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REMARKS ON SOME OF THE CLINICAL FEATURES OF TRICUSPID STENOSIS.

Remarks on some of the Clinical Features of Tricuspid Stenosis. By G. A. Gibson, M.D., D.Sc., F.R.C.P.Ed.

The interesting contributions of Professor Gairdner and Dr. Philip lead me to bring forward two observations on stenosis of the right auriculo-ventricular orifice, with the view of calling attention to some points in regard to the physical signs produced by this cardiac lesion, and more particularly to the characteristic phenomena revealed by auscultation.

The anatomical features of tricuspid stenosis have been known since the times of Morgagni, who described a case in which the lesion was discovered after death in association with a similar affection of the mitral orifice.

Corvisart carefully recorded the symptoms observed during life, and the lesions found after death in a case of combined tricuspid and mitral stenosis, and he further mentions a pathological specimen obtained from a patient with similar morbid changes, adding that he could have given several other instances of the same nature.

Horn shortly afterwards recorded a similar case in the person of a woman, æt. 25, whose heart was observed, post-mortem, to have obstruction of both right and left auriculo-ventricular orifices caused by valvular changes.

Burns, a year later, gave a full and clear account of the symptomatology and pathology of similar double lesions as they occurred in a young woman, 19 years of age.

Bertin describes an instance of stenosis of both right and left auriculo-ventricular orifices, and another in which the tricuspid orifice alone was the seat of obstruction. He further cites a case which had been recorded in *Le Journal de Médecine*, vol. xix. p. 468, by Corvisart, Leroux, and Boyer.

Bouillaud incorporated in his work the description of the case of the general officer, who died with symptoms of cardiac

disease, which was recorded by Corvisart, Leroux, and Boyer, and referred to by Bertin. The lesions found in this case after death were somewhat singular. The tricuspid valves were so united as to close the orifice with a diaphragm penetrated by three apertures, two of which opened from the auricle into the ventricle, while the third ended in the left ventricle. In this case the left side of the heart was otherwise in a normal condition.

Since the date of these observations many cases of tricuspid stenosis have been recorded, but comparatively few of them were recognised during life.

Before the date of Laennec's great discovery, Kreisig attempted to formulate rules by which tricuspid obstruction might be detected during life, and, after the introduction of auscultation, Hope drew up a clear statement of the local symptoms and physical signs, which he judged to be those which might be expected to occur in obstruction of the right auriculo-ventricular orifice.

In most of our systematic treatises, whether devoted specially to the diseases of the heart, or embracing the wider sphere of general medicine, the subject of tricuspid stenosis is dismissed with a few theoretical sentences. This is the case not only with the majority of our English works, but with those also by transatlantic and continental authors. A small number of writers, however, have laid before us some positive observations on this form of valvular disease, from which useful general principles as to diagnosis may be drawn.

The extremely interesting case which Professor Gairdner placed on record in 1862, and which he has fully described in the pages of the present volume, was the first instance of a diagnosis of obstruction of the right auriculo-ventricular orifice made during life, and verified by post-mortem examination. To some of the features of this case, reference will be made in the sequel.

The late Dr. Rutherford Haldane showed at a meeting of the Edinburgh Medico-Chirurgical Society, in 1864, two specimens of stenosis of the tricuspid orifice, in both of which mitral obstruction was also present. In one of these the patient during life presented no physical signs which could lead to a

diagnosis of the lesion, and in this respect it closely resembles the case which Dr. Philip has so clearly narrated in the present volume. In the other patient the lesions were recognised during life by the existence of an auricular-systolic, or pre-systolic, murmur heard at the apex, and another, of similar rhythm at the lower end of the sternum, between which points of maximum intensity the murmur was not so loud.

The late Professor Walshe would appear in his long experience only to have met with a single case in which this lesion probably existed, but in which there was no post-mortem examination, and he in consequence speaks very guardedly on the subject of diagnosis.

