A CLINICAL LECTURE ON PERICARDITIS.

Delivered at the Manchester Royal Infirmary,

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RHEUMATIC PERICARDITIS.

GENTLEMEN,—We had recently in the wards a case of rheumatic pericarditis in a young man who had been the subject of acute rheumatism three years ago, and who had again suffered from slight arthritis of the knees about a fortnight before admission to hospital on November 17th last. He had gone about his work, however, till November 10th. He did not seem to have had any chest pain till the 9th, when he had severe pain on the left side, specially on taking a deep breath, and he noticed that he was short of breath. He had had attacks of chorea at the ages of 8, 9, and 11 respectively. There could be no doubt, then, that he was a thoroughly rheumatic subject, and had had endocarditis in all probability.

When I examined him on November 18th, the day after admission, I was surprised to find, besides the physical signs of a moderate left pleural effusion, not so extensive as to prevent the left border of the cardiac dulness being defined so far, a huge precordial dull area, and such a dull area as would imply enormous enlargement of the heart, were it due to the size of the heart, and of the shape that is assumed by a true cardiac dulness only in rare cases, as Sir William Gull long ago pointed out, of general dilatation of the heart, in which the dilatation of the left ventricle, and that of the right auricle and of the infundibulum of the right ventricle are all extreme. But this lad's history pointed to his cardiac disability being comparatively recent. When I listened over the heart what struck me most was the feebleness of cardiac sound of any kind. All I could make out, in fact, was the presence of a slight systolic murmur in the mitral and tricuspid areas. I could hear no friction.

We had then a remarkable degree of silence over the heart, a greatly increased area of dulness, and a very feeble systolic murmur in the tricuspid and mitral areas. The natural inference to be drawn from this condition, of course, was that there was pericardial effusion. The absence of friction sound was of course remarkable, as there was every reason to believe that if pericardial effusion were present it was of inflammatory origin in view of the rheumatic but otherwise good history of the patient.

It was on Saturday (November 18th, 1899) that I examined the case with the result related. On the following Monday on my arrival I was informed that our resident medical officer, Dr. Melland, had detected pericardial friction sound, an observation most creditable to his acuteness of observation, because the sound, as some of you can bear me out in saying, was of the feeblest, and, moreover, was limited to a small area, I should think about an inch and a-half in its vertical and an inch in its transverse diameter. This area was situated at the left border of the lower part of the sternum. During the following couple of days the friction increased in loudness and extent of area of audibility, and then it declined and ceased. But if any doubt had remained as to the diagnosis of pericardial effusion, it was soon dissipated by the rapid diminution of the precordial area of dulness till it approached practically normal limits. The pleuritic effusion—which in this case was evidently genuine—moreover soon likewise receded. (You will see the meaning of my use of the word "genuine" later.) The mitral murmur remained, and became associated with a reduplicate diastolic sound. Evidently the murmur was old, and the result of the first rheumatic attack, or more likely of the earlier chorea. The tricuspid murmur was no doubt due to the debility of the cardiac muscle only. Unfortunately the patient absconded four days ago, but, to do him justice, he took this action in a good cause: to join his militia regiment.

PERICARDIAL EFFUSION WITHOUT FRICTION.

Sometimes, though rarely, you have to make the diagnosis

of pericardial effusion without any assistance whatsoever from auscultation, that is to say, pericardial friction may never be heard from first to last. Again, paradoxically, I shall relate to you a case in which friction was audible over the heart, and yet in which it could hardly be said there was pericarditis present at all, so trivial evidently was the process that temporarily annulled the smoothness of the gliding surfaces.

First, as to pericardial effusion without friction sound. When I was resident medical officer here, an old woman with chronic Bright's disease, but without any general dropsy, was admitted to the wards. It was noticed that she always sat up in bed, leaning forwards, and was evidently unable to lie down owing to the dyspnoea it occasioned her. Percussion of the chest showed a precordial dull area of enormous size, extending upwards and to both right and left sides. The heart sounds were feeble, but there was neither friction sound nor murmur audible. There was no febrile disturbance. In a few days the dull area had become greatly diminished, while neither friction nor murmur had been heard. The patient could now lie down comfortably. Evidently this was a case of one of the copious effusions into sacs or loose tissues that are so apt to occur in the course of Bright's disease. So much for effusion into the pericardium without any evidence of inflammation whatsoever.

