema, spinal cord injury, glomerulonephritis
  - Immunological disorders: Rheumatic disorders, skin disorders (for example eczema), allergic reactions, nephrotic syndrome, anti-rejection post transplantation
- Cancer
- Antenatal glucocorticoid therapy in women at risk for preterm delivery reduces the incidence of neonatal RDS, intraventricular hemorrhage, necrotizing enterocolitis, sepsis, and neonatal mortality by approx 50 percent.
- Glucocorticoids are used to establish the diagnosis and cause of Cushing's syndrome (the dexamethasone suppression test).
- Glucocorticoids may also be used as an anti-emetic agent in the periop period.

Perioperative Risks
- The underlying condition warranting the use of steroids may carry its own inherent risks in the periop period.
- Long-term corticosteroid therapy induces adrenal insufficiency (inability to mount an adequate stress response to surgery), osteoporosis (increased risk for fracture), impaired wound healing, increased risk of infection, increased fragility of skin and other tissues (mild pressure may cause skin ulceration, tape may tear skin, sutures may not hold), cushingoid body habitus (central obesity, buffalo hump, puffy and/or moon face may create a difficult airway), Htn, peptic ulcers, and psychoses.
- Oral contraceptives can increase risk for thromboembolism, thrombophlebitis, Htn, myocardial infarction, and cerebral thrombosis if aspirin is not used concurrently. These adverse effects are most common among women who smoke.

Worry About
- Fluid and electrolyte disturbances
- Hypo- and/or Htn and myocardial events
- Adrenal insufficiency
- Hyperglycemia
- Possible difficult airway with cushingoid body habitus

Overview/Pharmacology
- Steroids are lipids formed of a sterane core with varying functional groups and states of oxidation that alter their physiological effects.

Drug Class/Usual Dose
- Dosing of steroids will vary depending on biologic potency, pharmacokinetic properties, disease process, concurrent medications, route of administration, and type of surgery.
- Mineralocorticoid
  - Fludrocortisone: 0.05–0.2 mg/d (oral adult physiologic replacement)
- Glucocorticoid
  - Hydrocortisone: 20–30 mg/d (oral adult physiologic replacement), 100 mg bolus followed by 200–300 mg/d (IV adult stress dose), 1–2 mg/kg/dose (IV pediatric stress dose)
  - Cortisone: 25–35 mg/d (oral adult physiologic replacement)
- Antenatal glucocorticoid treatment for accelerating fetal lung maturity:
  - Betamethasone (two doses of 12 mg given IM 24 hr apart)
  - Dexamethasone (four doses of 6 mg given IM 12 hr apart)

<table>
<thead>
<tr>
<th>DRUG EFFECTS</th>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Excess: Htn, premature CAD</td>
<td>Headache, chest pain, SOB, diaphoresis, poor exercise tolerance</td>
<td>Blood pressure, auscultation</td>
<td>EKG, ECHO</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Deficit: Hypotension, decreased response to vasoconstrictors</td>
<td>Postural Sxs, syncope</td>
<td>Orthostatic VS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Decreased airway reactivity</td>
<td>Decreased use of rescue inhalers, decreased SOB and cough</td>
<td>Decreased wheezing</td>
<td>Spirometry</td>
<td></td>
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<tr>
<td>CNS</td>
<td>Changes in mood/behavior, brain excitability, insomnia, psychosis</td>
<td>Symptoms of depression, personality change, insomnia, psychosis</td>
<td>Mental exam</td>
<td></td>
<td></td>
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<tr>
<td>GI</td>
<td>Decreased Ca²⁺ absorption</td>
<td>Abd pain, GI bleed, N/V</td>
<td>Abd pain</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Gastritis, PUD, pancreatitis</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>ENDO</td>
<td>Central redistribution of body fat</td>
<td>Wt gain</td>
<td>Obesity, buffalo hump, moon faces, supraclavicular fat, thin extremities</td>
<td>Glucose, insulin levels</td>
<td></td>
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<tr>
<td></td>
<td>Increase blood glucose, insulin resistance</td>
<td>Polydipsia, polyuria</td>
<td>Refractory hypotension</td>
<td>Lipid panel</td>
<td></td>
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<tr>
<td></td>
<td>Adrenal insufficiency</td>
<td>Lethargy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Increase free fatty acids</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>RENAL</td>
<td>Excess: Positive Na⁺ balance, increased extracellular fluid, Ca²⁺ excretion, hypokalemia, alkalosis</td>
<td>Fluid retention</td>
<td>Edema</td>
<td>Lytes, ABG, BUN/Cr</td>
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<td></td>
<td>Deficit: Na⁺ wasting, decreased extracellular fluid, hyperkalemia, acidosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>MS/DERM</td>
<td>Excess: Muscle wasting/myopathy</td>
<td>Muscle wasting, weakness</td>
<td>Decreased muscle strength, bulk and tone</td>
<td>X-ray, bone density</td>
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<td></td>
<td>Osteoporosis/osteonecrosis</td>
<td>Hx of pathologic fractures/bone pain</td>
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<td></td>
<td>Skin thinning, acne, striae, alopecia, hirsutism, edema</td>
<td>Skin/hair changes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Deficit: Weakness, fatigue</td>
<td>Fatigue, weakness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Excess: Polycythemia, decreased lymphocytes, increased polymorphonuclear leukocytes, immunosuppression</td>
<td>Increased infections</td>
<td>Signs of infection</td>
<td>CBC, differential</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Deficit: Anemia</td>
<td>Fatigue</td>
<td>Pallor</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Perioperative Implications

Preoperative Concerns
- Correction of fluid, electrolyte, and metabolic disturbances
- Evaluation for hypo- and/or Htn, CAD and/or myocardial events, and DM and/or hyperglycemia
- Airway evaluation (for upper airway obstruction from excess facial tissue or edema, Mallampati, mouth opening, degree of neck extension with buffalo hump or cervical arthritis, obesity, dentition)
- Adequate IV access given superficial blood vessels and friability of tissues
- Co-morbid conditions

Induction/Monitoring/Maintenance
- No specific anesthetic drug or technique has advantages in a pt on steroids; however, hemodynamic instability may occur if standard dosing is used in a hypovolemic pt.
- Induction agents, maintenance, and monitoring should be tailored to the co-morbidities of the pt and the surgical needs.
- Consider avoiding etomidate, which can inhibit adrenocorticosteroid production for up to 8 hr
- Monitor for hypo- and/or Htn, hyperglycemia, myocardial events, fluid and electrolyte disturbances
- Administer stress doses of glucocorticoid to any pt who has clinical Cushing’s syndrome or any pt who has received more than 20 mg/d of prednisone or its equivalent (16 mg/d of methylprednisolone, 2 mg/d of dexamethasone, or 80 mg/d of hydrocortisone) for more than 3 wk in the past year:
  - For minor surgery: Usual steroid dose (no extra supplementation required)
  - For moderate surgical stress: In addition to usual steroid dose, administer hydrocortisone 50 mg IV on induction and 25 mg 8 q for 24 hr, then resume baseline dosing
  - For major surgery: In addition to usual steroid dose, administer 100 mg of IV hydrocortisone on induction and 50 mg every 8 q for 24 hr, then taper dose by half per day to baseline dosing
- Careful positioning and padding of pressure points (important because pts on steroids are predisposed to osteopenia and/or skin friability)
- Steroids can prolong the effect of muscle relaxants; therefore, ensure adequate strength prior to extubation.

Postoperative Period
- Monitor for hypo- and/or Htn and myocardial events
- Monitor for signs of adrenal insufficiency (hypotension)
- Continue exogenous steroid administration postop. Pts should revert to their baseline dose within 48 hr of surgery unless their clinical condition warrants otherwise.
- Consider compression devices to prevent venous thrombosis in nonambulatory pts (esp. smokers taking oral contraceptives who are not receiving concomitant aspirin)

Anticipated Problems/Concerns
- Fluid, electrolyte, and metabolic disturbances
- Adrenocortical suppression
- Strict attention to sterile technique and periop antibiotics (pts on steroids are at increased risk for infection to immunosuppression)
Tacrolimus (FK-506)

### Indications
- Rescue of 1st immunosuppressant Rx following liver, lung, heart, pancreas and limb transplant
- Approx candidates: 3000 liver and 9000 kidney transplants in USA; 15,000 living liver, 50,000 kidney transplant recipients chronically receiving immunosuppressants
- Has been used to suppress the inflammation assoc with ulcerative colitis

### Preoperative Risks
- Htn: Ca^2+-channel blockers may be effective in treating tacrolimus-assoc Htn, but care required. Interference with tacrolimus metabolism may necessitate a dosing reduction
- Nephrotoxicity: Do not administer concurrently with cyclosporine; administer cautiously with other potentially nephrotoxic drugs, e.g., aminoglycoside antibiotics

### Worry About
- Drug is metabolized by cytochrome P450 3A enzyme system. Other medications that inhibit or induce this enzyme may affect tacrolimus drug levels.

### Overview/Pharmacology
- General effect: Macrolide antibiotic with potent immunosuppressive properties, often used for rescue therapy in liver transplant pts with rejection refractory to other immunosuppressants
- Tacrolimus metabolized by liver; metabolites primarily excreted in bile; elimination T1/2 of 8.5 hr prolonged with hepatic dysfunction
- Ca^2+-channel blockers, cyclosporine, erythromycin, antifungal agents, metoclopramide may ↑ blood levels of tacrolimus as function of P450 inhibition

### Drug Class/Mechanism of Action/Usual Dose
- Macrolide antibiotic, highly protein bound (>75%), binds primarily to albumin and/or α1-glycoprotein
- Tacrolimus binds to calcineurin, blocking production of interleukin-2, thereby inhibiting further T-lymphocyte proliferation, immunosuppression
- Dosing: IV 0.05–0.1 mg/kg/d; PO 0.15–0.3 mg/kg/d in 2 divided doses

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENERAL</td>
<td>Hypersensitivity, rash</td>
<td>Observe 1/2 hr; have epinephrine 1:1000 available</td>
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<tr>
<td>CARDIO</td>
<td>Htn</td>
<td></td>
<td>BP/HR</td>
<td></td>
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<tr>
<td>RESP</td>
<td>Pleural effusion, dyspnea</td>
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<td></td>
<td></td>
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<tr>
<td>GI</td>
<td>Diarrhea, N/V, constipation, abn liver function, anorexia, abd pain</td>
<td></td>
<td>LFTs</td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Abn kidney function, oliguria</td>
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<td>BUN, Cr</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperkalemia, hypokalemia, hyperglycemia</td>
<td></td>
<td>K^+ glucose</td>
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</tr>
<tr>
<td>HEME</td>
<td>Anemia, leukocytosis, thrombocytopenia</td>
<td></td>
<td>CBC</td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Headache, tremor, insomnia, paresthesias, mental status changes, circumoral numbness</td>
<td></td>
<td>Preop neuro exam</td>
<td></td>
</tr>
</tbody>
</table>

### Key Reference:

### Perioperative Implications
#### Preoperative Preparation
- Continue all immunosuppressants through periop period
- Monitor levels: Therapeutic range 5–30 ng/mL; maintenance level 5–10 ng/mL

#### Monitoring
- Consider frequent NIBP or arterial catheter

### Induction/Maintenance
- Inducers of P450 system incl phenobarbital, phenytoin, isoniazid; some volatile anesthetics may result in ↑ metabolism of tacrolimus
- Inducers of P450 system incl phenobarbital, phenytoin, isoniazid; some volatile anesthetics may result in ↑ metabolism of tacrolimus

### Possible Drug Interactions
- Ca^2+-channel blockers, cyclosporine, erythromycin, antifungal agents, metoclopramide may ↑ blood levels of tacrolimus as function of P450 inhibition
- Anticonvulsants (carbamazepine, phenobarbital, phenytoin), rifampin may ↓ blood levels of tacrolimus 2° to induction of cytochrome P450 system
- Adverse effects requiring dose adjustments incl nephrotoxicity, neurotoxicity, alterations in glucose metabolism, infection, and susceptibility to malignancy

### Anticipated Problems/Concerns
- Hypersensitivity may occur with IV formulation
Terbutaline

**Uses**
- Prescribed for pts with bronchospasm caused by asthma, bronchitis, or emphysema
- Effective for acute asthmatic attacks, COPD
- Used as tocolytic for preterm labor (not FDA-approved for this and use in preterm labor has declined over the last decade)

**Perioperative Risks**
- Complications incl. Tachyarrhythmias
- Hypokalemia
- Hyperglycemia
- Hyperglycemia failure vs. noncardiogenic etiologies
- Pulm edema (unclear mechanism: myocardial
- Tachycardia/palpitations
- Decrease in BP and uterine tone
- Bradyarrhythmias and noncardiogenic etiologies
- Hypokalemia
- Rebound hyperkalemia has been described in OB pts who have terbutaline D/C

**Worry About**
- Tachycardia/palpitations
- Pulm edema (unclear mechanism: myocardial failure vs. noncardiogenic etiologies)
- Hyperglycemia
- Hypokalemia
- Fetal side effects are increased fetal heart rate and neonatal hypoglycemia.

**Overview/Pharmacology**
- Used for both acute bronchospasm, chronic management of COPD
- Tachyphylaxis possible with prolonged use
- 7–14% of delivered aerosol reaches circulation
- ½ SQ dose metabolized in liver to inactive sul-fate conjugates
- Metabolites I and II, unchanged drug excreted in urine

**Onset/Duration**
- SQ
  - Onset: Significant ↑ in FEV₁ in 15 min, peak in 30–60 min
  - Duration: 1.5–4 hr, T½ 3–4 hr
- IV (not FDA-approved route)
  - Onset: Immediate; T½ 3–4 hr
  - PO
  - Onset: Significant improvement in FEV₁ in 60–120 min
  - Duration: At least 4 hr
- Metered dose inhaler/nebulizer
  - Onset: 5 min, peak, 1–2 hr
  - Duration: 3–4 hr

**Drug Class/Mechanism of Action/Usual Dose**
- β₂-agonist (thought to be more specific for β₂ receptors)
- β₂ stimulation ↑ adenyl cyclase conversion of ATP to cAMP, this effect → cell hyperpolarization, ↑ inward Ca²⁺ flux, → relaxation of bronchial, uterine, vascular smooth muscle

**Usual Dose**
- SQ: 0.005–0.01 mg/kg to a max 0.25 mg/dose; inject every 15–20 min as needed
- PO: 5 mg tid; reduce to 2.5 mg tid if side effects occur. Not to exceed 15 mg/24 hr
- Metered dose inhaler: 2 inhalations every 4–6 hr (200 μg/actuation)
- Nebulizer: 0.01–0.03 mL/kg (1 mL = 1 mg); minimum = 0.1 mL, maximum = 2.5 mL; dilute in 1–2 mL N/S
- IV (not FDA approved) for tocolysis: IV 2.5–10 mcg/min; increased every 10–20 min. Maximum dosages from 17.5–30 mcg/min described.

**Perioperative Implications**

**Preoperative Concerns**
- Evaluate the disease being treated: Asthma, preterm labor
- For asthmatic pts, consider administering inhaled β₂-agonist before inducing anesthesia
- For pts in preterm labor, assess fetal well-being: FHR, wt, indices of lung maturity, etc.
- Evaluate VS, esp. HR, BP, R/O CHF
- Assess volume status (avoid excess hydration in obstetrical pts as it could increase the risk for pulm edema)
- Lab studies to check: Glucose, K⁺

**Induction/Maintenance**
- Increasing CO may prolong inhalation induction
- Intraop bronchospasm possible with inhaled or IV terbutaline; absorption after SQ injection possibly unreliable
- Tachycardia possible 2° to the effects of the drug, not necessarily as a result of light anesthesia

**Anticipated Problems/Concerns**
- Increased HR, decreased SVR possibly not tolerated well by pts with CAD, mitral or aortic stenosis
- Usually associ with hypokalemia, but rebound hyperkalemia in pts has been described in pts who have terbutaline therapy D/C
- Pulm edema may be a result of myocardial failure or of a noncardiogenic etiology.


**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Tachycardia, Htn, hypotension, arrhythmias, ↓ SVR</td>
<td>Palpitations</td>
<td>↑ HR, irregular rhythm, BP; ECG</td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Bronchodilation</td>
<td>Dyspnea</td>
<td>↓ Wheezing</td>
<td>O₂ sat, PFT, PEF</td>
</tr>
<tr>
<td>GI</td>
<td>Nausea</td>
<td>Nausea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperglycemia, hypokalemia*</td>
<td>Polydipsia, polyuria</td>
<td>Dehydration</td>
<td>Blood glucose, serum K⁺</td>
</tr>
<tr>
<td>CNS</td>
<td>CNS stimulation</td>
<td>Insomnia, anxiety, hyperactivity, drowsiness, headache</td>
<td>Tremor</td>
<td></td>
</tr>
</tbody>
</table>

*Plasma hypokalemia is due to intracellular transport of K⁺. Hypokalemia is seen most often with IV terbutaline Rx for preterm labor. K⁺ supplementation rarely required, serum levels usually normalize within 3 hr of D/C of infusion.

**Usual Dose**
- SQ: 0.005–0.01 mg/kg to a max 0.25 mg/dose; inject every 15–20 min as needed
- PO: 5 mg tid; reduce to 2.5 mg tid if side effects occur. Not to exceed 15 mg/24 hr
- Metered dose inhaler: 2 inhalations every 4–6 hr (200 μg/actuation)
- Nebulizer: 0.01–0.03 mL/kg (1 mL = 1 mg); minimum = 0.1 mL, maximum = 2.5 mL; dilute in 1–2 mL N/S
- IV (not FDA approved) for tocolysis: IV 2.5–10 mcg/min; increased every 10–20 min. Maximum dosages from 17.5–30 mcg/min described.

**Tetracyclines**

**Uses**
- Administered PO (most common), IV (fewer side effects), IM (rare, painful), topical (eyes only)
- Original broad-spectrum antibiotic with activity against gram-positive and gram-negative bacteria; species of *Chlamydia, Rickettsia, and Mycoplasma* (in adults); and some protozoa. One of few agents active against organisms without cell walls. Resistance worldwide.
- Secondary uses: Alternative drugs in the treatment of syphilis, tx of resp infections caused by susceptible organisms, prophylaxis against infection in chronic bronchitis, tx of leptospirosis, and in the tx of acne.
- Selective uses
  - Tetracycline used for the tx of GI ulcers caused by *Helicobacter pylori*.
  - Doxycycline for Lyme disease, the prevention of malaria, and the tx of amebiasis.
  - Minocycline for meningococcal carriers
  - Demeclocycline for the management of pts with ADH-secreting tumors.
- Incidence in USA: 20 million doses/y

**Perioperative Implications**
- IV tetracycline frequently → thrombophlebitis, lessens efficacy of oral contraceptives
- Decrease dose with age
- Decrease dose in those with poor renal/hepatic functions as tetracycline accumulates in such pts, and can lead to hepatic toxicity (instead in pts with renal dysfunction use doxycycline which has an unchanged elimination half-time in such pts)
- Barbiturates may ↓ T½ β, tetracycline will ↑ cone of digoxin, warfarin. Pts may exhibit GI distress, even *Clostridium difficile* colitis.

**Worry About**
- Tetracycline (esp. 1st generation) absorbed poorly if given within 3 hr of di-/trivalent cations (Ca²⁺, Al³⁺, Mg²⁺, Fe²⁺, Bi³⁺)
- The possibility of tetracycline-resistant bacterial endotoxins as well as GI distress limit the oral dose of these antibiotics
- Doxycycline should only be administered orally or by IV

**Overview/Pharmacology**
- Two generations: First (e.g., tetracycline); second (e.g., doxycycline)
- Classified as bacteriostatic (newest ones possibly bactericidal)
- First generation T½ β 6–12 hr, excreted in urine, feces
- Second generation more lipophilic, greater Vd, recirculation, T½ β 16–18 hr; doxycycline excreted 90%+ in feces; safe for anephric pts
- Adjust dose with age, impaired renal/hepatic functions
- PO uptake in duodenum (esp. first generation); peak level, 2 hr; IV peak level, 1 hr

**Drug Class/Mechanism of Action/Usual Dose**
- Original broad-spectrum antibiotic
- Effective against *Rickettsia, Mycoplasma, Chlamydia, Borrelia*, spirochetes, some fungi
- Local irritant (sclerotherapy)
- Normal dose: Impairs bacterial protein synthesis; binds via a Mg²⁺ bridge to single active site of 30 S subunit of bacterial ribosome; prevents binding of aminoacyl tRNA to the mRNA-ribosome complex. Without this codon–anticodon interaction, peptide chain formation cannot proceed.
- Inhibit collagenase (osteoarthritis), tumor-induced angiogenesis (chemoRx)
- Usual dose: Doxycycline, 100 mg PO bid

**ASSESSMENT POINTS**

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<td>HEENT</td>
<td>Frequently causes thrombophlebitis ↓</td>
<td>Hepatitis</td>
<td>LFIs</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Rare toxicity, esp. with ↑ dose, IV route, preg; usually reversible with drug cessation</td>
<td></td>
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<td>HEPAT</td>
<td>Disturbances in the normal flora lead to candidiasis (oral and vaginal)</td>
<td>Mild N/V, severe colitis</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>May inhibit/suppress antibody production, leukotaxis, complement system</td>
<td></td>
<td></td>
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<tr>
<td>HEME</td>
<td>May aggravate uremia in susceptible pt; crosses placenta, excreted in breast milk</td>
<td>BUN</td>
<td></td>
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<tr>
<td>CNS</td>
<td>Penetrates CNS; may ↑ ICP during Rx, esp. in infants</td>
<td>Vision change, headache</td>
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<tr>
<td>DERM</td>
<td>Phototoxic skin reaction, esp. 1st generation</td>
<td>Dizziness, nausea</td>
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<tr>
<td>MS</td>
<td>↓ Bone growth in preemies, ↓ collagenase in joints</td>
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**Perioperative Concerns**
- May ↑ digoxin levels, higher prothrombin time if pt on warfarin

**Possible Drug Interactions**
- Methoxyflurane, tetracycline may → renal failure
- Barbiturates may ↓ T½ β

**Anticipated Problems/Concerns**
- Although resistance is rising, drugs remain useful antibiotics, with nonantibiotic indications increasing
- Contraindicated in pregnancy and childhood

Thyroid Supplements

Uses
- Incidence in USA: >3 million chronic users
- $T_4$ prescribed for pts with chronic hypothyroidism
- $T_3$, successfully used as rescue therapy for cardiogenic shock post-CPB
- $T_4$, reported as favorably administered to brain-dead donors before organ harvesting for heart, heart-lung transplantation; N.B. prophylactic $T_3$

Worries About
- $T_4$ or $T_3$ can aggravate Sx of myocardial ischemia

Overview/Pharmacology
- Hypothyroidism (overt) estimated at 0.5–3.8% of adults, ↑ with age (over 15% of women at age 60+)
- Post-thyroidectomy <30% of pts euthyroid at 10 y due to inadequacy or D/C of therapy
- Reversal of clinical Sx of chronic hypothyroidism, including myocardial edfusions, requires 2–4 mo Rx
- $T_4$ for $T_3$: 7 d, $T_3$: 1.5 d
- $T_3$, relatively inactive prohormone undergoing monodeiodination in liver, kidney to biologically active $T_3$

ICD-9-CM Code: 244.9

Perioperative Implications/Possible Drug Interactions

Preoperative Concerns
- Thyroid hormones ↑ breakdown of vitamin K-dependent clotting factors—can alter coag status
- Chronic amiodarone therapy may produce hyper- or hypothyroidism

Induction/Maintenance
- Exaggerated Htn, tachycardia can occur with agents such as ketamine, exogenous catecholamines
- $T_3$ replacement can produce detrimental increases in O$_2$ requirements (esp. myocardial), protein catabolism without improving mortality rates

Adjuvants/Regional Anesthesia/Reversal
- Anticholinergics with minimal CV effects, e.g., glycopyrrolate, preferred over atropine
- Caution in the presence of spinal anesthesia, $T_3$ administration may produce aggravated hypotension

Postoperative Period
- Cirrhosis, sepsis, renal failure, surgery may all ↓ peripheral conversion of $T_4$ to $T_3$ (euthyroid sick syndrome), precipitate hypothyroidism

Drug Class/Mechanism of Action/Usual Dose
- Thyroid hormone replacement Rx
- $T_3$, binding to specific membrane receptor proteins augments membrane transport activity, mitochondrial oxidative phosphorylation, protein synthesis
- Extraneural effects of $T_4$ occur in min, ↑ myocardial mitochondrial and transmembrane transport activity
- Nuclear effects of $T_3$ occur within 0.5–1.0 hr, involve transcription, translation of myocardial enzymes, contractile proteins
- Direct effect of $T_3$, ↓ arterial smooth muscle tone
- Usual dosage of $T_3$ is 0.15 mg/d PO
- Acute Rx: $T_4$, 0.3–0.5 mg by slow IV infusion followed by 0.1–0.15 mg/d, or $T_3$, 0.005–0.01 mg IV

OVERVIEW
- $T_3$: Binding to specific membrane receptor proteins augments membrane transport activity, mitochondrial oxidative phosphorylation, protein synthesis
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Anticipated Problems/Concerns
- In critically ill pts, $T_3$ replacement can produce detrimental increases in O$_2$ requirements (esp. myocardial), protein catabolism without improving mortality rates
**Tissue Plasminogen Activator**

**Uses**
- Used for thrombolysis during Rx of pulm embolism, stroke, and myocardial infarction. Best used within 3 hr of incident or 6 hr if injected via. artery directly into the site of occlusion and after exclusion of intracranial hemorrhage via CT scan.
- Rapid clot lysis by t-PA offers advantages in comparison with streptokinase.
- May be used in combination with other anticoagulants such as heparin and aspirin. Also may be combined with β-blockers, morphine, nitroglycerin, and plt IIb/IIIa blockers.

**Perioperative Risks**
- Increased bleeding during surgery; if severe, possible need for blood transfusion, fresh frozen plasma, cryoprecipitate, and plt infusion therapy.
- Intraop Htn may increase the risk of intracranial hemorrhage (about 1%).
- Incomplete restoration of coronary flow and persisting thrombogenicity may lead to cardiac instability and risk for periop infarction.

**Overview/Pharmacology**
- Thrombolytic agent; natural t-PA is produced by vascular endothelial cells and is naturally released from the endothelium in response to venous occlusion, physical activity, stress, or vaso-active medications.
- Accelerates the conversion of plasminogen bound to fibrin, to plasmin, resulting in fibrinolysis as the site of clot.
- t-PA (alteplase) is commercially produced using cDNA for natural t-PA, transfected into a mammalian cell line.
- Initial thrombolytic response is seen within 30 min when given IV. T½ ~5 min; elimination T½ ~30–50 min. Eighty percent cleared from plasma within 10 min of stopping a standard infusion and clearance is via the liver.

**Drug Class/Mechanism of Action/Usual Dose**
- Thrombolytic agent. Binds to fibrin threads of a thrombus, converts enmeshed plasminogen to plasmin which initiates localized fibrinolysis. For this reason, unlike streptokinase, t-PA can be considered fibrin specific; t-PA lacks effect on circulating plasminogen thereby limiting systemic effects.
- In myocardial infarction, usual dose for pts 70 kg or more is a front-loading protocol, with 100 mg t-PA being given IV by bolus and infusion over 90 min, with heparin.
- Amount of salvaged myocardium is directly related to the time until the occluded artery is reopened. GUSTO I investigators showed 84% patency within 6 hr of front-loaded t-PA.
- More rapid lysis, less systemic fibrinolysis, and few if any anaphylactic reactions when compared to streptokinases—but t-PA is more expensive.
- In ischemic stroke, IV t-PA dose is lower; heparin is not used.

