removed, and, all being dry, the abdominal wound was closed with silkworm-gut sutures in the usual manner, the wound dressed with sublimate gauze, and a large pad of wool and a many-tailed bandage firmly applied. The patient was then returned to bed.

The patient bore the operation exceedingly well; the only time that her condition appeared to give anxiety was after the division of the stomach and stitching up the duodenal opening. During the latter part of the operation she rallied a great deal. The operation lasted one hour and forty minutes. The portion of stomach and pylorus removed measured about 6 in. in length and 4 in. from above downwards at the gastric end. The specimen was shown at the Clinical Society with the patient. It contained a mass of growth, which was firm and not ulcerated, and which almost completely obstructed the orifice of the pylorus. There were no adhesions. The patient was ordered to have enemata of brandy (2 oz.) and beef-tea (2 oz.) every two hours, and zymised suppositories every

FIG. 5.

two hours; if she expressed a wish for anything by the mouth she was to be allowed a tablespoonful of tepid water every hour.—Aug. 5th: Passed a good night; slept fairly well; no vomiting. The rectal feeding was now given every four hours; urine to be drawn off every six hours. To have two teaspoonfuls of peptonised milk and four teaspoonfuls of tepid water every hour by the mouth.—6th: Slept well. No vomiting; complains of very slight pain in abdomen; bowels opened. The milk-and-water by mouth to be increased to double the quantity. Enemata as before.—7th: Wound dressed. There has been no tympanitis. No vomiting or other bad symptom.—At 9 P.M. the patient complained of pain in the back and at the epigastrium. At 12 P.M. the pain had considerably increased. No tympanitis. Food by the mouth to be discontinued for awhile.—8th: Better; free from pain; no vomiting; bowels opened. One teaspoonful of peptonised milk to be taken every hour. At 2 P.M. she was ordered to take two teaspoonfuls of milk-and-water every hour, and one teaspoonful of Brand's essence every four hours.—9th: Patient decidedly stronger; pulse improved. Complaints of a little soreness at the right iliac and left hypochondriac regions.—12th (a week after the operation): The patient sleeps well and continues to improve daily. Allowed strong beef-tea, custard, and jelly by the mouth. She is now taking four ounces of beef-tea three times a day; three pints of milk, four eggs, and jelly and mutton broth in the twenty-four hours. All enemata discontinued; suppositories three times a day.—18th (a fortnight after the operation): Sutures removed from abdominal wound. Suppositories discontinued. Diet as on the 12th.—20th: The silk ligature loops which held the plates, and with it a portion of the chemicised gut ligature, have been passed per rectum. Diet to consist of boiled fish, tripe, milk, strong beef-tea, mutton broth, jelly, and eggs.—Sept. 17th: The patient has made uninterrupted progress, been out for drives, and takes her food well.—Oct. 23rd: The patient was shown at the Clinical Society. Since the operation she has increased nearly 2 st. in weight, which on Sept. 16th was 6 st. 4½ lb.; Oct. 1st, 7 st. 4 lb.; Oct. 21st, 7 st. 11 lb.

Remarks.—There have been, so far as I know, only six cases of pylorotomy reported as having been performed in Great Britain; five of these were operated on by the method of excising the pylorus and suturing the divided ends of the stomach and duodenum together, after the method practised on the Continent by Rydyger, Wölfler, and Bilroth. All