In the exhaustive work of Dr. Hayden, there is an account of a large number of cases recorded in the United Kingdom and America, including three observed by the author, two of which were recognised during life. In every one of these instances there was stenosis of the mitral as well as of the tricuspid orifice.

Dr. Bedford Fenwick, when showing two cases of tricuspid obstruction at the Pathological Society, more recently analysed the facts presented by 46 recorded instances. The most important conclusions to which he has been led are, that it is far more common in women than in men—41 of the 46 cases being of the female sex; that it is always associated with mitral stenosis; that there is an antecedent history of acute or subacute rheumatism in about 50 per cent. of the cases observed; and that, when found in adults, tricuspid stenosis is due to acquired disease, and very rarely, perhaps never, of congenital origin.

Down to the present time the case described by Bertin is the only one in which there was stenosis of the tricuspid orifice without coincident mitral obstruction.

The two cases which form the basis of this contribution came under my observation during the course of last year.

Isabella F., æt. 31, domestic servant, was admitted, on 1st April 1892, to Ward 25, then under my care, suffering from right-sided hemiplegia, with aphasia. Her father died at the age of 72, of bronchitis. Her mother, 65 years old, always enjoyed good health. She had three brothers and four sisters, who had

always been healthy. Her social surroundings were satisfactory. Ten years before she had suffered from a severe attack of acute rheumatism, since which time her health had not been so good as formerly.

The present attack began about nine months before admission. After undergoing some mental troubles, the patient suddenly found that she had lost the power of moving the right arm, and, on attempting to inform some one of the circumstance, discovered that she was unable to do so. She was told by another servant that her face was drawn to the left side. It was deemed advisable that she should go to bed, and, after betaking herself thither, the patient gradually lost the power of moving the right leg. After the lapse of a few weeks she partly regained the use of the leg and arm, and to a less extent that of the face, but speech was only restored in a very slight degree.

On examination it was found that the alimentary and hæmopoietic systems were in no way affected.

Inspection showed a faint double reflux — auricular- and ventricular-systolic in rhythm respectively—in the veins of the neck. The apex beat was very distinct on account of the thinness of the patient; it was situated in the sixth intercostal space, $4\frac{1}{4}$ inches from the middle line. There was some pulsation in the epigastrium.

On palpation it was noticed that there was at times a faint diffuse thrill immediately preceding the cardiac impulse. It was felt to be most distinct at the apex, but could be traced almost as far as the xiphoid cartilage. The radial pulse was of moderate fulness and tension; its rate was usually about 80, and its rhythm regular.

The cardiac dulness began above at the lower edge of the third left costal cartilage. The right border of the heart was $1\frac{1}{2}$, and the left $4\frac{1}{2}$ inches from the midsternal line.

At the apex there was a distinct, but not loud, murmur of presystolic rhythm and rough character, which was succeeded by a soft blowing systolic murmur, followed in its turn by a reduplicated second sound. When these murmurs were traced, it was found that the one of presystolic time could only be heard for about an inch to the left of the apex, but that it was

propagated as far as the ensiform cartilage, slightly diminishing in loudness for a certain distance, and, after that point had been passed, increasing in intensity and altering in character as the lower end of the sternum was approached. The systolic murmur was conducted as far as the axilla on the one hand, and to the xiphisternum on the other, but, as in the case of the presystolic murmur, there was a decrease of intensity on auscultating across the space from the apex to the sternum, followed by a gain in loudness when the ensiform process was reached. There were, in short, presystolic and systolic murmurs having maximum intensity both in the mitral and tricuspid areas. At the base of the heart the first sound was very feeble, and the second sound reduplicated, the pulmonary part of it being at once louder and later than the aortic. The position and extent of the abnormal sounds are shown on Fig. 1, in which the presystolic murmur is represented by vertical, and the systolic by horizontal lines.

