DETECTION OF FŒTAL HEART MURMUR IN GRAVIDA WITH REPORT OF A CASE.

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Presented by the Author.
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Osler mentions, in his Practice of Medicine, that foetal endocarditis has been diagnosed in gravida by the detection of a rough systolic murmur through the abdominal walls. He has kindly given me the references to these cases—two quoted by Hochsinger, and the third reported to the American Pediatric Society by Christopher of Chicago. Longstreth refers to a case, but it is probably one of those quoted by Hochsinger. The reported cases are briefly as follows:

H. Barth* saw a pregnant woman who presented, to the left and below the navel, instead of the normal foetal heart-sounds, a widespread, rough, systolic murmur. The child was still-born, and presented, upon section, enormous hypertrophy of the heart, especially upon the right side, with thickening of the tricuspid valves, and abundant endocarditic vegetation.

Hennig† reports the second case, occurring in a woman with contracted pelvis, who was artificially delivered in the eighth month. Before delivery it was found that both heart-sounds were replaced by murmurs, and this was confirmed by examination of the already cyanotic foetus after delivery. Section demonstrated the presence of extensive endocarditis about the aortic valves, giving rise to both stenosis and insufficiency.

The case reported by Christopher occurred in the practice of Frederick J. E. Ehrmann, of Chicago, who has kindly sent me the notes for this paper, an abstract of which I present:

A German woman, thirty-two years of age, presented herself in the eighth month of her third pregnancy, for examination, and everything was found practically normal. The foetal heart-sounds were pure, 150 per minute. At time of labor the

*Quoted by Hochsinger.
† Ibid.
fœtus presented O. L. A., and a murmur was found in the left iliac fossa, 140 per minute. The female child was born after a normal labor, and was normal excepting for a systolic murmur, heard over the cardiac region, and transmitted to the neck. Cyanosis appeared on the second day, and the child expired on the fourth, there having been no material change in the murmur. The remainder of the report I give in Dr. Ehrmann's words:

"Diagnosis before birth, possible intra-cardiac lesion, on account of the previous clear tones, but later only murmur. The umbilical souffle was thought of."

"Diagnosis after birth, aortic valvulitis, which, on account of the rarity with which inflammation occurs on the aortic valves, was thought to be possibly connected with some anomaly."

"Post-mortem.—Patent foramen ovale, verrucose thickening of aortic valves, and also tricuspid. No pulmonary artery arising from right ventricle, but from aorta just above sinus of valsalva."

My own case was as follows:

Mrs. P., American, primipara, seventeen years of age. Position, O. R. A. The foetal heart was heard with the ear, as I had then no stethoscope with me, 124 per minute, to the right of the navel, and nothing abnormal was noted. The placental bruit was marked just at the left of the navel.

The first stage progressed very slowly, pains being absent for many hours. The next day at noon they began again, and I examined the abdomen with the stethoscope.

The heart beat at the same rate, being heard about two inches downward and to the right of the navel. The first sound was rough and blurred, the second being normal. My assistant, Dr. S. S. Connacher, examined the case and noted, under my direction, the murmur replacing the first sound. I stated at this time that, in my opinion, we should find a systolic murmur over the child's heart after birth, probably originating from the right ventricle, because of the marked predominance of right-sided endocarditis in foetal life.

A boy weighing seven pounds was delivered at 4 p.m. with forceps. For about fifteen minutes he was decidedly cyanotic, although the heart could be seen at first, beating forcibly, over an area greater than usual. A rough, slightly musical systolic murmur, loudest at the junction of the third costal cartilage and
the sternum, was heard by both of us. It was not transmitted materially in any direction, and could not be heard in the back. The pulmonic second sound was accentuated. From the very unusual area of pulsation of the heart I had at first supposed the organ to be hypertrophied, but I apparently erred in this in not taking into account the temporary difficulty in respiration, on account of which the lungs had not expanded so as to cover the heart to the normal degree. A half hour after birth, respiration was well established, cyanosis had disappeared, and the area of cardiac dulness was normal.

On the third day Dr. S. D. Hopkins examined the case with me. The murmur was about as at the time of birth, but seemed to me somewhat weaker. The pulmonic second sound was thought to be three times as pronounced as the aortic. Unfortunately I failed to note whether the sound was transmitted into the arteries of the neck, although I believe that I should have noted it had such transmission existed.

During the next week the murmur grew progressively weaker, and on the tenth day the heart was entirely normal in its action so far as I could determine.

At the time of my report of this case to the Denver and Arapahoe Medical Society on May 25, 1897, I had not had opportunity to study up the possible modes of origin of the murmur, and especially had not had access to Hochsinger's invaluable work (Die Auscultation des kindlichen Herzens). I believe, however, that I am now able to offer a satisfactory explanation of the origin of the murmur.

It will be recalled that the ductus arteriosus botalli conducts the foetal blood from the pulmonary artery to the aorta, the artery being unable to conduct the blood to the lungs as after birth, because of the non-use of these organs during the foetal life. When the lungs begin to receive the blood from the pulmonary artery, upon the establishment of respiration, the duct begins to close, the closure becoming complete from the fourth to the tenth day after birth, according to Gray.

In persistence of the duct, the physical signs noted are, according to Hochsinger, a loud, whirring murmur, heard loudest over the region about the second left interspace, and often transmitted to the arteries of the neck; and marked accentuation of the pulmonic second sound. Upon this latter feature this author lays especial stress, the blood-pressure in the pulmonary artery
being decidedly raised by the inter-communication with the aorta, with its higher blood-tension, thus accounting for the accentuation.

The murmur in our case disappeared at the time of the normal closure of the duct. The child is at this time, four months after birth, well developed, and apparently entirely normal. Anomalies in structure of the heart, and persistent foetal endocarditis may apparently be properly excluded. We have only to suppose a structural defect or endocarditis, limited to the duct, and causing roughening or stenosis of its lumen, to account for a systolic murmur, which would, as in our case, disappear with the physiological closure of the duct by the tenth day. We assume in this case that the possible endocarditis is no longer active. The point of greatest intensity of the murmur in our case so nearly coincides with that mentioned by Hochsinger that we may overlook the discrepancy.

In closing it should be stated that Hennig believes that we may have single or double fetal heart murmurs, audible in utero, but not to be heard after birth, and this in cases in which the possibility of the location of the murmur in the cord is excluded.

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