these patients died within twenty-four hours of the operation. The sixth case was reported last year by Dr. Rawdon¹ of Liverpool, who adopted a novel method and utilised decalcified bone plates to unite the divided ends of the stomach and duodenum. The operation was thus performed in considerably less time than had hitherto been occupied. The patient made an uninterrupted recovery. I have on several occasions pointed out the difficulties that attend the operation of pylorotomy performed by uniting the divided ends of the stomach and duodenum, on account of the exceedingly short piece of duodenum covered with peritoneum that we have at our disposal, and, further, from the necessary traction upon the stitches which must ensue. To overcome this I have advocated the method of performing the combined operation of pylorotomy and gastro-enterostomy by means of approximate plates. This operation has been successfully performed by Dr. Bull of New York. His operation lasted three hours, as he spent one hour in ligaturing the omentum with a number of chain stitches before dividing it. He has operated in two other patients unsuccessfully, one dying from faulty suture and the other from a surgical disaster.² The case I am now reporting is the first in which the operation has been performed in this country, and the result is so brilliant that I am fully convinced that in the future it will be adopted by surgeons in all cases in which the diseased pylorus is sufficiently free from adhesion to neighbouring organs to allow of its removal. Moreover, by adopting this method, growths of very much greater magnitude than have ever yet been interfered with may be removed. In those cases where the disease is so extensive and adherent that it would be quite unjustifiable to interfere with it, the minor operation of gastro-enterostomy should always be had recourse to, as not only will the patient's sufferings be considerably alleviated thereby, but also life will be prolonged. I have now performed this operation on five occasions. One patient is still alive and well upon whom I performed gastro-enterostomy eighteen months ago, and showed at the Clinical Society last April; another is getting about and able to perform her duties twelve months after the operation; a third lived in comparative comfort for nine months. All these cases were *in extremis* when they came under my care, and could have lived a miserable existence for only a few weeks. The after-treatment I think of importance. In all my successful cases I have commenced to feed at once. If the union is perfect, as it should be, there can be no more harm in introducing warm water or peptonised milk than in the presence of the gastric secretions which are continuously being poured out; in fact, by diluting these I am sure the patient experiences considerable relief, and at the same time the strength is being maintained.

Wimpole-street, W.

THE CONDITION OF THE HEART IN DEBILITY.³

(THESIS FOR THE DEGREE OF M.D.)

BY ARTHUR FOXWELL, M.A., M.D. CANTAB.,
PHYSICIAN TO THE QUEEN'S HOSPITAL, BIRMINGHAM.

THE condition of the heart in debility is one of the *questiones vexatissimæ* of medicine. Thus will it probably remain for a long time to come, as the chances of post-mortem elucidation are but rarely afforded us. Hence one reads and hears many conflicting and even contradictory statements as to the causes of functional murmurs, and, moreover, as to their very character and existence. To have given merely a brief summary of the chief theories on the subject would have taken up more time than I have at my disposal, and I have thought it wise, therefore, simply to state the views which I have myself formed after five years' special thought and investigation. To this end I have divided my thesis into two parts, in one of which I shall

¹ Brit. Med. Jour., vol. i. 1890, p. 323.

² New York Medical Record, Jan. 10th, 1891, p. 39.

³ I have chosen the word "debility," rather than *anæmia*, of set purpose, as quite half my cases, and many of the most marked and instructive, have occurred in broken-down subjects, who are not suffering, nor have they ever suffered, from the special blood state styled *anæmia*.

point out the clinical facts as they have been gathered by me, and in the other I shall give what I consider to be the true explanation of these facts. The clinical facts are these :—

1. *The apex beat* is displaced upwards and outwards, usually being in the fourth left space nearly as far out as the nipple line; it is rarely displaced downwards, and I have no record of having found it in the sixth space, though it is

right to left, with rapid vermicular movement; in each space it starts close against the sternum, and in the fourth is occasionally perceived to the right of this bone; in rhythm it is entirely systolic, or, rather, it is one continuous movement occupying the whole period of the auricular and ventricular contractions.

2. *The area of the superficial cardiac dulness* is in debility characteristic, and is the most valuable of all the signs

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

not seldom in the fifth, even as far out as the nipple line. Its impulse is somewhat diffused, feeble, and sudden, the feebleness not seldom rendering its localisation almost impossible without the aid of the stethoscope. But there is another very evident impulse, chiefly perceptible in the second and third left spaces, but often also noticeable in the first and fourth; it spreads from above down, and from

which enable us to diagnose debility from organic disease. Taking the normal area of heart dulness to be the triangle (A, B, C, Fig. 1) made by the left edge of the sternum² from

