THE DIAGNOSTIC SIGNS OF INCOMPETENCE OF THE PULMONARY VALVE.

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Lesions of the pulmonary valve have been recognised anatomically since the description of an instance of this affection by Morgagni, but the clinical features presented by diseases of the orifice and cusps were not elucidated until long afterwards. Hope laid down rules for the recognition of pulmonary stenosis and incompetence in the first edition of his work, but does not seem by that time to have met with a case of the latter lesion. Perhaps the earliest instance on record of pulmonary regurgitation is that mentioned by Chevers, which was not, however, diagnosed during life. In the fourth edition of his work, Hope describes a most interesting case, in which, with other lesions, pulmonary incompetence was a prominent feature. Although a large number of cases have been placed on record since the date of these observations, the detection of pulmonary incompetence is still surrounded by serious difficulties, some of which it is the aim of this paper to consider. In the following pages it is not my intention to enter upon the historical aspect of the subject. This has been rendered unnecessary, seeing that, in one of the most valuable contributions to the study of cardiac disease during recent years, Barié has not only followed the development of our knowledge of this particular lesion, but has also given a brief statement of the facts recorded in 58 cases, two of which were observed by himself. His memoir on this subject will, for future workers, remain a storehouse of clinical and pathological facts, for which he deserves the cordial thanks of all interested in diseases of the circulation.

Several cases, more or less related to the subject of this paper, have lately come under my notice, and it will facilitate the discussion of the points which will arise, if, before attempting to enter upon their consideration, we cast a brief glance at their clinical and pathological features.

CASE 1.—Charles W., baker, æt. 45, married; was admitted to Ward 22 of the Royal Infirmary on 15th May 1893, complaining of breathlessness, and swelling of the feet and legs.

His father was 70 years old, and in good health. His mother had died, at the age of 64, of bronchitis. Of three brothers, one was in good health, another had died in infancy, and the remaining one had been killed by an accident. Of five sisters, four were alive and well, but one had died in infancy.

The patient's social conditions had always been fairly good.

He had in early years enjoyed very good health; but latterly, in consequence of three attacks of acute rheumatism, he had never felt well.

The illness for which he sought admission began about four months previous to his entrance into the hospital, with breathlessness, which was followed by the development of swelling of the lower limbs.

The patient was pale, with bloodless lips and gums, but the pallor was to some extent masked by a certain degree of cyanosis. There was considerable œdema of the lower limbs. The fingers, as well as the toes, were clubbed at their extremities, and the nails were distinctly arched.

The appetite was small and capricious. Constipation and diarrhœa frequently alternated. The teeth were much decayed; and the tongue, which was pale, was covered by a slight fur. The abdomen showed no ascites. The liver extended 6 in. in the mammary line.

The hæmopoietic system presented no abnormal features.

The radial arteries were soft and elastic, the vessels were rather empty, the tension was low; the rate of the pulse was from 70 to 80, it was perfectly regular, each pulse wave was of large size and short duration,—in short, the patient had a typical Corrigan's pulse. On producing a flush over the forehead by friction, it was seen to become paler and redder with the alternate systolic and diastolic phases.

On inspection of the neck, a violent pulsation was seen in the carotid arteries, and some undulation in the external jugular veins, while over great part of the præcordia there was an extremely distinct systolic impulse, which gave to the hand, however, the sensation of short duration. The apex beat was felt in the sixth intercostal space, 4 in. from the midsternum. No thrill could be detected on palpation. The deep cardiac dulness in the left parasternal line began in the second intercostal space, nearer the second than the third cartilage; it extended, at the level of the fourth cartilage, from $2\frac{1}{4}$ in. to the right to 4 in. to the left of the midsternal line. On auscultation, two murmurs were heard: one accompanied the first, the other replaced the second sound. Both of these murmurs were loudest at the base; the systolic, which was loud and rough, having its point of maximum intensity at the junction of the third left costal cartilage with the sternum; while the diastolic, which was quite as loud, though somewhat less rough, had its point of maximum intensity over the third left costal cartilage and the adjacent part of the fourth intercostal space, $1\frac{1}{2}$ in. from midsternum. Both murmurs were heard with diminishing intensity over the entire præcordia,—the systolic extending further downwards and to the left; the diastolic slightly further upwards and to the right. A slightly increased intensity of the systolic murmur could be discovered both in mitral and tricuspid areas, and a distinct systolic murmur was heard in the carotid arteries.