**Assessment Points**

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<td>Bleeding from vascular puncture sites</td>
<td>Hematoma</td>
<td>Hgb</td>
<td>Manual compression</td>
</tr>
<tr>
<td></td>
<td>Severe bleeding during surgery</td>
<td>Check if heparin or plt IIb/IIIa blockers are being given</td>
<td>Hgb, Pts, APTT</td>
<td>Rarely is blood transfusion necessary</td>
</tr>
<tr>
<td></td>
<td>Effects of ancillary treatment</td>
<td>Check for ongoing β-blocker, nitroglycerin, or morphine treatment</td>
<td></td>
<td>Transfusion of blood, FFP, cryo Factor VIII and plt may be needed, consider using TEG to guide therapy</td>
</tr>
<tr>
<td></td>
<td>Reperfusion arrhythmias</td>
<td>Can occur on restoration of blood flow to ischemic myocardium</td>
<td>CV stability</td>
<td>D/C if necessary; however, β-blockade has considerable benefit with little risk in most pts</td>
</tr>
<tr>
<td>CNS</td>
<td>Intracranial hemorrhage</td>
<td>Signs of stroke or raised intracranial pressure</td>
<td>Neurologic assessment, Urgent CT, MRI</td>
<td>Supportive BP control (risk is increased in presence of heparin)</td>
</tr>
</tbody>
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**Perioperative Implications**
- Danger of bleeding with central line placement, arterial cannulations.
- Risk of hypotension on anesthetic induction with adjuvant nitroglycerin infusion.
- Severe Htn may predispose to or exacerbate hemorrhagic stroke.
- Residual thrombus is highly thrombogenic, posing risk of rethrombosis.
- Regional anesthesia should not be used with caution.
Tranexamic Acid

Indications
- To prevent bleeding due to fibrinolysis after surgery or trauma (cardiac surgery with and without cardiopulmonary bypass; liver transplantation; orthopedic incl spine; GU surgery; peripartum hemorrhage). Can be diagnosed clinically or via laboratory tests (prolonged thrombin time, reduced fibrinogen levels, increased d-dimer levels, classic teardrop shape on thromboelastography).
- For short-term use (2–8 d) in hemophilia and von Willebrand disease pts to reduce or prevent hemorrhage and to reduce the need for replacement therapy during and following tooth extraction.
- To treat 1st menorrhagia, gastric and intestinal hemorrhage, urinary tract bleeding, recurrent epistaxis, and hereditary angioneurotic edema. The drug also inhibits induced hyperfibrinolysis during thrombolytic treatment with plasminogen activators.
- Used in pts with hemophilia or those receiving anticoagulation, about to undergo oral surgery.

Perioperative Risks
- Side effects of drug: Nausea, diarrhea, vomiting, and abdominal pain are the most common adverse effects (approx 30% with oral use)
- Giddiness has been reported
- Hypotension (if the drug is injected too rapidly)

Worry About
- Potential for thrombotic complications 2–4 weeks after the inhibition of fibrinolysis.

Overview/Drug Class
- A synthetic lysine analogue. Prevents plasminogen conversion and therefore fibrinolysis by occupying plasminogen’s lysine-binding site for fibrin.
- Has a structure similar to that of lysine, and reversibly binds to lysine-binding sites for fibrin on plasminogen, thereby blocking the binding of plasminogen to fibrin. Plasminogen activators are located on the fibrin clot. Without localized binding of plasminogen to fibrin, it cannot be converted to plasmin.
- Because fibrinolysis requires plasminogen (and plasmin) binding to fibrin, fibrinolysis is inhibited.
- A competitive inhibitor of plasminogen activation and at much higher concentrations, a noncompetitive inhibitor of plasmin. Suppresses fibrinolysis by inhibiting activation of plasminogen.
- Other antifibrinolytic medications incl epsilon-aminocaproic acid (lysine analog) and aprotinin (serine protease inhibitor).
- Reductions in mortality rates with TXA doses of 4.5 grams to 6 grams daily for 5 to 7 d (in most studies) produced statistical significance between TXA and placebo.
- TXA was assoc with reductions in mortality of 5%-54% in pts with upper GI bleeding compared with placebo. Meta-analysis indicated a reduction of 40%.
- Administered either PO 25 mg/kg every 6–8 hr or IV 10 mg/kg 6–8 h beginning the day prior to surgery
- Absorption after oral use is 30–50%; bioavailability is not affected by food.
- An antifibrinolytic concentration of drug remains in serum up to 7–8 hr.
- The protein binding to plasminogen is approx 3% at therapeutic plasma levels; it does not bind to serum albumin.
- The T1/2 of elimination when administered orally is 120 min.
- Urinary excretion is the main route of elimination via glomerular filtration.
- Overall renal clearance is equal to overall plasma clearance, and >90% of the dose is excreted unchanged in 24 hr.
- Pts with renal insufficiency should have their doses reduced according to creatinine clearance. Only a small fraction of tranexamic acid is metabolized.
- TXA is 6 to 10 times more potent in terms of binding to plasminogen/plasmin than epsilon-aminocaproic acid (EACA).
- Concurrent administration of heparin does not influence the activity of TXA.
- Pharmacokinetic properties: Maximum plasma concentrations of TXA can be attained within 3 hr after an oral dose. Elimination after IV administration is triexponential, and over 95% of each dose is eliminated unchanged in the urine.

ASSESSMENT POINTS

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<tr>
<td>HEENT</td>
<td>Retinal degeneration is assoc with prolonged use; incidence 25–100% and dose-dependent (animal studies)</td>
<td>Visual changes</td>
<td>Ophthalmologic exam in pts receiving tranexamic acid &gt;4 or 5 d</td>
<td>Visual acuity</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Hypotension (with rapid infusion)</td>
<td>Mental status changes, nausea</td>
<td>BP monitoring, heart rate, ECG</td>
<td>Color vision, Eyeground</td>
</tr>
<tr>
<td>RENAL</td>
<td>Reduce dose in pts with renal insufficiency</td>
<td></td>
<td></td>
<td>BUN/Cr, CrCl</td>
</tr>
<tr>
<td>GI</td>
<td>Nausea, diarrhea, vomiting, abd discomfort</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OB</td>
<td>Category B</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>IMMUNO</td>
<td>Male mice receiving tranexamic acid up to 5 g/kg/d have been found to develop leukemia</td>
<td></td>
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Perioperative Implications

Airway
- No interactions known

Preinduction/Induction
- If given IV, inject slowly to avoid hypotension

Maintenance
- No interactions known

Emergence
- No interactions known

Adjuvant/Regional Anesthesia/Reversal
- No interactions known

Contraindications
- Acquired defective color vision: Prohibits measuring one endpoint of toxicity
- Subarachnoid hemorrhage: Cerebral edema and cerebral infarction may be caused by tranexamic acid in pts with subarachnoid hemorrhage.

Active Thromboembolic disease
- Caution in the setting of disseminated intravascular coagulation (DIC), in which inhibition of fibrinolysis may aggravate the hypercoagulable state
- Reduced dose in renal insufficiency

Anticipated Problems/Concerns
- Potential for increased thrombotic events.
**Trimethaphan**

**Uses**
- Production of controlled hypotension during surgery to reduce bleeding into the surgical field
- Rapid reduction of BP in the treatment of hypertensive emergencies.
- Acute dissecting aortic aneurysm, particularly when pre-existing conditions make β-blockers a relative contraindication
- Emergency treatment of pulm edema in pts with pulm Htn assoc with systemic Htn
- May serve as an alternative to sodium nitroprusside for pts who are resistant to this drug, or can be mixed with nitroprusside to decrease risk of cyanide toxicity from nitroprusside
- May serve as a cost efficient alternative to nicardipine or clevidipine

**Perioperative Risks**
- High doses may cause profound hypotension and, rarely, resp arrest
- QRS prolongation has been seen during treatment
- Tachycardia, angina, or syncope may occur without warning
- Due to trimethaphan’s ability to cross the placenta, its ganglionic blocking effects may decrease GI motility in the fetus, resulting in meconium ileus or neonatal paralytic ileus
- CNS exam limited by production of mydriasis

**Worry About**
- Contraindicated in pts with shock, anemia, hypovolemia, uncorrected resp insufficiency, or neonates at risk for paralytic or meconium ileus
- Orthostatic hypotension; may cause severe hypotension
- Difficult to obtain as no longer manufactured for use in USA

**Overview/Pharmacology**
- Rapidly acting ganglionic acetylcholine blocker, onset 1–3 min
- Peak response 5–10 min
- Duration of action: 10–15 min for single dose
- Affects both parasympathetic and sympathetic pathways
- Renally excreted, mostly unchanged
- Most side effects are due to parasympathetic blockade and respond to dose reduction or D/C
- Cardiac output may increase in pts with CHF, or decrease in pts with normal heart function
- Tachyphylaxis may occur during continuous IV infusion

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**Perioperative Implications**

**Preoperative Concerns**
- Assess Hx of CAD, check baseline EKG
- Assess volume status
- Consider arterial line if trimethaphan infusion anticipated

**Induction/Maintenance**
- May prolong block from succinylcholine or non-depolarizing neuromuscular blockers

**Postoperative Period**
- Mydriasis from drug may interfere with neuro checks for postop neuurosurgery pts
- Risk for paralytic ileus is increased when drug infusion continued for longer than 48 hr
- Pts continued on trimethaphan infusions postop should be monitored in the ICU
- Oral antihypertensives should be instituted and timethaphan D/C as soon as pt can take oral medication and BP has stabilized

**Anticipated Problems/Concerns**
- Not ideal for prolonged infusions as tachyphylaxis may develop within first 48 hr of therapy, although this may be attenuated by concommitent use of a diuretic

**Drug Class/Mechanism of Action/Usual Dose**
- A short-acting ganglionic blocking agent
- Prevents stimulation of postsynaptic receptors by competing with acetylcholine for these receptor sites
- Hypotensive effect is primarily through sympathetic blockade by lowering SVR
- Hypotensive effect is also mediated through direct vasodilation and histamine release (esp. at higher rate of administration)
- Usual adult dosage
  - For controlled hypotension during surgery: Initial: IV infusion, 3 to 4 mg per min, adjusted according to response; Maintenance: IV infusion, 0.3 mg to 6 mg per min.
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  - Pts on concommitent antihypertensive medications require lower doses
**Indications**
- Incidence in USA: 16 million, esp. in elderly 5% under 55; 10% 55 to 64; 10 to 15% of 65 to 74, and 24% of 74 to 80 year olds. 75% of those over 64 with this do not have anemia or even RBC abn.
- Prescribed for pernicious anemia
- Lack of gastric secretion of intrinsic factor → malabsorption of vitamin B₁₂; therefore IM route preferred. Strict vegetarian diet-induced deficiency state; responds to oral supplementation.
- Until you reach midlife, you probably get all the B₁₂ you need from food (unless vegetarian). But sometime around 50, the stomach begins making less of the digestive fluids and intrinsic factor needed to absorb B₁₂. Pts are also almost certainly low on B₁₂ if taking a heartburn med called a proton pump inhibitor for a long time, which seriously diminishes B₁₂ absorption.
- Also assoc with *Helicobacter pylori* infection, chronic alcohol ingestion, and pancreatic exocrine deficiency conditions

**Worry About**
- Permanent neurologic injury (classic combined system disease with paresthesias, balance problems with loss of position and vibratory sense, and lack of myelination in long tracts, in long-term deficiency states)
- Interactions and neurologic injury with folate, methionine synthetase inhibitors, N₂O, which can produce rapid neurologic deterioration

**Overview/Pharmacology**
- Vitamin B₁₂ binds to intrinsic factor (gastric glycoprotein from parietal cells) in GI tract, is absorbed from ileum, bound to transcobalamin II in plasma for transport to tissues. Approx 3 μg of cobalamin secreted into bile/d.
- Excess vitamin B₁₂ admin ↑ urinary excretion
- Vitamin B₁₂ enzymatically converted to two active forms: deoxyadenosylcobalamin, methylcobalamin
- Deoxoyadenosylcobalamin is a cofactor for mitochondrial mutase enzyme that catalyzes t-methylmalonyl CoA to succinyl CoA.

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<td>Achlorhydric or gastrectomy pts at risk; assoc with atrophic glossitis</td>
<td>Burning and tingling of mouth</td>
<td>Small, slick, glistening tongue</td>
<td>Schilling test (for vit B₁₂ absorption)</td>
</tr>
<tr>
<td>HEME</td>
<td>Megloblastic anemia</td>
<td>Apathy, lassitude, fatigue</td>
<td>Pale skin, mucous membranes, esp. nailbeds, palmar surfaces</td>
<td>Peripheral blood smear: Macrocytic hyperchromic RBCs Bone marrow: Megaloblasts, megalaryocytes Plt count</td>
</tr>
<tr>
<td>CNS</td>
<td>Degeneration of dorsal, lateral columns of spinal cord</td>
<td>Numbness, tingling in extremities, difficulty walking</td>
<td>Loss of vibration, vibration, position sense; ataxia, Romberg’s sign, muscle flaccidity</td>
<td>Plasma B₁₂ &lt;150 pM suggests B₁₂ deficiency</td>
</tr>
<tr>
<td>PNS</td>
<td>Neuropathy</td>
<td>Paresthesias, dysesthesias of lower extremities</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications/Possible Drug Interactions**
- Folate admin reverses megaloblastic anemia but does not prevent (may precipitate) spinal cord degeneration.
- N₂O oxidizes vitamin B₁₂, reduces activity of methionine synthetase
- Effect of N₂O can be reversed by large doses of folic acid

**Anticipated Problems/Concerns**
- Extensive interaction between folate and vitamin B₁₂ makes it imperative that pernicious anemia be treated with B₁₂ at same time as folate to prevent CNS degeneration.

**Drug Class/Mechanism of Action/Usual Dose**
- H₂O-soluble B vitamin complex
- Cyanocobalamin administered IM or deep SQ route in doses of 1–1000 μg
- Oral dose to 80 μg can be administered with purified intrinsic factor; 1 U binds 15 μg of cyanocobalamin
- Need glycoprotein (intrinsic factor 60,000 MW) produced by gastric parietal cells for its absorption
- RDA: 2 μg/d for adults
- Vitamin B₁₂ is a quiet, conscientious type; doesn’t get much hype, yet works overtime to keep your brain, your immune system, and your ticker in tip-top shape, even protects against Alzheimer’s, depression, strokes, and vision loss.
- Therapeutic 100 μg SQ every month
**Warfarin (Coumadin)**

**Indications**
- Management of thromboembolic disorders: For prophylaxis, Rx, and prevention of recurrence of thromboembolic event incl DVT, pulm embolism, thrombosis of grafts. Prevention of arterial emboli assoc with prosthetic heart valves, nonvalvular AFib, acute MI. Prevention of MI, stroke, and recurrent MI. Rx for antithrombin III, protein C, protein S deficiency
- Newer indications: After angioplasty, for pts who have had coronary graft thrombosis when taking only ASA or ASA and dipyridamole
- Number of individuals receiving the drug: Unknown

**Perioperative Risks**
- Hemorrhage (minor to major life risk)
- Purple-toe syndrome, or warfarin necrosis
- Teratogenicity in pregnancy (decreases synthesis of vitamin K–dependent clotting factors by fetus)

**Worry About**
- Major drug interactions
- Multiplicity of drugs affecting action of warfarin. List extensive, continually expanding (see later). Be concerned with other drugs that potentiate bleeding (e.g., antiplatelet agents, ASA, NSAIDs); and drugs that displace warfarin from protein-binding sites or ↓ or ↑ vitamin K levels.

**Overview/Pharmacology**
- General effect: Anticoagulant with dose-dependent effect on coagulation

**Pharmacokinetics/Pharmacodynamics**
- Warfarin is a racemic mixture of R and S isomers (R-warfarin; S-warfarin)
- Warfarin warfarin absorbed rapidly from GI tract, reaches max plasma conc in 90 min, has T½ of 36–42 h; time to peak effect 36–72 h; duration after D/C 2–5 d at least
- In circulation, bound to plasma proteins, accumulates in liver. R-warfarin metabolites excreted in urine; S-warfarins eliminated in bile.
- Warfarin resistance or ↓ warfarin effect: When warfarin absorption from GI tract impaired by malabsorption syndromes, concurrent use of liquid paraffin laxatives, cholestyramine resin, or excessive amounts of certain antacids (e.g., Mg trisilicate)
- Vitamin K intake ↑ through diet or administration of vitamin K IM or IV
- With induction of hepatic enzymes, increasing metabolism of warfarin. Enzyme inducers incl anticonvulsants, barbiturates, primidone, carbamazepine, antimicrobials (e.g., griseofulvin, rifampin, nafcillin, ethanol) and smoking
- Increased warfarin effect, or warfarin sensitivity
- Drugs displacing warfarin from albumin ↑ its bioavailability (NSAIDs, ASA, phenytoin sodium, oral hypoglycemic agents, sulfa drugs, nalidixic acid, estrogen, miconazole)
- Deficiency of vitamin K enhances; occurs with malabsorption syndromes and during administration of liquid paraffin laxatives, and clofibrate; after long-term use of oral antimicrobials that deplete intestinal bacterial source of vitamin K. Large doses of vitamin E antagonize action of vitamin K; anabolic steroids, danazol impair synthesis of vitamin K–dependent clotting factors; olestra removes vitamin K.
- Metabolism blocked by phenytoin, chloramphenicol, erythromycin, clofibrate, TCAs, cimetidine, sulfipyrazone, sulfamethoxazoletrimethoprim, thus increasing warfarin effect. Disulfiram (Antabuse) significantly slows metabolism.
- Certain cephalosporins have a warfarin effect themselves—thus contraindicated.
- Elderly, febrile, debilitated pts and those with hepatic dysfunction, hyperthyroidism, or heart failure may have increased warfarin effect.

**Drug Class/Mechanism of Action/Usual Dose**
- Interferes with synthesis of 6 vitamin K–dependent proteins involved in coagulation sequence: Factors II, VII, IX, X; proteins C and S. Before these proteins are released into circulation, they undergo reactions converting glutamic acid residues to carboxylglutamic acid residues and require presence of reduced form of vitamin K.
- Inhibits cyclic interconversion between reduced form of vitamin K and its 2,3-epoxide (vitamin K epoxide)
- Defective clotting factors lacking “carboxyl tail” are produced, impairing coagulation
- Factor II has T½ of 48 h; requires 3–4 d before drops to level when PT significantly prolonged
- No urgent need for anticoagulation: Adult with average body mass, 5 mg/d PO prolongs PT to 1.5 × control value in 36–48 h, if not achieved by third day, daily dose may be adjusted by ↑ or ↓ of 2.5 mg; goal: PT = 1.5–2 × control. Increases bleeding complications when PT is 2.5x control. Once anticoagulation stabilized, warfarin dose should be adjusted to maintain INR of 2–3 for all indications, except mech prosthetic cardiac valves, which require higher level of anticoagulation.
- More urgent need: Heparin anticoagulation first; start warfarin, 10 mg for 2 d

### Table: ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>Vit K deficiency may result from a poor diet, extrapathic biliary obstruction, malabsorption, sterile gut</td>
<td>GI bleeding</td>
<td>Wt/height ratio (BMI)</td>
<td>Hct</td>
</tr>
<tr>
<td>ENDOWT</td>
<td>Malnourished</td>
<td>PT/PTT</td>
<td>Fecal occult blood</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Diuresis, pregnancy ↓ effect; warfarin teratogenic</td>
<td>PT/PTT</td>
<td>INR</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Arthritis pain medications that affect pts—e.g., ASA, NSAIDs—potentiate bleeding</td>
<td>PT/PTT</td>
<td>INR</td>
<td></td>
</tr>
</tbody>
</table>

**Perioperative Implications/Possible Drug Interactions**

**Preoperative Concerns**
- Anticoag: Consider Rx with vitamin K (oral, IM, IV, SQ: 2.5–5 mg/70 kg) or FFP (15–20 mL/kg)
- Monitor this drug: PT, INR

**Possible Drug Interactions**
- Regional: Risk of spinal or epidural hematoma when performing a regional when pt is anticoagulated. Risk theoretically ↑ with anticoagulant. Epidural catheter thought to be assoc with greater risk of spinal or epidural hematoma if no measurably anticoagulant effect from warfarin (e.g., PT nml), but if receiving warfarin, not known if risks of spinal or epidural hematoma significant.

Androstenedione

Overview
- Growing sales trend of 20–30% in USA for both medical and nonmedical use of anabolic-androgenic steroid (AAS)
- Available since 1996 as an OTC nutritional supplement
- Estimates that 10% of anabolic-androgenic steroid (AAS) users are teens
- Estimate that 4.9% of male and 2.4% of female adolescents in USA have used legal androgenic/anabolic steroids
- Current estimates indicate that there are as many as 3 million anabolic-androgenic steroid (AAS) users in USA
- Surveys among community wt trainers attendant gyms and health clubs indicate that AAS use is between 15% and 30%
- As a major precursor to testosterone that is available without a prescription, it is purported to increase strength and athletic performance.
- Used to endogenous testosterone production to enhance athletic performance and recovery from exercise, to keep RBCs healthy, and to heighten sexual arousal and function
- Popularity related to society's preoccupation with sustaining the male libido.

Medical Use
- Testosterone replacement therapy
- Treatment of hypogonadal men
- Age-related sarcopenia
- HIV-related muscle wasting
- Increase in bone mineral density
- Prevention of age-related frailty and falls

Perioperative Risks
- Coagulopathy
- Polycythemia

Pharmacology/Mechanism of Action
- As a member of a group of compounds known as anabolic-androgenic steroids (AAS), these synthetic derivatives of testosterone are thought to possibly restore sex drive and boost muscle mass.
- Testosterone enters the cell by passive diffusion and is converted by 5α-reductase to 5α-dihydrotestosterone, which binds to intracellular androgen receptors.
- It ↑ protein anabolism and ↓ protein catabolism. Nitrogen balance is improved only when there is sufficient intake of calories and protein.
- It stimulates the production of RBCs by enhancing the production of erythropoietic stimulating factor.
- Supplementation of androstenedione in the setting of a rigorous 12-wk resistance-training program resulted in a return of baseline levels of testosterone levels and significant increases in estrone and estradiol levels. No increase in measurable lean body mass or muscular strength when compared with placebo.
- Androstenedione is produced in the gonads and adrenal glands of both male and females.
- It is synthesized from dehydroepiandrosterone and then converted to testosterone by the enzyme 17β-hydroxysteroid dehydrogenase or to estrone by the aromatase enzyme complex.

### DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>↓ HDL, atherosclerosis</td>
<td>Angina</td>
<td>ECG, cholesterol</td>
</tr>
<tr>
<td>GI</td>
<td>Cholestasis, hepatocellular tumors, hepatitis, nausea</td>
<td></td>
<td>Liver enzymes, bilirubin</td>
</tr>
<tr>
<td>HEME</td>
<td>Polycythemia, chronic usage, Suppression of clotting factors, sodium and water retention</td>
<td>Easy bruising</td>
<td>PT, PTT, Lytes</td>
</tr>
<tr>
<td>CNS</td>
<td>Depression, anxiety, behavioral changes, headache</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


#### Usual Dose
- Androstenedione is a direct precursor of testosterone and estrone in both males and females; it might ↑ testosterone levels.
- Marketing claims for increased strength, greater fat-free mass, and improved libido; recommended doses of 100–300 mg/d or 50–100 mg twice daily taken 1 hr before exercise or upon awakening.

#### Contraindications
- Males with carcinoma of the breast, prostate gland
- Women who are or may become pregnant
- Pt with serious cardiac, hepatic, or renal disease

#### Adverse Effects
- Several AAS-induced CV concerns reported incl Htn, left ventricular hypertrophy (LVH), impaired diastolic filling, arrhythmias, erythrocytosis, altered lipoprotein profile, and thrombosis.
- AAS-induced elevations in liver enzymes (alanine- and aspartate-aminotransferases).
- Dermatologic chances such as acne, striae, alopecia, and hirsutism are possible results induced by the action of the AAS on the skin and sebaceous glands.
- Endocrine and/or reproductive effects incl a dose-dependent depression of levels of luteinizing hormone and follicle-stimulating hormone due to the negative feedback loop of the hypothalamic-pituitary-gonadal axis.
- Feminization (gynecomastia) in males due to the aromatization of exogenous testosterone to estrogen metabolites.
- Male users may have their endocrine suppression lead to hypogonadotrophic hypogonadism, testicular atrophy, sperm morphology, infertility, and changes in libido.
- Female-specific side effects of AAS incl hirsutism, increased facial hair, voice deepening, clitoral hyper trophy, oligomenorrhea, reduced breast tissue, and male-pattern baldness.
- Restoration of hypothalamic-pituitary homeostasis, endogenous testosterone, and spermatogenesis may take between 3 and 12 mo after using AAS.

#### Perioperative Implications
- Retention of sodium, chloride, potassium, calcium, inorganic phosphate, and water
- N/V, rarely hepatocellular neoplasms or hepatitis
- Suppression of clotting factors II, V, VII and X; bleeding in pts on concomitant anticoagulant therapy
- Polycythemia
- Increased serum cholesterol, ↓ HDL
- Pts with osteolytic lesions or who are semi-ambulatory may develop nephrocalcinosis.
- In geriatric pts, high risk of prostate hypertrophy and prostate carcinoma

#### Possible Drug Interactions
- Metabolic effects of androgens may ↓ blood glucose level and insulin requirements.
- Androgens ↓ levels of thyroxin-binding globulin, resulting in ↓ total T₄ serum levels and ↓ resin uptake of T₃ and T₄.
- Might interfere with androgenic or estrogenic drug therapy

**Overview**

- β-Sitosterol is one of the major plant sterols found in humans. Its chemical structure is similar to that of cholesterol with an ethyl group added at position 24.
- β-Sitosterol is available in many nonprescription supplements and with dietary plant consumption.

**Uses**

- CHD and hypercholesterolemia
- Benign prostatic hyperplasia and prostatitis
- Gallstones
- Enhances sexual activity
- Prevents colon cancer
- Boosts immune system
- Topically for treating wounds and burns
- Migraine headache, chronic fatigue syndrome, and symptoms of menopause
- Asthma, allergies, bronchitis, SLE, and alopecia

**ASSESSMENT POINTS**

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<thead>
<tr>
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<th>Effect</th>
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<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>CAD</td>
<td>Angina MI</td>
<td></td>
<td>ECG</td>
</tr>
<tr>
<td>RESP</td>
<td>Asthma</td>
<td>Wheezing</td>
<td></td>
<td>Wheezing</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

- Obtain adequate Hx to determine indication, since may have significant co-morbidity
- No known periop implications

**Side Effects**

- May cause N/V, indigestion, gas, diarrhea, or constipation
- Interactions: Ezetimibe (Zetia) may reduce absorption of β-sitosterol
- Anti-hyperlipidemic drugs such as atorvastatin (Lipitor), cholestyramine, and gemfibrozil have additive effects in lowering cholesterol level
- Pravastatin (Pravachol) can lower the blood level of β-sitosterol
- Increased risk of deficiency of fat-soluble vitamins. β-Sitosterol may reduce absorption and blood level of α- and β-carotene and vitamin E.
- Erectile dysfunction and loss of libido have been reported in pts on β-sitosterol.

**Pharmacology**

- With a low absorption rate, it inhibits intestinal absorption of cholesterol by competing for limited space with cholesterol in mixed micelles and also accelerates the esterification rate of the lecithincholesterol acyltransferase (LCAT) enzyme
- In benign prostatic hyperplasia, it binds to prostatic tissue, inhibits prostaglandin synthesis in the prostate, and has anti-inflammatory activity.
- Enhances proliferative responses of T cells in vitro
- Inhibits colon cancer growth in vitro
- Alternative for pts seeking modest reductions in LDL-C (<15%)

**Contraindications**

- Sitosterolemia, which is an inherited lipid storage disease with ↑ absorption of cholesterol and β-sitosterol from diet. Elevated liver β-sitosterol competitively inhibits cholesterol catabolism, which will lead to hypercholesterolemia.


**Usual Dose**

- For hypercholesterolemia, usual dosage is 800 mg–6 g before meals; for severe cases up to 15 g
- For benign prostatic hyperplasia and prostatitis, 60–130 mg tid
- About 175–200 mg is consumed daily in typical diet

**Contraindications**

- Sitosterolemia, which is an inherited lipid storage disease with ↑ absorption of cholesterol and β-sitosterol from diet. Elevated liver β-sitosterol competitively inhibits cholesterol catabolism, which will lead to hypercholesterolemia.
**Blue Cohosh (Caulophyllum Thalictroides)**

**Uses**
- Orally used for inducing labor
- Orally used as an abortifacient
- Orally used as an emmenagogue
- Orally used as an antispasmodic

**Risk**
- Ingestion of the leaf or seeds can lead to severe toxicity.
- Case reports document seizures, renal failure, and resp distress after use.
- Avoidance is advised in diabetic pts due to concern for hyperglycemia
- Reports of stroke, aplastic anemia, acute MI and CHF in infants following maternal use
- Should not be used by women with estrogen-sensitive conditions or cancers, and in pts with diarrhea

**Perioperative Risks**
- Coronary artery vasoconstriction that can lead to myocardial ischemia

**Worry About**
- Differentiating from black or white Cohosh, which have other physiological effects
- Product safety and efficacy profiles differ among manufacturers
- Usage in pregnancy due to concern of uterine stimulation, teratogenicity, and neonatal multisystemic complications
- Usage in pts with diabetes, Htn, or acute Hx of tobacco use

**Overview/Pharmacology**
- Several alkaloids and saponins are considered responsible for the pharmacological effects
- Anagyrine, N-methylcytosine, and taspine are constituents identified likely to be teratogenic
- N-methylcytosine acts similarly to nicotine, which can increase BP, stimulate the small intestine, and produce hyperglycemia in the developing fetus.

