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MULTIPLE CONGENITAL CARDIAC LESION.

A Clinical Lecture Delivered at the University Hospital,
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[Reported by Dr. A. L. Benedict.]

G. aged 5. Even when a baby, he seemed to have an abnormal amount of palpitation about the heart. He has had none of the diseases of childhood. Last winter he had a little fever, and was found to have heart disease.

His cheeks are a little flushed but his fingers are not clammy. There is marked pulsation of the vessels of the neck and the supra-sternal notch. His chest is symmetrical and so are the respiratory movements. The præcordia is a little prominent, and there is a visible heaving pulsation over it. The apex-beat of the heart is in the fifth interspace; it is very forcible in the nipple line and extends beyond it. There is an evident thrill occupying nearly the whole of the diastolic period of the pulsation, especially marked in the second interspace from the sternum outward. The cardiac dulness begins just below the left sterno-clavicular articulation and extends in a curved line to the beginning of the right second interspace, and thence downward three-quarters of an inch to the right of the sternum till it is lost in the liver dulness. From the left sterno-clavicular junction the dulness extends a little beyond the nipple line and thence to the apex.

The cardiac dulness extends too high and too widely; it goes to the right of the sternum and is felt outside the nipple. There is a transverse diameter of 4½ inches and a vertical diameter of 4 inches.

Such a cardiac dulness we would suppose would be produced by a general hypertrophy involving the ventricles and auricles of both sides of the heart. We would say then, without going any further in the examination, that this heart which gives us in a child five years old such an area of dulness should be the seat of a general hypertrophy.

Auscultation reveals a distinct systolic murmur at the apex. A second sound is heard, but it is accompanied by a slight diastolic murmur. The systolic murmur is transmitted into the arm-pit, and the diastolic murmur is also faintly heard there. At the xiphoid cartilage both murmurs are audible. At the aortic cartilage there is a short systolic murmur; this is followed by the diastolic murmur which is more clearly heard at this spot. Both murmurs are heard very distinctly over the sternum. At the pulmonary cartilage both systolic and diastolic murmurs are excessively loud. In fact the diastolic murmur there is louder than the systolic.

Thus we have a double murmur, and this double murmur, though audible over a very extensive area, is strongest at the pulmonary cartilage. The murmurs are transmitted beneath the left clavicle ; less distinctly under the right clavicle. In the left axilla, the diastolic murmur is loudest to the fourth interspace, while below this the systolic murmur is loudest and the diastolic murmur is scarcely heard.

Liver, spleen, and lungs normal. Pulse regular, of fair volume, of low tension. There is a decided tendency to the abrupt receding of the pulse—the so-called trip-hammer or water-hammer pulse of aortic regurgitation.

As I have said, there have been no evidences of serious trouble, no frequent pulse, no more coughing than other babies have, no cyanosis.

In January, he began to have severe coughs, and since then, I understand, he has had more or less bleeding from the nose and spitting of blood.

The diagnosis of the exact condition present is important. It is also important to determine in what way this began.

You see that it evidently dates back to a very early period of life if it is not congenital. This is especially important and interesting, because, as you know, congenital lesions of the heart are so frequently on the right side, whereas postnatal lesions more commonly involve the left side of the heart.

You see at a glance the conditions of which I read you the account—a very trifling prominence of the præcordia, a very strong thrill, and this thrill most strongly marked over the region of the pulmonary artery.

I will not spend time in studying the percussion sounds, for percussion would have to be made so gently that it would be scarcely audible at a distance.

In the next place, you see that the apex-beat of the heart is heaving and moving the 3d, 4th, 5th, and 6th ribs, extending outside the line of the nipple, although the true apex-beat is just about in the nipple-line in the 5th interspace.

Now, as to these murmurs of which I have said so much. At the base we have the loud systolic murmur and the slight diastolic murmur. We go to the apex and they are still audible, though they change their quality. We go to the xiphoid, and they are louder than at the apex. We go to the aortic cartilage, and they are very large and loud. There is an enormous humming murmur which occupies the whole revolution of the heart's action, heard over the upper piece of the sternum, but it is most intense in the pulmonary area where the thrill is felt most intensely, and is carried up very strongly along under the left clavicle where it becomes only a single systolic murmur. The diastolic murmur is heard best along the right border of the sternum.

