Clinical Lecture

ON

THREE CASES OF CARDIAC FAILURE WITHOUT EVIDENT DILATATION OF THE HEART.

BY

T. STACEY WILSON, M.D. EDIN., F.R.C.P.,

PHYSICIAN TO THE BIRMINGHAM GENERAL HOSPITAL.

THE three cases recorded in this paper illustrate muscular failure of the heart without marked enlargement and without valvular disease, and as cardiac failure without dilatation is apt to cause some embarrassment as regards diagnosis and treatment, their publication may be of interest.

The association of these three cases together is a natural one, for they were all admitted to the hospital within the same thirty-six hours. The first of the three was a fairly simple and common case of heart failure following rheumatism. A man, aged 25, was admitted to the hospital with acute rheumatism, from which he had been suffering for a fortnight. The attack was not a severe one and yielded to sodium salicylate, the temperature coming down on the third day, only rising above normal once afterwards. The heart never seemed very strong, and three weeks after admission he was apparently convalescent and was feeling quite well, and wanting to go out of the hospital and back to work. An examination of the heart at this time showed the following condition:

The apex was in the fifth interspace, well within the nipple line, and there was a very feeble impulse to be felt. There was no enlargement of the heart to the right, the liver dullness was smaller than normal, and there was no epigastric pulsation.

There was no fullness of the veins of the neck. There was a localized systolic murmur, audible at the base of the heart, over the sternal end of the second left interspace, and also in the third interspace, but no murmur at the apex or elsewhere.

The pulse was rather weak, but otherwise normal in rate, character, and rhythm.

So far, therefore, there was little evidence of any contra-indication to the man getting up and walking about. One portion of the heart, however, has not yet been referred to, and, as a matter of experience, this part is one of the first to give evidence of weakness in a numerous class of cases of cardiac failure. I refer to that portion which lies under the third rib and the third space just to the left of the sternum.

Anatomically, this part is the anterior wall of the right ventricle just below the origin of the pulmonary artery, and it is sometimes conveniently called the "conus arteriosus" of the right ventricle. On studying a heart *post mortem* it will be seen that in this situation the muscular wall of the ventricle is thinner than it is elsewhere. If, therefore, the power of resisting dilatation depended upon the muscular wall alone, we should expect this to be the first part of the ventricle wall to become overstretched in cases where excess of intraventricular pressure was tending to produce dilatation of its cavity. Now in the adult there is evidence that the fibrous tissues of the heart and pericardium play an important, and perhaps the most important, part in supporting the heart wall and counteracting any tendency to dilatation. In the heart, therefore, with strong fibrous tissues we do not find that the portion of the anterior wall of which we are speaking shows any exceptional tendency to dilate. In puberty and adolescence, however, where the fibrous tissues are developing, and have not attained to their adult resisting power, the heart has to rely mainly upon its muscular wall, and under these circumstances we do not find that this thin portion of the anterior wall is the first part of the right ventricle to dilate when the intraventricular pressure becomes excessive.

On carefully percussing this patient's heart when the lungs were emptied during full expiration it was possible to detect some upward increase of the cardiac area, for there was relative cardiac dullness for about three-quarters

of an inch in the second space, and 2 in. (as against a normal $1\frac{1}{2}$ in.) in the third interspace. In the fourth interspace there was only about 2 in. of relative dullness instead of the normal $2\frac{1}{2}$ or 3 in., showing not only the absence of any left ventricular dilatation but suggesting the presence of some diminution in the size of that part of the heart.

Coupled with this increase of the right ventricle upwards and to the left, the systolic pulmonary murmur which is usually associated with it was audible in the second and third left interspaces near the sternum. The theory of the production of this murmur, which is associated with the name of the late Dr. Foxwell, is so well known that I need not refer further to it here. This small amount of upward dilatation and the accompanying pulmonary murmur, associated with a weak pulse and a small left ventricle, were distinctly suggestive of myocardial damage as a result of the rheumatic fever. The second sound of the heart was not abnormally loud in the pulmonary area, and there was nothing to suggest any greatly increased resistance to the passage of blood through the lungs from failure of the left ventricle or other cause.

The condition, therefore, is suggestive of a right ventricle so weak that it is on the point of becoming dilated in its attempt to carry on the normal circulation while the patient is confined to bed. If he were to get up it would be certain to produce a very marked dilatation. In a precisely similar case I have seen the heart rapidly dilate when the patient began to walk about, and show some inches of dullness with well-marked pulsation in the second left interspace.

The treatment here must consist of rest in bed and tonics, etc., thus giving the heart muscle time to recover from the effects of the rheumatic poison. After two weeks the relative cardiac dullness lessened in the second and third interspaces and increased in the fourth, and the heart got stronger, and the patient, though not fit for work, was well enough to leave the hospital for a month in the country.