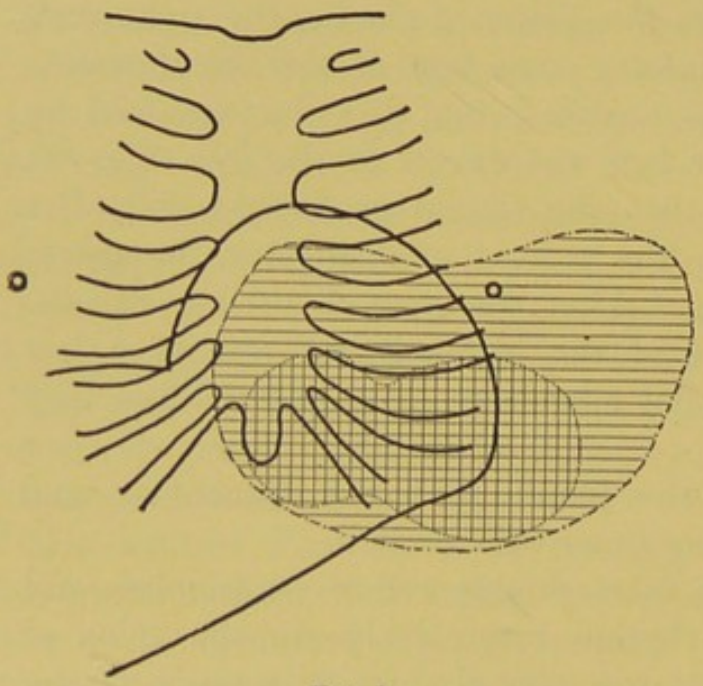

Fig. 1.

The respiratory system had but moderate deviations from the standard of healthy conditions, there being a little muffling of the percussion sound, a slight increase in the roughness of the respiratory murmur, and a few scattered crepitations over the whole of the thorax.

Neither the integumentary nor urinary functions were in any way affected.

With regard to the nervous system, it was found that there was no alteration in sensibility, ordinary or special. The right leg and arm were able to perform most movements, but in a weak and sluggish manner, and the toes and fingers moved

very imperfectly. The mouth was drawn slightly to the left side when the patient smiled, and the tongue tended towards the right on protrusion. The muscles of the right leg and arm were wasted, and the face on that side was rather expressionless. The right half of the entire muscular system showed diminished reaction both to galvanism and faradism. The temperature of the paralysed side was lower than that of the other. The plantar and other superficial reflexes were exaggerated on the right side of the body, and on that side there was great increase in the knee jerk, and a marked ankle clonus, as well as exaggeration of the elbow jerk and a distinct wrist clonus.

Even from the date of the paralytic seizure, the patient had been able to understand everything said in her presence, and to read with perfect ease; she had, in point of fact, spent most of her time in reading. At the time of the attack she was unable in any way to express her ideas, but she had gradually regained the power of saying a few words, and had taught herself to write answers to questions with her left hand.

The diagnosis arrived at in this very interesting case was stenosis of both auriculo-ventricular orifices, with incompetence of their respective valves, and embolism of a branch of the left middle cerebral artery, involving the motor tracts corresponding to the centres for the leg, arm, face, and speech.

After some gradual improvement in the general condition, and in the state of the nervous system, the patient's circulation showed symptoms of failure. The lungs became œdematous, and some anasarca of the lower extremities followed. Under appropriate treatment these symptoms, especially as regards the limbs, lessened to some extent, but on the 27th April death occurred suddenly from cardiac failure.

The post-mortem examination was performed on the day after death by Dr. William Russell. The following is the description of the morbid anatomy recorded in the books of the pathological department :—

External appearances.—Body spare. Rigor and lividity present. Face and neck, and upper part of thorax livid. No anasarca.

Thorax.—There were a few ounces of fluid in each pleural cavity. No adhesions were found. There were about two

ounces of clear fluid in the pericardium, and a patch of pericardial adhesion over the base of the right ventricle.