You will observe at once, therefore, that this is an exceedingly peculiar case of heart disease. It is peculiar in the very early age at which palpitation of the heart has developed itself. It is peculiar in that the child has not had any spells that can be regarded as rheumatic ; it has not had scarlatina ; there has been no attack since birth which could be regarded as the cause of this lesion. It is peculiar in the distribution of the physical signs and especially in regard to the large area of the pulmonary artery which is involved also by the murmur.

We come back, therefore, to the feeling that this must be a congenital

lesion. This is confirmed, not only by the early date at which the symptoms arose, but by the character of the lesion—for what is that lesion?

We may say with confidence, whatever else does or does not exist, that this certainly exists—a high degree of stenosis of the orifice of the pulmonary artery. This is a congenital lesion. In consequence, the right ventricle in endeavoring to propel blood through the pulmonary artery, finds the orifice greatly narrowed. There is a tremendous friction and fremitus produced at the orifice by the violent effort of the right ventricle to drive blood through this contracted opening, hence the thrill and hence the intense systolic murmur carried along the line of the pulmonary artery. Hence the enlargement, the dilated hypertrophy of the right ventricle, giving an added area to that of normal cardiac dulness.

But what becomes of the blood that cannot get out through the pulmonary artery and fails to secure its proper aeration in the lungs? How has this child preserved such good health; how has he escaped serious evidence of the backward pressure—general œdema and venous stasis? How has the equilibrium been maintained? It has been maintained by a widely patulous ductus arteriosus between the pulmonary artery and the aorta. I have no hesitation in saying that as the ductus arteriosus is a vessel of considerable size leading from the concavity of the arch of the aorta to the bifurcation of the pulmonary artery, and that a large amount of blood that cannot get into the pulmonary vessel by means of the stenosed orifice, gets to it by going first into the aorta and then back into that vessel. Hence I say that a large amount of this murmur is a ductus arteriosus murmur, and the extension across the sternal region and into the aortic area is, I think, dependent upon this fact.

But is not the aorta also involved? I think it not at all improbable. Certainly, from the character of the pulse there is, in some way, a cut-off which causes an abrupt recession of the wave. The aorta is filled by a large volume of blood thrown from the left ventricle. This blood is not all of it propagated throughout the arterial system. The pulse passes on, the blood falls back. This gives it the receding trip-hammer, water-hammer pulse, the pulse of Corrigan. But is it necessary that there should be an actual valvular insufficiency of the aortic orifice? Suppose this failure of the blood column is not due to failure of the valves, but due to being shunted off into the pulmonary artery. Would not that produce a Corrigan pulse as well as an aortic insufficiency? That is a point that I think has never been raised. The explanation that I suggest to you is one that, of course, would occur to any one, but I cannot say whether it is adequate.

It is not at all impossible that we have in addition to this, some lesion on the left side of the heart. In fact, the very wide distribution of these murmurs would indicate that the whole valvular apparatus of the heart is working badly. I think it extremely likely, from the strong diastolic murmur which is transmitted to the xiphoid cartilage that there is some insufficiency of the aortic leaflets, some aortic regurgitation.

As to the character of the systolic murmur at the apex, I speak with much less confidence. The systolic murmur over the pulmonary artery is so intense, so extraordinarily and almost unprecedentedly intense, that I think it is not at

all improbable that the systolic murmur heard over the body of the heart is largely the transmitted pulmonary murmur. Therefore, in view of the fact that this child is apparently so well, and has been free from colds, coughs, congestions, œdemas, and the like, I hesitate to say that there is a mitral regurgitation in addition to aortic insufficiency and pulmonary stenosis. My impression is that we must regard the pulmonary stenosis as the chief lesion, that we must regard the left cavities of the heart as fairly sound, that we must explain the aortic murmur, as far as possible, by the condition of the ductus arteriosus, and that we must be forced to admit some aortic implication, but that we must fight against the admission of mitral disease.