² Most writers state that the mid-sternal line marks the right boundary of absolute cardiac dulness, but I have found it to be the left edge in at least nineteen cases out of twenty.

the upper border of the fourth cartilage to the sixth, and by lines joining the extremities of this one to the apex beat in the fifth space at a point midway between the parasternal and nipple lines, then the similarly dull area in a typical debilitated heart would be an irregular figure made by joining the following points by lines slightly curved outwards (D, E, F, G, H, Fig. 1)—viz., D, at the lower border of the second left cartilage half an inch to the left of the sternum; E, a point over the third cartilage one and a half inches from the left edge of the sternum; F, the apex beat in the nipple line and fourth space; G, the right edge of the sternum at the upper border of the sixth cartilage; and H, a point in the right fourth space half an inch to the right of the sternum. If we compare this with the similarly dull area occurring in mitral regurgitation from organic disease, which has led to secondary right-sided dilatation as evidenced by tricuspid regurgitation, we find a striking difference: the irregular figure produced by a typical instance of this condition would have the following points as indices of its circumference (I, K, L, M, Fig. 1)—viz., I, the lower border of the third left cartilage at its junction with the sternum; K, the apex-beat in the sixth space and anterior maxillary line; L, the sixth right cartilage at its junction with the sternum; M, in the fourth right space one and a half inches to the right of the sternum.³ The three points, then, where the depressed upper surface of the liver would be evident in the epigastrium, shape of the heart in debility differs from that of left-sided organic disease are—1. The apex beat is raised and displaced to the left, but this latter change is only slightly in excess of what is required by the arc of the circle which it traverses. 2. The proportionate extension of the dull area to the right is much less. 3. The proportionate extension upwards is much greater, this last being by far the most important of the three.

3. *Systolic murmurs are often present.*—These are produced at three points, which I state in the order of frequency—viz., the pulmonary artery, the tricuspid valve, and the mitral valve. Their rhythm is always systolic.⁴ (1) The pulmonary murmur (Fig. 2) is heard best where the upper border of the third left cartilage joins the sternum and in the adjacent second and third spaces; it is also often conducted down to the fourth and up to the first left space near the sternum. It is seldom heard to the right of this bone or at the apex, but its line of best conduction passes up and out along the second left space towards the left shoulder. (2) The tricuspid murmur is best heard over the sternum between the third spaces, and from this spot it is conducted down spreading fan-like on both sides of the sternum as low as the fifth or even sixth spaces. In the third left space it is continuous with the pulmonary murmur, to which it is nearly always secondary. It is also sometimes well conducted up to the second and first right spaces, and may even be heard in the vessels of the neck. Though often harsh it may be very soft in character. It nearly always strikes one as being superficial, just beneath the chest piece of the stethoscope, bearing some faint resemblance to the whisper in pectoriloquy. Not seldom it is absent in the erect position, but becomes distinct when the patient lies supine; in any case, it becomes much more marked in this latter position. The pulmonary murmur also is often increased on assuming the supine position; but here the excess of loudness occurs chiefly in the second and third left spaces, whilst with the tricuspid the excess is most marked in the fourth and fifth spaces on both sides. In the erect position patients with tricuspid murmurs have systolic thrill or impact in their external jugulars, but when they lie down these vessels fill from below with distinct pulsation. (3) The mitral murmur is heard best at the apex; usually it is soft and not well conducted to the axilla; it

seldom replaces the first sound entirely, but either takes the place of the first half of it, or is more of the nature of an addendum to it. It is of varying intensity, and during some beats is often indistinguishable.