**Etiology**
- Berberidaceae or Leonticaceae family
- Listed in the United States Pharmacopeia 1882–1905 as a labor inducer
- Typically the dried rhizome/root parts are used

**Assessment Points**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Mucous membrane irritation</td>
<td>Complaints of oral irritation</td>
<td>Oral mucosa exam</td>
<td>----------------</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Ischemia, Htn, Tachycardia</td>
<td>Complaints of angina, dyspnea, or palpitations</td>
<td>Cardiac exam</td>
<td>EKG ± ECHO</td>
</tr>
<tr>
<td>GI</td>
<td>↑ Gastrointestinal motility, abd cramping</td>
<td>Changes in bowel movements (i.e., frequency, consistency, etc.), abd discomfort</td>
<td>Abd exam</td>
<td>----------------</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperglycemia</td>
<td>Fatigue, polydipsia, polyuria, vision changes, wt loss</td>
<td>Visual acuity exam</td>
<td>Blood glucose</td>
</tr>
<tr>
<td>OB/GYN</td>
<td>Uterine stimulation, estrogenic effects</td>
<td>Changes in contractions or menstruation</td>
<td>OB exam</td>
<td>Biophysical profile US, LH levels</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Concerns**
- Reliable self-reporting of use by pts
- Enhanced hyperglycemia in diabetics
- Can be assoc with coronary vasoconstriction

**Monitoring**
- Use of standard ASA monitors
- Intraop blood glucose level

**Airway/Maintenance**
- No known effects

**Preinduction/Induction**
- Coronary vasoconstriction

**Adjuvant**
- May accentuate the response to vasopressors
- May attenuate effectiveness of antihypertensive medications
- Possible drug-drug interactions due to inhibitory effects on hepatic enzymes

**Postoperative Period**
- Monitor CV status (i.e., BP, pulse, etc.) and blood glucose levels
**Carnitine**

**Uses**
- Treatment of 1° carnitine deficiency and deficiency 2° to complications of several inborn errors of metabolism, such as organic acidemia and fatty acid oxidation defects in children and adults, and acquired medical or iatrogenic conditions such as valproate and zidovudine treatment, cirrhosis, chronic renal failure on dialysis, etc.
- Treatment of valproic acid poisoning and/or overdosage and prevention of valproic acid-induced hepatotoxicity.
- Used for attention-deficit/hyperactivity disorder (ADHD), erectile dysfunction and male infertility, cardiomyopathy, PVD, CHF, chronic cardiac dysrhythmias; senile dementia, metabolic nerve diseases, in HIV infection, tuberculosis, myopathies, renal failure anemia, neuropathy, and neuropathic pain, etc. However, additional studies are needed to confirm these benefits.
- L-carnitine can effectively block the neuronal apoptosis caused by inhalational anesthetics in the developing rat brain. Clinical applications of this finding are unknown.

**Perioperative Risks**
- These are related to carnitine deficiency rather than carnitine itself.
- Hypoglycemia, lactic acidosis, and muscle weakness related to carnitine deficiencies and D/C of carnitine supplement
- Case report indicated that pts with carnitine deficiencies may develop symptoms similar to those assoc with propofol infusion syndrome when large doses of propofol are used.

**Worry About**
- Individuals with L-carnitine deficiency should continue this medication as scheduled preop to avoid acute hypoglycemia, lactic acidosis etc. IV carnitine or dextrose-containing solutions may be needed for fasting individuals with L-carnitine deficiencies.

**Overview/Pharmacology**
- Carnitine (3-hydroxy-4-trimethylamino-butyric acid or β-hydroxy-γ-methylaminobutyric acid) is a quaternary ammonium compound biosynthesized from the amino acids lysine and methionine.
- It exists in two stereoisomers: L-carnitine, biologically active form and D-carnitine, the biologically inactive form that may be harmful.
- About 75% comes from the diet, particularly from red meat and dairy products. Endogenous synthesis combined with high tubular re-absorption is enough to prevent deficiency in healthy people. Thus, carnitine deficiency is uncommon in the healthy and well-nourished adult population.
- Most of the body's carnitine is stored in skeletal muscle, but it is also found in other high energy demanding tissues such as those in the myocardium, liver, and adrenal glands. Carnitine is excreted in urine. Thus, carnitine and its metabolite may accumulate in renal failure pts.

**Pharmacokinetics**
- **Formula**: C7H15NO3
- **Mol. Mass**: 161.199 g/mol
- **Bioavailability**: <10%
- **Protein binding**: None
- **Metabolism**: Slightly
- **Half life**: 15 hrs
- **Excretion**: Urine (>95%)

**Drug Class/Usual Dose**
- Carnitine is available both as a prescription drug and as a food supplement.
- Pregnancy: Category B. Studies in bacteria found no evidence of mutagenicity. No human data is available. Carnitine occurs naturally in human breast milk.
- Dosing: The usual supplementation dose is 2–6 g/d for a period ranging from 10 d to 10 wk. For infants and children, recommended dosage is between 50 and 100 mg/kg/d in divided doses with a max of 3 g/d. IV L-carnitine is used for treatment of lactic acidosis and cardiomyopathy 2° to L-carnitine deficiency. The recommended dosage is a 50 mg/kg bolus injection over 2–3 min, followed by an equivalent dosage over the next 24 hr (divided every 3–4 hr). Subsequent dosages would be based on responses.
- Overdosage: There have been no reports of toxicity from L-carnitine overdosage. Oral doses of 15 g/d have been well tolerated.

<table>
<thead>
<tr>
<th>ASSESSMENT POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>System</strong></td>
</tr>
<tr>
<td>CNS</td>
</tr>
<tr>
<td>GI</td>
</tr>
<tr>
<td>DERM</td>
</tr>
</tbody>
</table>


**Possible Drug Interactions**
- Carnitine has not been thoroughly tested for interactions with other herbs, supplements, drugs, or foods.
- L-carnitine might decrease the need for certain drugs such as glycosides, digoxin, diuretics, beta-blockers, channel blockers, hypolipidemic (cholesterol-altering) drugs, and nitro derivatives.
- L-carnitine might increase the effects of warfarin (Coumadin) and heparin.

**Anticipated Problems/Concerns**
- None
Chitosan

Uses
- Excellent hemostatic agent
- Antibacterial properties useful for periodontal disease
- Pharmaceutical sustained-release drug carrier (chitosan glutamate)
- Wt loss agent (modest)
- Decreases cholesterol and triglycerides and increases (improves) HDL:total cholesterol ratio
- Cleaning petrochemical spills
- Water purification agent

Risk
- None known

Perioperative Implications
- None known or studied

DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Improved cholesterol</td>
<td>Lipid profile</td>
</tr>
<tr>
<td>HEME</td>
<td>Improved hemostasis</td>
<td>None</td>
</tr>
<tr>
<td>GI</td>
<td>Stomach upset, steatorrhea, loss of fat soluble vitamins</td>
<td>None</td>
</tr>
</tbody>
</table>


Perioperative Risk
- None known

Worry About
- Theoretical inhibition of absorption of fat soluble vitamins A, D, E and K

Etiology
- Chitosan is a completely indigestible fiber source with the ability to electrostatically attract and bond with negatively charged dietary lipids thus prohibiting their absorption.
- The hemostatic activity of chitosan is from an ionic interaction between the positively charged chitosan polymer and the negatively charged cell membrane of the red blood cell, and works irrespective of the presence of fibrin.

Overview
- Chitosan is a naturally occurring marine polysaccharide fiber derived from a common byproduct of shellfish processing. (Chitosan is the de-acetylated form of chitin, a sugar from the shells of crustaceans).
- Recently, ingenious medical applications have been developed which use chitosan as a pharmaceutical sustained-release drug carrier effectively encapsulating various anti-inflammatory and chemotherapeutic agents allowing it to function as a moiety for safe sustained release.
Red Yeast Rice (Cholestin)

**Uses**
- Hypercholesterolemia
- Prevention of coronary events, stroke, and TIA
- Treatment of dyslipidemia in statin intolerant pts
- Prostate and colon cancer

**Perioperative Risks**
- Obtain adequate Hx to determine indication for taking red yeast rice

**Worry About**
- Chemical composition of red yeast rice is not controlled by the FDA, and may vary by manufacturer.
- Relatively contraindicated in liver disease. Hepatotoxicity is worsened in combination with other hepatotoxic drugs.

**Overview**
- Prepared by growing red yeast (monascus purpureus) on rice to produce a red product
- Contains 10 mevinic acids incl monacolin K, also known as lovastatin

**Drug Class/Mechanism of Action/Usual Dose**
- HMG-Co-A reductase inhibitor
- Inhibits conversion of HMG-Co-A to mevalonic acid, an early precursor of cholesterol
- Usual dose 600–2400 mg daily

**Perioperative Implications**

**Preoperative Concerns**
- Lovastatin has been designated as pregnancy category X by the FDA. Red yeast rice should be avoided in pregnancy and lactation.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIAC</td>
<td>Reduces VLDL, LDL and triglyceride levels</td>
<td>VLDL, LDL, HDL, triglycerides</td>
</tr>
<tr>
<td>HEPAT</td>
<td>Rare hepatocellular damage and cholestasis</td>
<td>AST, ALT</td>
</tr>
<tr>
<td>MS</td>
<td>Rare myopathy, myalgia, and rhabdomyolysis</td>
<td>CPK</td>
</tr>
</tbody>
</table>


**Preinduction/Induction**
- Succinylcholine is contraindicated in myopathies assoc with elevated serum creatine phosphokinase (CPK) values.
Uses
• The consensus of expert and industry opinions supports the use of chondroitin sulfate (CS) for improving symptoms and stopping, possibly reversing the degenerative process of osteoarthritis.
• CS is relatively free of side effects when used at the recommended daily dosage, at least for short periods of time, and thus offers an attractive alternative to traditional treatments mainly consisting of NSAIDs.
• As CS has gained in popularity, researchers have recognized the need to conduct more scientific studies to validate or refute its efficacy, as well as its safety.
• Most trials testing the efficacy of CS have demonstrated significant improvement of pts’ OA, although the level of improvement continues to be debated, and is about the level of placebo in recent trials at about 60% gaining significant pain relief in trials too short to show joint remodeling.
• Its common partner, glucosamine, has resulted in significant reduction in moderate and severe pain over 24 wk greater than placebo and NSAIDs, and either CS or itself alone.

Perioperative Risks
• No known significant complications of CS and no known anesthetic implications. Use cautiously in pts with shellfish allergy. Worsening of previously well-controlled asthma has been reported. CS has a structural similarity to heparin and may need to be reconsidered in certain health conditions where heparin is contraindicated.

Overview/Pharmacology
• Major component of extracellular matrix and important in its role in forming proteoglycans of which it is a part. The tightly packed and highly charged sulfate groups of CS generate electrostatic charges that provide much of the resistance of cartilage to compression. Loss of CS from the cartilage is a major cause of OA.
• Oral CS is a less than perfectly absorbed nutritional supplement for the treatment of OA.
• Shown to have activity and capable of ↑ proteoglycan synthesis in articular cartilage; mechanism of action may be related to local inhibition of interleukin 1β.
• CS usually takes 1 mo to exert any effect, with maximum benefits seen after 4 wk of therapy; conversely, benefits are sustained for 1–2 mo after D/C of therapy.

Drug Class/Usual Dose
• CS is a glycosaminoglycan found in the proteoglycans of articular cartilage.
• Currently manufactured from natural sources (shark and/or beef cartilage or bovine trachea)
• Dose is 400 mg three times daily or 600 mg two times daily, by mouth (18 yr and older)
• Most studies have tested doses of 1–2 g CS daily, although a higher dose has not clearly been linked to a greater effect.
• Optimal dose when used in combination with glucosamine is not clear.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Drug Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>Anti-inflammatory effect, slows joint degeneration</td>
<td>Pt assessment of pain scores</td>
<td>Joint tenderness and function</td>
<td>Joint narrowing on x-rays</td>
</tr>
<tr>
<td>GI</td>
<td>Low incidence of nausea and diarrhea</td>
<td>Subjective reports of nausea and diarrhea</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications/Drug Interactions
• None
Chromium

**Uses**
- Body building (ineffective)
- May aid in glycemic control of type II DM and gestational DM
- Hyperlipidemia
- Hypoglycemia—reactive
- Obesity

**Perioperative Risks**
- Risks minimal
- Chronic ingestion assoc in one case with thrombocytopenia, hepatic dysfunction, renal dysfunction

**Overview**
- A trace mineral
- Improves glucose tolerance in type II DM and gestational DM (in some studies)
- Shown to ↑ insulin sensitivity and ↓ serum triglycerides
- Shown to alleviate symptoms of reactive hypoglycemia
- Popular as wt loss and body building supplement, but effect not supported in clinical trials

**Worry About**
- Nephrotoxicity

**Perioperative Implications**
- No known interaction

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>RENAL</td>
<td>Nephrotoxicity</td>
<td>Cr</td>
</tr>
<tr>
<td>ENDO</td>
<td>Insulin sensitivity</td>
<td>Glucose</td>
</tr>
</tbody>
</table>


**Drug Class/Mechanism of Action/Usual Dose**
- Hypothesis: In normal functioning, it ↑ circulating insulin, results in binding of chromium to peripheral, insulin-sensitive tissue; ↑ insulin receptor number and activates insulin receptor kinase
- Usual dosage recommended: 50–200 μg/d
  - Available orally or IV
  - Taken as supplement of 200–1000 μg/d
- Mixed results in randomized clinical tests
Cranberry

Uses
- Many cranberry juice consumers are aware of a beneficial link between cranberry juice and urinary tract and of valuable polyphenol activity
- Most common as alternative agent for prevention of UTIs
- Also, believed to be beneficial for prevention of upper GI ulcers, reducing the risks of CV disease and improving oral hygiene
- Native Americans and Early American sailors used cranberries for treating wounds and blood poisoning, urinary illnesses, diarrhea, diabetes, and as antiscorbutic agent

Perioperative Risks
- A few case reports suggested possible interaction with warfarin resulting in prolonged INR and bleeding. Subsequently, two small randomized controlled trials could not confirm clinically significant enhancement of anticoagulation.

Worry About
- Theoretical risk of oxalate urinary stone formation (if large volumes consumed daily)
- Contentious issue of interaction with anticoagulation effect of warfarin

Overview/Pharmacology
- Cranberries are a fruit native to New England and belong to the Vaccinium macrocarpon
- The most popular form for consumption is the cranberry-juice cocktail, containing about 27% cranberry juice, sweetener, water, and vitamin C
- Also available as juice concentrate, tablets, or capsules

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Drug Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Reduces dental plaque, periodontal and gum disease</td>
<td>Toothache</td>
<td>Dental exam</td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Improves ability of LDL to resist oxidative stress (antioxidation)</td>
<td>Frequency and urgency and painful urination</td>
<td>ECHO of arteries</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Prevents UTI; stone formation</td>
<td>Cloudy urine, low back pain</td>
<td>UA culture of urine</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Concerns
- Hx of recurrent UTI, possible urolithiasis, the need for antibiotics

Induction/Maintenance
- Routine monitoring
- Consider antibiotic coverage if UTI is present

Postoperative Concerns
- Immediate resumption not necessary

Anticipated Problems/Concerns
- Cranberries consist of 90% water and various organic substances such as quinic acid, malic acid, and citric acid as well as glucose and fructose.

Drug Class/Mechanism of Action/Usual Dose
- Increased concentration of hippuric acid and increased acidification of urine
- Inhibits bacterial adherence to mucosal surface by at least two kinds of inhibitors: fructose and proanthocyanidins
- Fructose and proanthocyanidins in cranberries inhibit type I–fimbriated Escherichia coli adhesion
- Cranberry products reduced the incidence of UTIs in women at 12 mo
Creatine

Uses
- Medical: Historically used to lower cholesterol and treat rare conditions of heart failure due to creatine deficiencies; has proposed benefits to decrease myalgias and myositis with statins.
- Fitness: Usage over past decade to ↑ muscle mass and enhance physical performance. Initially used by professional athletes, now used as nutritional supplement in almost all areas of exercise fitness (in both casual and competitive athletes).
- Incidence: Unknown incidence in population

Perioperative Risks
- Unknown. Theoretical problems in pts with impaired renal function. Potential for drug interactions, though no definitive studies (see Possible Drug Interactions below)

Worry About
- Hypovolemia and/or dehydration if inadequate nutrition

Overview/Pharmacology
- Commercially available as creatine citrate, creatine monohydrate, and creatine phosphate
- Creatine exists intracellularly in skeletal muscle, cardiac muscle, brain, and testes as creatine phosphate, otherwise called phosphocreatine. Phosphocreatine contains a high-energy phosphate bond, used for short, intense muscle activity via the phosphagen energy system.
- Studies in animal and human subjects have demonstrated ↑ of cellular phosphocreatine levels in skeletal muscle following creatine ingestion. Few studies demonstrating ↑ in muscle strength or endurance.
- Recent randomized trials have shown neither increased strength nor increased stamina
- Increase in muscle mass is thought related to increase in intracellular H₂O content brought about by influx of phosphocreatine into myocyte.
- Creatine is eliminated from the body by renal excretion as creatinine, the anhydrous form of creatine.
- Creatine is usually ingested dissolved in fluid.
- Use creatine to ↑ muscle mass and performance. (Special concern should be paid to athletes desiring wt loss, i.e., wrestlers, gymnasts, body builders, football players, in examining renal function.)

Drug Class/Usual Dose
- Creatine is classified as a nutritional or dietary supplement; therefore, it is unregulated by the FDA.
- Typical usage: Initially 20–25 g ingested daily for 5–7 d, followed by 5–10 g daily for 10–12 wk. However, some individuals take higher dosages continually.

ASSESSMENT POINTS

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<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypovolemia/hypotension</td>
<td>Exposure</td>
<td>BP/HR</td>
<td>Lytes</td>
</tr>
</tbody>
</table>


Possible Drug Interactions

Preoperative Period
- Because of the assoc risk of hypovolemia/dehydration in pts using creatine, there are theoretical problems when used with the following classes of medications: diuretics, H₂ antagonists (e.g., cimetidine), NSAIDs, probenecid, and trimethoprim, or when taken near the time of exercise.

Induction/Maintenance
- No known interactions. May need bolus of intravascular fluids and careful attention to BP at time of induction.

Adjuvants/Regional Anesthesia/Reversal
- No known interactions. Consider pro/cons of NSAID usage intraop, esp. if no assessment of renal function.
Uses
• Rx for liver disease (e.g., liver congestion, bile duct inflammation, hepatitis, gallstones, and jaundice)
• Rx for kidney disease
• Rx for fluid retention
• Rx for diabetes with specific hypoglycemic effects
• Less commonly used for mastitis, heartburn, boils, and fevers, among other uses
• Dietary supplement as a source of vitamins and minerals (leaves contain the highest concentration of vitamin A at 14,000 IU/100 g raw) in addition to vitamin D, vitamin B complex, vitamin C, iron, silicon, Mg, Na, K, zinc, manganese, copper, and phosphorus

Perioperative Risks
• No clinical trial to date on hemodynamic instability
• No clinical trial to date but may potentially cause bleeding 2° to ↓ the clotting

Worry About
• If used in combination with prescription diuretic drugs, effects of either or both drugs may be enhanced, leading to a hypovolemic state.
• Multiple minerals in dandelion may ↓ systemic absorption of PO-administered drugs (e.g., ciprofloxacin, famotidine, and esomeprazole)
• Too much vitamin A

Overview/Pharmacology
• 1° effect in relieving dyspepsia disorder is caused by taraxerol.
• Stimulation of bile release by the liver and gallbladder, hence, improving both bile flow (choleretic effect) and release (cholagogue effect)
• Diuretic activity comparable to that of furosemide has been demonstrated in mice; however, because dandelion replaces potassium lost through diuresis, metabolic complications occur only rarely.
• Insulin, a polysaccharide fiber composed of long chains of fructose-containing molecules contained in the plant, may act to buffer fluctuations in blood sugar levels.

Usual Dose
• Root used for general tonic and mild liver remedy up to tid
  • Dried root: 2–8 g by infusion, or decoction
  • Fluid extract: 4–8 mL
• Tincture, alcohol based: Not recommended 2° to high dosage required
  • Juice of fresh root: 4–8 mL
  • Powdered solid extract: 250–500 mg
• Leaf preparations used for diuretic effects tid
  • Dried leaf by infusion: 4–10 g
  • Fluid extract: 4–10 mL

Toxicity
• Generally considered one of the safest medicinal plants used
• May be potentially toxic because of the high content of K, Mg, and other minerals, and vitamin A

ASSESSMENT POINTS

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</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Orthostasis, polyurea, polydipsia</td>
<td>↓ Skin turgor, hypotension, tachycardia, orthostasis</td>
<td>Orthostatic BP, HR</td>
</tr>
<tr>
<td>GI</td>
<td>↑ Gastric secretion</td>
<td>Diarrhea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RENAL</td>
<td>Prerenal failure</td>
<td>Polyuria, polydipsia</td>
<td>As for CV</td>
<td>BUN/Cr</td>
</tr>
<tr>
<td>METAB</td>
<td>Hypoglycemia</td>
<td>Lightheaded, clammy, shaky</td>
<td>Sweaty</td>
<td>Blood glucose</td>
</tr>
</tbody>
</table>


Perioperative Implications

Preoperative Concerns
• Unknown effects in pediatric and pregnant pts
• Rely on pt self-report

Monitoring
• Routine

• May require fluid bolus if there is an indication of hypovolemia and UO

Regional Anesthesia
• Not clear but can potentially affect plt function

Emergence/Extubation
• No known complications to date

Postoperative Period
• Continue to assess volume status and treat accordingly
• Potentially increased bleeding
Dehydroepiandrosterone (DHEA)

USES
- Unproven benefits and uses:
  - Adrenal insufficiency
  - Aging, slowing
  - Alzheimer's disease
  - Anorexia nervosa
  - CV disease
  - Chronic fatigue
  - Chron's disease
  - Depression
  - Diabetes
  - Heart failure
  - Obesity
  - Osteoporosis
  - Perimenopause issues incl increasing bone mineral density and for vaginal atrophy
  - Sexual dysfunction
  - Sleep disorders
  - SLE
  - Well-being
- Marketed as a dietary supplement because DHEA can be manufactured from natural sources, such as soy and wild yam. However, many of these products, depending on source and metabolism, are not converted into DHEA in humans and are not recommended or preferred.

Pharmacodynamics
- Hepatic, adrenal gland, testes, and in minute quantities, the brain endogenously produce
- Hepatic metabolism, urinary excretion with a 12-hr half-life
- Steroid hormone produced by adrenals, interconverted to testosterone, estrone, estradiol, androsterone
- Considered a prohormone, so effects similar to those of anabolic steroids.
- Increased protein synthesis in skeletal muscle, however, increase in serum testosterone or enhancement of strength during resistance training is controversial. A placebo-controlled, randomized clinical trial reported in the New England Journal of Medicine in 2006 found that supplementation in the elderly had no significant beneficial effects on body composition, physical performance, insulin sensitivity, or quality of life.
- Inhibits glucose-6-phosphate, which theoretically accounts for postulated antiatherogenic properties.


Overview
- Popularized after a New England Journal report that high levels correlated with fewer cardiac events (Rancho-Bernardo study); later, not found so in larger Rancho-Bernardo study
- FDA categorized DHEA as unapproved drug in 1985; reclassified as dietary supplement by 1994
- Banned by NCAA, the National Football League, and Olympics
- Contraindicated in breast, ovarian, and prostate cancers
- May cause hirsutism, acne, headache, insomnia, wt gain, alopecia, deepening of voice and abn menses in women, or gynecomastia in men
- No data indicate benefit greater than long-term risk

Usual Dose
- 25–50 mg/d for angioedema
- 50 mg tid for CV disease
- 30–90 mg/d for depression, memory improvement, or cognition
- 200 mg/d for SLE

DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Hx Assessment</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Hirsutism</td>
<td>Anabolic steroids assoc with sudden cardiac arrest, Htn; DHEA rarely causes arrhythmias</td>
<td>Determine chronic and acute dose and duration of self-administration; palpitations</td>
<td>HR</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Antiglucocorticoid actions</td>
<td>Inhibits plt aggregation in vivo</td>
<td>Ecchymoses</td>
<td>Bleeding time; Preoperative glucose for diabetics</td>
</tr>
<tr>
<td>GI</td>
<td>Anabolic steroids assoc with hepatitis, cholestatic jaundice</td>
<td>Inhibits plt aggregation with hepatitis, cholestatic jaundice</td>
<td>Prostate exam</td>
<td>PSA</td>
</tr>
<tr>
<td>HEME</td>
<td>Anabolic steroids may cause aggressiveness; DHEA binds to NMDA, sigma, GABA receptors</td>
<td>Anabolic steroids may cause aggressiveness; DHEA binds to NMDA, sigma, GABA receptors</td>
<td>↑ Pituitary tumor growth</td>
<td>ACTH</td>
</tr>
</tbody>
</table>


Possible Drug Interactions

Preoperative Period
- Insulin resistance, check preop glucose
- Synergism with corticosteroids

Induction/Maintenance
- Unknown effects of inhibition of steroid synthesis if combined with etomidate, or immuno-suppressives for transplantation

Postoperative Concerns
- Unknown effects on stress response

Anticipated Problems
- Unpredictable CV effects
Echinacea (American Coneflower, Purple Coneflower: E. Angustifolia, E. Purpurea, E. Pallida)

Uses

- Purported immunostimulation; the prevention and treatment of resp tract infections.
- As an adjuvant in the treatment of other bacterial, viral, or fungal infections of the urinary and resp tract.
- Antinflammatory action when used topically for conditions such as eczema, psoriasis, and herpes simplex.
- Promotes wound healing when used topically, i.e., in leg ulcers and burns.
- As an adjuvant for cancer therapy, and in the treatment of the chronic fatigue syndrome.

Overview

- The most common side effects are GI symptoms, allergic reactions, and rashes.
- Allergic reactions are more common in atopic individuals and individuals with a Hx of sensitivity to the Asteraceae-Compositae family of plants (ragweed, chrysanthemums, marigolds, daisies etc.), and can be serious.
- Echinacea may exacerbate autoimmune diseases such as MS, SLE, rheumatoid arthritis, AIDS, tuberculosis, and pemphigus vulgaris.
- Echinacea may inhibit Cytochrome P450 (CYP 1A2, 3A4) enzymes, altering levels of drugs metabolized by these enzymes.
- Tachyphylaxis may occur with prolonged, uninterrupted use.

Perioperative Risks

- No known drug interactions or toxicities.
- No known sedative, CV, or coagulation effects relevant to anesthesia.

Worry About

- Immunostimulation may counteract the effect of steroids and immunosuppresant drugs in transplant recipients and pts with autoimmune disease.

Overview

- Anti-inflammatory activity by inhibition of cyclooxygenase and 5-lipogenase.
- Promotes wound healing by protecting Type 3 collagen from free radical damage and inhibiting bacterial hyaluronidase
- Concentration of active ingredients varies widely according to species and preparation used.
- 1–3 mL of the fluid extract or cold-pressed juice of plant (or root) three times daily.
- 1 gm of powdered root three times daily (capsules, tablets)
- Echinacea appears to modestly inhibit cytochrome P450 1A2 (CYP1A2), and to induce hepatic cytochrome P450 3A4 (CYP3A4), but inhibit intestinal CYP3A4 (opposing effects).

Drug Class/Mechanism of Action/Usual Dose

- Increases phagocytosis and lymphocyte activity, possibly by release of tumor necrosis factor (TNF), interferlin-1 (IL-1), and interferon.

Perioperative Implications

- Possible antagonism of antirejection drugs used following bone marrow or organ transplantation.

**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>IMMUNE</td>
<td>Immunostimulation, anti-inflammatory activity</td>
<td>Phagocytic activation, IL-1 and TNF activity</td>
</tr>
<tr>
<td>HEPAT</td>
<td>P450 CYP1A2 inhibition</td>
<td>Caffeine clearance test</td>
</tr>
</tbody>
</table>

Ephedra (Ma-Huang)

**Uses**
- Ephedra is a plant that contains a variety of ephedrine alkaloids, incl ephedrine and pseudoephedrine.
- Dietary supplements containing ephedra were marketed in the USA as agents that may aid in wt reduction and energy enhancement. Ephedra may be used in the manufacture of methamphetamine.
- Several governmental agencies inquired into the safety of ephedra and regulated the use of dietary supplements containing ephedra in response to reported pt adverse reactions. A 2004 U.S. ban on the sale of ephedra-containing supplements currently continues.
- Some supplements have marketed “ephedrine-free” or legal ephedra products, in which the ephedra is replaced with other herbal stimulants such as bitter orange.