The second case is also a fairly simple one. The patient was a stoutish, red-faced man, aged 44, whose work as an agent involved a good deal of bicycling. For many months he had noticed an increasing breathlessness, and he had been unable to do much bicycling for more than a year. Within the last few weeks his breathlessness had been so troublesome that he came to the hospital and was admitted. There had been no swelling of the ankles and no fainting attacks. On admission the most evident sign of cardiac failure was fullness of the veins of the neck with well-marked pulsation in them as far up as the angle of the jaw. The pulse was small in volume and wanting in vigour, the tension was rather high, and the blood pressure as tested by the sphygmomanometer was 160 mm. of mercury. As regards the heart, instead of finding distinct dilatation and perhaps a tricuspid murmur, as might be expected under such circumstances, percussion showed the cardiac area to be normal, and on palpation and inspection the cardiac action and impulse were found to be more feeble than normal. Auscultation confirmed this, for the heart sounds were decidedly weak, and those at the apex were short and sharp, suggesting feebleness, and there was also a very faint systolic murmur present.

The right ventricular sounds, while rather better than those at the apex, were not so loud as normal. As regards the lungs there was no emphysema, but there was a good deal of bronchial wheeze, suggestive of bronchial catarrh, with some bronchial spasm. Here, again, we were dealing with a case of heart failure without manifest dilatation, although some dilatation of the right auricle, and possibly of the right ventricle, might nevertheless exist. The explanation of such a case is probably as follows: The man had been suffering more or less for some time from a gradual rising blood pressure, the result probably of failing kidney action or an injudicious dietary, and the nutrition of the heart had not been sufficient (owing to faulty coronary arteries) to enable it to keep pace with the extra work thus thrown upon it. Had it done so there would have been a measure of hypertrophy with heart sounds louder than normal. The final cause of his breakdown was doubtless his bronchial trouble. The fact of having a heart of normal size and weak action in face of a blood pressure of 160 mm. of mercury suggests myocardial degeneration from malnutrition. The distension and pulsation of the

veins shows that the right ventricle was relatively strong and was endeavouring to compensate for the defective action of the left heart. As regards the treatment: The bronchial catarrh and spasm, though only slight, had to be dealt with, and also small doses given of such a drug as strophanthus, which, while not increasing arterial contraction, does increase the vigour of the cardiac action, and so tend to improve the cardiac nutrition. Digitalis would, of course, not be desirable. A very important part of the treatment of such a case is the lessening of the peripheral resistance by means of drugs such as the salicylates. In such a case as this, however, the prognosis is not very hopeful. This was shown by his rate of progress, for, although the heart sounds gained in vigour after three weeks' rest in bed combined with the treatment described, the man progressed but slowly.

The third case of heart failure without any obvious cardiac dilatation or murmur was that of a woman aged 61. She had been short of breath for several years, and of late had been gradually getting worse. Recently she had rapidly got weaker, and the day before admission she had a serious fainting attack—the first of the kind she had had. There was no history of any previous illnesses, and her present condition had developed gradually. Her state on admission was as follows: A rather stout woman, very pale, not breathless when lying in bed with a moderately high bed-rest, but breathless on exertion, and unable to lie flat in bed. There was no dropsy and no cyanosis.

The pulse was decidedly small, but of good strength, and was of the strikingly collapsing character usually associated with aortic regurgitation. Although its volume was small it came up under the finger with suddenness and force, and subsided again with equal suddenness, leaving the artery empty to the touch between the heart-beats. On testing it with the sphygmomanometer, it was found that the pulse wave would withstand a pressure of nearly 160 mm. of mercury. On testing the collapsibility of the pulse when the arm was raised or lowered it was found that elevation of the arm did not (as in aortic regurgitation) increase the collapsing character of the pulse. On looking at the neck there was not only no pulsation of the veins, but there was a hollowing above the clavicles which suggested undue emptiness of the vessels.

As regards the heart there was no visible or palpable impulse anywhere, the cardiac area was small, decidedly less than normal, and the liver was not enlarged. The cardiac sounds were decidedly weak, and there was no murmur. The lungs and other organs appeared quite healthy.

This patient was evidently suffering from weakness of the heart muscle, and its degree was worse than in the second case described, for the heart had not sufficient strength to keep the superior cava and neck veins distended and pulsating as in the last case.

The diagnosis was heart weakness due to fatty degeneration, the result probably of diseased coronary arteries. A more complete analysis of the symptoms would have led to an even fuller diagnosis, but the importance of the collapsing pulse and the systolic pressure of 160 mm. coupled with the empty veins was not recognized till after the *post-mortem* examination revealed the true nature of the case.