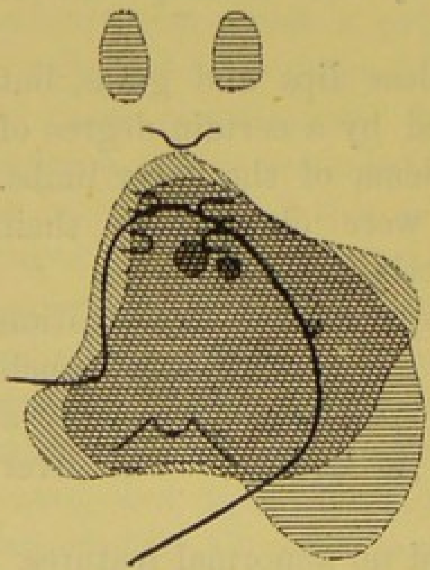

Fig. 1.

The distribution of these murmurs, as well as the area of cardiac and hepatic dulness, is seen in Fig. 1., the method of producing which was described in the volume of the *Edinburgh Hospital Reports* for last year. In these figures presystolic murmurs are shaded vertically, systolic horizontally, and diastolic diagonally. The points of maximum intensity are marked by the darker lines within the circles.

The patient was very breathless, and coughed a good deal, the cough being accompanied by a frothy serous expectoration. The only physical sign connected with the respiratory system was a rather coarse crepitant râle over the bases of both lungs.

The urine, which was small in amount, contained a trace of albumin.

The patient was restless and sleepless, but those symptoms were in great part due to the dyspnœa.

The diagnosis in this case presented, in one respect, some difficulty. There could be no doubt that there was stenosis of the aortic orifice; the propagation of a systolic murmur up the carotid arteries was sufficient proof of this, and there could also be just as little question in regard to the fact of aortic incompetence, since there was the characteristic water-hammer pulse, and the significant capillary pulsation. The considerable hypertrophy of the left ventricle also supported this. The point around which some difficulty hung was with regard to the condition of the pulmonary orifice and its valves. The position of the maximum intensity of the murmurs seemed to indicate a probability that both stenosis and incompetence existed, and this seemed the more likely, seeing that there was some cyanosis, with distinct clubbing of the fingers and toes. On the other hand, the small degree of cyanosis might have been the result simply of cardiac failure, and if this had recurred from time to time, it might also have given rise to the change in the ends of the phalanges. It was my opinion that, while the diagnosis of aortic stenosis and incompetence was absolutely established, and while admitting the probability of a pulmonary lesion, it was better to leave it an open question whether such an affection was present. It seemed to me, however, extremely likely that, if the pulmonary valves were healthy, there was such a pulmonary regurgitation, as will be described in the sequel, produced by a functional inability of the pulmonic cusps to close completely, in consequence of the high pressure within the pulmonary circuit.

In spite of every method of treatment, the patient became steadily worse. The dependent parts developed more œdema; ascites and hydrothorax came on; the urine fell off in quantity, and contained more albumin; and the dyspnœa and cyanosis increased. Death occurred quietly on 7th July.

The post-mortem examination was performed the following day by Dr. Leith, whose notes, for which my thanks are due to Mr. J. Stuart Gallie, are appended.

External appearances.—Rigidity absent. Lividity well marked. There is considerable dropsy.

Thorax.—There are extensive old adhesions in both pleural sacs.

Heart.—The pericardium is adherent all over; the adhesions are slender, and easily broken through. They are extensive both in front and behind, chiefly consisting of fine threads attaching the two surfaces together. There are two large irregular nodulated masses of osseous consistence, one to the right of the pulmonary artery, which is the larger, about the size of a hazel nut; the other to the left of the pulmonary artery. Both are very irregular in shape, very jagged and nodular on their external surfaces, and lie in both layers of the pericardium, the two layers being here inseparable.

Pulmonary valves are incompetent.
Aortic „ „
Cone diameter of pulmonary = ·85.
„ „ aortic = ·6.

All the pulmonary cusps are thickened. This is especially marked at their attachments, where it is almost cartilaginous in consistence. The lunule also shows considerable thickness, although it is not so pronounced. Their attachment to the artery is also thickened. The condition is one of well-marked chronic endocarditis. There are some recent vegetations at junction of anterior and right posterior cusps.