**Perioperative Risks**
- Risks assoc with an ↑ in the sympathetic nervous system activity and dysrhythmias and Htn
- Lethal cardiac arrhythmias, Htn, myocarditis, MI, angina, ↑ thermogenesis
- Hemorrhagic and/or ischemic stroke, subarachnoid hemorrhage, cerebral vasculitis, seizures
- Bronchial dilation, acute hepatitis
- Preterm labor

**Overview/Pharmacology**
- Mechanism of action is via increases in sympathetic stimulation
- Ephedrine-containing substances are also known as ma-huang, Mormon tea, squaw tea, and herbal ecstasy.

**Perioperative Implications**

**Preoperative Period**
- Ephedra may produce adverse pt reactions with medications such as MAO inhibitors, digoxin, cold medications containing ephedrine, diuretics, and antihypertensives
- Assess preop BP, HR, and ECG
- Consider as a potential cause of preterm labor

**Preinduction/Induction Period**
- Control hemodynamics before induction
- Observe ECG for arrhythmias

**Maintenance Period**
- Response to ephedrine may be hampered 2° to tachyphylaxis; therefore, control hypotension with direct-acting adrenergic agonists, like phenylephrine
- Ephedrine is an indirect-acting sympathomimetic that exerts its effects mainly by stimulating release of norepinephrine.
- Other ephedrine alkaloids in ephedra have direct-acting effects on both α- and β-adrenoceptors
- Ephedra is often packaged with guarana-derived caffeine, which may synergistically augment adrenergic stimulation.

**Drug Class/Mechanism of Action/Usual Dose**
- Works via stimulation of sympathetic nervous system

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
</table>
| CARDIO | Arrhythmias, Htn, myocarditis, MI, angina thermogenesis | Chest pain | BP | BP/HR ECG, cardiac enzymes
| GU     | Acute hepatitis | ↑ Temp | Temp probe |
| CNS    | Stroke, subarachnoid hemorrhage, vasculitis, seizure | Decreased mental status | Neuro exam | CT, vascular biopsy, EEG |
| PULM   | Bronchial dilation | Headache | LFTs |

**Effect**
Reduces platelet aggregation when subject fed an atherogenic diet.

**ALTERNATIVE MEDICINE**
phenothiazines.
Seizures have not been seen in pts not taking known epileptogenic drugs such as phenothiazines.

**IMMUNO**
GI
CARDIO

**Overview/Pharmacology**
EPO is a rich source of the essential fatty acids LA and GLA. These essential fatty acids are involved in prostaglandin biosynthetic pathways.

**Uses**
- Evening primrose oil (EPO) is obtained from the seed of the plant species *Oenothera biennis*
- EPO is also known as fever plant, huile d’onagre, king’s cureall, night willow-herb, seabush, and sundrops
- EPO may be used as a food supplement for the essential fatty acids linoleic acid (LA) and γ-linolenic acid (GLA).
- Infusion of the whole plant has been used for asthma, GI disorders, whooping cough, and as a sedative pain killer
- EPO had been licensed in Britain for treatment of atopic eczema and cyclic and noncyclic mastalgia; but the license was withdrawn upon a conclusion that there was not enough evidence of effectiveness.
- Other uses for EPO incl PMS, psoriasis, MS, hypercholesterolemia, rheumatoid arthritis, Raynaud’s phenomenon, Sjögren’s syndrome, postviral fatigue syndrome, asthma, and diabetic neuropathy, without solid evidence it is effective but with recurrent anecdotal evidence of beneficial outcomes.

**Overview/Pharmacology**
• EPO is a rich source of the essential fatty acids LA and GLA. These essential fatty acids are involved in prostaglandin biosynthetic pathways.
• DGLA, a metabolite of GLA, is a precursor of both the inflammatory prostaglandin series via arachidonic acid (AA), and the less inflammatory series (PGE1).
• Actions of PGE1 incl anti-inflammatory, immunoregulatory, and vasodilatory properties, inhibition of platelet aggregation and cholesterol biosynthesis, hypotension, and elevation of cyclic AMP.
• GLA has been shown to have a favorable effect on the DGLA:AA ratio. The ↑ in AA is smaller and less consistent when compared with the ↑ in DGLA. This is beneficial because DGLA leads to the less inflammatory prostaglandin series PGE1.

**Drug Class/Mechanism of Action/ Usual Dose**
- Dose of EPO is specific for each condition being treated, e.g., the EPO dose for atopic eczema is 6–8 g for adults or 2–4 g for children.

**EPO studies are in a preliminary phase; its effects have been proved only in animal models. The effects mentioned here have yet to be proved in humans.**

**Uses**
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**Table: Drug Effects**

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<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Inhibits the ↑ of serum total cholesterol + VLDL + IDL + LDL cholesterol concentrations in the presence of excess cholesterol in the diet. Serves as an antioxidant in hyperlipemic states. Reduces oxidative stress by inhibiting lipid peroxidation and reinforcing the glutathione-dependent antioxidant defense system.</td>
</tr>
<tr>
<td>GI</td>
<td>Has anti-ulcer and cytoprotective effects on experimentally induced gastric lesions</td>
</tr>
<tr>
<td>HEME</td>
<td>Reduces platelet aggregation when subject fed an atherogenic diet</td>
</tr>
<tr>
<td>DERM</td>
<td>May be used for Rx of atopic eczema. Treatment of atopic eczema with EPO is controversial. Clinical studies have been equivocal on whether symptoms of atopic eczema benefit from EPO. May be used for the treatment of limited scleroderma, or CREST syndrome. Clinical studies have been equivocal in relation to fatty acid placebos but have shown qualitative improvement in symptoms of Raynaud’s phenomenon.</td>
</tr>
<tr>
<td>GU</td>
<td>Has been used for PMS and to help reduce frequency of nighttime hot flashes during menopause. Treatment is controversial because clinical studies have not shown a clear benefit of EPO for PMS and menopause. Has been shown to be no better than fatty acid placebo or topical NSAIDs for treatment of mastalgia. Has been used by many midwives to hasten cervical ripening in an effort to shorten labor and ↓ incidence of postdate pregnancies. One retrospective study showed that EPO does not shorten gestation or ↓ length of labor. Moreover, it was found that EPO may be assoc with above-mentioned adverse effects on labor.</td>
</tr>
<tr>
<td>CNS</td>
<td>Significantly reduced headache in women with PMS. Pts given both EPO and fish oil had fewer symptoms assoc with headache, such as depression and fatigue. Animal studies suggest EPO may be useful in the treatment of diabetic neuropathy, although the exact physiological mechanism remains to be demonstrated.</td>
</tr>
<tr>
<td>IMMUNO</td>
<td>In pts with mild RA, EPO has been shown to improve morning stiffness, and there was also improvement in the Ritchie articular index for each pt. Pts with severe RA did not exhibit improvement. Although not scientifically proved, EPO has been taken by asthmatics to gain the anti-inflammatory effects of PGE1.</td>
</tr>
</tbody>
</table>

**Perioperative Implications**

**Preoperative Concerns**
- EPO may cause an ↑ risk of developing temporal lobe epilepsy, specifically in pts taking known epileptogenic drugs such as phenothiazines. Seizures have not been seen in pts not taking phenothiazines.

**Worry About**
- Obstetrics: Orally administered EPO may be assoc with an ↑ in the incidence of prolonged rupture of membranes, oxytocin augmentation, arrest of descent, and vacuum extraction

**Drug Class/Mechanism of Action/ Usual Dose**
- Dose of EPO is specific for each condition being treated, e.g., the EPO dose for atopic eczema is 6–8 g for adults or 2–4 g for children.

**Perioperative Implications**

**Preinduction/Induction**
- No known interactions

**Maintenance**
- No known interactions

**Postoperative Period**
- No known interactions

Fish Oil

Uses
- Active ingredient for brain and retinal health (more than 40% of brain and retina is structural fat) is DHA (more than 50% of fat in brain and retina is DHA)
- Decreases arrhythmias and deaths related to heart disease
- Important component for cell signaling
- Data from MIDAS trial indicate restoration of memory to that of 3.5 y younger with 900 mg of DHA (about 3 gm of fish oil) a day in pts with minimal cognitive dysfunction
- Data from trial in non-breastfed infants indicate better IQ by about 16 points in babies formula fed with 20 mg of DHA per day compared to those fed in formula without DHA
- To ↓ plasma concentrations of triglycerides. Reduces elevated VLDL and chylomicrons, causes slight ↑ in HDL. To ↓ risk of death from CAD and to ↑ risk of stroke.
- Lowers BP (minimal)
- Decreases the risk of arrhythmias
- Myocardial reinfarction prevention
- Beneficial antithrombogenic from EPA (DHA has no anticoagulating effect) and anti-inflammatory effects from DHA or EPA
- May prevent immunologic injury in pts with IgA nephropathy; to retard renal function loss.
- May benefit renal transplant recipients treated with cyclosporine. Significant beneficial effects on diabetic nephropathy and macroangiopathy.
- Beneficial in chronic and severe mental disorders (bipolar disorder, depression)
- To reduce inflammatory Sx assoc with inflammatory bowel diseases
- Other uses: Dysmenorrhea, kidney stones, diabetic neuropathy, gout, migraine headaches, male infertility, osteoporosis, multiple sclerosis, cancer-related cachexia, reduce modestly cataract risks, may improve risk of depression

Perioperative Risks
- Risks of long-term use not known. Variable ↑ in bleeding time if given EPA (not a risk from DHA).
- Worry About
  - Coagulation disorders, greater than 3 grams per day can inhibit blood coagulation and potentially reduce plt aggregability and increase risk of bleeding.

Overview/Pharmacology
- Omega-3 fatty acids: Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)
- Also known as cod liver oil, marine oils, menhaden oil, N-3 fatty acids, N3-polyunsaturated fatty acids, omega 3, Omega-3 fatty acids, PUFA, salmon oil, W-3 fatty acids, algal DHA
- May reduce blood clotting and increase risk of bleeding (not an effect of DHA alone)—can switch pts on 3 gm of fish oil a day to 900 mg of DHA a day with perhaps same anti-arrhythmic and brain function preserving effects—half-life variable depending on preparation, ideally pt having surgery or pain procedure should be off fish oil 7 d, allowing enough time for fish oil-induced blood thinning effects to be gone, but switch to DHA at same time.
- May benefit renal transplant recipients treated with cyclosporine. Significant beneficial effects on diabetic nephropathy and macroangiopathy.
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Induction/Maintenance
- No interactions known

Adjuvants/Possible Drug Interactions
- Caution if receiving heparin, warfarin, dihydropyrimidone, ticlopidine, sulfnyprazone, or aspirin
- Fish oil can reduce vitamin E levels. Caution with herbs that have antiplatelet and/or anticoagulant constituents (angelica, clove, danshen, garlic, ginger, ginkgo, Panax ginseng, red clover, turmeric, willow, and others) with EPA, not DHA.
- Dietary supplements available in capsules or oil by brand names: Coromega, Solgar Omega 3 700, Nature Made, Spring Valley, Bounty, Barleans, LifeFitness DHA, NatureMade DHA and others
- Fish oil and DHA supplements are not regarded as drugs and are not regulated by the FDA medication rules except for Lovaza.
- Fish oils produce biologic effects on prostaglandins, thromboxanes, and leukotrienes; ↑ TXA, levels and ↓ TXA, levels stimulate the formation of prostaglandin E, moderately reduce formation of TXB, in plt, inhibiting aggregation and adhesion
- Results in reduced plt aggregation (EPA) and vasoconstriction (DHA)
- Recent studies show small increase in LDL levels with large doses
- Improves large artery endothelium-dependent dilation of hypercholesterolemic (both EPA and DHA) without affecting endothelium-independent dilation
- Reduces blood viscosity by ↑ RBC deformability
- Substantial ↓ of triglyceride levels, variable effects on cholesterol levels

Drug Class/Usual Dose
- Not clear: Usual dosage is 2–9 g/d of fish oil or 20mg per year of life up to age 45 (900 mg) where dose stays constant (DHA)


ASSESSMENT POINTS

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<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>Abd distention, belching, halitosis, heartburn, flatulence, diarrhea</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Prolongs bleeding time, inhibits plt aggregation (EPA only)</td>
<td>Anticoagulant Rs, fatigue, weakness, bleeding problems</td>
<td>VS</td>
<td>Bleeding time, Hct</td>
</tr>
<tr>
<td>ENDO</td>
<td>Mild glucose intolerance in pts with NIDDM</td>
<td></td>
<td>FBS</td>
<td></td>
</tr>
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Perioperative Implications

Preoperative Concerns
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Drug Class/Usual Dose
- Not clear: Usual dosage is 2–9 g/d of fish oil or 20mg per year of life up to age 45 (900 mg) where dose stays constant (DHA)

Garlic (Allium sativum)

Uses
- Administered orally and topically as a powder, tablet, and raw clove. Allicin is pharmacologically active component.
- Potential beneficial activity as an antihyperlipidemic (conflicting results in recent clinical trials), antimicrobial (Mikrosporum canis, sporotrichosis, tinea pedis), antiplatelet (via ↑ thrombocyte levels), fibrinolytic, antioxidant (↑ catalase and glutathione peroxidase), anti-diabetic, and vasoprotective agent (i.e., antithrombotic agents and agents to protect elastic properties of the aorta).
  - Note: These indications are not FDA approved, but garlic is generally recognized as safe (GRAS). Interpretation of data must take into account publication bias (preferential publication of positive findings).

Perioperative Risks
- Increased bleeding diathesis

Overview/Pharmacology
- Intact cells of garlic bulbs contain alliin, an odorless, sulfur-containing amino acid. Crushed garlic causes the enzyme allinase to convert alliin to allicin—a potent antibacterial agent that is odoriferous and unstable. Ajoenes, a self-condensation product of allicin, has antithrombotic activity. Fresh garlic releases allicin in the mouth during the chewing process. Dried garlic preparations lack allicin but contain allin and allicin; they should be enteric-coated so they pass through the stomach into the small intestine where allicin can be enzymatically converted to allicin. Allicin is unstable in oil. Allinase is inactivated by heat (cooking) and acid.

APPLICATIONS

Anticoagulant use, coagulopathy, dysfunctional plts, bleeding disorders

Lipid profile

Prolonged PT (INR) Pit Hgb/Hct

CT scan

Perioperative Concerns/Possible Drug Interactions
- High consumption may cause significant antiplatelet activity; ASA, NSAIDs, other pt inhibitors, thrombolytic agents, or certain herbs may cause risk of bleeding, but no clinical data are available
- Hypoglycemia may be ↑ for individuals receiving antidiabetic agents

Monitoring
- Preop PT (INR), blood glucose levels

Postoperative Period
- Theoretical ↑ risk of bleeding and hypoglycemia

Anticipated Problems/Concerns
- See Postoperative Period
- Pts who are avid garlic consumers should not double up doses to make up for missed doses while undergoing surgery
- If on coumadin postop, should be warned against heavy consumption

Garlic oil contact dermatitis

Facial/tongue swelling

Postoperative Period
- ↑ risk of bleeding and hypoglycemia

Perioperative Implications


ASSESSMENT POINTS

Moderate daily consumption has no effects on normal individuals. Effects not seen with cooked garlic.

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<tbody>
<tr>
<td>CARDIO</td>
<td>Reduced BP, reduced LDL cholesterol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RESP</td>
<td>Hypoglycemia</td>
<td>Insulin, oral hypoglycemic use</td>
<td>FSBG</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Bleeding</td>
<td>Anticoagulant use, coagulopathy, dysfunctional plts, bleeding disorders</td>
<td>Hematomas; poor surgical hemostasis</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Low dose: Enhanced peristalsis</td>
<td>&gt;5 cloves/d</td>
<td>Dyspepsia, eructation, pyrosis (heartburn), flatulence</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Large doses: Inhibited peristalsis; possible reduction in stomach cancer</td>
<td>Constipation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Spontaneous spinal epidural hematoma</td>
<td>Headache, paralysis</td>
<td>Neuro exam</td>
<td></td>
</tr>
<tr>
<td>ALLERGY/IMMUNO</td>
<td>Allergic reaction</td>
<td>Garlic oil contact dermatitis</td>
<td>Facial/tongue swelling</td>
<td></td>
</tr>
</tbody>
</table>
Ginger (Zingiber officinale)

Sanup Pathak
Wei Pan

Overview/Pharmacology
- Pungent constituents: Gingerol, shogaol, ginge-
erdiols, vanilloids, and diarylheptanoids
- Plasma concentration curve defined by a two-
compartment model with a terminal half life
of 7.2 min and a total body clearance of 16.8
mL/min/kg
- 92.4% serum-protein binding with elimination
by the liver and gut flora

Mechanism of Action
- Anti-5-HT, mediates anti-emetic effects
- Direct cholinergic agonist of post-synaptic M,
receptors and inhibitor of pre-synaptic muscar
inic autoreceptors: May mediate GI prokinetic
effects.
- Cyclooxygenase and lipoxygenase inhibition:
Mediates anti-inflammatory and anti-thrombotic
effects by decreasing levels of thromboxane B,
prostaglandin E, and leukotrienes
- Inhibition of cytokine and chemokine
induction in vitro: Mediates anti-inflammatory
effects
- Insulin sensitization mediates hypoglycemic
and lipid-lowering effects
- Calcium channel inhibition mediates decrease
in BP

Uses
- Ginger ranks 18th in herbal supplement sales
- Has long been used in Ayurvedic and Chinese
medicine for a wide variety of conditions incl
arthritis, rheumatism, constipation, indigestion,
nausea, vomiting, motion sickness, and diabetes
- In vivo human studies show ginger effective
in management of pregnancy assoc and postop
N/V
- In vivo animal studies show ginger has signifi-
cant anti-inflammatory, anti-thrombotic, hypoten-
sive, glucose-lowering, and lipid-lowering effects.
- In vitro studies show ginger has significant
antioxidant, antitumorogenic, anti-inflammatory,
antiviral, and antimicrobial effects.

Perioperative Risks
- No toxic or unpleasant side effects reported
in human studies with therapeutic doses
- High doses may prolong bleeding time due to
inhibition of thromboxane synthetase and stimu-
lation of prostacyclin
- High doses may lower BP

Worry About
- Potential additive or synergistic effects with
antiplatelet agents, heparin, or warfarin, which
may increase bleeding risks
- Potential hypotensive effect

Possible Drug Interactions

Preoperative Period
- Possible interaction with antiplatelet agents or
warfarin

Induction
- May potentiate hexobarbital
- May potentiate hypotension

Postoperative Concerns
- May increase bleeding complications

Anticipated Problems/Concerns
- May increase bleeding complications when
used with antiplatelet drugs, warfarin, or heparin
- May consider avoiding use in the presence of
gallstone conditions
- May potentiate periop hypotension

Ginkgo

Uses
- Used for its antioxidant and polyphenol properties, improvement of cognitive performance in demented pts with Alzheimer’s disease or vascular dementia, and improvement in symptoms of intermittent claudication, Raynaud’s phenomenon and acrocyanosis. Evidence for effectiveness debated.
- Used in pts with macular degeneration, sexual dysfunction, symptoms of vertigo, depression, anxiety, and vitiligo.
- Also used but ineffective in prevention of age-related memory impairment, dementia, and altitude sickness.

Perioperative Risks
- Increased risk of bleeding and drug interactions.
- Lack of safety data in certain populations. Therefore, not recommended for use in pregnancy, breastfeeding, and children younger than 12 y of age.
- Commonly reported side effects incl N/V/D, headache, and bleeding.

Worry About
- Spontaneous bleeding can occur due to the inhibition of plt aggregation.
- Risk of bleeding is further increased if combined with antithrombotic drugs (aspirin, NSAIDs, clopidogrel, dipyridamole), anticoagulant drugs (heparin, warfarin, enoxaparin), and other herbal medicines known to increase bleeding (ginger, garlic, ginseng).
- Can decrease the effectiveness of anticonvulsants (valproate, carbamazepine)
- May enhance the effects of MAO inhibitors (phenelzine, selegiline, tranylcypromine) and increase the risk of serotonin syndrome when taken with SSRIs.
- Interactions have also been reported with calcium channel blockers, trazadone, acetylcysteinerase inhibitors, blood glucose lowering medications, insulin, erectile dysfunction drugs, and thiazide diuretics.

Overview
- Ginkgo (Ginkgo Biloba) is one of the oldest tree species and is one of the most common supplements used worldwide.

Drug Class/Mechanism of Action/Usual Dose
- Active elements responsible for ginkgo’s medicinal effects incl ginkgo flavone glycosides and terpine lactones, both obtained from dry leaves of the tree.

Extracts standardized to contain 24% to 27% ginkgo flavone glycosides and 6% terpines, are commonly found in 40–80 mg oral capsules and recommended TID.
- Ginkgo has a wide range of properties. Hemodynamic effects incl antagonism of plt activating factor (PAF), lowering serum fibrinogen levels, stimulation of endothelium derived relaxing factor (EDRF), facilitating prostacycline release and inhibiting nitric oxide.
- CNS effects are mainly attributed to its antioxidant characteristics. Decreasing superoxide release and acting as a scavenger of free radicals results in prevention of hypoxic brain tissue damage and improvement of cerebral metabolism. O2 utilization in the brain may be improved and age-related changes in the animal hippocampus may be prevented.
- Additional studies have indicated that ginkgo reversibly inhibits MAO-A and MAO-B, inhibits acetylcysteinerase, and decreases adrenal benzodiazepine receptors.

Perioperative Implications
Preoperative Concerns
- Outside of the potential increased risk of bleeding, periop concern with ginkgo intake revolves around drug interactions.
- Minimal data on effects in pregnancy, breastfeeding, and pediatrics.
- Many pts do not account for alternative medicines when asked for medication lists by their physician.
- Inhibition of plt aggregation can result in significant intraop bleeding, thus D/C ginkgo at least 36 hr before elective surgery is recommended.

Monitoring
- Routine
- Airway
- Avoid nasal intubation to minimize intranasal bleed

Preinduction/Induction
- Avoid excessive hypotension with induction agents, because ginkgo’s subtle vasodilatory effects can further decrease BP and ginkgo’s effects on the adrenal receptors minimizes a normal stress response. Hence, prolonged and excessive hypotension can jeopardize vital organ perfusion.

Postoperative Period
- No known concerns

Maintenance
- Side effects can be amplified with concomitant use of interacting drugs. Such concerns incl bleeding, hypotension, seizures, sedation, serotonin syndrome, and cholinergic crisis.

Extubation
- Avoid administering classes of drugs that may interact and potentiate the effects of ginkgo as previously mentioned.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>↑ Ocular blood flow</td>
<td>Bleeding by Hx</td>
<td>Mucosal bleeding</td>
<td>BP/HR</td>
</tr>
<tr>
<td>CARDIO</td>
<td>Vasodilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Inhibition of platelet aggregation</td>
<td>Bleeding, bruising</td>
<td>Mucosal bleeding</td>
<td>Petechiae</td>
</tr>
<tr>
<td>GI</td>
<td>N/V/D</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>↑ Cerebral blood flow</td>
<td>Headache</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Contact dermatitis</td>
<td>Exposure</td>
<td>Rash</td>
<td></td>
</tr>
</tbody>
</table>


Ginseng

Uses
- Ginseng has been used for more than 2000 years in Chinese herbal medicine for a variety of proposed health benefits.
- Used as an adaptogen, it is believed to increase the body’s resistance to stress and fatigue.
- Reputed to have antioxidant properties
- Reputed to have antihypoglycemic properties. (Some studies have shown Asian ginseng may lower plasma glucose.)
- Used as a stimulant to enhance physical, sexual, and mental performance (it has not been easy to quantify these effects, because scientific studies have not returned consistent results).
- Thought to stimulate the immune system, and to have anticancer properties

Perioperative Risks
- Ginseng has the ability to lower postprandial blood glucose in both pts with diabetes type 2 and non-diabetic pts. This effect may lead to unintended hypoglycemia, particularly in pts who have fasted prior to surgery.
- Ginseng may promote bleeding in surgical pts. Ginsenosides (the active ingredient) in American ginseng has been shown to inhibit plt aggregation. Studies in laboratory rats show prolongation of the coagulation time of thrombin and activated partial thromboplastin. One study suggests that the antiplatelet activity of panaxynol, a constituent of ginseng, may be irreversible in humans. Given these findings, it may be prudent to recommend that pts D/C ginseng use at least 7 d prior to surgery.

Worry About
- The development of hypoglycemia, esp. in diabetic pts taking insulin or oral antihyperglycemic agents
- May have additive effects when used with corticosteroids and may intensify the side effects of corticosteroids
- May lead to development of headache, tremors and manic episodes when used in pts receiving MAO inhibitors such as phenelzine
- Interferes with the pharmacodynamics and drug level monitoring of pts taking digoxin and may increase digoxin levels. The underlying mechanism is unclear.
- May increase the risk of surgical bleeding due to its antiplatelet effects and inhibition of the coagulation cascade
- Ginseng taken together with anticoagulants or with aspirin or other NSAIDs may increase the risk of bleeding.
- Ginseng may have estrogen-like effects and should be avoided in pregnant or breastfeeding women and in children. Avoid the use of ginseng in pts with hormone sensitive conditions, such as breast cancer, uterine cancer, or endometriosis.
- Consumption of ginseng can increase and/or decrease BP. Caution should be used in those with Htn or low BP.

Overview
- The term ginseng refers to several species of the genus Panax and comprises a family of plants (American ginseng, Asian ginseng, Chinese ginseng, Korean red ginseng, Panax ginseng: Panax spp., incl P. ginseng C.C. Meyer and P. quinquefolius L., excluding Eleutherococcus senticosus).
- Dietary supplements are typically derived from American ginseng (Panax quinquefolius) or Asian ginseng.
- Siberian ginseng (Eleutherococcus senticosus) is a different genus and does not contain the ingredients believed to be active in the two forms used in supplements.
- Ginseng can be taken as fresh or dried roots, extracts, solutions, capsules, tablets, sodas, and teas, or used as cosmetics.

Drug Class/Mechanism of Action/Usual Dose
- The active ingredients in American ginseng are panaxosides (saponin glycosides). The active ingredients in Asian ginseng are ginsenosides (triterpenoid glycosides).
- Most of the pharmacological actions of ginseng are attributed to the ginsenosides that belong to a group of compounds known as steroidal saponins.

System | Effect | Test
--- | --- | ---
CARDIO | Tachycardia, palpitations, Htn with other cardiac stimulants, edema | HR, BP
HEME | Decreases effectiveness of warfarin, inhibition of coagulation cascade | INR, PT, PTT
NEURO | Excessive use: Somnolence, hypertonia, nervousness, and excitability | |
ENDO | Hypoglycemia | Blood glucose
REPROD | Mastalgia, postmenopausal bleeding | Hct


Perioperative Implications

Preoperative Concerns
- Check coagulation studies, monitor blood glucose

Monitoring
- Standard

Induction
- No specific concerns

Postoperative concerns
- Monitor blood glucose level, monitor for signs of excessive postop bleeding

Airway
- No specific concerns
**Glucosamine Sulfate**

**Uses**
- For pain assoc with osteoarthritis (OA), particularly of the knee
- IBD
- Other inflammatory disorders such as rheumatoid arthritis, psoriasis

**Perioperative Risks**
- No convincing evidence of increased periop risk due to glucosamine therapy
- No known significant interactions with commonly administered anesthetic drugs

**Worry About**
- Increase in INR in pts on warfarin who initiate glucosamine therapy, or increase glucosamine dose

**Overview**
- Classified as a food additive, not FDA regulated, made from crustacean skeletons
- As monotherapy, little consistent evidence of therapeutic effect
- Side effect profile indistinguishable from placebo and better than NSAIDs
- In combination with chondroitin may prolong the time to total knee replacement in those with severe OA

**Drug Class/Mechanism of Action/Usual Dose**
- Glucosamine is a component of the extracellular matrix of articular cartilage.
- Recommended oral dose is 1500 mg qd, may have anti-inflammatory effects
- The mechanism of action of glucosamine is unknown, presumably aids in cartilage repair

**Perioperative Implications**
- Glucosamine therapy has no significant periop anesthetic implications. No need to interrupt therapy for a surgical procedure, no reason to modify an anesthetic plan due to glucosamine, and there is no urgency with regards to restarting therapy postop.

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</tr>
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<tr>
<td>COAG</td>
<td>May potentiate warfarin</td>
<td>PT/INR if pt on warfarin</td>
</tr>
<tr>
<td>ENDO</td>
<td>No consistent effect</td>
<td>Glucose if otherwise indicated</td>
</tr>
</tbody>
</table>

**Key References**
# Glycine

**Uses**
- Glycine is an inhibitory neurotransmitter in the brainstem and spinal cord.
- Glycine and GABA receptors may mediate the effects of inhaled anesthetics.
- It is a nonessential amino acid sold as a natural sugar substitute, a sedative, an antacid; to promote muscle growth, ↓ Sx of BPH, as an polyphenol, and antipsychotic.
- Glycine 1.5% used as a nonhemolytic irrigation solution during TURP.
- Antagonists of glycine binding to NMDA receptor complex are used as anticonvulsants.
- Attemps to use glycine and other NMDA agonist in schizophrenia have had little success.
- Intrathecal glycine is not different than placebo in CRPS treatment.