In the second place, I would consider the causes of these abnormal facts. The chief fundamental condition is this—the left ventricle undergoes little or no enlargement. The best proof of this is the position of the apex beat; but there are other cogent reasons why this should be so. First, we must remember that debility is not a special cardiac condition. The body fails as a whole; and the heart muscle no more than other muscles, nor the heart nerves more than the nervous system generally. The whole constitutional state is below par, and every action, mental and physical, the beating of the heart included, is performed feebly; but there is no evidence to show that the feeble left ventricle is unable to supply the diminished wants of the feebly acting body.⁵ Secondly, should the left ventricle from any particular cause be unable to send on all the blood delivered to it by the right ventricle, there is no necessity here for enlargement; it may simply adjust matters by but partially emptying itself and then refusing to admit all the blood from the lungs, for the wants of the debilitated body are not imperious, and will not clamour for their due quantity of blood, but will weak-heartedly content themselves with a smaller amount and so lead to further anæmia. The next fundamental condition is this: the right ventricle in debility nearly always becomes enlarged. The reasons for this are not far to seek. In debility, continuous work tires the body as a whole, and the heart is subject to no particular liability to fail, for the debilitated body is incapable of prolonged exertion of any kind. But the condition in *sudden* exertion is very different. In exercise the initial strain falls on the right ventricle; the first need created is for more rapid oxidation of the blood, and the labour of producing this oxidation is the right ventricle's; should it, through debility, fail in complying with a *sudden* demand for extra labour dyspnoea is induced, the individual abandons his task, and the rest of his body, including the left ventricle, is saved the undue strain by the failure of the right ventricle, which thus acts as a buffer to the left. Even if the *left* ventricle fails, the right again becomes the scapegoat. As we have seen, failure of the left does not necessitate its enlargement; it merely induces it to receive less blood from the left auricle and thus increases the tension in the pulmonary circuit and the work of the right ventricle. Thus the left ventricle has no extrinsic causes for failure, while the right has two—viz., the initial strain of sudden exertion and increased pulmonary tension from weakness of the left. Both these are important and frequently occurring causes, and quite account, in my opinion, for the enlargement of the debilitated heart on the right side only.

These statements granted, it is easy to explain (1) The position of the apex beat. The aortic valves may be regarded as the fulcrum of the heart. If the left ventricle does not enlarge, it is evident that its apex must move in the arc of a circle of which the aortic valves form the centre; that is, either down and to the right, or up and to the left. In debility the enlargement of the right ventricle naturally pushes the apex to the left, and therefore up, so as to obtain more room for its apical portion between the left ventricle and the liver. (2) The peculiar dullness is due to a dilatation of the right ventricle unaccompanied by a corresponding increase in size of the left. Hence the right ventricle's dilatation takes place chiefly in the upward direction, as it is here freest from the restraining influence of the stout left ventricular walls. The two ventricles may be regarded as a cone, which is shown in section at Fig. 3; if one half of the cone be rigid (L) and the other half uniformly distensible (R), it is evident that with a uniformly distending force the chief dilatation of the distensible side will occur about the free semicircular border of its base—will, in fact, occur along the line C C', which bisects the angle A, B, D. Such seems to me the condition of the right ventricle in debility, and this pushing upwards of the free semicircular

³ There would also be epigastric pulsation, and the convexity of the

⁴ I have never yet been able to satisfy myself as to the necessary existence of an aortic murmur. That a murmur is often loudly heard over the aortic area, and is at the same time widely distributed to the root of the neck, there can be no doubt; but I have never had any difficulty in explaining it as part of a tricuspid or pulmonary murmur according as I thought it venous or arterial. For (a) the right auricle not seldom occupies a portion of the second right space. (b) There is no valid reason for supposing that the audible vibrations of tricuspid regurgitation cannot be conducted as far as the internal jugulars. Again, (c) the pulmonary artery branches to the apices of both lungs, and its vibrations should be heard over these. (d) The pulmonary artery and aorta are two closely apposed vessels, each tensely distended with the same fluid; it is only reasonable to believe that vibrations arising in the one are transmitted to the fluid in the other. In proof of this I might add that the murmur of aortic regurgitation is not seldom most audible over the third left space—that is, at a point where its vibrations reach the ear through the pulmonary artery.