At the right margin of the right posterior aortic cusp there is an irregular perforation larger than a crow quill;

the margins are irregular, and stained pinkish. Above it, and slightly to the right, there is a small aperture in the commencement of the intima of the aorta, *i.e.* just at the upper part of the sinus of Valsalva. This aperture leads into an aneurismal sac a little to the right, about the size of a bean.

The left ventricle is considerably dilated, its wall being but slightly thickened. The muscle is somewhat paler than usual, and, besides some fatty change, is probably the seat of a chronic endocarditis.

There is some fatty change in the wall of the right ventricle, and its muscular substance is somewhat hypertrophied.

The cone diameter of the tricuspid orifice is 1·3. The cusps are slightly thickened, but uniformly so. The cavity of the right auricle is dilated, and its walls a little hypertrophied. The cone diameter of the mitral orifice is ·88. The cusps are chronically thickened, the anterior markedly so. The thickening is not uniform. It is greatest at the contiguous margins of the valves. The wall of the left auricle is hypertrophied, but the cavity is not dilated.

Heart, with pericardium, weighs 1 lb. 10 oz.

Lungs.—Left weighs 1 lb. 15 oz. There are old pleuritic patches over both lobes and between them. There is marked œdema in both lobes, and brown induration in the lower lobe. Right weighs 1 lb. 14 oz.; shows old pleurisy at base, apex, and between lobes. There is œdema and congestion of all the lobes.

Liver weighs 3 lb. 6 oz. Gall bladder contains blackish green bile, somewhat inspissated. The liver substance is firm. The surface is granular towards the anterior margin. On section, there is a nutmeg condition present, considerable fatty change of the peripheral portion of the lobules, but not much bile staining.

Spleen weighs 14 oz.; shows some perisplenitis. It is large and firm. On section, the Malpighian bodies are distinct, and stand out as white points upon a dark purple. There is no reaction with iodine.

Kidneys.—Left weighs 7½ oz., and is firm. On section, it is pale. The superficial cortex shows irregular areas, and is diminished in places. There is a cyst about the size of a pea in it. The capsule strips off badly. There is some superficial cirrhosis and some slight waxy change. Right weighs 6½ oz. It resembles its fellow.

Stomach.—The stomach contains some brownish fluid, and shows a chronic catarrh, but is otherwise healthy.

The result of the post-mortem examination in this case shows that it is possible to be too cautious in the diagnosis of rare lesions.

Another patient presenting many features similar to those just described was in the hospital about the same time.

Esther C., æt. 46, housewife; was admitted to Ward 25 of the Royal Infirmary on 3rd July 1893, suffering from dropsy and dyspnœa.

The patient was so ill that it was not possible to disturb her by any prolonged examination, but it was ascertained that she had suffered more than once from acute rheumatism, and that four years before admission she had been attacked by hemiplegia of the right side.

The patient was deeply cyanosed, and could only breathe when sitting upright in bed. The fingers and toes were most distinctly clubbed, and there was much arching of the nails. The right side of the face was rather vacant in expression, and its muscles were weak, while the right arm and leg were almost powerless. The feet, legs, and thighs were very œdematous, and there was some ascites.

The pulse was irregular, and did not give any definite indications, although the pulse wave was of brief duration. The cardiac impulse was feeble, but the apex beat was felt in the sixth intercostal space. No thrill could be discovered. On account of the patient's condition, it was not considered right to trouble her by percussing the præcordia, and the size of the heart was not therefore ascertained. For the same reason, it was not possible to study the auscultatory phenomena so fully as we could have wished, but the following facts were definitely established. At the apex of the heart there was a rough first sound, accompanied by a blowing systolic murmur propagated into the axilla, and also, but to a less degree, towards the sternum. At the base a rough systolic was followed by a softer, although still somewhat harsh, diastolic murmur. Both of these murmurs were heard over the whole of the upper part of the chest, but their maximum intensity was to the left of the sternum, in the neighbourhood of the third costal cartilage. The diastolic murmur, in particular, had its point of maximum loudness in the third left intercostal space, about an inch to the left of the external edge. The systolic murmur was distinctly propagated by the carotid arteries.