**Perioperative Risks**
- TURP syndrome incidence is 0.5–8% and mortality rate is 0.2–0.8% up to 25% in severe TURP syndrome.
- Glycine metabolized to ammonia can lead to hyperammonemic encephalopathy.

**Worry About**
- Glycine irrigation is contraindicated in pts with anuria.
- TURP syndrome is thought to be due to hypotension, hypomosmolality, and elevated glycine levels due to absorption of irrigation fluid. Onset from 15 min after starting irrigation up to 24 hr postop. Manifestations likely related to the glycine load incl myocardial depression, hemodynamic changes, and visual disturbances. Other Sx incl burning sensations in the face, N/V, weakness, confusion, seizure, coma.
- Glycine irrigation should be used with caution in pts with CHF.

**Overview/Pharmacology**
- Glycine is inhibitory on ligand-gated, strychnine-sensitive Cl– channel receptors but excitatory on strychnine-insensitive NMDA receptors where it is a cofactor for activation of the NMDA receptor by L-glutamate.
- Glycine metabolism: Primarily transamination to serine, and deamination to ammonia which is converted to urea and excreted by the kidneys. A portion of absorbed glycine is excreted unchanged by the kidneys.

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<tr>
<td>CARDIO</td>
<td>T wave depression or inversion, increased long-term risk of MI</td>
<td>Homeopathic use or glycine 1.5% irrigation</td>
<td>BP, HR, ECG</td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>Antagonists assoc with aplastic anemia</td>
<td>Homeopathic glycine use</td>
<td>Skin/mucous membranes for infection/petechiae</td>
<td>CBC, peripheral smear, bone marrow Bx</td>
</tr>
<tr>
<td>GU</td>
<td>Metabolizes oxalate and glycolate may produce renal failure</td>
<td>TURP with glycine irrigation or homeopathic use</td>
<td>Chem 7</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Gastric antacid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Glycine accumulates in cells, which ↑ cerebral edema, hyponatremia, hypotonicity, and direct toxicity account for neurologic Sxs in TURP syndrome</td>
<td>Headache, N/V, visual changes, seizure, weakness, encephalopathy, lethargy</td>
<td>Mental status, visual acuity, strength</td>
<td>Serum, Na serum osmolality</td>
</tr>
<tr>
<td>PULM</td>
<td>Pulm edema (↑ with spinal anesthesia)</td>
<td>TURP</td>
<td>SOB, wheeze, frothy sputum</td>
<td>CXR, SpO2</td>
</tr>
</tbody>
</table>

**Drug Class/Usual Dose**
- Glycine 1.5% solution is used as an irrigation solution during endoscopic procedures, esp. TURP. It is nontoxic, has a refractive index close to that of water, and is nonhemolytic despite a hypotonic osmolality of 200–220 mOsm/L.
- Homeopathic use for BPH at 780 mg/d × 2 wk and then 390 mg for the next 3 mo.
- Glycine 30–60 g/day improves negative symptoms of schizophrenia.
- NMDA receptor allows influx of Na+ and Ca2+. Overstimulation of this channel leads to Ca2+ overload in neurons, which has been shown to be neurotoxic. Glycine site antagonists ↓ the release of excitatory amino acids, such as glutamate, which are known to potentiate cerebral ischemic injury.

**Possible Drug Interactions**
- Clozapine, haloperidol, olanzapine, risperidone

**Perioperative Implications**

**Preoperative Period**
- Elicit recent Hx of glycine use as homeopathic treatment.
- Anuria is a contraindication to use of glycine irrigation. Caution in oliguric pts.

**Induction/Maintenance**
- No known interactions with homeopathic doses of glycine.
- If pt is using glycine as a homeopathic antipsychotic, affect/mental status may be problematic given underlying disease and side effect profile of drug.

- Risk of TURP syndrome. Be aware of degree of blood loss and amount of irrigation used. If regional anesthetic used, monitor for symptoms of glycine toxicity: N/V, visual changes, weakness. Also monitor for hemodynamic instability; ECG changes, hypo- or Htn.
- With intraop onset of TURP syndrome, surgery should be terminated as soon as possible.

**Postoperative Concerns**
- TURP syndrome may occur within 15 min of beginning irrigation or as late as 24 hr postop. Monitor for signs of changing mental status, hemodynamic instability, seizures.
- Seizures, if caused by glycine activity on NMDA receptors, could be treated with NMDA receptor antagonists or glycine antagonists. Mg2+ exerts a negative effect on NMDA receptors and Mg2+ may be low after TURP, so a trial of Mg2+ therapy may be warranted.


**FDA Professional Drug Information. Glycine. www.drugs.com/pro/glycine.html.**
Goldenseal (Hydrastis Canadensis)

Lynn A. Fenton

Overview/Mechanism of Action/Pharmacology

Compounds: Isoquinoline alkaloids are the main bioactive constituents. The chief alkaloids are berberine, hydrastine, and (−)-canadine (no information available).

In investigational uses: Antineoplastic and anti-HIV treatment.

Historical uses: Native American Indians have used GS for multiple medicinal purposes and for the production of a yellow dye.

Perioperative Risks

Prolonged use can cause digestive disorders, constipation, excitatory states, hallucinations, and occasional delirium.

Side effects of drug (large doses): Mucocutaneous irritation, GI tract upset (N/V), cardiac and uterine contractility, bradycardia, cardiac damage, vasoconstriction/Htn, spasms, neonatal jaundice, hyperbilirubinemia, and death.

See section on Perioperative Implications/Drug and Herbal Interactions.

Anticipated Problems/Concerns

Not to be used by pregnant or lactating women.

Oral: Used for treating gastritis, peptic ulcers, colitis, atonic dyspepsia with hepatic Sx, jaundice, anorexia, UTIs, menorrhagia, dysmenorrhea, gonorrhea, postpartum and internal hemorrhage, cancer, conjunctivitis, tinnitus, catarrhal deafness, earaches, malaria, nasal congestion, upper resp tract inflammation, canker sores, and sore gums.

Topical: Used for eczema, itching, acne, dandruff, ringworm, herpes labialis, and wounds.

Goldenseal has been used orally to create false negative urine tests for several illicit drugs.

Other Names

• Eye balm, eye root, goldenroot, golden seal, goldsiegel, ground raspberry, Indian dye, Indian plant, Indian tumeric, jaundice root, orange root, Sceau D’Or, tumeric root, Warnera, wild curcuma, yellow Indian paint, yellow paint, yellow psocoon, yellow root

Indications/Uses

• Goldenseal (GS) is considered POSSIBLY SAFE for short-term, appropriate use.

• GS may be purchased in salve, tablet, bulk powder, or tincture forms.

• Oral: Used for treating gastritis, peptic ulcers, colitis, atonic dyspepsia with hepatic Sx, jaundice, anorexia, UTIs, menorrhagia, dysmenorrhea, gonorrhea, postpartum and internal hemorrhage, cancer, conjunctivitis, tinnitus, catarrhal deafness, earaches, malaria, nasal congestion, upper resp tract inflammation, canker sores, and sore gums.

• Topical: Used for eczema, itching, acne, dandruff, ringworm, herpes labialis, and wounds.

• Goldenseal has been used orally to create false negative urine tests for several illicit drugs.

Drug Class/Usual Dose

Oral doses: 0.5–1 g tid of the dried root or rhizome × 1 wk.

Liquid extract: (1:1, 60% EtOH) – 0.3–1 mL TID × 1 wk.

Tincture: (1:10, 60% EtOH) – 2–4 mL tid × 1 wk.

Oral rinse and topical solution: 6 g dried root/150 mL H2O tid – qid × 1 wk.

LD50 of berberine in humans is reported to be 27.5 mg/kg.

Doses in excess of 500 mg of berberine (or 8–100 g of dry root depending on the berberine concentration) can lead to significant toxicity.

ASSESSMENT POINTS

System | Effect | Assessment by Hx | PE/Test
--- | --- | --- | ---
CARDIO | Htn | CV response to Rx | HR, BP, ECG
 | Hypotension | | |
 | Bradycardia | | |
 | Inotropic effect | | |
PULM | Resp failure | SOB, DOE | Wheezing/SaO2, ABG, CXR
GI | Mucosal irritation | N/V, diarrhea | |
GU | Oxytocic | Abortion | |
 | Diuretic | Polyuria | |
ENDO | Increase effectiveness of insulin | Hypoglycemic SNS, Sx | |
 | | Tachycardia, Hypotension | |
 | | Hypokalemia | |
CNS | CNS stimulation | Muscle spasms, excitatory state, hallucinations, seizures, occasional delirium | EMG, EEG
 | Central paralysis | | |
HEME | Hemostatic | Oppose anticoagulation | INR, PTT

Perioperative Implications/Possible Drug-Herb Interactions

• Acid-inhibiting drugs: Might increase stomach acid thereby interfering with antacid-type meds.

• Antihypertensive agents: Vasoconstrictive action of hydrastine might interfere with BP control.

• B vitamins: Prolonged use can decrease B vitamin absorption.

• Berberine: Thought to act intraluminally; exerts antimicrobial activity against numerous bacteria, fungi, and protozoa; blocks adhesion of bacteria to epithelial cells to inhibit intestinal secretory responses to cholera and E. coli toxins; may increase effectiveness of insulin; may act as a vasoconstrictor to decrease uterine bleeding.

• Hydrastine: At low doses is hypotensive agent; at higher doses is a peripheral vasoconstrictor.


ALTERNATIVE MEDICINE

678
Licorice (Glycyrrhiza Glabra)

R. Blaine Easley

Uses
• Incidence in USA: One of the top ten herbal medications
• Medical: Historically used to improve immune function and treat a variety of conditions incl PUD, duodenal ulcers, cough and/or bronchitis, attherosclerosis, chronic fatigue syndrome, various cancers, AIDS, and Addison’s disease. Most recently a study has demonstrated its effectiveness in relieving constipation.

Perioperative Risks
• Unknown. Theoretical problems in pts with impaired renal function, Htn, chronic liver disease, cardiac arrhythmias, and hypertension.
• Potential for drug interactions. Pseudoaldosteronism has been produced experimentally in healthy subjects taking >100 g/wk.

Worry About
• Pseudoaldosteronism: Documented mineralocorticoid effects that result in fluid retention, hypernatremia, hypokalemia, and edema
• Hypertension: Direct effects on vascular smooth muscle tone independent of mineralocorticoid properties
• Vasospasm and/or headache: Case reports of cerebral artery spasm causing severe headache, visual disturbances, and potential ischemia have recently been published.

• Hypokalemia and/or muscle weakness: Chronic usage related to hypokalemia myopathies, muscle cramps, and skeletal muscle spasms
• Arrhythmias: Rare side effect, but more worrisome in pts with Hx of arrhythmias requiring medication (e.g., digoxin)
• Paresthesias: Numbness in extremities may be a sign of licorice toxicity

Overview/Pharmacology
• Licorice is the common name given to various substances derived from the plant root Glycyrrhiza glabra (Spanish licorice). This plant is a perennial that grows 3–7 ft high and originated from Europe and Asia. Also called sweet root and licorice root.
• Glycyrrhizin and/or glycyrrhizic acid (the glucoside form) and glycyrrhetinic acid (the glycoside form) are the most important substances or metabolites found in licorice. The roots also contain coumarins, flavonoids, volatile oils, and plant sterols.
• Licorice and its components are metabolized and excreted by the liver and kidneys.
• Mineralocorticoid effects of licorice, via glycyrrhetinic acid, result from the inhibition of 11-β-hydroxysteroid dehydrogenase (an enzyme that normally inactivates cortisol by converting its C11 alcohol to a ketone). Excess glucocorticoids then bind to mineralocorticoid receptors and produce a mineralocorticoid response, as evidenced by ↑ sodium retention and Htn. Thereby, licorice ingestion creates a syndrome of hyperaldosteronism characterized by hypernatremia, Htn, hypokalemia, and suppression of the renin-angiotensin system.
• Glycyrrhetinic acid also inhibits 15-hydroxyprostaglandin dehydrogenase and prostaglandin reductase. These two enzymes are important in the metabolism of prostaglandin E and F₂, perhaps explaining licorice’s immunologic benefits, effects on reducing cough and/or bronchospasm, protection of gastric mucosa, and benefit by decreased plt aggregation.

Drug Class/Usual Dose
• Made from peeled and unpeeled dried root compounded and sold as powders, dry extracts, and liquid extracts. Some preparations such as deglycyrrhizized licorice (DGL) have removed harmful compounds. Unfortunately, preparation and advertising of these compounds is unregulated by the FDA.
• Licorice is taken in the following manner
  • Dried root: 1–5 g PO tid, up to 6 wk (indication: general use)
  • Extract: (1:1 preparation) 2–5 ml PO tid, up to 6 wk (indication: general use)
  • DGL extract: 1.5–3 g/d for peptic ulcer
  • DGL extract: 380–760 mg PO 20 min before meals for peptic ulcer

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Headache</td>
<td>Exposure/use of licorice</td>
<td>Visual acuity</td>
<td>Neuro consult, possible MRI</td>
</tr>
<tr>
<td></td>
<td>Visual changes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Paresthesia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Hypovolemia</td>
<td>Exposure/use of licorice</td>
<td>BP/HR, consider orthostatics</td>
<td>ECG rhythm strip</td>
</tr>
<tr>
<td></td>
<td>Hypervolemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Htn</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Arrhythmia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Black stools (rare)</td>
<td>Report of loose, dark stool</td>
<td>Abd exam</td>
<td>Stool guaiac</td>
</tr>
<tr>
<td></td>
<td>Laxative effect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HEME</td>
<td>↓ Clothing (rare)</td>
<td>Bleeding problems</td>
<td>Pts, PT/PTT</td>
<td>Serum chemistries</td>
</tr>
<tr>
<td>ENDO</td>
<td>Hyperglyceria</td>
<td>Exposure/use of licorice</td>
<td>Wt gain, ↑ Urination</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypernatremia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypokalemia</td>
<td></td>
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</tr>
</tbody>
</table>


Possible Drug Interactions

Preoperative Period
• Multiple adverse drug interactions reported in pts using licorice preparations and prescription medications. Licorice can interfere with the function of hormone supplements (e.g., birth control pills), oral hypoglycemic agents, and corticosteroids. Electrolyte imbalances and GI symptoms can be worsened by usage of licorice with diuretics and laxatives. Digoxin usage and licorice-induced hypokalemia can be potentially arrhythmogenic.
• Electrolyte abn of hypokalemia, hypernatremia, and metabolic alkalosis should be sought and corrected before surgery in high dose frequent users.
• Pt should be instructed to D/C use of the herbal medicine approx 2 wk before elective surgery.

Induction/Maintenance
• No known interactions with licorice metabolites. However, pseudoaldosteronism should be considered and anesthetic management directed at the problems of hypokalemia, Htn, and fluid status. Placement of an arterial line and/or central venous line should be considered in symptomatic pts. (See Hyperaldosteronism [secondary] in Diseases section.)

Adjuncts/Regional Anesthesia/Reversal
• No known interactions. Consider pros and cons of NSAI D use intraop, esp. if no assessment of renal function. Careful attention to neurologic exam and/or paresthesias before initiation of regional technique.

Emergence/Extubation
• No known interactions. Acute topical administration has been used to prevent postop sore throat without adverse effect. However, hypokalemia with or without a Hx of muscle weakness could potentially modify nondepolarizing muscle relaxant response.

Postoperative Concerns
• Failure of resolution of preop symptoms attributed to licorice use with D/C of licorice-containing compound should prompt investigation of other causes.
• Continued monitoring of fluid and electrolyte status. If problems with hypokalemia continue, despite potassium supplementation, then consider potassium-sparing diuretics (e.g., triamterene), a competitive aldosterone antagonist (e.g., spironolactone), and investigating other causes.
Melatonin (N-Acetyl-5-Methoxytryptamine, Bevitamel, Vitamist, Melatonex)  
Ori Gottlieb

**Risks**
- May interact with other CNS-acting medications such as hypnotics, sedatives, or psychotropics
- Contraindicated during pregnancy and when breastfeeding
- May cause excessive somnolence
- Not recommended in infants and children due to insufficient data
- Not FDA controlled—quality/potency may vary
- The use of animal-source melatonin products is not recommended due to the risk of viral contamination or infection.

**Overview/Pharmacology**
- Secretion modulated by enzymes secreted by hypothalamus in response to dark
- Exogenous routes of administration: Oral tablets, capsules, lozenges, teas, sprays
- Unlike endogenous melatonin, oral doses undergo first-pass hepatic metabolism with a bioavailability of 30–50%
- Crosses the blood-brain barrier
- The mean elimination $T_{1/2}$ is 45 min. Only 0.01% of melatonin is excreted unchanged in urine.

**Endogenous Actions**
- Secreted by the pineal gland in response to the absence of photic stimuli (known as the ‘Darkness Hormone’)
- Reduces the body’s core temp in preparation for sleep
- Secretion peaks during the pediatric years and decreases with age
- In some way, melatonin is involved in reproductive function. Receptors found in reproductive tissues.
- Endogenously produced melatonin may have a significant role in deferring a number of free radical-related diseases and some pathophysiological changes assoc with aging.

**Exogenous Actions**
- Resets the body to the environmental clock and allows pts to normalize physiologic and behavioral sleep patterns
- Used commonly as a preventive and therapeutic anti-jet lag agent
- Useful in individuals with poor circadian synchrony such as the visually impaired


**Perioperative Implications**
- May be used as a preop anxiolytic agent. Comparable anxiolysis to midazolam without amnestic effect. Questionable effects in the elderly.
- Studied in children as a premedicant; shown to be comparable to midazolam but with faster recovery and a lower incidence of postop cognitive impairment and delirium.
- Melatonin ↑ benzodiazepines binding to receptor sites, enhancing activity
- Methamphetamine users may also be taking melatonin to offset the insomnia brought on by the drug.

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Nutraceuticals

**Risks**
- Approx 64% of pts have taken or are taking a nutraceutical agent.
- Incidence in USA: More than 50% take dietary supplements and in a study of over 1000 outpa-
tients, approx 33% were using herbs at the time of surgery or pain procedures.
- 31% use herbal drugs in combination with prescriptions
- Up to 70% of pts do not inform the physician about use of these products at the time of routine anesthesia preop assessment.
- Some of these agents have the potential to cause serious drug interactions, incl bleeding, prolonged sedation, and hemodynamic instability periop.

**Perioperative Risks**
- Hemodynamic and/or CV instability or collapse
- Increased risk of bleeding (in particular many of the “G” herbs and EPA [not DHA] component
  of fish oil, so not a problem for people supplementing with algal DHA)
- Potential for prolongation of anesthetic and barbiturates
- Electrolyte imbalance and/or renal dysfunction
- Atn thyroid function

**Worry About**
- Oversedation from prolonged effects of barbiturates
- Hemodynamic instability

**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>Name</th>
<th>Common Uses</th>
<th>Possible Side Effects/Drug Interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatine</td>
<td>Endurance/strength, bodybuilding</td>
<td>Exacerbation of pre-existing renal dysfunction</td>
</tr>
<tr>
<td>Ephedra (Ma-Huang)</td>
<td>OTC diet aids</td>
<td>Severe intraop hypotension; arrhythmias; enhanced sympathomimetic effects with MAOI; Htn with oxytocin</td>
</tr>
<tr>
<td>Echinacea</td>
<td>Common colds, coughs</td>
<td>May cause hepatotoxicity or increase barbiturate duration</td>
</tr>
<tr>
<td>Feverfew</td>
<td>Migraine prophylactic</td>
<td>Inhibition of plt activity with ↑ bleeding, ↑ hemodynamic instability</td>
</tr>
<tr>
<td>Garlic</td>
<td>↓ BP and lipids; antioxidant and antithrombolytic</td>
<td>May potentiate warfarin; will see increased INR (PT), hypotension</td>
</tr>
<tr>
<td>GBL/GHB</td>
<td>Bodybuilding, sleep inducement, wt loss</td>
<td>GHB is “date rape” drug; may cause death, seizures, vomiting, bradycardia, slowed breathing, prolonged anesthetic</td>
</tr>
<tr>
<td>Ginger</td>
<td>Antinauseant</td>
<td>May ↑ bleeding time, hemodynamic instability</td>
</tr>
<tr>
<td>Ginkgo biloba</td>
<td>Circulatory stimulant</td>
<td>May enhance bleeding with anticoagulants or antithrombotics, decrease effectiveness of barbiturates</td>
</tr>
<tr>
<td>Ginseng</td>
<td>Energy-level enhancer, antioxidant</td>
<td>Tachycardia, Htn, mastalgia, postmenopausal bleeding, potential for ↑ bleeding with warfarin, hypoglycemia, Htn</td>
</tr>
<tr>
<td>Garlic</td>
<td>↓ BP and lipids; antioxidant and antithrombolytic</td>
<td>May potentiate warfarin; will see increased INR (PT), hypotension</td>
</tr>
<tr>
<td>Goldenseal</td>
<td>Diuretic</td>
<td>Works as an aquaretic (no sodium excreted); may worsen edema/Htn</td>
</tr>
<tr>
<td>Kava-Kava</td>
<td>Anxiolytic</td>
<td>Potentiates barbiturates and benzodiazepines; portion of kava can cause liver toxicity and liver failure</td>
</tr>
<tr>
<td>Licorice</td>
<td>Gastric/duodenal ulcers</td>
<td>May cause ↑ BP, hypokalemia, edema; contraindicated with chronic liver conditions, renal insufficiency</td>
</tr>
<tr>
<td>St. John’s wort</td>
<td>Mild/moderate depression</td>
<td>May prolong anesthesia (anecdotal); pseudoephedrine, MAOIs, SSRIs should be avoided; can potentially interact with meperidine and cause serotoninergic crisis</td>
</tr>
<tr>
<td>Triax metabolic</td>
<td>Wt loss aid</td>
<td>Contains potent thyroid hormone; may cause heart attacks, strokes; altered thyroid function</td>
</tr>
<tr>
<td>accelerator (Triax)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valerian</td>
<td>Mild sedative or anxiolytic</td>
<td>May potentiate barbiturates, and other gabbaergic sedatives</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>Antioxidant, anticlotting agent</td>
<td>↑ BP in Htn pts with too high a daily dosage (400 IU/d); ↑ bleeding; may worsen vitamin K deficiency; may ↓ thyroid hormone levels</td>
</tr>
</tbody>
</table>


**Perioperative Risks**
- The American Society of Anesthesiologists (ASA) recommends that all herbal medications be D/C 2–3 wk prior to elective surgery, because it takes 5–6 half lives for an agent to leave the body, and these substances lack uniform uptake, distribution, and elimination as they are not considered drugs by the Food and Drug Administration.
**Phytosterols**

**Uses**
- Naturally occurring in human diet
- Used as a supplement, esp. in margarines, to reduce cholesterol levels
- May also possess anti-inflammatory, anti-pyretic, antineoplastic, and immune-modulating properties
- Some recent evidence questions the beneficial effect of phytosterols and potential for increased CV risk

**Perioperative Risks**
- None known

**Worry About**
- Pts may be taking phytosterols because of hypercholesterolemia and occult CAD

**Overview/Pharmacology**
- Phytosterols (incl plant sterols and stanols) are natural components of edible vegetable oils such as sunflower seed oil and, as such, are natural constituents of the human diet.
- It is difficult to incorporate free sterols into edible fats and/or oils because of their insolubility, whereas sterols esterified to fatty acids are more fat soluble.
- In the intestine, most sterol esters are hydrolyzed to free sterols as part of the normal digestive process.
- Plant stanols are hydrogenation products of the respective plant sterols, e.g., campestanol and/or campesterol and sitostanol and/or sitosterol, and are found in nature at very low levels.
- Enrichment of foods such as margarines with plant sterols and stanols is one of the recent developments in functional foods to enhance the cholesterol-lowering ability of traditional food products.
- May reduce the absorption of some fat soluble vitamins. Randomized trials have shown that plant sterols and stanols lower blood concentrations of β-carotene by about 25%, concentrations of α-carotene by 10%, and concentrations of vitamin E by 8%.

**Drug Class/Usual Dose**
- Consumption of plant sterols and plant stanols lowers blood cholesterol levels by inhibiting the absorption of dietary and endogenously produced cholesterol from the small intestine, and the plant sterols and/or stanols are only very poorly absorbed themselves.
- This inhibition is related to the similarity in physicochemical properties of plant sterols and stanols and cholesterol and may be related to two mechanisms:
  - The greater the amount of plant sterols and/or stanols, the lower the solubility and perhaps the greater the amount of cholesterol precipitated. Cholesterol in the crystalline form cannot be absorbed.
  - Competition for space in mixed micelles
- Being marketed in new margarine formulations

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hypercholesterolemia</td>
<td>CAD, angina</td>
<td>Chest pain</td>
<td>ECG</td>
</tr>
<tr>
<td>GI</td>
<td>Malabsorption of some vitamins</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Possible Drug Interactions**
- No known drug interactions

**Anticipated Problems/Concerns**
- None known

**Pseudoephedrine**

**Uses**
- An OTC sympathomimetic commonly used as a nasal decongestant or for opening obstructed eustachian ostia
- Used in the symptomatic treatment of reactive airway disease; however, appears to be ineffective as a bronchodilator
- Also used for treatment of ejaculatory dysfunction, and as a starting material for illicit drug manufacturing
- Abuse and addiction to OTC stimulants does occur, particularly in those with eating disorders or erratic work hours such as truck drivers. Assoc with myocardial injury and withdrawal symptoms in this setting.