⁵ It is true that when there is high tension in anæmia the left ventricle has extra work to do; but most of these high-tension anæmias are not severe ones, while in nearly all the debilities of middle life the condition is one of low tension. Moreover, if the tension is high and the ventricle too feeble to fight against it, there is no need for it to enlarge. It may content itself with a still more imperfect systole, and so still further handicap the right ventricle, both by raising the tension in the pulmonary circuit, and also by more feebly supplying the body (including the right ventricle) with arterial blood.

border takes place to such an extent that the pulmonary valves are sometimes found as high as the first left space.⁶ As we shall see directly, the pushing up of the pulmonary valves shortens the distance between the extremities of the pulmonary artery, and thus renders its dilatation easy, which may also help to explain the high range of the dulness, a range which I have occasionally found extending as far as the clavicle. (3) The murmurs: The pulmonary murmur is the result of a complicated change in the shape and position of the pulmonary artery. This change consists in: (a) The carrying upward of the pulmonary valves till they are vertically over the bifurcation⁷ of the artery (or even higher). This necessarily brings the valves nearer the bifurcation; for the artery may be compared to a U-tube which has its ends widely apart (a, Fig. 4); the change in position approximates these ends (b, Fig. 4), and thus obliges the curve of the artery to be the circumference of a smaller circle—in other words, sharpens the curve, and at the same time carries it higher up, and presses its superior wall against the arch of the aorta. As the tension in the aorta is so much greater and its wall so much thicker, it may be looked on as a rigid body; the pressure of the pulmonary artery against its arch will therefore flatten the superior wall of this vessel. (b) The plane of the right auriculo-ventricular septum ceases to lie in the same plane as the left, but becomes tilted from left to right upwards and backwards (*vide* Fig. 3), owing to the dilatation of the right ventricle.⁸ This will prevent the axis of ventricle and artery being in the same straight line, and hence the blood during systole will impinge upon the side of the vessel. (c) The condition mentioned under (b) is still further increased by the union of the pulmonary artery to the aorta, which will not allow the posterior wall of the former to rise to anything like the same extent which the free anterior one does. (d) It follows from this that the anterior semi-circle of the arterial coat is also more pushed up than the posterior, and must therefore become more lax, and hence an additional reason for its compression against the aortic arch. It will, moreover, assume a more or less anterior bulge or aneurysmal state, which must render the production of vibrations more easy. (e) The movement of the apex to the left and somewhat upwards still further alters the angle at which the ventricle lies to the pulmonary artery. This movement of itself, when of considerable degree, is sufficient to produce a murmur, as we know from the temporary murmurs arising in left pleuritic effusion, and which vanish at once on the withdrawal of the fluid by a syringe. (f) Dr. Russell's theory (*loc. cit.*), that it is due to pressure by an abnormally distended left auricle, is no doubt also true, though I do not think it a main cause, nor an always present one; for I have not found evidence of an over-distended left auricle in those cases where the left ventricle was not notably enlarged. Besides, the tension in the left auricle is considerably less than in the pulmonary artery necessarily, and therefore its power of flattening this must be small. At the same time I quite agree with Dr. Russell that mitral regurgitation is by no means essential to the production of high tension in the left auricle. All that is required is that the left ventricle, from its debility, should be incapable of emptying itself during systole, and that it is so incapable we have absolute proof in the small pulse of debilitated subjects.

The tricuspid murmur has for its causes: (a) The increase in the circumference of the valve ring, which is one result of the general giving way and enlargement of the right ventricle. This is more especially the case, as the valve ring is situated at a portion of the right ventricle, which we have seen is liable to disproportionate stretching. (b) Professor Roy and Mr. Adami, in their valuable and extremely suggestive papers in the *Practitioner* for 1890, have shown