The respiratory system showed symptoms and physical signs of hydrothorax and œdema of the lungs.

The urine was scanty, and contained albumin.

The patient had almost complete paralysis of the right leg and arm, with weakness of the facial muscles of the right side, and on the same side there was a considerable degree of wasting of the muscles, with contracture.

In this case the original clinical features were to a great extent modified by the obvious failure of the cardiac muscle. It could not be doubted that there was a double lesion at the mitral orifice, producing stenosis and incompetence. It was clear also that there was aortic stenosis, and very probably incompetence as well. But, as has been noted, the maximum intensity of the diastolic, as also of the systolic murmur, was to the left of the left sternal border, which fact, taken along with the clubbing of the fingers and toes, together with the profound respiratory disturbance, caused some doubt whether there might not be some lesion of the pulmonary valve or orifice. The conclusion arrived at, however, was that, in consequence of the backward pressure resulting from the mitral lesion, the clubbing of the fingers had resulted, while the combined effect of the mitral and aortic affections had been to cause the serious condition of cardiac failure.

In spite of all treatment, the state of the patient became gradually worse, and she died somewhat suddenly on the 12th July. The post-mortem examination was performed on the 14th July by Dr. Muir, whose notes, for which my thanks are also due to Mr. Stuart Gallie, are appended.

External appearances.—Body well nourished. Some dropsy of lower limbs. Rigidity well marked.

Thorax.—Left pleura, 24 oz.; serous fluid, with fibrous adhesions here and there. Right pleura about 15 oz. Pericardium contains about 3 oz. serum.

Heart is considerably enlarged on both sides. The right auricle is somewhat dilated, and filled with dark clot. Aortic orifice distinctly incompetent. Pulmonary orifice competent.

Aortic = ·65 (stenosed).		Pulmonary = 1.
Mitral = ·7	„	Tricuspid = 1·7.

The aortic cusps show extensive chronic vegetative endocarditis—(*a*) The two posterior cusps are much thickened at their junction, and also calcareous. The margins are also thickened. (*b*) The anterior cusp is considerably ulcerated, and there are large irregular vegetations hanging down from it.

The orifice is stenosed from the chronic endocarditis, and also markedly incompetent.

The mitral segments are much thickened and indented, and have become adherent for a considerable distance, giving rise to stenosis. The chordæ tendineæ are also thickened and contracted somewhat, as are also the apices of the papillary muscles. There is also evidence of more recent endocarditis along the margins of the segments.

The tricuspid and pulmonary valves are normal.

Left ventricle, 4 in. in length by $\frac{5}{8}$ to $\frac{3}{4}$ in. in thickness; slightly dilated and hypertrophied. Right ventricle is much hypertrophied; its walls measure $\frac{5}{16}$ in. in thickness.

The inner surface of both ventricles shows commencing fatty change in the myocardium. No ante-mortem thrombi.

Lungs.—Right, 1 lb. 12 oz.; shows chronic venous congestion. The bronchi are deeply congested, and contain frothy secretion. The posterior part of lower lobe is going on to hypostatic pneumonia. There is also slight interstitial change in places. Left, 1 lb. 8 oz.; at the posterior part about middle line there is anasarca, in which part the bronchi are considerably dilated, with considerable interstitial change around. Some of the bronchi contain a purulent fluid. There is a similar patch in the lower lobe. The general condition resembles that of the other lung.

Abdomen.—Peritoneum contains about 40 oz. of serous fluid.

Liver.—2 lb. 10 oz.; shows chronic venous congestion. The tissue is somewhat indurated, and the capsule at places is thickened.

Spleen.—10 oz.; shows typical chronic venous congestion.

Kidneys, each 7 oz.; they show fœtal lobulation, and also several irregular depressions owing to old infarcts. The infarcts have been in both kidneys, but are more numerous in the left.

The other organs show chronic venous congestion. There are no recent infarcts.

Brain.—There is nothing abnormal to see on the surface, and the vessels at the base are practically normal; none appear to have been the site of embolism.

On opening the brain, there is found an old area of softening in the left side, which implicates the following structures. At its lowest level, the posterior part of the lenticular nucleus, the external capsule and the claustrum, with the convolution (in part) of the island of Reil, are softened. The posterior end of the internal capsule is also affected, and there are one or two minute points of softening in the optic thalamus.