**Perioperative Risks**
- Concern about the coadministration of other sympathomimetic agents because of the possibility of additive effects and increased toxicity

**DRUG EFFECTS**

<table>
<thead>
<tr>
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<th>Assessment by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>Hm, dysrhythmias, cardiac irritability</td>
<td>Palpitations</td>
<td>BP/HR</td>
<td>ECG</td>
</tr>
<tr>
<td>HEENT</td>
<td>Mucosal vasoconstriction</td>
<td>Nasal congestion</td>
<td>Absence of hyperemia of nasal mucosa</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Reduction of vol of nasal mucosa</td>
<td>Head stuffiness</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Drainage of sinus secretions, opening of obstructed ostia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEURO</td>
<td>Nervousness, excitability, restlessness, dizziness, weakness, insomnia, headaches, drowsiness</td>
<td>Tremors, anxiousness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GU/RENAL</td>
<td>Urinary retention</td>
<td>Difficulty voiding, emptying bladder completely</td>
<td>Tachycardia, Htn</td>
<td>Bladder US, postvoid residuals</td>
</tr>
<tr>
<td>GI</td>
<td>N/V</td>
<td>Abdominal tenderness</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Concerns**
- Oral administration of usual doses to normotensive pts usually produces minimal effects
- Hm, tachycardia in those sensitive
- Those with concomitant hyperthyroidism, ischemic heart disease, or prostatic hypertrophy may be more at risk
- May ↑ the irritability of the heart muscle and result in multifocal PVCs
- May be teratogenic; avoid use in pregnant pts if possible; avoid use in breastfeeding women
- Geriatric pts may be esp. sensitive

**Monitoring**
- Routine

**Induction**
- Increased absorption of pseudoephedrine with antacid administration

**Airway**
- Improvement of airway edema and congestion related to mucosal hyperemia is often seen

**Maintenance**
- Careful administration/titration of other sympathomimetic drugs

**Regional Anesthesia**
- Pts may be more prone to urinary retention with regional techniques that block sacral roots.

**Postoperative Concerns**
- Resumption of drug should not pose particular problems once vital signs are stable.

**Anticipated Problems/Concerns**
- Caution in administering other sympathomimetic agents
- β-Adrenergic blocking drugs may ↑ pressor effect
- Antihypertensive effects of reserpine, methyldopa may be diminished

**Overview/Pharmacology**
- Acts directly on α- and, to a lesser degree, β-adrenergic receptors. Has an indirect effect by releasing NE from its storage sites.
- α-Adrenergic effects result from the inhibition of the production of cAMP by inhibition of the enzyme adenyl cyclase, whereas β-adrenergic effects result from stimulation of adenyl cyclase activity

**Drug Class/Dose**
- T½ 6 h for standard preparation and 12 h for extended release
- Adults and children >12 y of age: 60 mg q 4–6 h with a maximum dosage of 240 mg/d
- Children 6–11 y of age: 30 mg q 4–6 h with a maximum dosage of 120 mg/d
- Children 2–5 y of age: 15 mg q 4–6 h with maximum dosage of 60 mg/d
- Children <2: No FDA approved dosing

- Pressor effects of pseudoephedrine are more pronounced in
  - Hypertensive pts
  - Pts taking β-adrenergic blocking drugs
  - Pts taking serotonin/norepinephrine reuptake inhibitors (SNRIs)
- May ↑ heart irritability
- MAO inhibitors, by increasing the quantity of NE, potentiate pseudoephedrine’s indirect pressor effects; infrequently, a hypertensive crisis may result
- May also reduce the antihypertensive effects of reserpine and methyldopa
- May relax bronchial smooth muscle by stimulation of β-adrenergic receptors, but this effect is not consistent
- Readily and completely absorbed; elimination is predominantly renal and pH dependent

**DRUG EFFECTS**

- Overdose may cause hallucinations, CNS depression, seizures, and death
  - Monitoring
  - Routine
  - Induction
  - Increased absorption of pseudoephedrine with antacid administration
  - Airway
  - Improvement of airway edema and congestion related to mucosal hyperemia is often seen
  - Maintenance
  - Careful administration/titration of other sympathomimetic drugs
- Acts directly on α-receptors in the mucosa of the resp tract producing vasoconstriction, therefore shrinking mucous membranes, reducing edema and congestion
- May relax bronchial smooth muscle by stimulation of β-adrenergic receptors, but this effect is not consistent
- Readily and completely absorbed; elimination is predominantly renal and pH dependent
Psyllium, Bulk-Forming Laxatives (*Plantago Isphagula, Plantago Ovata*)

**Uses**
- Chronic constipation (requires adequate hydration)
- Diarrhea (bulk-forming agent)
- IBS
- Management of hemorrhoids
- Regulates the effluent for pts with colostomies
- Cholesterol-lowering agent for mild hypercholesterolemia

**Perioperative Risks**
- Allergic reaction (rare) from ingested or inhaled powder
- Do not use if vomiting, intestinal obstruction, abdominal pain, nausea, or fecal impaction is present
- Do not give other oral drugs for 2 hr before or after psyllium

**Worry About**
- Stenosis or obstruction of esophagus or GI tract
- Adequate hydration
- Constipation, impaction, or obstruction can result without adequate hydration
- Loss of diabetic control (use sugar-free preparations in diabetics)
- Decreases absorption of oral medications

**Overview/Pharmacology**
- Psyllium causes retention of water, which ↑ fecal bulk (expands 8–14 × normal size in water). This provides a mechanical stimulus for peristalsis and the rate of bowel transit.
- Effective within 12–24 hr
- Maximum effect after several days
- May stabilize postprandial glucose levels in NIDDM
- Other names
  - Trade names: Metamucil, Hydrocil, Fiberall, Citrucel
  - Herbal names: Psyllium seed, black; psyllium seed, blonde

**Drug Class/Usual Dose**
- Daily dose 10–30 g total in divided doses PO
- Available in powder, wafer, cereal, capsules, chewable pieces
- Must be taken with adequate fluid

**DRUG EFFECTS**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>ENDO</td>
<td>Altered blood sugar</td>
<td>Symptoms of hyperglycemia/hypoglycemia</td>
<td>Glucose</td>
</tr>
<tr>
<td>GI</td>
<td>If not consumed with adequate fluid, esophageal/intestinal impaction and obstruction</td>
<td>Dysphagia, odynophagia, inability to swallow, abdominal pain and distention</td>
<td>Imaging studies</td>
</tr>
<tr>
<td>CARDIO</td>
<td>↓ Cholesterol levels</td>
<td></td>
<td>Cholesterol profile</td>
</tr>
<tr>
<td>RESP</td>
<td>Inhalation-induced allergy/asthma</td>
<td>Exposure</td>
<td>Wheezing, skin rash, itching, hives</td>
</tr>
<tr>
<td>GU</td>
<td>Unknown effect on pregnancy and lactation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Emollient</td>
<td></td>
<td>Dermatitis, pruritus</td>
</tr>
</tbody>
</table>


**Perioperative Implications**

**Preoperative Concerns**
- Underreported due to impression that agent is not a drug
- Inquire into use of other herbal drugs
- Possible decrease in absorption of oral drugs
- Concern with hypovolemia

**Monitoring**
- Routine
- Consider NG tube

**Airway**
- None

**Induction**
- Hypotension a concern if hypovolemic

**Maintenance**
- Hypotension a concern if hypovolemic

**Extubation**
- None

**Postoperative Period**
- Worry about constipation postop if chronic use is not continued postop (can be added to almost any smoothie as long as can take smoothies and water)
Pyruvate

**Uses**
- Orally, used for wt loss, improving exercise endurance, hyperlipidemia, antioxidant protection, and inhibiting tumor growth
- Topically, pyruvic acid used for signs of aging skin and photodamage
- Inhaled as sodium pyruvate may act as an anti-inflammatory agent to improve some chronic lung diseases (in prelim clinical trials)
- Intracoronary infusion, used to improve hemodynamics in pts with CHF due to dilated cardiomyopathy

**Risks**
- Available OTC without regulation
- Orally large doses, >6 g/d, can cause GI symptoms incl abd discomfort, bloating, gas, and diarrhea
- Topically, applying pyruvic acid as a facial peel can cause an intense burning sensation. Apply in adequate ventilation because the vapors have been reported to cause resp irritation.
- One child receiving IV pyruvate for restrictive cardiomyopathy died.
- There is insufficient reliable studies or evidence about the effectiveness of pyruvate for most uses.

**Overview/Pharmacology**
- Anionic form of the three-carbon organic acid, pyruvic acid
- Present in red apples (∼450 mg), red wine and dark beer (∼75 mg), and cheeses.
- Serves as a biological fuel, converted to acetyl-coenzyme A, which enters the Krebs cycle and is metabolized to produce ATP aerobically
- Anaerobically energy is obtained when pyruvate is formed as an end-product of glycolysis and then reduced to lactate
- Known for infomercial with Steve Garvey where drug effects were misrepresented and resulted in $10 million settlement against the company

**Worry About**
- Possible hypoglycemia via blood glucose extraction into muscle cells and decreased insulin resistance
- May potentiate inotropic effects of catecholamines and phosphodiesterase inhibitors
- Insufficient data about safety in pediatrics, pregnancy and lactation, and pts with liver and kidney disease

**Mechanism of Action**
- Increases contractile function of heart muscle
- Increases generation of ATP and ATP phosphorylation potential
- Reduces free radical production, increases lipid oxidation, and decreases carbohydrate oxidation resulting in greater loss of fat mass
- In combination with dihydroxyacetone, increases arm and leg exercise endurance
- Topically exfoliates the surface layers of skin
- Repairs injured mitochondria to inhibit tumor growth by suppressing the Warburg effect
- Up-regulates NO that can kill viruses, infections, and tumors and reduce inflammation in lungs

**Usual Dose**
- Oral: For wt loss, 22–44 grams/d as a supplement to a low-cholesterol, low-fat diet with exercise
- Topical: For aging skin, a 50% pyruvic acid peel applied once weekly for 4 wk

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**PERIOPERATIVE IMPLICATIONS**

**Preoperative Concerns**
- Reliable self report of drug use and amount
- Check labs for glucose level

**Monitoring**
- Routine

**Airway**
- No special concerns

**Preinduction/Induction**
- No special concerns other than considerations of co-morbidities

**Postoperative Period**
- Routine

---

**Drug Effects**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>↓ Pulm artery pressure and pulm vascular resistance</td>
<td>PA cath calc., ECHO, heart cath</td>
</tr>
<tr>
<td></td>
<td>↓ Pulm capillary wedge pressure</td>
<td>ECG, &quot;</td>
</tr>
<tr>
<td></td>
<td>↓ Heart rate</td>
<td>&quot;</td>
</tr>
<tr>
<td></td>
<td>↑ Cardiac output and cardiac index</td>
<td>&quot;</td>
</tr>
<tr>
<td></td>
<td>↑ Left ventricle ejection fraction</td>
<td>&quot;</td>
</tr>
<tr>
<td></td>
<td>↑ Inotropy and lusitropy</td>
<td>&quot;</td>
</tr>
<tr>
<td></td>
<td>↓ Plasma cholesterol and LDL</td>
<td>Chol, LDL</td>
</tr>
<tr>
<td>ENDO</td>
<td>↓ Insulin resistance</td>
<td>Glu, ltes</td>
</tr>
<tr>
<td></td>
<td>↑ Levels of thyroxine</td>
<td>TFT</td>
</tr>
<tr>
<td></td>
<td>↑ Resting metabolic rate</td>
<td>Calorimetry</td>
</tr>
<tr>
<td>IMMUNE</td>
<td>Anti-inflammatory and anti-oxidant</td>
<td>Poss. CBC</td>
</tr>
</tbody>
</table>

S-Adenosyl-L-Methionine (SAMe)

Uses
- Anti-aging, anti-disease therapeutic agent
- May protect against hepatotoxic effect of certain drugs (e.g., alcohol, acetaminophen, phenobarbital, and steroids)
- Depression, anxiety, premenstrual syndrome (PMS)
- Heart disease
- Liver disease, cirrhosis, intrahepatic cholestasis, disorders of porphyrin, and bilirubin metabolism
- Osteoarthritis, tendinitis, bursitis, chronic low back pain
- Dementia, Alzheimer’s disease, Parkinson’s disease
- MS, migraine, seizure, spinal cord injury
- Chronic lead poisoning
- Disorder of porphyrin and bilirubin metabolism
- Chronic fatigue syndrome (CFS)
- Intellectual enhancement, attention deficit-hyperactivity disorder (ADHD)

Perioperative Risks
- N/V, flatulence, diarrhea, irregular or accelerated HR
- Anxiety

Overview/Pharmacology
- SAMe is produced endogenously by adenosine triphosphate (ATP) activation of methionine that is produced by the body from dietary protein.
- SAMe is required in numerous transmethylation reactions involving nucleic acids, proteins, phospholipids, amines, and other neurotransmitters. The synthesis of SAMe is linked with folate and cyanocobalamin metabolism, and deficiencies of both these vitamins have been found to reduce CNS SAMe concentrations.
- May improve methylation by different mechanisms in several neurologic and psychiatric disorders
- Is well tolerated with oral use and free of serious side effects. The oral supplement was developed in the 1970s and has been touted as a multipurpose treatment ever since.
- Exogenously administered SAMe has a low bioavailability due to rapid first-pass metabolism by the liver
- Peak plasma concentration in 3–5 hr
- ↓ ↑ 100 min
- Excreted in urine and feces
- Crosses the blood-brain barrier
- Metabolized to homocysteine, which is remethylated to form methionine, which can form more SAMe
- Tosylate salt has 1% oral bioavailability
- Butane disulfonate salt has 5% oral bioavailability

Mechanism of Action
- Contributes to the synthesis, activation, and metabolism of hormones, neurotransmitters, nucleic acid, proteins, phospholipids, and some drugs
- SAMe crosses the blood-brain barrier and is involved in transmethylation and folate and monamine metabolism as well as in membrane function and neurotransmission.
- SAMe plays a role in more than 100 biochemical reactions: increases levels of serotonin, dopamine, norepinephrine, phosphatides, proteoglycans.
- Improves intrahepatic cholestasis. SAMe supplementation seems to improve hepatic function and reverse imbalances of various enzymes. In liver disease deficiencies of methionine adenosyl transferase (MAP) often leads to reductions in cysteine and choline which can lead to depletion of glutathione. SAMe restores levels of glutathione, decreases inflammation and increases methylation of DNA.
- Stimulates articular cartilage growth
- Relieves joint pain possibly due to analgesic or anti-inflammatory effects. Possibly stimulation of articular cartilage growth and repair as a result of chondrocyte proteoglycan synthesis. May antagonize tumor necrosis factor-alpha (TNF-alpha) which may be beneficial in arthritic pts.
- Antidepressant effect is probably due to ↑ serotonin turnover and elevated dopamine and norepinephrine levels or due to alteration of cellular membrane fluidity, which facilitates signal transduction across membranes and ↑ the efficiency of receptor-effector coupling.
- In liver disease, it restores the biochemical factors that are depleted.
- In AIDS myelopathy, it replenishes depleted endogenous SAMe.

Usual Dose
- For depression, 400–1600 mg PO/d or 200–400 mg IV/d to speed the onset of tricyclic antidepressants
- For osteoarthritis, 200 mg tid PO or 400 mg IV
- For alcoholic liver disease, cirrhosis, or intrahepatic cholestasis, 1200–1600 mg PO or 800 mg IV/d
- For AIDS myelopathy, 800 mg IV/d for 14 d
- For fibromyalgia, 800 mg PO/d

Interactions
- Additive serotonergic effects and serotonin syndrome-like effects with antidepressants incl SSRIs
- Due to serotonergic properties, the following should be avoided with SAMe due to the risks of serotonin syndrome-like effects: dextromethorphan (Robitussin DM, other cough syrups), meperidine (Demerol), pentazocine (Talwin), tramadol (Ultram), sumatriptan (Imitrex) and other 5-HT1B/1D receptor agonists.
- Additive side effects like hyperthermia, agitation, confusion, coma when used with MAO inhibitors
- Other side effects may incl dry mouth, nausea, gas, diarrhea, headache, anxiety, nervousness, restlessness, and insomnia.
- Large doses of SAMe may cause mania (abn elevated mood). People with bipolar disorder (manic depression) should not take SAMe because it may worsen manic episodes.

Contraindications
- pts taking MAO inhibitors or within 2 wk of their D/C
- Concurrent use with antidepressant drugs incl MAOIs could result in additive stimulatory effects. Agitation, tremor, insomnia, nervousness, irregular or accelerated heart rate are theoretical concerns.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment by Hx</th>
<th>PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>N/V, diarrhea</td>
<td>GI complaints</td>
<td>KUB</td>
</tr>
<tr>
<td>MS</td>
<td>Ostearthritis</td>
<td>Stiff joints</td>
<td>ROM</td>
</tr>
</tbody>
</table>

Adam M. Kaye
Alan Kaye

ALTERNATIVE MEDICINE
Saw Palmetto

Overview
- Saw palmetto extract is an extract of the fruit of Serenoa repens from the American dwarf palm tree. Saw palmetto’s active ingredients incl fatty acids, plant sterols, and flavonoids.
- Saw palmetto has hormonal (estrogenic) effects, as well as direct inhibitory effects on androgen receptors. There are possible anti-inflammatory effects (from the berries of the plant).
- Saw palmetto has not been evaluated by the FDA.
- Saw palmetto is possibly ineffective for its intended use—BPH (NEJM, 2006).

Etiology
- Mechanism of action: Saw palmetto exhibits antiestrogenic and antiandrosterone effects by inhibiting the actions of 5-alpha reductase enzyme (thereby preventing the conversion of testosterone to dihydrotestosterone (DHT)—a cause of BPH and baldness).

DRUG EFFECTS

<table>
<thead>
<tr>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI Occasional upset, hepatitis, and cholecystitis (very rare)</td>
<td>LFTs</td>
</tr>
<tr>
<td>HEME Bleeding, iron deficiency</td>
<td>None, iron studies, Hb</td>
</tr>
<tr>
<td>GU Improved urinary symptoms (conflicting data)</td>
<td>None</td>
</tr>
<tr>
<td>ENDO Breast enlargement (unproved) Prevent hair involution due to DHT (unproved)</td>
<td>None</td>
</tr>
</tbody>
</table>


Possible Drug Interactions
- Any medication that alters male sex hormones should not be taken with saw palmetto. Examples incl finasteride and flutamide.
- Drugs that effect coagulation should also not be consumed with saw palmetto: Coumadin and anti-inflammatory agents (clopidogrel, ibuprofen, aspirin).
- Because saw palmetto may have hormone-like effects, it may make oral contraceptives less effective, raising the risk of unplanned pregnancy.
- Tannins in saw palmetto may interfere with iron absorption.
- Tinctures may contain large amounts of alcohol and cause N/V when taken with metronidazole or disulfiram.

Uses
- Benign prostatic hypertrophy
- Urinary tract inflammation (prostatitis)
- Underactive bladder
- Male and female pattern baldness
- Aphrodisiac
- Breast augmentation

Perioperative Risks
- No established interactions with anesthetic agents

Worry About
- Saw palmetto has been implicated in hepatitis, cholecystitis, bleeding diatheses, conduction defects, and erectile dysfunction. No studies confirm these effects.
- Unsubstantiated pharmacological effects increasing action of benzodiazepines.

Perioperative Implications

Preoperative Concerns
- Self reporting of other herbal supplements
- Unknown effects in children, interference with birth control, and in lactating mothers

Intraoperative Concerns
- None known

Postoperative Period
- Routine
Soy

**Uses**
- Soy is used to prevent or treat high LDL cholesterol, menopausal symptoms, osteoporosis, memory problems, HTN, breast cancer, and prostate cancer.
- Human and rodent studies support the hypothesis that soy-based phytoestrogens may decrease age-related diseases (CV disease, osteoporosis), prevent obesity, lower cholesterol, and favorably alter glycemic control; however, further research is required.

**Perioperative Risks**
- None known

**Worry About**
- Minor stomach and bowel problems such as nausea, bloating, and constipation are possible.
- Allergy to soy, though rare, can occur.

**Overview**
- Phytoestrogens are plant-derived compounds with a structure and function similar to estradiol.
- Isoflavones, a class of phytoestrogens, have the highest concentration in soybeans and act as an estrogen receptor (ER) agonist or antagonist.
- The two major isoflavones found in soy are genistein and daidzein. As with estrogens, these isoflavones may affect glucose and lipid metabolism.
- Dietary soy may inhibit atherosclerosis by mechanisms yet unknown.

**Drug Effects**

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>Potential cognitive improvement</td>
<td>Cognitive testing</td>
</tr>
<tr>
<td>CARDIO</td>
<td>May inhibit development of atherosclerosis</td>
<td>HDL, LDL, Cholesterol levels</td>
</tr>
<tr>
<td>GI</td>
<td>N/V and bloating may be possible</td>
<td></td>
</tr>
<tr>
<td>GU</td>
<td>Reduces menopausal hot flash severity and decreases vaginal dryness</td>
<td></td>
</tr>
<tr>
<td>ENDO</td>
<td>Inhibits adipose tissue deposition and stimulates lipolysis</td>
<td>Glucose</td>
</tr>
<tr>
<td>HEME</td>
<td>Allergic reactions incl anaphylaxis may occur</td>
<td></td>
</tr>
</tbody>
</table>


**References**
St. John’s Wort (Hypericum Perforatum)

Uses
- More than 3% of presurgical pts report using St. John’s wort
- Taken mainly for depression, although pts may take for a variety of reasons incl anxiety, viral and bacterial infections, menstrual cramps, HIV, cancer, chest congestion, hemorrhoids, skin wounds, and burns.
- May be as effective as low-dose first-generation tricyclic antidepressant for treating mild depression
- Most integrative medical specialists will use every other alternative first due to drug interactions; this is at best a third-line medication.

Worry About
- Drug interactions: May prolong sedative effects of other drugs incl anesthetics and sedatives. Case reports of severe hypertensive response to vaso-pressors such as ephedrine or phenylephrine in pts taking St. John’s wort
- Induces cytochrome P450 enzymes; and promotes metabolism and decreased blood levels of warfarin, cyclosporine, digoxin, Ca²⁺-channel blockers and steroids; even renders birth control pills and menopausal drug therapies ineffective (watch for unplanned and sometimes unwanted pregnancies due to this effect)
- Serotonin-like syndrome (Htn, tachycardia, agitation, restlessness)
- Unpredictable effects due to lack of strict regulation

Overview/Pharmacology
- Classified as a dietary supplement and not subject to FDA drug regulation; pharmacologic activity can be unpredictable and highly variable in different preparations. Marketed PO 300 mg hypericum extract (0.3% hypericin) tid.
- Contains many complex chemicals, but hypericin and hyperforin are responsible for the antidepressant effects
- Absorbed within 40 min of oral administration
- Mainly metabolized by the liver and cleared by renal excretion; elimination T½ 43 hr

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Assessment/by Hx</th>
<th>PE</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEENT</td>
<td>Photosensitivity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CARDIO</td>
<td>Rarely, Htn, tachycardia, and serotonin-like syndrome</td>
<td>Dosage taken; also taking SSRI</td>
<td>BP/HR</td>
<td>ECG</td>
</tr>
<tr>
<td>GI</td>
<td>Nausea</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERM</td>
<td>Rarely, rash</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Restlessness, fatigue, antidepression</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications
Preoperative Period
- Hx can incl dose, duration, and preparation taken and the reasons for its use
- Well advised to D/C preop at least 1 wk in advance for the drug to be cleared from body.
- May see as much as a 50% decreased effect of warfarin. Consider alternatives to warfarin.
- Can decrease digoxin levels, possibly by induction of a P-glycoprotein transporter
- Serotonin-like syndrome, esp. when combined with an SSRI, tricyclics, or MAOIs

Induction/Maintenance/Emergence
- May prolong anesthesia via potentiation of central effects of inhaled agents, sedatives, and opioids.
- May prolong the sedative effects of anesthetics
- Watch for serotonin-like syndrome (Htn, tachycardia, agitation, restlessness)

Anticipated Problems/Concerns
- Effects may be variable among different preparations due to lack of standardization.
- Anticipate decreased effects of certain drugs such as warfarin, cyclosporine, beta blockers, Ca²⁺-channel blockers, steroids, and digoxin.
- May prolong the sedative effects of anesthetics

Mechanism of Action/Usual Dose
- May act as a nonspecific reuptake inhibitor of serotonin, norepinephrine, and dopamine
- Appears to work differently from conventional antidepressants
- MAO inhibition reported in early studies. Not confirmed in follow-up studies.
- Usually taken as a capsule consisting of the plant extract; typical dosage is 300–500 mg of hypericum extract tid.
Valerian (Valeriana officinalis)

Richard M. Layman
Lisa Caplan

Uses
• Insomnia (present in virtually all herbal sleep aids)
• Anxiety
• Depression
• HT
• GI hyperactivity
• Headaches
• Muscle spasms
• Benzodiazepine withdrawal

Perioperative Risks
• Potential for valerian withdrawal exists if acute cessation after chronic high-dose administration occurs. This withdrawal can present as delirium, tachycardia, and diaphoresis.
• Chronic high dose valerian has been linked with cardiac failure and emergence delirium.

Worry About
• No direct drug interactions are reported.
• Valerian may act synergistically with sedative anesthetics leading to prolonged emergence.

Overview
• Valerian is a native herb of temperate regions whose name is believed to be derived from the Latin word “valere,” meaning to be healthy or strong. It has been used for centuries as a sleep aid by Greeks, Romans, Chinese, American Indians, and Europeans.
• Prior to the introduction of barbiturates to the U.S. National Formulary, valerian was indicated for treatment of unrest and nervous sleep disturbance. It has since been dropped from the U.S. National Formulary.
• Valerian contains many constituents working synergistically, incl volatile oils, valepotriates, monoterpene alkaloids, and furanofuran lignans.
• Volatile oils: These oils give valerian a pungent odor due to the release of isovaleric acid. The sesquiterpene skeleton present on volatile oils such as valerenic acid, valeranone, and kessyl glycol is a proposed primary source of pharmacological effects. These components have been shown to act on the amygdaloid body in the brain, and inhibit GABA breakdown thus leading to sedation.
• Valepotriates: Valepotriates have a furanopyranoïd monoterpene skeleton which can be found in glycosylated forms known as iridoids. The compounds have been shown in mice and cat experiments to decrease spontaneous motility after oral administration.

Mechanism of Action/Usual Dose
• Produces dose-dependent sedation and hypnosis mediated mainly through GABA_A receptor, the adenosine A_1 receptor, and recently noted, the 5-HT_1A receptor.
• Tablets: 300–400 mg PO 30 min to 1 hr prior to sleep.
• Tea: 1 cup of boiling water over 1 to 2 teaspoons (2–3 g) of the root and infuse for 10 to 15 min. May drink up to 2 cups daily.
• Tincture: 2 to 6 mL (½ to 1 teaspoon) up to three times daily.

DRUG EFFECTS

<table>
<thead>
<tr>
<th>System</th>
<th>Effect</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIO</td>
<td>High output cardiac failure</td>
<td>Rule out other causes of high output cardiac failure: Sepsis, beriberi, cardiac shunt, or Paget disease.</td>
</tr>
<tr>
<td></td>
<td>Hypotension</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Arrhythmias</td>
<td>EKG, ECHO</td>
</tr>
<tr>
<td></td>
<td>Dilates coronary arteries</td>
<td></td>
</tr>
<tr>
<td>HEPAT</td>
<td>CYP4A inhibitor</td>
<td>Baseline LFTs</td>
</tr>
<tr>
<td>CNS</td>
<td>Sedation</td>
<td>Sleep studies: May improve sleep latency and slow wave sleep</td>
</tr>
<tr>
<td></td>
<td>Hypnosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anticonvulsives</td>
<td>EEG</td>
</tr>
<tr>
<td></td>
<td>Headache</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Restlessness</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hallucinations</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ataxia</td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Nausea</td>
<td>Decrease dose or stop ingestion.</td>
</tr>
<tr>
<td></td>
<td>Intestinal irritability</td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Muscle relaxation</td>
<td></td>
</tr>
</tbody>
</table>


Perioperative Implications
• Valepotriate component of valerian may alkylate DNA, which could be potentially cytotoxic or carcinogenic. It has been recommended that valerian not be used in pregnancy or while breastfeeding.
• Cessation of valerian consumption prior to surgical intervention should be made on an individualized basis. If a 2-to 3-wk taper is not feasible, then pts should continue taking valerian. Benzodiazepines can be used to treat withdrawal symptoms should they develop.
### Autonomic Function

#### Overview
- Measuring BP and HR are inexpensive.
- Equipment costs for manometer and isometric handgrip, minimal
- Simple bedside tests incl orthostatic stress, deep breathing, Valsalva maneuver, cold pressor or a slightly more involved test incl atropine administration, and isometric handgrip give valuable information on autonomic function.
- Multiple tests are usually performed; presence of two or more abn results indicates some degree of autonomic dysfunction

#### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Test</th>
<th>Method</th>
<th>System</th>
<th>Abnormal Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthostatic stress</td>
<td>Pt supine 10 min; then stands or is tilted 80° head up; BP measured at 2 min</td>
<td>Sympathetic</td>
<td>↓ in SBP by &gt;20 mm Hg &lt;1.2 (&lt;1.15 if &gt;65 y) in DBP by &gt;10 mm Hg &lt;5 bpm if &gt;65 y</td>
</tr>
<tr>
<td>30:15 Ratio</td>
<td>From continuous ECG strip; ratio of longest R-R interval (~30th beat) to shortest R-R interval (~15th beat) after assuming standing position</td>
<td>Parasympathetic</td>
<td>&lt;1.03 (&lt;1.01 if &gt;65 y) &lt;1.2 (&lt;1.15 if &gt;65 y) BP does not exceed baseline after release of blowing</td>
</tr>
<tr>
<td>Deep breathing</td>
<td>Difference between mean HR at max inspiration and mean HR at max expiration for 6 breathing cycles over 1 min</td>
<td>Parasympathetic</td>
<td>&lt;10 bpm if &lt;40 y &lt;5 bpm if &gt;65 y</td>
</tr>
<tr>
<td>Valsalva maneuver</td>
<td>Pt blows into manometer to maintain intrathoracic pressure at 40 mm Hg for 15 sec; ratio of highest HR during blowing to lowest HR during first 20 sec after release of intrathoracic pressure; repeat for reproducibility. Same maneuver with directly measured arterial pressure</td>
<td>Sympathetic</td>
<td>HR does not change by &gt;20 bpm</td>
</tr>
<tr>
<td>Atropine</td>
<td>1.8 mg IV over 3 min</td>
<td>Parasympathetic</td>
<td>SBP ↓ by &lt;20 mm Hg at peak (1 min) HR ↑ &lt;10 bpm</td>
</tr>
<tr>
<td>Cold pressor</td>
<td>Immerse hand in ice water for 1 min</td>
<td>Sympathetic</td>
<td>↑ DBP by &lt;10 mm Hg after 3 min</td>
</tr>
<tr>
<td>Isometric handgrip</td>
<td>Isometric contraction at 30% max strength for 3 min</td>
<td>Sympathetic</td>
<td>↑ DBP by &lt;10 mm Hg after 3 min</td>
</tr>
</tbody>
</table>

#### Key Reference:

### Perioperative Implications

#### Preoperative Preparation
- Gastroparesis: Consider premedication with agents to ↑ gastric motility (e.g., metoclopramide), and ↓ consequence of aspiration (e.g., antacids, H₂ blockers)
- Abn sensitivity to anesthetic agents and apnea tendencies: Minimize narcotics or benzodiazepines as premedication; monitor intensively periop
- Orthostatic hypotension treated by volume expansion, which may cause supine Htn

#### Induction
- Consider rapid-sequence induction
- Consider etomidate
- Titrate agents with CV and resp effects

#### Monitoring
- Consider arterial line
- Consider ECG monitor
- Consider BP monitors
- Titrate agents with CV and resp effects

#### Maintenance
- Aggressively treat blood loss, keep well hydrated
- Denervation supersensitivity: Unexpected Htn responses to adrenergic agonists used to treat hypotension; if vasopressors are required, use direct-acting agents; indirect-acting agents have unpredictable effects.