Heart.—There was a large and tough pinkish clot in the left ventricle, which extended into the aorta. There was a similar clot in the right ventricle extending into the pulmonary artery, accompanied by a little post-mortem clot in the same chamber. Diameters: aorta, ·97; pulmonary artery, 1·2; mitral, 1·1; tricuspid, 1. The aorta was normal, with the cusps slightly thickened, but not at all shrunken. Both cusps of the mitral valve were thickened and fibroid, as also were the chordæ tendineæ and apices of papillary muscles. From the anterior cusp there was suspended an irregular elongated mass of vegetation, which evidently hung free in the blood stream, and the tendinous cord with which it was united showed a small warty mass of vegetation. The auricular aspect of the posterior segment, and the auricular endocardium continuous with it was the seat of an extensive warty growth, covering an area about 1½ in. square. The left ventricle was dilated, and its muscle soft, pale, cloudy, and somewhat fatty. The left auricle was dilated and slightly thickened. The pulmonary artery was somewhat dilated. The right ventricle was elongated, but not much dilated; its wall was not hypertrophied, and its muscle was in the same condition as that of the left ventricle. The tricuspid valves were so united as to form a ring which admitted two fingers, forming stenosis. The ring was adherent to the endocardium ¾ of an inch below the pulmonary valves. The right auricle was much dilated, and the foramen ovale was closed. The heart weighed 1 lb. 6 oz.

Left lung weighed 1 lb. 4 oz., was emphysematous, anæmic, and œdematous.

Right lung weighed 1 lb. 15 oz. It was emphysematous, and showed slight chronic venous congestion, with œdema.

Head.—Brain weighed 3 lb. 2 oz., and was extremely pale and bloodless. A vertical section through the ascending parietal convolution, revealed in the parietal fasciculus above the island of Reil an area of brownish pigmentation, with a degree of softening which was no doubt due to embolism. The other parts of the brain and the upper part of the cord were normal to the naked eye. No embolism in large vessels.

Abdomen.—Liver weighed 4 lbs. 1 oz., was congested, and the margins of the lobules fatty. The gall bladder was œdematous, and contained a little greenish bile. Spleen weighed 1 lb. 2 oz. The capsule was tight and thickened. On section the organ was firm, the fibrous tissue was very prominent and thickened, and the Malpighian bodies were somewhat enlarged. Left kidney weighed 6½ oz. This was very anæmic and mottled with a diffused grey colour. The capsule was adherent, and the markings in cortex were obscured and broken up. Right kidney weighed 6½ oz.; in the same condition as its fellow.

In this case the diagnosis of the double cardiac lesion, which we were led to form in consequence of the distribution of the murmurs, was justified by the result of the post-mortem examination. Some points in the clinical features of the case will be adverted to in the sequel.

Marion R., æt. 21, machinist, presented herself as an out-patient on the 5th November 1892, complaining of pains in her thighs, and was sent by me to Ward 25, under the care of Professor Grainger Stewart.

Her father, æt. 62, had for some time suffered from asthma; her mother, æt. 58, was in good health. Two brothers and two sisters were in perfect health. One brother had died of peritonitis, and a sister of exophthalmic goitre. The patient had always been in comfortable circumstances. She had enjoyed good health throughout most of her life, but had once been in the Royal Infirmary for a few weeks, on account of hæmoptysis. The pains for which she sought advice had troubled her for three weeks. On making a routine examination of the physical condition of the patient, it was found that, with the exception of the circulatory organs, every system presented phenomena in all respects normal.

No symptoms were present that could be referred to the heart.

On inspection, a slight oscillation was seen in the veins of the neck, preceding in time the pulsation of the carotid arteries. The patient was somewhat plump, and no impulse of any kind could be seen in the præcordia.

Palpation determined that the apex beat was in the fifth intercostal space 3 in. from midsternum. It revealed in

addition another fact, viz. that there was a very distinct thrill, preceding the cardiac impulse, and not confined to the region of the apex, but felt widely spread in every direction round that area. The radial pulse, 76 per minute, was full, of moderate tension, and perfect regularity.