Higher up, the lenticular nucleus is more destroyed, but the internal capsule is practically unaffected. The softening runs up for some distance along its outer aspect.

The convolutions (with the exceptions mentioned) are unaffected. No change could be seen on naked-eye examination in the pons or medulla.

In this second case, so closely resembling the first in many of its features, the pulmonary orifice and valve were perfectly healthy; the two cases in this respect standing in strong contrast to each other.

The consideration of the clinical and pathological facts presented by the two patients whose cases have been described, leads to the conclusion that the mere position of the maximum intensity of murmurs in itself may lead to erroneous conclusions; and that even when definite appearances, strongly suggestive of pulmonary lesions, are present, they may be susceptible of other explanations.

That the murmurs produced at the aortic orifice may be loudest in, or even to the left of, the pulmonary area, is a well-known fact. As one of several patients presenting this feature, which have come under my notice, the clinical features of the following case may be shortly described:—

Case 3.—Bella W., æt. 19, domestic servant; presented herself as an out-patient at the Royal Infirmary on 29th May 1893, complaining of a rash on her skin.

Her family history was in every respect satisfactory, and her surroundings had always been favourable.

She had suffered from acute rheumatism some years ago, but had otherwise enjoyed good health.

The affection of the skin—well-marked psoriasis—had for some months been gradually increasing; with the exception of slight uneasiness in the præcordia, it was the only symptom of which she complained.

The alimentary and hæmopoietic systems showed no abnormal phenomena. The pulse was usually about 80 per minute, and perfectly regular; the vessel was somewhat empty, and the tension low; the pulse wave was large and bounding, but short and collapsing,—in a word, it was a well-marked Corrigan's pulse. The arteries of the neck pulsated rather strongly. Inspection of the præcordia showed no abnormality, and on palpation the apex beat could be felt beating strongly in the fifth intercostal space. No thrill was present: the deep cardiac dulness extended from $2\frac{1}{2}$ in. to the right, to $2\frac{1}{2}$ in. to the left of the mid-sternal line. On auscultation, two very distinct murmurs were heard, systolic and diastolic in time. The systolic murmur was loudest at the junction of the second left costal cartilage with the sternum, and was propagated up the neck, out to the shoulders, and around the thorax on both sides to the vertebral column behind. The diastolic murmur had its maximum intensity in the second left intercostal space rather more than $1\frac{1}{2}$ in. to the left of the sternal border, and it was conducted to the upper end of the manubrium sterni, along the clavicles, and over most of the præcordia. The distribution of these murmurs is shown in Fig. 2.

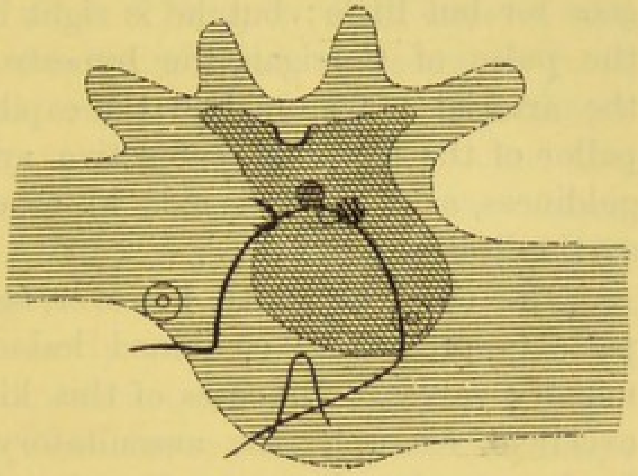

Fig. 2.

There was no clubbing of the fingers, or any symptoms of backward pressure; in fact, no other point of clinical importance, except the psoriasis.

This case shows very well how, in an undoubted instance of aortic disease, the auscultatory phenomena may be most distinct in the pulmonary area. Such instances might be multiplied almost indefinitely, were it necessary to do so.