Why has not this child cyanosis? Why is there not blue discoloration and clammy fingers? Is it the case that, in spite of this tremendous murmur, the roughness of the pulmonary orifice, is, after all, not so very great? You know you cannot possibly tell by the intensity of the murmur how great a valvular lesion is. Some of the loudest murmurs that you hear are murmurs that are caused by small patches of roughness with very strong heart muscle driving the blood rapidly and powerfully over them. Some of the most serious lesions, where it seems that life can scarcely be prolonged, are not attended by marked murmurs, because the heart is not able to drive the blood forcibly enough through the opening to produce such a murmur. Now, it may be that, while the pulmonary orifice is diseased, and while there is a calcareous rough ring or hard fibrous ring with little vegetations, and while the enlarged right ventricle, driving the blood through this, causes this transmitted murmur, the amount of actual obstruction is not so extreme, and this would explain why the admission of a certain amount of blood through the ductus arteriosus to the pulmonary artery, would keep up the equilibrium of circulation, and preserve the child from cyanosis or serious congestions.

Lastly, the auricles of this heart are enlarged, and there is a mechanism in the auricles, as you very well know, the foramen of Botal, with its valve, which is in fœtal life open, and which is an expedient to permit the excessive pressure of the right side of the heart to relieve itself by the passage of the blood into the left auricle. Now, if this be a congenital lesion, would not the excessive tension of the right auricle have prevented the closure of this foramen? You know that the foramen ovale closes a few days or weeks after birth because the balance of pressure becomes equal, the valve is held vertically between the auricles, it secures an attachment, and closes the foramen. But, in a case like this where there must be an excessive tension of the right cavities of the heart due to obstruction at the pulmonary orifice, this occurring in fœtal life must have kept that foramen from closing, must have prevented the valve from becoming adherent. Hence, I would add unhesitatingly here, that we have a patulous foramen ovale, and I think very likely, some of this diffuse humming murmur heard here over the upper segment of dulness, is due to a current passing from the right auricle into the left auricle.

So that the total diagnosis of this case is—congenital cardiac lesion; the initial lesion, endocarditis of the pulmonary orifice with stenosis and roughening; consecutive changes, the inability of the ductus arteriosus or foramen ovale

to close ; the current passing from the aorta into the pulmonary artery ; the current passing from the right auricle into the left auricle ; thus the distended right cavities are relieved ; thus the insufficient supply of pulmonary blood is aided by an installment from the arorta with every stroke ; hence nutrition is fairly well preserved, and the equilibrium thus far so well maintained that cyanosis has not resulted ; all the cavities of the heart are in a state of dilated hypertrophy.

Now as to prognosis. I need not say to you that the prognosis is unfavorable. This dates back to a period of life when plastic processes are so rapid and when tissues organize until they become in firmness almost like the original tissues of the body We cannot hope, therefore, actually to remove or to relieve the stenosis of the pulmonary orifice. Our prognosis must be that this lesion is essentially incurable. As to the duration of life which is compatible with such a lesion, I should put no limit at all upon it. As long as the equilibrium of circulation between the two sides of the heart is as well maintained as at present, there is no reason why this child should not live with comparative comfort indefinitely. It is therefore wholly uncertain, but hopeful as to duration of life ; yet clinical experience shows that as puberty approaches, and the subjects of congenital heart disease becomes self-willed and difficult to control and their ambitions and impulses stir within them, nearly always they overdo and bring on rupture of compensation, so that the instances are rare in which congenital lesion of the heart, of as serious a grade as this has been, have endured for more than twenty-five or thirty years, at most.

The treatment is, of course, absolutely hygienic. It is by strict obedience to all the rules of health, careful diet, extreme care in dress, extreme care in avoidance of damp, in avoidance of chilling, in avoidance of excitement and over-exertion and the like, that safety to life lies. But if this plan be carried out faithfully, there is no reason why a long period of comparative health shall not be enjoyed.