#### Additional Tests
- Additional tests incl plasma catecholamines, atrial vasopressor protein, pancreatic polypeptide determinations in response to standing or other maneuvers to resolve site of lesion, and responses to α₁-adrenoceptor agonist for peripheral denervation superfetation.

#### ICD-9-CM Codes: 337.0 (Autonomic nervous system disorder); 337.1

#### Indications
- Ps with symptoms of autonomic failure (e.g., intolerance to standing, bladder and/or sphincter disturbances, impaired sweating) may have autonomic failure due to 1° causes such as Shy-Drager syndrome, or disorders such as DM, chronic alcoholism, chronic renal failure, advanced age, vitamin deficiency (e.g., B₁₂), HIV infection, or prescription drugs (e.g., TCAs)
- Diabetic autonomic neuropathy is the most common autonomic neuropathy characterized by both CV and pseudomotor autonomic dysfunction

#### Diabetic autonomic neuropathy
-增加的血流动力学异常
- 中枢神经系统
- 肾功能不全
- 糖尿病
- 贫血
- 酗酒
- 慢性肝病
- 中枢神经系统疾病
- 全身性血管炎
- 全身性血管炎
Chest X-Ray

Risk
- Radiation dose is small (0.02 mSv) and so is radiation risk (cardiac CT gives radiation of 300–800 chest x-rays).
- The risk from misinterpretation could be significant.

Overview
- It is the most frequently performed radiographic study in the USA.
- The cost is <$250 (incl equipment, execution, and interpretation).
- Most common used views are PA and lateral; portable CXR is almost always AP view.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Aspect of Test</th>
<th>Positive Result</th>
<th>Confounding Factors</th>
<th>Dx Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac shadow</td>
<td>CT ratio &gt;0.5</td>
<td>Inspiratory film, when perihilar markings present cardiac silhouette could be difficult to determine</td>
<td>Cardiomegaly, CHF</td>
</tr>
<tr>
<td>Perihilar markings</td>
<td>1-Increased markings in one area or whole lung fields 2-Absent lung markings in one area or whole field</td>
<td>1-Underexposed film, female breast tissue 2-Over exposure</td>
<td>1-CHF, fluid overload (e.g. renal failure, excess fluid administration, etc.) 2-Pneumothorax</td>
</tr>
<tr>
<td>Diaphragmatic placement</td>
<td>Flattened hemidiaphragms</td>
<td>Improper posture, splinting due to pain</td>
<td>Extent of COPD, extent of air trapping from exacerbation of reactive airway disease or FB</td>
</tr>
<tr>
<td>ETT and lines</td>
<td></td>
<td></td>
<td>Proper position of ETT, CVL, and chest tubes is confirmed.</td>
</tr>
</tbody>
</table>


Perioperative Implications
- CHF: Further evaluation with EKG, echo may be needed; consider invasive monitoring with arterial line, CVP monitoring, may consider PA catheter or TEE. Judicious use of fluids and minimize myocardial stress.
- COPD: Further evaluation with PFTs could be necessary; may consider invasive monitoring. Avoid interscalene block (hemidiaphragm paralysis).

Indications
- Cardiomegaly if cardiothoracic (CT) ratio >0.5. Fluid overload if increased perihilar markings both cardiac and noncardiac.
- Consolidation could be pneumonia; hyperinflation, increased radiolucency and flattened diaphragm indicate COPD.
- Lobular atelectasis is opaque; absent lung markings and mediastinal shift is tension pneumothorax.
- Trachea or esophageal FB could be visible; tracheal FB may show air trapping.

Special Considerations
- Reactive airway disease: Check for oral steroid therapy; if so, stress dose may be indicated. Avoid intubation or if necessary to intubate ensure adequate depth to avoid bronchospasm.
- Maintain spontaneous breathing in case of airway FB.

In pts with advanced COPD avoid depressing intrinsic resp drive; consider epidural for postop pain control. Consider delaying elective surgery if an acute exacerbation.
- Arrangements for ICU bed if pt's condition or surgical procedure (e.g., thoracotomy, lung resection) warrants.
## Diagnostic 12-Lead ECG

**Overview**
- Cost: $15–$50 with physician charges
- ECG assesses myocardial ischemia, MI, rhythm and conduction disorders (intrinsic myocardial disease), electrolyte and metabolic disorders, and medical/systemic effects (extrinsic disorders).

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Positive Result</th>
<th>Confounding Factors</th>
<th>Dx Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial ischemia</td>
<td>ST-segment depression &gt;1 mm</td>
<td>Baseline ST-T wave changes</td>
<td>ST-segment changes correlate poorly with site of CAD Magnitude of depression weakly related to severity</td>
</tr>
<tr>
<td></td>
<td>Deep T-wave inversion</td>
<td>BBB (esp. LBBB)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Digoxin/drug effects</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abn autonomic tone</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Q waves</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>↑ ST segment due to pericarditis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intracranial pathology</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>New Q waves ≥40 msec, amplitude &gt;25% of R wave</td>
<td>Q waves in V₁ and aVL or isolated inferior leads may be normal</td>
<td>Q waves are sensitive and specific indicators</td>
</tr>
<tr>
<td></td>
<td>↑ ST during acute stage</td>
<td>BBB (esp LBBB)</td>
<td></td>
</tr>
<tr>
<td>Rhythm disorders</td>
<td>Abn timing of P wave</td>
<td>Depends on chronicity, Rx, hemodynamic consequences</td>
<td></td>
</tr>
<tr>
<td></td>
<td>QRS or absence of normal P wave and P-R interval</td>
<td>Atial dysrhythmias usually less dangerous than ventricular</td>
<td></td>
</tr>
<tr>
<td>Conduction disorders</td>
<td>Axis deviation</td>
<td>Atrial fibrillation associ with increased risk periop stroke</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PR &gt; 120 msec</td>
<td>LAFB—usually benign</td>
<td></td>
</tr>
<tr>
<td></td>
<td>QRS &gt; 100 msec</td>
<td>LPFB—likely myocardial or conduction damage</td>
<td></td>
</tr>
<tr>
<td></td>
<td>J point elevation</td>
<td>RBBB—more likely associ with CAD and/or impaired ventricular function</td>
<td></td>
</tr>
<tr>
<td>Metabolic disorders</td>
<td>Hypokalemia—flattened T waves, ST ↓</td>
<td>Other nonspecific changes</td>
<td>Chemistry Laboratory</td>
</tr>
<tr>
<td></td>
<td>Hyperkalemia—peaking T waves, wide QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypocalemia—lengthen QT interval</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypercalémia—shorten QT interval</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV hypertrophy</td>
<td>Multiple criteria</td>
<td></td>
<td>Asov with severe</td>
</tr>
<tr>
<td></td>
<td>Sum V₁ + V₅ ≥ 35 mm</td>
<td></td>
<td>Htn or aortic stenosis</td>
</tr>
</tbody>
</table>


**Perioperative Implications**
- $Q$ waves diagnostic of prior MI associ with elevated risk of postop cardiac morbidity.
- Number of $Q$ waves on ECG tracing negatively correlated with the ejection fraction
- LBBB more likely associ with significant CAD and impaired ventricular function than RBBB.
- However, intraventricular conduction delay, with very wide and bizarre QRS morphology, may have similar significance as LBBB.

**Indications**
- Known or suspected (i.e., multiple risk factors or abn on Hx or PE) CAD
- Major surgery regardless of clinical Hx in pts with known or at high risk for CAD or other cardiac disease.

**Confounding Factors**
- Appropriate, cost-effective starting point for more extensive, costly evaluation of cardiac diseases
- Predictive value, cost-effectiveness of preop 12-lead ECG are controversial. Incidence of ECG abn ↑ with age, concurrent medical illness (esp. Htn, CAD, diabetes).

**Special Considerations**
- Non specific ST–T wave changes, T-wave flattening/inversion, and QT-interval prolongation markedly influenced by autonomic tone and common in the early postop period
- Accuracy of computerized interpretation varies among manufacturers
- Sensitivity and specificity of ECG are poor following cardiac surgery

**Additional/Alternative Tests**
- Exercise treadmill testing with or without thallium imaging, static or stress ECHO, dipyridamole or adenosine thallium imaging, coronary angiography to diagnose CAD, ischemia
- Holter monitoring for arrhythmias, conduction defect, ischemia

**Risk**
- None except misinterpretation

**Sensitivity/Specificity**
- Varies according to specific clinical indication, population. For rhythm and conduction disorders, 100% sensitive. Sensitivity of $Q$ waves for autopsy-proven MI is 33–62%, with a specificity of 88–98%. Sensitivity, specificity of ST-T changes on resting ECG for myocardial ischemia in absence of clinical $Sx$ are low.

**Laboratory**
- Magnitude of depression weakly related to severity
- Depends on chronicity, Rx, hemodynamic consequences
- Atial dysrhythmias usually less dangerous than ventricular
- Atrial fibrillation associ with increased risk periop stroke
- LAFB—usually benign
- LPFB—likely myocardial or conduction damage
- RBBB—usually benign
- LBBB—more likely associ with CAD and/or impaired ventricular function
- J point elevation inferior or lateral leads >0.1 mm associ with increased risk long-term cardiac death

**Diagnosis**
- Newer forms: These incl computerized vectorcardiography and late- and mid-QRS signal-averaged electrocardiography utilizing a different lead system (the Frank Lewis XYZ leads) and signal-averaging techniques. Their periop value is currently unknown although recent data suggests that abn spatial QRS–T angle is a strong predictor of cardiac death in high-risk medical pts.
**Dibucaine Number (Atypical Cholinesterase)**

### Risks
- Requires a serum sample for in vitro testing.
- Risks assoc with blood sample collection (i.e., venipuncture) incl pain, bruising, infection, and/or syncope.
- Periop risks of not receiving the test or misinterpretation of test: prolonged muscle paralysis and apnea following succinylcholine (SCH) or mivacurium (MIV). Possible ↑ risk of toxicity of ester-linked local anesthetics.

### Overview
- Cost: Approximately $50
- Dibucaine number is used to determine if a pt has a genetically determined form of atypical cholinesterase (BHCHE; butyrylcholinesterase, benzoylcholinesterase, acetylcholine acylhydrolase, plasma ChE, pseudo-ChE, serum ChE) that is resistant to dibucaine inhibition. There is a number of phenotypes and genotypes of serum ChE, incl the normal gene, dibucaine-resistant genes, fluoride-resistant genes, and the silent gene. The silent gene has no ChE activity when in the homozygous state; the dibucaine and fluoride-resistant forms have reduced ChE activity.

### Variants of Butyrylcholinesterase

<table>
<thead>
<tr>
<th>Name</th>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usual</td>
<td>U</td>
<td>Normal</td>
</tr>
<tr>
<td>Atypical</td>
<td>A</td>
<td>Reduced activity, dibucaine resistant</td>
</tr>
<tr>
<td>Fluoride resistant</td>
<td>F</td>
<td>Reduced activity, fluoride resistant</td>
</tr>
<tr>
<td>Silent</td>
<td>S</td>
<td>No activity</td>
</tr>
<tr>
<td>H</td>
<td>H</td>
<td>Approx 10% reduced concentration</td>
</tr>
<tr>
<td>J</td>
<td>J</td>
<td>Approx 33% reduced concentration</td>
</tr>
<tr>
<td>K</td>
<td>K</td>
<td>Approx 66% reduced concentration</td>
</tr>
</tbody>
</table>

### Key Reference:

### Assessment Points

<table>
<thead>
<tr>
<th>Variant</th>
<th>Approx Duration of SCH-Induced NMB</th>
<th>Dibucaine Number (% Inhibition of Enzyme Activity)</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homozygous</td>
<td>5–10 min</td>
<td>70–80</td>
<td></td>
</tr>
<tr>
<td>Heterozygous</td>
<td>20 min</td>
<td>40–60</td>
<td>1/480</td>
</tr>
<tr>
<td>Homozygous atypical</td>
<td>60–180 min</td>
<td>20–30</td>
<td>1/3200</td>
</tr>
</tbody>
</table>

### Key Reference:

### Perioperative Implications
- Avoid use of, or use cautiously, SCH, MIV, or ester-linked local anesthetics in pts confirmed or suspected to be homozygous for atypical ChE.
Dipyridamole Thallium Imaging

Lee A. Fleisher

Risk
- In pts with CAD, risk of MI and death 1/100,000

Sensitivity and Specificity
- Sensitivity: 70–80%
- Specificity: 80–90%
- Pos predictive value: 20–50%
- Neg predictive value: 85–99%

Overview
- Cost: $1200–$1500, depending on laboratory
- Test to assess presence of coronary artery stenosis in pts unable to exercise

Indications
- Dx of CAD in pts unable to exercise
- Quantification of area at risk for ischemia

Tests
- Holter monitoring for silent ischemia
- Dobutamine thallium imaging
- Dobutamine stress ECHO
- Coronary angiography
- Coronary calcium score
- Coronary CT scan

ICD-9-CM Code: 414.0 (Coronary atherosclerosis)

Overview

- Cost: $1200–$1500, depending on laboratory
- Test to assess presence of coronary artery stenosis in pts unable to exercise

Dipyridamole used to dilate normal coronary arteries, resulting in flow heterogeneity
- Thallium taken up by viable myocardial cells
- Obtain stress and at-rest images
- Areas of myocardial necrosis demonstrate fixed defect
- Areas at risk demonstrate reversible defect
- Able to quantify area at risk

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Aspect of Test</th>
<th>Positive Result</th>
<th>Confounding Factors</th>
<th>Dx Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV thallium imaging</td>
<td>Reversible defect</td>
<td>Breast artifact</td>
<td>Area of myocardium at risk</td>
</tr>
<tr>
<td></td>
<td>Fixed defect</td>
<td>Delayed imaging or reinjection needed to determine if severe ischemia or scar present</td>
<td>Area of old scar or severe ischemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LV dilation</td>
<td></td>
</tr>
<tr>
<td>Lung imaging</td>
<td>♤ Lung uptake</td>
<td>LV dysfunction</td>
<td></td>
</tr>
<tr>
<td>ECG</td>
<td>ST-segment changes</td>
<td>Baseline abn</td>
<td>Indicates dipyridamole results in myocardial ischemia— ♤ risk</td>
</tr>
<tr>
<td>Sx during test</td>
<td>Chest pain</td>
<td>Multiple causes</td>
<td>May be ischemia or nonspecific cause</td>
</tr>
</tbody>
</table>


Perioperative Implications
- A reversible defect suggests the presence of a critical coronary artery stenosis; larger defects are assoc with a greater area at risk and a higher incidence of periop cardiac morbidity.
- Increased lung uptake or LV dilation identifies those pts at risk for LV dysfunction with ischemia.
- Fixed defects represent old scar and are assoc with reduced function and increased long-term risk.

Special Considerations
- Pts with fixed defects may require reinjection or 24-hr delayed imaging to differentiate scar from severe ischemia.
**Risk**
- The incidence of potentially life threatening complications (i.e., cardiac rupture, acute MI, cerebrovascular accident, ventricular fibrillation, and sustained ventricular tachycardia) of dobutamine echocardiography and alternative tests
  - Dobutamine echocardiography 1:475
  - Exercise stress testing 1:1100
  - Dipyridamole echocardiography 1:1400
  - Dipyridamole scintigraphy 1:1600
- Dobutamine induces myocardial ischemia, contraindications are: Symptomatic severe aortic stenosis, unstable coronary syndromes, obstructive hypertrophic cardiomyopathy, and acute aortic dissections.

**Sensitivity/Specificity**
- Sensitivity for detection of CAD: 85–90%
- Specificity for detection of CAD: 80–85%
- Positive predictive value (for any periop event): 25–45%
- Positive predictive value (for a hard event): 15–25%
- Negative predictive value: 95–100%

**Overview**
- Cost: $600–$900
- Dobutamine infused in incremental doses to ↑ HR and contractility (e.g., myocardial O₂ demand)
- Normal response is dose-dependent improved wall thickening motion at every stage

---

### Tests

#### Recommendations on Stress Testing Prior to Surgery

<table>
<thead>
<tr>
<th>Class</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-risk surgery</td>
<td>I C</td>
</tr>
<tr>
<td>Stress testing is recommended in pts ≥3 clinical risk factors</td>
<td>I</td>
</tr>
<tr>
<td>Stress testing may be considered in pts &lt;2 clinical risk factors</td>
<td>IIb B</td>
</tr>
<tr>
<td>Stress testing may be considered in intermediate-risk surgery</td>
<td>IIb C</td>
</tr>
<tr>
<td>Stress testing is not recommended in low-risk surgery</td>
<td>III C</td>
</tr>
</tbody>
</table>

Class I Conditions for which there is evidence for and/or general agreement that the procedure or treatment is beneficial, useful, and effective.

Class II Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class IIa Usefulness/efficacy is less well established by evidence/opinion.

Class III Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective, and in some cases may be harmful.

---

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Test</th>
<th>Positive Result</th>
<th>Confounding Factors</th>
<th>Diagnostic Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wall motion analysis</td>
<td>Resting abn</td>
<td>Cardiomyopathy, LBBB β-blockers; image quality</td>
<td>Prior MI</td>
</tr>
<tr>
<td></td>
<td>Induced abn</td>
<td>Cardiomyopathy</td>
<td>Ischemia</td>
</tr>
<tr>
<td></td>
<td>Multiple abn</td>
<td></td>
<td>Multivessel disease /left main</td>
</tr>
<tr>
<td>ECG</td>
<td>ST-segment depression</td>
<td>Baseline abn</td>
<td>Ischemia</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Chest pain</td>
<td>Nonspecific; multiple causes</td>
<td>Angina or other causes</td>
</tr>
</tbody>
</table>

---

**Perioperative Implications**
- A normal stress ECHO confers favorable prognosis, very low risk of periop morbidity (high negative predictive accuracy).
- Inducible wall motion abn identifies pts at ↑ risk for periop event; but many pts with positive test can still undergo surgery without serious complications (e.g., MI or death low positive predictive accuracy), provided optimal medical therapy is prescribed (e.g., beta-blockers, statins, and aspirin) and administered in immediate periop and operative periods.
- Resting wall motion abn suggests prior infarction
- Stress-induced wall motion abn indicates current ischemia

**Indications**
- Assessment of left ventricular function, heart valve abn, and stress-induced myocardial ischemia, all major determinants of adverse postop cardiac outcome. Myocardial ischemia is defined by the numbers of affected segments and the HR at which ischemia is induced; markers of the extent and severity of CAD.

---

Exercise Stress Testing

**Risk**
- Mortality risk of EST is <0.01%.
- Risk of inducing a myocardial infarction (MI) with EST is 1%.

**Sensitivity/Specificity**
- Accuracy of exercise ECG testing is dependent on the pretest probability of coronary heart disease which reflects pt gender, age, coronary risk factors, and the characteristics of the chest pain.
- Overall sensitivity is 68%; sensitivity for multi-vessel disease is 81%. Sensitivity is reduced when exercise workload is submaximal.
- Overall specificity is 77%; specificity for multi-vessel disease is 66%.
- Negative predictive value for periop cardiac events is 90–100%.
- Positive predictive value for periop cardiac events is 6–67%.

**Overview**
- Cost: $100–$300, depending on lab
- Exercise stress testing (EST), is a less invasive diagnostic test to:
  - Evaluate the extent of coronary artery disease (CAD)
  - Permits stratification of pts who are deemed to be at intermediate risk for periop cardiac events.
  - Assess a pt’s functional capacity.
  - Determine the effects of treatment or to determine an appropriate exercise prescription for cardiac rehabilitation.
  - EST attempts to induce coronary ischemia by the stress of vigorous exercise (treadmill) while detecting ischemia by monitoring the ECG and pt’s vital signs.
  - Exercise-induced ST-segment elevation in a previously non-infarcted territory, profound ST-segment depression, fall in exercise systolic BP and low exercise capacity (inability to achieve greater than 85% of predicted maximal HR during treadmill exercise testing) are assoc with a poor prognosis and also with multi-vessel CAD.
  - In pts who cannot exercise nor have abn on the baseline ECG that interfere with interpretation, pharmacologic stress testing and alternative ischemia imaging techniques are used. (see Tests)

**Indications**
- Evaluate pts with angina to assess likelihood of CAD.
- Evaluate pts with palpitations to detect arrhythmias.
- Provide a prognostic estimate of the risk of a periop cardiac event.
- Provide an objective estimate of functional capacity.
- Determine a safe level of exercise for pts who are considering beginning a new exercise regimen or who require cardiac rehabilitation after an myocardial infarction.

**Contraindications**
- Acute myocardial infarction within 48 hr
- Unstable angina not yet stabilized with medical therapy
- Uncontrolled arrhythmia having possible hemodynamic compromise (e.g., ventricular tachycardia)
- Symptomatic severe aortic stenosis
- Aortic dissection
- Pulm embolism
- Pericarditis

**Additional/Alternative Tests**
- EST may be inappropriate for risk assessment or diagnosis of coronary disease in pts having an abn ECG at baseline (>1 mm depression, LBBB, paced ventricular rhythm or pre-excitation syndrome [WPW])

**TEST**

<table>
<thead>
<tr>
<th></th>
<th>Nonanginal Pain</th>
<th>Atypical Angina</th>
<th>Typical Angina</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–39</td>
<td>4</td>
<td>34</td>
<td>76</td>
</tr>
<tr>
<td>40–49</td>
<td>13</td>
<td>51</td>
<td>87</td>
</tr>
<tr>
<td>50–59</td>
<td>20</td>
<td>65</td>
<td>93</td>
</tr>
<tr>
<td>60–69</td>
<td>27</td>
<td>72</td>
<td>94</td>
</tr>
</tbody>
</table>


**Perioperative Implications**
- High risk of periop cardiac morbidity and mortality is predicted by profound ST-segment ECG changes, poor exercise capacity (<4 METs), exercise-induced drop in BP or exercise-induced angina.
- Low periop cardiac morbidity is predicted by the absence of the findings listed above.
- Intermediate results may require additional noninvasive testing (i.e., exercise myocardial perfusion imaging and/or echocardiography) to more accurately estimate prognosis.

**Special Considerations**
- EST is less accurate in pts who fail to achieve 85% of age-predicted maximum HR.
- Diagnostic accuracy of EST also depends on the pretest clinical risk estimate.
- Interpretation of EST results requires integration of all information acquired during the test. Although often reported as simply positive or negative based on the presence or absence of exercise-induced ECG changes suggestive of ischemia, this over-simplification can be misleading.

- Pharmacologic stress testing with dobutamine: Often used when a pt has bronchospastic lung disease (asthma or severe COPD), severe carotid stenosis, second or third degree atrioventricular block or unable to perform exercise protocol. Dobutamine mimics exercise stress physiologic by increasing HR, increasing contractility, and decreasing systemic afterload.
- Pharmacologic stress testing with adenosine or dipyridamole: Often employed in pts with poorly controlled Htn, glaucoma, or a LBBB. These agents are coronary vasodilators that result in hyperemia to detect differences in coronary flow reserve between stenosed and normal vascular territories.
- Myocardial perfusion imaging: IV administration and subsequent radiologic imaging of a radioactive tracer (e.g., thallium-201 or 99-technetium) is taken up by healthy myocardial cells but not by infected, ischemic, or under perfused tissues. The areas of reduced tracer uptake appear as relative perfusion defects.
- Echocardiography: Can be performed to supplement both EST and pharmacologic stress tests. Echocardiography provides useful additional information about ventricular function, areas of ischemia and valvular heart disease.
- Holter monitoring: Preop ambulatory ECG monitoring enables the detection of significant ischemic ECG changes, the duration and severity of which have been correlated with periop cardiac morbidity in vascular surgery pts.
- Coronary angiography: Remains the gold standard for definitive Dx and specific anatomic characterization of coronary disease.

- Additional imaging modalities (echocardiography or myocardial perfusion) and pharmacologic challenge (dobutamine, adenosine, or dipyridamole) increase the diagnostic specificity and sensitivity.
Flow-Volume Loops

**Risk**
- Virtually no risks associated with flow-volume loops (PFTs)
- Risk from bronchodilator use and misinterpreting data

**Sensitivity/Specificity**
- Flows depend on pt factors, incl body size (ht, wt); habits; gender; age; ethnicity. The 95% confidence interval incl values 20–30% above and below mean for given healthy population. This wide range of normal values limits interpretation of PFTs; interpretation of PFTs critically depends on prior probability of disease. The Dx of COPD does not usually require PFTs; it is based on clinical criteria. Results within given pt reproducible to within 5% or less in cooperative subjects. Repeated measurements of PFTs over time are sensitive to changes in health or disease status. Some pts with confusing Hx and physical findings may need PFTs to diagnose lung disease.

**Overview**
- Cost variable $250–650 (State of Maryland Health Services Cost Review Commission)
- Flow-volume loops show relationship between airflow, with max effort starting from position of either max inspiration or exhalation, and volume (exhaled or inspired, respectively)

**Indications**
- Confirm Dx of suspected obstructive lung disease
- Suggest presence of restrictive lung disease
- Intra- versus extrathoracic obstructions

**Tests**
- CT images of sites of airway obstruction

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Test</th>
<th>Positive Result</th>
<th>Confounding Factors</th>
<th>Dx Information</th>
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<tbody>
<tr>
<td>Measurements suggest OLD (obstructive lung disease)</td>
<td>Flow-volume loops show exaggerated upward concavity of descending limb of flow-volume curve with ↓ peak flows, ↓ volume; inspiratory flows relatively preserved</td>
<td></td>
<td>Causes of OLD: acute (asthma), chronic (bronchitis, emphysema), or related to upper airway lesions</td>
</tr>
<tr>
<td>Measurements suggest RLD (restrictive lung disease)</td>
<td>Flow-volume loops show preservation or ↑ of peak expiratory flow but ↓ volume; flow-volume curve has normal shape but reduced in all dimensions; inspiratory flows relatively preserved</td>
<td></td>
<td>Suggest RLD</td>
</tr>
<tr>
<td>Measurements suggest central airway obstruction (flow-volume loops)</td>
<td>Predominant ↓ in expiratory flow with relatively normal inspiratory flow; expiratory flow curve often has plateau (same flow at all lung volumes) rather than downward bowing normally seen Predominant ↓ in inspiratory flow with relatively normal expiratory flow</td>
<td>Flow-volume loops have role in screening, but confirmation of location, size of lesion may be obtained from imaging studies—e.g., CT of chest</td>
<td>Variable intrathoracic obstruction: Pleural pressure variations during inspiration, exhalation influence magnitude of obstruction so it is less during inspiration, indicating that site of lesion is in thorax (e.g., tracheal tumor) Variable extrathoracic obstruction: Pleural pressure variations during inspiration and exhalation influence magnitude of obstruction so that it is less during exhalation, indicating that site of lesion is in upper airway (e.g., laryngeal tumor) Fixed central obstruction (e.g., tracheal stenosis): Pleural pressure variations during inspiration and exhalation do not influence magnitude of obstruction</td>
</tr>
</tbody>
</table>


**Perioperative Implications**
- Flow-volume loops can distinguish intrathoracic from extrathoracic lesions (see Mediastinal Masses in Diseases section for intrathoracic lesions)
HIV Testing
Tests for Antibodies
• Antibody testing is the most efficient and routine method.
• After a person becomes infected with HIV, an antibody response is detectable within 2–8 wk (average of 25 d) in 97% pts. There is a chance that some people have a longer window period of seroconversion. Therefore if there is a definitive risk event, pts should get retested in 6 mo as in rare cases it may take that long for antibodies to develop.
• Routine testing is based on antibody detection using the enzyme-linked immunosorbent assay (ELISA) or enzyme immunoassay (EIA). In these tests the pt’s serum is diluted and applied to a plate which has HIV antigens attached. If the pt has anti-HIV antibodies they will attach. The plate is then washed and covered with a 2° antibody with an enzymatic function that binds to human antibodies. The plate is again washed and finally a substrate, which reacts with the 2° antibody with the enzymatic function is added producing a catalytic change in color or fluorescence.
• The Western blot is a confirmatory test when the initial antibody testing is positive. False positives are sometimes seen in pts who have other infections, particularly hepatitis. The procedure takes cells that may be infected, lysis them and applying the proteins to a gel to which electric current is passed. Naturally each protein band has a different velocity. The proteins are separated out and then applied to a membrane with antibodies similar to the ELISA test. The specific viral bands that are sought are for the GAG, POL, and ENV gene products. It is possible to have an indeterminate result. These people should be re-tested 1 mo later.
• Rapid point of care testing tests for antibodies using immunoassay technique. False positives are possible if the test is administered too early and antibodies are not formed. Although highly specific, false positives are possible with co-existing illness.