The consideration of these three cases shows how difficult it may be to arrive at a definite diagnosis in doubtful instances of pulmonary disease. In the thoughtful paper already referred to, Barié, in analysing the cases he has reviewed, fully acknowledges this difficulty. "Le diagnostic," he says, "de l'insuffisance des valvules de l'artère pulmonaire est difficile; dans 7 cas seulement, justifiés par l'autopsie, la maladie fut reconnue; dans trois autres faits, également vérifiés a l'ampithéâtre, le diagnostic fut discuté et regardé comme extrêmement probable, mais, vu la rareté grande de la maladie, on n'osa point porter un diagnostic ferme; enfin dans un dernier groupe de 8 observations, les symptômes cliniques furent assez nets pour autoriser le diagnostic d'insuffisance de l'artère pulmonaire: mais les malades n'ayant pas succombé, la preuve anatomique reste encore à établir." Barié points out that cases of pulmonary incompetence have most frequently been taken for instances of aortic valvular disease or aortic aneurism.

Dismissing aneurism of the arch of the aorta, which should hardly be a real cause of dubiety, we may briefly consider the difficulty of distinguishing cases of aortic valvular disease from affections of the pulmonary orifice. Barié is a little too sanguine as to the diagnosis of these affections by the results of auscultation, for, as shown in Cases 2 and 3, the mere position of the greatest loudness of the murmurs goes for but little; but he is right in strongly insisting upon the pulse of Corrigan, the hypertrophy of the left ventricle, the arterial murmurs, and the capillary pulsation, along with pallor of the face, and subjective symptoms like angina and giddiness, as the great aids in establishing the diagnosis of aortic disease.

The chief difficulty, however, seems to me to be found in patients presenting combined lesions of the aortic and pulmonary valves. In cases of this kind, of which Case 1 is an excellent example, the auscultatory phenomena may be all in favour of pulmonary disease, while many physical signs pathognomonic of aortic disease may be present. We may be able to fall back upon the secondary results of lesions of the pulmonary valve in such cases, but even these are somewhat indefinite, from the fact that they may be counterfeited by the results of cardiac failure. The dyspnœa, œdema of lungs, hydrothorax, and enlargement of the right side of the heart, may be produced by simple cardiac failure, and the clubbing of the fingers may result from old standing backward pressure in consequence of a mitral lesion. To this may be added that the thrill over the præcordia, and the turgescence or pulsation of the cervical veins, sometimes found in pulmonary cases, may be absent.

It has long seemed to me to be probable that regurgitation is possible at the pulmonary orifice without any lesion of the valve. In a contribution made by me to the study of affections of the right side of the heart some years ago, the conclusions arrived at from some experiments on the pulmonary valves of the dead heart were given, which may be quoted here. As the average result of a series of experiments made with a column of fluid, it was there stated "that from the semilunar valves of the pulmonary artery of the sheep a strong jet escaped until the column of fluid reached $14\frac{1}{2}$ in., from which height it trickled until the valves became competent with a column of 9 in. In the ox a strong jet was emitted down to 12 in., and dropping of the fluid reduced the superincumbent column to the height of 6 in., when competence was established. In the healthy human heart a jet escaped down to 13 in., and the valves were competent with 8 in. of fluid resting on them. Now, in each case of the pulmonary valves, with a column of fluid exceeding 6 ft. in height, perfect competence was obtained in a very simple manner by constricting the pulmonary artery. A cord tied round the artery, exactly at the attachment of the valves, gave the means of perfect control over the escape, so that, by varying the amount of tightening, the jet was converted into a drop falling quickly or slowly, and this in turn was totally stopped. The whole diminution of circumference only amounted to a few lines. This shows clearly that the escape is caused by distension of the elastic artery, and relative incompetence of the valves." It may be added that, tested in the same way, it was found that the aortic valves never allowed any escape, and the paper referred to goes on to say: "Such experiments show with certainty that the orifices of the right side of the heart are not closed so perfectly as those of the left, and point to the ease with which they probably allow escape with any supernormal pressure."

Although these observations convinced me that there was probably some escape at the pulmonary valve, whenever there was any considerable increase of pressure in the pulmonary artery, no case came under my notice until comparatively recent years, in which there was absolute evidence in proof of the correctness of the view based on them; but in the interval other observers, who have successfully devoted much attention to the diseases of the circulation, have independently advanced the opinion that a diastolic murmur occurs in consequence of high pressure in the pulmonary artery.

In his little work on the heart, Graham Steell, after referring to the diastolic murmur of aortic dilatation, says: "I am inclined to believe that a murmur of similar mechanism occurs on the right side of the heart, when there is much obstruction to the pulmonary circulation with a dilated pulmonary artery."