The patient was given small doses of strophanthus and strychnine, and seemed to be improving when suddenly she had another fainting attack, five days after admission, and died a few hours later in a second attack. The earlier of the two attacks came on at 4 p.m. There was marked dyspnoea and cyanosis, with pain over the heart and left chest. The pulse was almost imperceptible. She rallied somewhat, but became worse an hour later. In this second attack she suddenly sat up in bed and then fell back dead.

At the *post-mortem* examination there was fatty degeneration involving the right ventricle, due to atheroma mainly involving the one coronary artery. The condition of the muscles of the left ventricle was good, and the artery of supply was patent. Here, then, was the explanation of the small powerful collapsing pulse.

The right side of the heart was too feeble to force the proper amount of blood through the lungs, and, as the condition had evidently developed slowly, the amount of blood in circulation had presumably diminished *pari passu* with the diminution in the strength of the right ventricle, and thus venous plethora, which might have been ex-

pected, had the failure of the right ventricle been less gradual, was not produced. The left ventricle was therefore underfilled at each beat, and, in order to get as large an amount as possible into the capillaries of the brain and elsewhere, the blood was thrown out of the ventricle suddenly and with force, the arteries being kept as contracted as possible. The rapid collapse of the pulse wave and the emptiness of the artery between the beats were due to the abnormally small volume of blood causing the pulse wave.

This case illustrates well the main signs upon which diagnosis of a fatty right ventricle with a fairly strong left ventricle is to be based—namely, great dyspnoea and weak right ventricular sounds coupled with absence of well-marked venous pulsation in the neck in combination with a regular and not very rapid pulse of small volume and fair strength. Where some amount of arterio-sclerosis is present, as in a case I saw some months ago, the rapid and great collapse of the pulse may not be noticeable, but where present, as in the case here noted and in one I have subsequently seen, its diagnostic importance is great. The prognosis, it is needless to add, is in such a case extremely grave.

These, then, are the three cases. The first showing heart weakness with diminution in the size of the cardiac area from damage to the heart muscle from the toxins or micro-organisms of acute rheumatism. In this case the left ventricle was rather weaker than the right, and the latter showed a slight amount of the adolescent type of dilatation and a pulmonary artery murmur. The second case was one of the muscular failure of the heart, without evident dilatation, due to malnutrition presumably from atheroma of the coronary arteries. In this case the left ventricle was more affected than the right.

The third case was exceptionally interesting owing to the limitation of the fatty degeneration to the right ventricle as well as from the absence of physical signs of cardiac failure except those associated with a relative emptiness of the heart and great vessels, and the weakness of the cardiac sounds, especially those heard over the right ventricle.

A CASE OF OBLITERATIVE ENDO-ANEURYSMORRHAPHY FOR POPLITEAL ANEURYSM.

BY

ARTHUR E. BARKER, F.R.C.S.,

SURGEON TO UNIVERSITY COLLEGE HOSPITAL AND CONSULTING SURGEON
TO THE QUEEN ALEXANDRA MILITARY HOSPITAL, MILLBANK, S.W.

ENDO-ANEURYSMORRHAPHY in its most recent form, as elaborated by Dr. Matas of New Orleans, is still so uncommon an operation in this country that the publication of even a single case in which it was employed needs no apology. The reasons for abandoning the old method of Antyllus in the treatment of aneurysm in favour of the classical Hunterian operation are familiar to all British surgeons. But the grounds for a return, more or less, to the direct operation on the aneurysm practised by Antyllus require some justification. These are so ably set forth in the writings of Dr. Matas that they need not be dealt with in any detail here. I will only recall the fact that in the Antyllian operation the artery was tied above and below the aneurysmal sac, and that the latter was laid widely open, packed, and allowed to heal slowly from the bottom by granulation. In the days before the Listerian era the risks of this procedure were obviously very great. In the first place sepsis at the point ligated was almost certain to be followed by severe haemorrhage, and sepsis in a ragged cavity such as the open sac, whatever material it was stuffed with, was most likely to occur in communication with an open wound. Moreover, severe haemorrhage could occur from the branches opening into the sac which belonged to the free anastomosis around it. The probability that under modern aseptic conditions these chief risks would disappear, as well as his experiences in the case of several traumatic aneurysms treated by direct opening of the sac and closure of the entering arteries from within, led Dr. Matas to the conclusion that much would be gained if aneurysms could be immediately obliterated on the principles of Antyllus, but without leaving the sac open to granulate up as he did, with all the risks of sepsis and of haemorrhage from the ligated main