Tests for Antigen (the actual HIV virus particle)
• p24 test uses monoclonal antibodies. When p24 antigen is present it sticks to the antibody and the enzyme linked antibody causes a color change. The test is generally positive from about 1–3 wk following infection. Once the pt starts to produce their own antibodies, the p24 will usually be negative although they are infected. Later in the course, the p24 will often again be detectable if the disease is untreated.
• Nucleic acid amplification tests (NAAT or HIV PCR or viral load) are quantitative measurement of the HIV RNA. The test looks for and amplifies a 142 base sequence of the HIG GAG gene. Nucleic acid-based tests are used to test donated blood. Since these are costly tests they are first screened using a pooled sample of 18–20 units at a time. If a pool is positive the individual units are then tested. In addition, these types of tests are used clinically in conjunction with CD4 counts to monitor the progress of HIV pts.
• PCR tests uses RNA extracted from the plasma and is treated with reverse transcriptase to create cDNA. The polymerase chain reaction is then applied to amplify, hybridized, and then enzyme link identified.
• Quantiplex method centrifuges the virus, releases the RNA and bound with oligonucleotides with an enzymatic reaction.
• The results of these exams report the number of HIV copies in copies/milliliter. A low viral load of 40–500 copies/mL indicates the HIV is not reproducing. Undetectable levels does not imply cure, but rather it means the levels of circulating RNA are below the detectable amounts. The HIV will still persist in cells and tissues throughout the body.

Monitors
• CD4 and CD8 lymphocyte counts are important markers in monitoring immune function in pts with HIV. CD4 cells also known as T-helper cells identify, attack, and destroy infectious agents. HIV virus attaches to the CD4 cells, enters them and either replicates thus killing the CD4 cell or lies dormant. Eventually with unchecked infection the CD4 cell count declines. Once the CD4 count is less than 200cells/mL a person is said to have AIDS. The 200 level was selected as it correlates with increased likelihood of contracting opportunistic diseases. Sometimes the CD4 count is expressed in absolute numbers as above or as a percentage of lymphocytes or as a ratio to CD8 cells.

Risks
• There are no known risks of testing except for possible false positive or false negative.

Indications
• Recent advances in antiviral therapy whereby mother to fetus transmission can be blocked suggests routine testing of all pregnant mothers. While this is not current practice it is certainly has gained support.

Perioperative Precautions
• Positive state should be transmitted to OR and other staff so appropriate universal precautions can be vigorously maintained.
**Liver Function Tests (LFTs)**

**Risk**
- No risk with testing other than misinterpretation of data or misdiagnosis
- False-positive results, esp. with routine testing not guided by Hx/physical.
- Abn values can be due to causes unrelated to the liver; tests may be normal even in severe disease.

**Overview**
- Costs
  - Alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactate
  - Dehydrogenase (LDH), gamma glutamyl transpeptidase (GGT), alkaline
  - Phosphatase (AP), total bilirubin, direct bilirubin, albumin: $11–$40 each
  - 5' nucleotidase (5NT): $30–$50
  - Prothrombin time (PT): $8–$49
  - Multiple chemistries often available as panel $17–$49
- Liver function tests is somewhat misleading because most do not directly indicate hepatic function.

**ASSESSMENT POINTS**

<table>
<thead>
<tr>
<th>Test (Normal Result*)</th>
<th>Significance</th>
<th>Abnormal Response</th>
<th>Extrahepatic Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT (10–55 U/L)</td>
<td>Hepatocellular injury</td>
<td>Mild ↑ nonspecific; marked ↑ in extensive damage (acute viral, toxic hepatitis, or shock liver)</td>
<td>Most specific for liver injury</td>
</tr>
<tr>
<td>AST (10–40 U/L)</td>
<td>Hepatocellular injury</td>
<td></td>
<td>Myocardium, skeletal muscle, kidney, brain, pancreas, lung, leukocytes, erythrocytes</td>
</tr>
<tr>
<td>LDH (&lt;250 U/L)</td>
<td>Hepatocellular injury</td>
<td>Transient ↑↑ with shock liver; persistent ↑ in malignant infiltration</td>
<td>Myocardium, skeletal muscle, kidney, brain, erythrocytes</td>
</tr>
<tr>
<td>AP (45–115 U/L)</td>
<td>Cholestasis</td>
<td>Slight ↑ nonspecific; ↑↑↑ in cholestasis and infiltrative disease (i.e., metastasis)</td>
<td>Bone growth or ↑ turnover in bone disease states; pregnancy (placental origin)</td>
</tr>
<tr>
<td>GGT (&lt;30 U/L)</td>
<td>Cholestasis</td>
<td>Most sensitive indicator of biliary tract disease; poor specificity</td>
<td>Widely distributed in many tissues. induced by ethanol and drugs (i.e., anticonvulsants, warfarin)</td>
</tr>
<tr>
<td>SNT (&lt;11 U/L)</td>
<td>Cholestasis</td>
<td>Can be used to confirm hepatic source of ↑ AP</td>
<td>Widely distributed but serum ↑ specific for hepatic origin</td>
</tr>
<tr>
<td>Bilirubin (&lt;1.0 mg/dL)</td>
<td>Excretory function</td>
<td>Mild-moderate ↑ in many disease types; ↑↑↑ in obstructive, viral, alcoholic, drug-induced, or congenital disease</td>
<td>Hemolysis, hematoma resorption, muscle injury</td>
</tr>
<tr>
<td>Albumin (3.5–5.0 g/dL)</td>
<td>Biosynthetic capacity</td>
<td>Low in chronic disease, long half-life (20d); not a reliable indicator of acute disease</td>
<td>↓ In burns, enteropathy, malnutrition, fluid retention, nephrotic syndrome, etc.</td>
</tr>
<tr>
<td>PT (10.9–12.5 sec)</td>
<td>Biosynthetic capacity</td>
<td>Sensitive indicator of acute hepatic dysfunction due to short half-life of Factor VII.</td>
<td>Prolonged with warfarin therapy, vitamin K deficiency (may result from obstructive jaundice and ↓ uptake), consumptive coagulopathy</td>
</tr>
</tbody>
</table>

* Normal values may change depending on assay method.


**Perioperative Implications**
- Periop morbidity and mortality increased with acute or chronic liver disease
- Impaired hepatic function has implications for metabolism and elimination of some anesthetic drugs.
**Pregnancy Testing**

### Ethical/Legal Considerations
- **Who to test:** All female pts of childbearing age versus selected populations
- **If pt refuses after risks explained, should they be required to sign a waiver to legal rights relating to undetected pregnancy?**
- **Individual state legislature regarding rights of adolescents to keep results private from family**
- **Routine testing may create pt trust issues**
- **Important to protect the physician from unwarranted litigation**

### Risk
- **Risk to pt is false positive or false negative test or misinterpretation**
- **Increases risks in anesthesia, surgery in pregnant women of spontaneous abortion, low birth wt, and premature labor, and may be cognitive dysfunction of offspring if repeated anesthetics** (see SafeKidsAnesthesia.org at IARS site)

### Overview
- **Cost:** Numerous tests available; cost varies, both to pt and to institution. Average cost to pt
  - Urine hCG: $5–$25
  - Serum hCG: $15–$85
- **Importance of ruling out pregnancy before surgery**
- **Preop tests should be done or required on a selective basis for purposes of guiding and optimizing periop management**

### ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Pregnancy Test Technique</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Test Duration</th>
<th>Postconception Age 1st Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRMA (immunoradiometric assay)</td>
<td>150 mIU/mL</td>
<td>No cross-reactivity to LH, FSH, or TSH</td>
<td>30 min</td>
<td>18–22 d</td>
</tr>
<tr>
<td>IRMA less sensitive</td>
<td>1500 mIU/mL</td>
<td>Same as above</td>
<td>2 min</td>
<td>25–28 d</td>
</tr>
<tr>
<td>ELISA (enzyme-linked immunosorbent assay) more sensitive</td>
<td>25 mIU/mL</td>
<td>Same as above</td>
<td>80 min</td>
<td>14–17 d</td>
</tr>
<tr>
<td>ELISA less sensitive</td>
<td>50 mIU/mL</td>
<td>Same as above</td>
<td>5–15 min</td>
<td>18–22 d</td>
</tr>
<tr>
<td>RIA (radioimmunoassay)</td>
<td>5 mIU/mL</td>
<td>Cross-reactivity with LH (0.5%), FSH (0.2%)</td>
<td>4 hr</td>
<td>10–18 d</td>
</tr>
<tr>
<td>Fluoroimmunoassay</td>
<td>1 mIU/mL</td>
<td>No cross-reactivity to LH, FSH, or TSH</td>
<td>2–3 hr</td>
<td>14–17 d</td>
</tr>
<tr>
<td>Urine pregnancy test</td>
<td>25–50 mIU/mL</td>
<td>Same as above</td>
<td>1–5 min</td>
<td>&gt;22 d</td>
</tr>
</tbody>
</table>

False positives/negatives can occur with any of the above tests secondary to confounding factors including phantom hCG, pituitary hCG, exogenous hCG, trophoblastic neoplasms, nontrichoblastic neoplasms, molar pregnancy, ectopic pregnancy, hydatidiform mole, and delivery or abortion within a few weeks.

For borderline results, repeat test in 48 hr if hCG doubles; correlate hCG with LMP, PE, US.


### Perioperative Implications
- **1–2% of pregnant women undergo surgery for reasons unrelated to parturition.**
- **Literature is inadequate to determine whether or not anesthesia causes harmful effects on pregnancy.**
- **Intraop concerns incl the effect of surgery and anesthesia on the developing fetus from teratogenic effects of medications, maternal physiologic changes (hypoxia and/or acidosis), and changes in uteroplacental blood flow.**
- **Numerous studies on fetal outcome post surgery, post anesthesia demonstrate ↑ incidence of spontaneous abortions, LBW, esp. if surgery performed in first trimester**
- **No ↑ incidence of congenital anomalies (even with N2O), although recent concern with doubling of behavioral abn and cognitive dysfunction if repeated anesthesia exposure prior to age 3 y (see SafeKidsAnesthesia.org at IARS)**
- **If possible, local or regional anesthesia used in first trimester.**
- **During first trimester, thiopental, muscle relaxants, narcotics, propofol safely used.**
- **Use of benzodiazepines, N2O, inhalation agents more controversial**

### Test Indications
- **Pts should be offered, not required, pregnancy testing unless there is a compelling medical reason to know the pt is pregnant.**
- **To diagnose pregnancy in periop period; to quantify gestation.**
- **Medical Hx alone often unreliable, esp. in adolescents**

### Recommendations
- **Age <13: No pregnancy test unless indicated by history.**
- **Age 13 until 1 y after last menses: Preop pregnancy test should be offered to all pts.**
Renal Function Testing

Overview
- Cost
  - Urine indices
    - Basic analysis (SG, pH): $6–$12
    - Lytes (Na⁺): $8–$36
    - Cr: $10–$29
    - Osm: $16–$46
  - Serum chemistries
    - BUN: $8–$29
    - Cr: $8–$29
    - Lytes (Na⁺): $8–$36 (29)
    - Osm: $16–$46
  - Combination indices
    - Cr clearance: $60–$75
    - Free water clearance: $90
    - Fe Na⁺: $120

Positive Result

Confounding Factors
- <25 mL/min
- Dx Information
  - Changing hydration states, inaccurate volumes
  - Renal function testing save inappropriate Rx based on misleading data or data misinterpretation
  - Test to predict periop renal function reserve, predict or Dx renal morbidity during high-risk surgery (trauma, vasc, cardiothoracic) in pts at high risk for renal failure (preop renal insufficiency, low CO syndrome, etc.)

Test Indications
- Urine formation begins with glomerular ultrafiltration and progresses to tubular reabsorption and tubular secretion. All anesthetic agents have the potential to alter renal function by altering BP and cardiac output so that renal blood flow is redistributed. This redistribution is accompanied by sodium and water conservation and decreased urine formation. Dx, evaluate extent of renal tubular function, GFR in pts to assess periop risk and/or morbidity.

ASSESSMENT POINTS

<table>
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<tr>
<th>Test</th>
<th>Positive Result</th>
<th>Confounding Factors</th>
<th>Dx Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinalysis</td>
<td>Hematuria (&gt;1–2 RBC)</td>
<td>Multiple causes</td>
<td>Glomerular disease, free Hgb or myoglobinuria, UTI, interstitial nephritis, pyelonephritis, glomerular disease</td>
</tr>
<tr>
<td>Urine Na⁺</td>
<td>&lt;20 mEq</td>
<td>Hormonal secretion (ADH, aldosterone), Na⁺-avid states (CHF, cirrhosis), saline infusion, diuretics, dopamine</td>
<td>Prerenal azotemia sensitivity 50% (PPV 50%), ATN sensitivity 55% (PPV 50%)</td>
</tr>
<tr>
<td>Urine Osm</td>
<td>&gt;500 mOsm/kg H₂O</td>
<td>Proteins, glucose, mannitol, diuretics, advanced age, temp extremes</td>
<td>Prerenal azotemia sensitivity 30% (PPV 60–90%), ATN sensitivity 80% (PPV 65–95%)</td>
</tr>
<tr>
<td>Serum Cr</td>
<td>&gt;2 mg/dL</td>
<td>↑ N halance, tissue breakdown, basal metabolism, diet, activity, hepatic disease, hematoma, GI bleeding, drugs</td>
<td>Assoc with ↑ risk of postop renal insufficiency, LOS, and cost of care after CABG surgery when &gt;1.4 mg/dL. Nml variant or ↓ renal function reserve GFR ↓ by &gt;50%</td>
</tr>
<tr>
<td>Fe, Na⁺, urine Na⁺/plasma Na⁺</td>
<td>&lt;1%</td>
<td>Vol depletion</td>
<td>Only helpful after ATN Does not allow prediction</td>
</tr>
<tr>
<td>Cr clearance</td>
<td>Urine Cr/Plasma Cr &gt;25 mL/min</td>
<td>Changing hydration states, inaccurate vol collection, nml day-to-day variation</td>
<td>Predicts ↑ periop renal morbidity, renal failure</td>
</tr>
</tbody>
</table>

Additional/Alternative Tests
- A plain KUB film may be used to identify renal disease with hematuria, pain, and/or fever to R/O trauma
- US to discriminate renal masses (cyst versus mass), locate obstructive nephropathy source
- Doppler US can facilitate finding cause of allograft dysfunction when evaluating renal flow following transplant
- Renal flow scan (¹²⁵I–DTPA) also useful for RBF analysis esp when comparing one kidney with the other
- Renal angio can be used to visualize medium and/or small artery anatomy.
- Alternatives to above may incl MRI and contrast US.

Perioperative Implications
- Periop renal failure following high-risk procedures has a reported incidence of 0.1–50% depending on population analyzed and methods used to define renal failure; is assoc with a reported mortality of 20–95%
- Periop renal failure accounts for half of all pts requiring acute renal dialysis
- No simple, inexpensive test adequately determines renal function
- Advanced biomarkers of renal tubular cell damage
- Urinary glutathione S-transferase (GST)
- β-N-acetyl-[14C]-glucosaminidase (NAG)
- Test to predict periop renal function reserve, predict or Dx renal morbidity during high-risk surgery (trauma, vasc, cardiothoracic) in pts at high risk for renal failure (preop renal insufficiency, low CO syndrome, etc.)


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<td>Glomerular disease, free Hgb or myoglobinuria, UTI, interstitial nephritis, pyelonephritis, glomerular disease</td>
</tr>
<tr>
<td>Urine Na⁺</td>
<td>&lt;20 mEq</td>
<td>Hormonal secretion (ADH, aldosterone), Na⁺-avid states (CHF, cirrhosis), saline infusion, diuretics, dopamine</td>
<td>Prerenal azotemia sensitivity 50% (PPV 50%), ATN sensitivity 55% (PPV 50%)</td>
</tr>
<tr>
<td>Urine Osm</td>
<td>&gt;500 mOsm/kg H₂O</td>
<td>Proteins, glucose, mannitol, diuretics, advanced age, temp extremes</td>
<td>Prerenal azotemia sensitivity 30% (PPV 60–90%), ATN sensitivity 80% (PPV 65–95%)</td>
</tr>
<tr>
<td>Serum Cr</td>
<td>&gt;2 mg/dL</td>
<td>↑ N halance, tissue breakdown, basal metabolism, diet, activity, hepatic disease, hematoma, GI bleeding, drugs</td>
<td>Assoc with ↑ risk of postop renal insufficiency, LOS, and cost of care after CABG surgery when &gt;1.4 mg/dL. Nml variant or ↓ renal function reserve GFR ↓ by &gt;50%</td>
</tr>
</tbody>
</table>

Fe, Na⁺, urine Na⁺/plasma Na⁺ | <1% | Vol depletion | Only helpful after ATN Does not allow prediction |

Cr clearance | Urine Cr/Plasma Cr >25 mL/min | Changing hydration states, inaccurate vol collection, nml day-to-day variation | Predicts ↑ periop renal morbidity, renal failure |

PPV = positive predictive value.

Spirometry

Risk
- Virtually no risks assoc with spirometry PFTs; risk can occur with use of bronchodilators or mis
interpretation of data

Overview
- Cost: Variable: $250 spirometry; $100
diffusing capacity for CO; $650 lung volumes
(State of Maryland Health Services Cost Review
Commission)
- Spirometry is relationship between exhaled
volume (starting from position of maximum
inspiration) with maximum effort (as forceful as
possible—i.e., forced) and time. Quotient of FEV
in first sec of exhalation (FEV,) and FVC (known
as FEV,%) may be used to define obstructive lung
disease and to suggest restrictive lung disease (see
table following)
- PFTs reflect airway resistance, elastic properties
test of lungs, chest wall
- Airway resistance not measured by PFTs; presence
of increased airway resistance inferred from
decided exhalation airflow; assumes max effort
was made by pt
- Accuracy, interpretation of PFTs highly dependen
t on pt cooperation, pt effort. Results must be
reproducible to be valid.

ASSESSMENT POINTS

<table>
<thead>
<tr>
<th>Test</th>
<th>Positive Result</th>
<th>Confounding Factors</th>
<th>Dx Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measurements suggestive of OLD</td>
<td>FEV, &lt; FVC = 0.8</td>
<td>Requires pt's cooperation, max effort, measurements must be reproducible</td>
<td>Normal ratio</td>
</tr>
<tr>
<td></td>
<td>1) FEV, &lt; FVC = 0.66–0.8</td>
<td></td>
<td>Moderate OLD</td>
</tr>
<tr>
<td></td>
<td>2) FVC &lt; predicted</td>
<td></td>
<td>Severe OLD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Causes of OLD: Acute (asthma), chronic (bronchitis,</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>emphysema), or related to upper airway lesions</td>
<td></td>
</tr>
<tr>
<td>Measurements suggestive of reversible OLD</td>
<td>In FEV, and FVC of at least 15% with administration of inhaled bronchodilator</td>
<td>Lack of response to inhaled bronchodilator does not exclude reversible airflow obstruction in pts with severe obstruction</td>
<td></td>
</tr>
<tr>
<td>Measurements suggestive of RLD</td>
<td>FEV, &lt; 85% and FVC &lt; predicted</td>
<td>Requires lung vol measurement to confirm</td>
<td>Suggests RLD, incl NM disease; chest wall disease (kyphoscoliosis); infiltrative or destructive interstitial diseases (interstitial fibrosis, ARDS); space-occupying lesions; or pleural disease</td>
</tr>
<tr>
<td>Measurements suggestive of mixed OLD/RLD</td>
<td>FEV, &lt; 0.8 and FVC &lt; predicted or significantly ↓ VC assoc with ↓ FEV, /FVC ratio</td>
<td>When mixed defect considered, lung volume determination must be made</td>
<td>Suggests presence of two processes—e.g., COPD and NM disease, or COPD and tumor; sarcoidosis</td>
</tr>
</tbody>
</table>


Perioperative Implications
- Routine use of PFTs not indicated; consider
PFTs if Dx of obstructive lung disease not pos-
sible on clinical basis
- Peak flow during max exhalation useful as sim-
ple bedside test to follow response of broncho-
spasm to Rx. Peak flow determined primarily by
diameter of large airways; is ↓ in moderate-to-
severe obstruction.
**Indications**

- **Cardiac Indications**
  - Cardiac function: Especial useful to assess preload, systolic, diastolic function
  - Ischemia: Regional wall motion abn, defined as changes in wall thickening, wall motion, indicative of ischemia
  - Valvular function: valvular abn identified using imaging, Doppler exam; intraop assessment allows ↑ use of valve repairs rather than replacements, vascular route placement of replacement valves
  - Aortic disease: TEE is the gold standard for Dx of aortic disease dissection; used by some to select cannulation site for A-cannulae to ↓ risk of emboli
  - CHD: TEE allows assessment of adequacy of valve repairs intraop
  - Cardiac devices: Placement of intracardiac devices and monitoring of their position during port access and other cardiac surgical interventions

- **Non-cardiac Indications**
  - Assess: Volume status, ventricular function, valve pathology, pericardial effusions, constrictions
  - Monitor: Ischemia, assessment response to therapy
  - Rapid Dx; trauma, pericardial pathology, hemodynamic instability and hypoxia
  - Aorta: Endovascular stent, aortic dissection, aortic plaque
  - Heart transplant: Right heart function

**Contraindications**

- **Absolute**
  - Extensive esophageal or gastric disease, esophagitis, strictures, advanced carcinoma, surgery.
  - Unstable cervical spine: Neck, flexion to introduce the probe a sure way of ‘pithing’ a pt with a fractured odontoid peg, or severe atlanto-axial subluxation

- **Relative**
  - Esophageal varices, Zenker’s diverticulum, Barrett’s esophagus
  - Postradiation: Mediastinal irradiation therapy
  - Upper airway disease, very tenuous airway
  - Poor dentition, recent dental surgery
  - Pt hasn’t fasted for 4 to 6 hr

**Complications**

- Esophageal tear and/or perforation, Mallory-Weiss tear related to TEE, bleeding from esophageal varices, esophageal and/or gastric erosion
- Burns, electrical shock
- Swallowing difficulties, dysphagia and/or odynophagia
- Skin staining, damage of oropharynx, dental
- Airway obstruction, tracheal extubation, mainstem intubation, vocal cord paralysis, bronchospasm, desaturation and resp distress
- Sedation in awake pts: Aspiration or apnea
- Other complications: Htn, hypotension, angina, vascular compression, bradycardias due to vagotomy, AV block, tachyarrhythmias (atrial or ventricular)

**Training**

- Development of competence in TEE requires acquisition of numerous cognitive and technical skills; a period dedicated to intensive training under direct supervision of expert is highly recommended.
- Advanced periop TEE certification since 2003
- Basic perioperative TEE certification 2010

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**Recorders:** Hard copy, videotape, or digital

- **Ehoscanner:** Analyzes reflected echoes, generates images or flow tracings

**Equipment**

- **Esophageal probe:** Single-plane, transverse images; biplane, transverse, longitudinal images; multiplane, transverse to longitudinal to transverse images (180°)
- **Echoscaner:** Analyzes reflected echoes, generates images or flow tracings
- **Recorders:** Hard copy, videotape, or digital

**Overview**

- Cost: $300–$500/pt use
- Imaging technique utilizing US to examine structure, function of heart, great vessels, to gain information on blood flow within these structures
- US crystals mounted on gastroscope inserted into esophagus/stomach; placed behind heart
- Tomographic images constructed from intensity of reflected signals, analyzed electronically, converted to image by echoscanner
- Flow from frequency shift between emitted and reflected US using Doppler equation

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**Risk**

- Esophageal injury or bleeding, vocal cord paralysis, dysrhythmias, hypotension, seizures, cardiac arrest (occur in less than 3% of exams) Mortality rate (0.01–0.03%)
- Minor injuries: Lip injuries (13%), hoarseness (12%), dysphagia (1.8%), ET intubation (0.3%), bradycardia (0.2%), dental injuries (0.1%)
- Bacteremia (0–4%), little evidence that pts on anticoagulation increases risk of bleeding during TEE.
- No evidence that anticoagulation increases risk of bleeding during TEE.
- Erroneous interpretation, distraction from other anesthetic duties (unknown incidence)

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V/Q Scan (Nuclear Ventilation-Perfusion Scintigraphy)

**Risk**
- Essentially the same for CXRs and IV access
- Small exposure to radiation from the radioisotope which is gone in 2–3 d and should be avoided in pregnancy and breastfeeding as well as those with allergies to radioisotope.
- Pulm embolism, the most common reason for ordering a V/Q scan, has an incidence of 94,000 cases annually in the USA.

**Sensitivity/Specificity**
- For evaluation of pulm embolism, the sensitivity of a high-probability (PE present) scan is 77.4% (95% CI 96.4–98.9%) while the specificity of a very low probability or normal (PE absent) scan finding is 97.7% (95% CI 96.4–98.9%).

**Overview**
- Cost: $800 to $1000 for CPT reimbursement (CPT code 78580, 78585); Total cost per test to hospital is roughly $250 to $350 per pt
- Nuclear medicine test to evaluate the flow of blood (perfusion) through the lungs and the flow of gas (ventilation) through the lungs
- The ventilation scan has radioactive tracer gas (\(^{133}Xe\), \(^{127}Xe\), \(^{99m}Te\)) inhaled into lungs with bright spots being where gas is ventilated and dark spots where gas is not.
- The perfusion scan has radioactive tracer substance (\(^{99m}Te\) MAA) injected into a vein where it travels to the lung and shows up as bright where blood is being perfused and dark where it is not.

**Indications**
- Evaluation of disturbances between ventilation and perfusion incl pulm embolism, non-thrombotic emboli, lung cancer, intravascular disturbances (tumor, fat, parasite), pulm stenosis/arteritis, COPD, pneumonia and for performance quantification pre- and post- lung lobectomy/pneumonectomy surgery

**Additional Tests**
- It is common to have a routine two-view CXR before receiving a V/Q scan
- Chemistries, cultures, and blood counts are not routine; however, may be helpful in further ruling in or out a disease process.

**ASSESSMENT POINTS**

**Interpretation involves looking for mismatch for ventilation>perfusion, ventilation=perfusion and ventilation<perfusion by comparing tracer brightness of ventilation images to perfusion images**

**Interpretation for pulmonary embolism**

**High Probability**
- At least 2 large (>75% of a segment) segmental perfusion defects without corresponding ventilation or CXR abn
- At least 4 moderate segmental (25–75% of a segment) perfusion defects without corresponding ventilation or CXR abn

**Intermediate Probability**
- 1 large and less than 2 moderate segmental perfusion defects without corresponding ventilation or CXR abn
- Single moderate mismatched defect with normal CXR findings
- Corresponding V/Q defects and CXR parenchymal opacity in lower lung zone
- Corresponding V/Q defects and small pleural effusion

**Low Probability**
- Multiple matched V/Q defects, regardless of size, with normal CXR findings
- Corresponding V/Q defects and CXR parenchymal opacity in upper and middle lung zone
- Corresponding V/Q defects and large pleural effusion
- Any perfusion defects with substantially larger CXR abn
- Defects surrounded by normally perfused lung (Stripe sign)
- More than 3 small (<25% of a segment) segmental defects with a normal CXR
- Nonsegmental perfusion defects

**Very Low Probability—Up to 3 Small Matching Segmental Perfusion Defects with Normal CXR**

**Normal**
- No perfusion defects and perfusion outlines the shape of the lung seen on CXR


**Preoperative Preparation**
- Management of anesthesia for pts with V/Q abn is to support vital organ function and to minimize anesthetic-induced myocardial depression
- If operation is not emergent and not aimed at correction of V/Q abn, strong consideration should be made to postpone operation to allow correction of V/Q abn if possible.
- Not uncommon for pts with severe V/Q mismatch to arrive in OR intubated and mechanically ventilated, often with high FIO2.
- May be necessary to support cardiac output with inotropic drugs incl catecholamines to increase myocardial contractility or phosphodiesterase inhibitors to increase myocardial contractility and vasodilate pulm arteries
- Maintenance of NMB should be done to minimize histamine release.

**Induction**
- Monitoring of intra-arterial pressure and cardiac filling pressures often necessary if V/Q mismatch severe
- Right atrial filling pressure can be a guide to optimize IV fluid administration

**Maintenance**
- Must avoid any accentuation of arterial hypoxemia, systemic hypotension, and pulm Htn
- Can be maintained with any drug or combination of drugs that avoid significant myocardial depression
- Nitrous oxide not a likely selection due to often high FIO2 requirement as well as its potential to increase pulm vascular resistance worsening the perfusion of the lungs
- Often significant hemodynamic improvements are made post embolectomy or operations aimed to correct V/Q abn.
- Postop care should aim to continue supporting vital organ function and minimize cardiac injury which may incl remaining intubated or pressor support.