Duckworth described a case of mitral and tricuspid stenosis, in which there was a temporary pulmonary diastolic murmur, which he considered as due to the dilated state of the vessel. It disappeared before death, and at the autopsy the pulmonary cusps were not found to be markedly altered.

The whole subject was again reviewed more recently by Graham Steell, who proposes to call this diastolic murmur, occurring independently of disease or deformity of the valves, "the murmur of high pressure in the pulmonary artery."

Within the last few weeks these views receive emphatic support from some remarks by Barr on mitral stenosis, in which, writing on the obstructive diastolic murmur of mitral stenosis, he says: "I am convinced that it is often confounded with a short, soft-blowing diastolic murmur, which not infrequently occurs in this disease at the pulmonic valve, and which arises from slight regurgitation into the right ventricle, owing to the high pulmonic tension."

Before making any further remarks on this murmur, it will be helpful to describe its mode of occurrence, as observed in a recent case:—

Case 4.—Maggie G., æt. 18, engaged in household duties, was admitted to Ward 25 of the Royal Infirmary on 5th June 1893, complaining of pains in her wrists and elbows.

Her father and mother, both aged 42, had always been in good health. She had four brothers and one sister, all very strong, but three brothers had died in infancy. The patient's social surroundings had always been good.

She had never been very robust, and four years before admission had suffered from a rheumatic attack, since when she had never felt quite well. About four months before entering the hospital, pains had begun in the joints, and had persisted ever since.

On her admission, the patient was found to be somewhat pale, with a bright spot on each cheek. The skin was moist. The tongue was slightly furred, but the digestive system was otherwise healthy. There was no symptom connected with the hæmopoietic system.

She complained of some palpitation and a slight degree of breathlessness. The pulse was of low tension and moderate volume, perfectly regular, and varying in rate from 80 to

90. There was some pulsation in the veins of the neck, and a very distinct impulse in the second left intercostal space. On palpation, the apex beat was found to be in the fifth left intercostal space, 3¼ in. from midsternum. The pulsation, systolic in time, in the second left intercostal space, was found to be most distinct 1¼ in. from the midsternal line. A tracing obtained from it by means of a revolving cylinder is given in

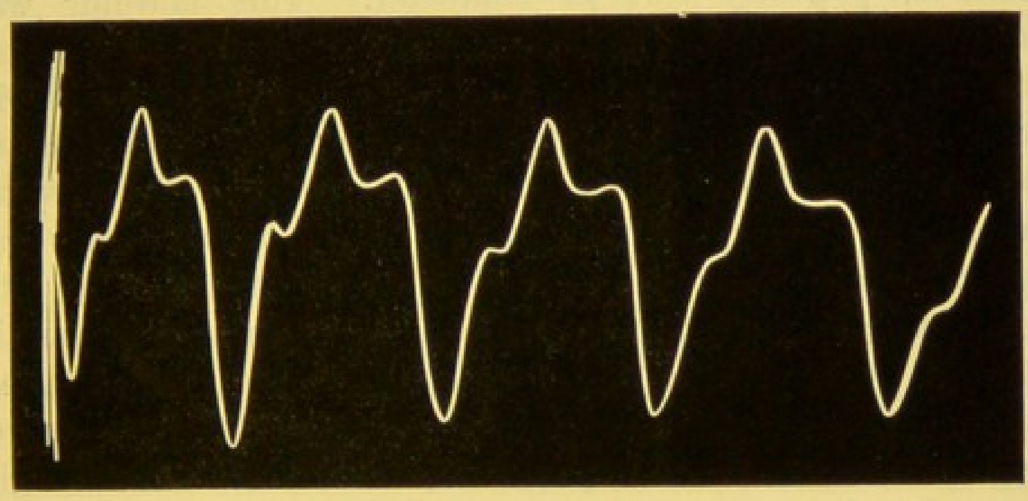

Fig. 3.