that the muscoli papillares acted independently of the heart walls; further, that with high tension the contraction of the wall is somewhat delayed, whereas the papillary muscles contract as sharply as ever. When the right ventricle is dilated, one would judge that its walls similarly procrastinate their contraction; moreover, the bases of the papillary muscles are further than normal from the valve plane. If, then, these begin their contraction before the walls of the ventricle, we can easily see that at the commencement of this latter contraction the valve flaps might be too far pulled down into the cavity of the ventricle, and hence regurgitation might ensue till the ventricle had contracted sufficiently to allow the valve flaps to reattain their proper position. (c) There is the change in the angle of the plane of the valve to the axis of the ventricular outflow, which change, as was shown in the case of the pulmonary valve, will tend to produce a murmur if any outflow, owing to the action of (a) and (b), take place. (d) Hesse and Ludwig have shown that the circumference of the base of the heart during diastole is to its circumference during systole as 13 to 7.2. This change is due to muscular contraction. In debility it seems more than probable that this contraction is less marked; that is, the auriculo-ventricular openings are less diminished in area than is the case in systole of the normal heart. Here, then, would appear to be a further cause of regurgitation.

The mitral murmur is by far the least common of the three; it is very seldom a prominent one. I think dilatation of the ventricle is rarely its cause; it certainly cannot be in that large proportion of cases of functional apical systolic murmur where the position of the apex beat is not displaced downwards or outwards. Its most frequent causes are: (a) Lack of due systolic contraction of the valve ring; (b) want of due coördination between the muscoli papillares and the ventricle walls. This latter has been shown to occur by Professor Roy and Mr. Adami with large doses of straphanthus, and they suggest that in certain diseased conditions it may also arise. This incoördination may act in two ways: it may cause regurgitation at the commencement of the systole by shortening the tendinæ before the ventricle has shortened, or by the muscoli finishing their contraction too soon and lengthening when the ventricles are shortest, and so inducing regurgitation by allowing the valves to penetrate too far into the auricle. Probably this latter incoördination is the more common, for very often a short systolic apical murmur is heard following immediately the first sound and before the second.

Birmingham.

A CASE OF SUCCESSFUL REMOVAL OF A FRAGMENT OF STEEL FROM THE RETINA BY MEANS OF THE ELECTRO-MAGNET.

By J. TATHAM THOMPSON, M.B.,

OPHTHALMIC SURGEON TO THE CARDIFF INFIRMARY AND TO THE SOUTH WALES INSTITUTE FOR THE BLIND, CARDIFF.

THE patient, D. T. J.—, a blacksmith, was sent to me on Dec. 10th by Mr. Shaw Lyttle of Pontypridd, with the following history. On Dec. 8th, two days before his admission, he was engaged at the colliery stamping some new tools by means of a steel stamp and hammer, when a fragment of steel flew off and struck him on the left eye. He at once went to the foreman and asked him to see if the piece of steel was in the eye. The foreman "wiped a drop of blood from the white of the eye, but could see nothing there." He had little pain, and little or no disturbance of vision. Two hours after the accident he saw his club surgeon, Mr. Shaw Lyttle, who reported that he found a small clean cut in the sclerotic at the inner side, just beyond the ciliary region. He failed to make out the presence of any foreign body. The next day (Dec. 9th) he had dull pain in the injured eye and a good deal of haziness in the sound one, which next day increased, and was accompanied by slight photophobia. Mr. Lyttle then sent him to me at the Cardiff Infirmary. When examined there, the left eye showed general congestion of the conjunctiva, with slight ciliary congestion; pupil regular, dilated with atropine;

⁶ Vide W. Russell. Investigations into some Morbid Cardiac Conditions, p. 37, et seq. In two patients at the Queen's Hospital who died of pneumonia, and in whom there was no cardiac lesion except right-sided enlargement, I found the pulmonary valves in the one to be beneath the second left cartilage, and in the other beneath the first space.

⁷ This bifurcation is practically a fixed point, for the descending arch of the aorta lies in its fork. To the aorta it is fastened by the ductus arteriosus; it has attachments to the pericardium, and its two branches are fixed by the roots of the lungs.

⁸ This is almost impossible to prove by direct evidence, as post mortem the active distension of the cavities is absent; but I have found it to be so when acting on the heart of a debilitated subject experimentally—that is, by forcing water by means of a syringe into the right ventricle, having previously tied the pulmonary artery.