The upper limit of cardiac dulness was found to be at the level of the upper border of the third costal cartilage. The right border was 2½ and the left 3½ in. from the middle line.

On auscultation, a loud rough presystolic murmur was heard over an area larger than and embracing the whole of the præcordia. The point of maximum intensity was exactly at the junction of the fourth left costal cartilage with the sternum, and from this spot it was conducted in every direction to different distances. It was audible 3½ in. upwards, 3½ in. downwards, 4½ in. horizontally to the right, 5½ in. horizontally to the left, 4½ in. diagonally downwards and to the right, and 6 in. downwards to the left. Although the maximum intensity was at the junction of the fourth left costal cartilage with the sternum, there was very little change in loudness for about a couple of inches in every direction around this spot; the murmur, for instance, was almost equally loud at the ensiform process, and at the cardiac apex. There was absolutely no change in the character of the murmur when the mitral and tricuspid areas were carefully compared. The first sound was everywhere perfectly closed, the second sound was reduplicated, and the pulmonary element was both later in time and relatively louder than the aortic.

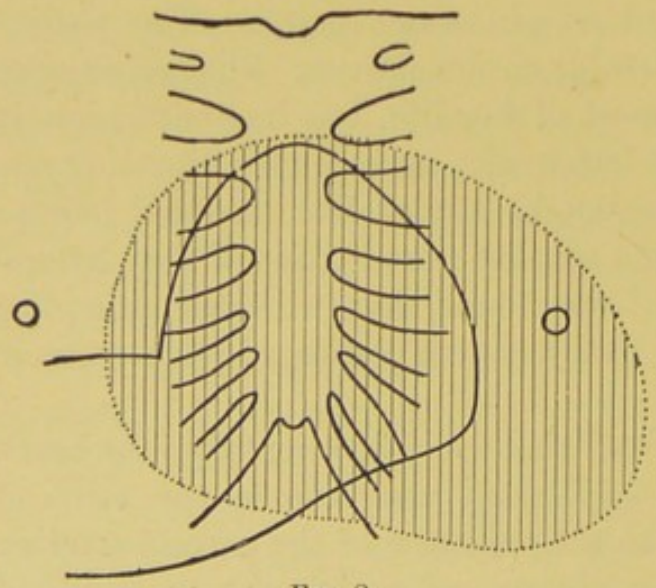

Fig. 2.

Now the results of auscultation in this case, which are shown in the tracing (Fig. 2), are not perhaps absolutely free from the possibility of different, even antagonistic,

interpretations. It may be held, on the one hand, that the presystolic murmur was entirely due to a stenosis of the tricuspid orifice, or, on the other, that it was caused solely by obstruction of the mitral orifice. It seems to me that the possibility of such a view as this latter hypothetical opinion may be at once dismissed, for if, with a presystolic murmur having its point of maximum intensity in the situation above described, there should be no tricuspid stenosis, "we shall," as Professor Gairdner has so well put it, on p. 229 of the present volume, "have to rewrite our whole cardiac diagnosis and pathology of murmurs; for it is impossible to find a stronger case than this for the *absolute* diagnosis of tricuspid obstruction."

Dr. Hayden, in his remarks on tricuspid obstruction, previously referred to, mentions a case in which he had diagnosed aortic and mitral obstruction. These lesions were found on post-mortem examination, but there was also tricuspid stenosis which had not been diagnosed. The author reproduces some observations which he made at the Dublin Pathological Society, which may well be quoted here. He remarked that "diagnostically the case is of considerable interest. It is perfectly novel to me, and, with the light it affords, I should have no difficulty in diagnosing, in a similar case, the existence of constriction of the two auriculo-ventricular openings. The point on which the diagnosis turns is this, that whereas the murmur of mitral constriction is always at the apex of the heart, and, in the great majority of cases, strictly limited to the area of the mitral opening, in this case a murmur of the same rhythm was audible to the left of the sternum. Between these two points there was a portion of the chest over which no murmur was distinctly audible." Profiting by the experience thus gained, the author just quoted was able, as we saw previously, to diagnose during life and verify after death two later cases of tricuspid stenosis.