the accompanying figure (Fig. 3). No thrill could be detected over any part of the præcordia. The cardiac dulness extended to 1 in. to the right and 4 in. to the left of the middle line at the level of the fourth rib. On auscultation, a venous hum was heard in the neck, and there were murmurs, systolic in rhythm, over the whole præcordia, which, on careful analysis, proved to be twofold. Around the region of the apex beat, and with its maximum loudness in the fourth interspace 3½ in. from midsternum, there was a harsh blowing systolic murmur, conducted as far as the edge of the sternum to the right, and beyond the anterior axillary line to the left. Over almost the entire sternal region there was a soft blowing systolic murmur, quite different in character from that heard at the apex. It had the same tone throughout the whole sternal region, but it seemed to have two points of maximum intensity; to be more exact, it was loudest in the pulmonary region, exactly over the area of pulsation, from which point it waned in its intensity in every direction until near the lower end of the sternum, when it became louder, again culminating at the point where the left side of the sternum was joined by the sixth costal cartilage; but in this situation the murmur was not quite so loud as over the area of pulsation in the pulmonary region. The second sound was frequently reduplicated, and the later of the two second sounds, which could by auscultation be determined to be that due to the pulmonic cusps, was instantly followed by a short, sharp, high-pitched murmur, perfectly soft in character. This murmur was extremely restricted in its distribution, being only heard over a small triangular area, 2¼ in. in vertical, and 2 in. in horizontal measurement, extending along the left border of the sternum, from the lower border of the third costal cartilage to the upper border of the fifth. This murmur was perfectly soft in character, and was absolutely unlike the obstructive diastolic murmur which is found in mitral stenosis. It could not be due to aortic disease, of which there was no indication, and it could only, therefore, be a murmur of regurgitation, from the pulmonary artery into the right ventricle, due to the increased pressure and consequent dilatation of the orifice, with relative and transient incompetence of the cusps. All these murmurs are shown in Fig. 4.

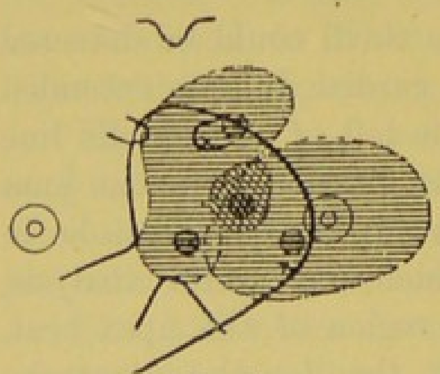

Fig. 4.

The other systems presented no symptom of disease, with the sole exception of a few crepitations at the bases of both lungs.

The diagnosis was cardiac dilatation, with mitral and tricuspid regurgitation, produced by the febrile affection, but it was considered probable that some stenosis of the mitral orifice might be insidiously progressing, although this was a mere supposition, not based on any direct evidence. The crepitations at the bases of the lungs were regarded as the expression of passive congestion from mitral incompetence; and the diastolic murmur was assumed to be one of pulmonary escape, in consequence of the strain on the artery from the high pressure within it.

By means of salol and similar remedies, the patient was relieved of her rheumatic symptoms, and the administration of iron with other tonics greatly improved the cardiac condition. The diastolic murmur disappeared, and the lungs cleared up, but at the time of the patient's departure from the Royal Infirmary, on 17th July 1893, she still had the systolic murmurs, and the pulsation in the second intercostal space. She presented herself at the hospital on the 2nd March 1894, when the diastolic murmur was found to be still absent, but the systolic murmurs were present as before. The first sound in the mitral area, preceding the systolic murmur, was, however, loud and clanging in character, which seemed to bear out the view that a stenosis of the mitral orifice was gradually and insidiously developing.

Cases closely resembling this might be repeated in not inconsiderable numbers from the facts observed and recorded by myself, but it is not necessary to describe any others on this occasion. That which has been chosen was selected on account of the absence of such disturbing elements as the physical signs of mitral stenosis, and it may be said in passing, that the diastolic pulmonary murmur is by no means confined to cases of mitral obstruction, but occurs also in many instances of simple cardiac debility, with regurgitation at the left auriculo-ventricular orifice.

This diastolic pulmonary murmur, occurring without any lesion of the cusps, is, in my opinion, the possible explanation of the position of the maximum intensity of the diastolic murmur in such cases as have physical signs resembling those of organic pulmonary incompetence.

For the opportunity of observing and recording these cases I am indebted to Professor Sir Thomas Grainger Stewart, to whom it is a pleasure to return my thanks for the obligations under which he has thus placed me.

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