Dr. George W. Balfour speaks of a boy who, he says, "had a presystolic murmur so loud and rough that I have selected it as a measure of the extent to which such murmurs could be propagated. In mapping out the propagation of his murmur, I found it to extend so much further to the right than usual

that the thought struck me, Is it possible that we can have in this case not only a mitral but also a tricuspid stenosis? But I dismissed the idea as in the highest degree improbable, and referred the great propagation to the loudness and roughness of the mitral murmur. The result shows that in this I was mistaken, though unquestionably there were no other symptoms present but the excessive propagation of the murmur which could countenance the former idea." In this case considerable stenosis of both mitral and tricuspid orifices was found on post-mortem examination. The result of this case was such as to lead Dr. Balfour to diagnose in a similar case combined mitral and tricuspid stenosis, but, as down to the time of the publication of the second edition of his work the patient was alive, the diagnosis was not absolutely confirmed.

The results of such valuable experience seem to me absolutely conclusive with reference to the case of Marion R., in so far as concerns the diagnosis of tricuspid stenosis. But with a presystolic murmur of practically the same intensity at the apex as at the sternum it is impossible to avoid the conclusion that there was also in this patient an obstruction of the mitral orifice. To doubt this is to fly in the face of every principle of the physical diagnosis of cardiac valvular lesions, and the case was therefore assumed to be one of stenosis of both auriculo-ventricular orifices.

No doubt another consideration tends to support the view that in this case there is a double lesion. If it be diagnosed as at any rate one of tricuspid stenosis, the extreme rarity of this lesion, apart from coincident mitral obstruction, is a strong argument, when unopposed by the facts of physical diagnosis, in favour of combined mitral and tricuspid obstruction.

With reference to this very interesting case, one final remark must be made. It will be remembered that, with the sole exception of an attack of hæmoptysis on a former occasion when she was an inmate of the Royal Infirmary, her health has always been good. She has, in particular, never had a rheumatic symptom, unless the pains for which she presented herself in November are of this nature. It appears to me as in the highest degree probable that the valvular lesions are congenital. Two of the greatest authorities on cardiac disease,

Pollock and Rosenstein, believe that the majority of cases of tricuspid obstruction occur as the result of intrauterine causes, and, although one half of the cases collected by the industry of Fenwick do not support their views, there are many other facts which lend aid to them.

From the total absence of any symptoms, the stenosis may probably be caused by some roughening of the auricular surfaces of the auriculo-ventricular valves, without any considerable narrowing of the orifice, and there cannot be any interference with the closure of the valves, as there is no evidence of regurgitation.

The two cases which have been narrated in the foregoing pages offer in certain respects a marked contrast. In the first, there was not only incompetence of both auriculo-ventricular valves, shown by the distinct murmurs of systolic rhythm in the tricuspid as well as mitral area, but an easy means of differentiation of the mitral and tricuspid presystolic murmur was afforded by their separate points of maximum intensity. This case also differed from the second in the presence of some definite results of valvular disease, *i.e.* the venous congestion of the lungs, and the œdema of the lower extremities. The second case, as we have seen, presented considerable difficulty on account of the maximum intensity of what almost seemed to be a single presystolic murmur, and this murmur was absolutely free from anything tending to point in the direction of regurgitation.

The last point to which reference need be made in connection with these interesting cases is to recommend the adoption of such a system of mapping the exact area over which murmurs are heard, as well as the limits of the cardiac dulness. If this be done on the præcordia, an exact copy may be obtained by tracing the lines upon transparent paper, and by photography a reduction can be produced on a uniform scale. The two figures which accompany this paper have been reduced in this way on the same scale.

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