I next relate to you a case to show that there may be severe pericarditis with great effusion and fibrinous exudation, and yet, again, there may be no friction sound audible throughout the period of observation, extending over weeks, while the debility of the heart muscle in consequence of the contiguous inflammation and the embarrassment of its movement occasioned by the effusion may determine the ordinary effects of heart failure on the general circulation.

History.—Mrs. M. A. T., aged 27, was admitted to the Manchester Royal Infirmary on September 24th, 1891, suffering from dyspnoea, dropsy of the lower extremities and of the right upper extremity, and from ascites. Her illness, which had commenced several weeks before admission, had followed recent confinement. She had never had rheumatic fever or chorea. The temperature was moderately febrile.

Condition on Admission.—The upper right extremity was found to be much swollen, and the swelling was stated to have been preceded by pain referred to the region of the corresponding clavicle and shoulder. The area of precordial dulness was enormously increased, but its extension upwards was proportionately less than that to the right and left sides, especially the latter, the left border of the dull area passing in a slanting direction across the axillary region, the base of the left lung being at the time resonant. Both sounds of the heart were audible and free from murmur. There was no trace of friction sound. The liver was enlarged and tender—no doubt from venous stasis—and there were moist sounds at the bases of the lungs. There was no albumen in the urine. The patient was anæmic and cachectic-looking.

After-History.—Slight hæmoptysis occurred from time to time, and the pulmonary bubbling sounds became more abundant, while the bases of the lungs became impaired.

Post-mortem Examination.—The pericardium was hugely distended with light amber-coloured fluid. Both the visceral and parietal layers were the seat of diffuse hæmorrhagic fibrinous pericarditis. The heart itself had a patchy, shaggy, rough, brownish appearance, due to the irregularly deposited flaky lymph which, adherent below, had its upper layers blood stained. This lymph was deposited on both surfaces of the pericardium, and was particularly marked around the origin of the large vessels at the base of the heart and over the auricles and around the pulmonary veins. The fibrinous deposit was fairly firmly adherent and had evidently been of some standing. The heart was small and presented no valve lesion. There was some fluid in both pleuræ and infarcts in both lungs. The pelvic organs were matted together and surrounded by inflammatory material. Thrombi were found in several veins, including the right subclavian. The abdomen contained ascitic fluid, the liver was enlarged and presented appearance of well-marked passive congestion. Kidneys normal in size and general appearance, except for slight congestion.

This case, then, was an instance of the fact that even fibrinous pericarditis may be present, run a subacute course, and occasion great effusion and yet be unaccompanied by any friction sound. In the case narrated the septicæmic source seemed to lie in the pelvis.

The next fact with regard to pericarditis to which I wish to call your attention is that cases of the worst type of pericarditis not rarely run their fatal course without a trace of friction sound from first to last. I refer to suppurative cases of septicæmic origin.

When I was House-Physician in the Fever House of the Edinburgh Royal Infirmary a convalescent from typhus, a man about 40 years of age, abruptly developed an extreme degree of irregularity of his pulse along with some pyrexia and *malaise*. His cardiac dulness coincidentally increased, though it hardly assumed the characteristic pyramidal form of the dulness of pericardial effusion. No friction sound or

murmur was ever audible, and the heart sounds, though remarkably weak, were quite pure. He died, and necropsy revealed purulent pericarditis.

But here is another case of the kind from our own records.

History.—A man, aged 41, was admitted to the Manchester Royal Infirmary on October 20th, 1892, suffering from arthritis of the right ankle and wrist, dyspnoea (evidently not of laryngeal origin though the patient was hoarse), delirium, and great prostration. He was too ill to give any account of his illness, but we afterwards ascertained from his friends that ten days before admission he had struck his right ankle against a piece of iron without, however, breaking skin. He thought little of the injury at the time, but later the ankle became painful and swollen, and a week before admission he complained of feeling unwell, of aching pains over his body, and of shortness of breath. For three weeks before the accident he had been troubled with diarrhoea; thus had been ailing.

Condition on Admission.—When I saw him on October 31st the right wrist and right ankle were swollen and red, and in the neighbourhood of the latter there was fluctuation, and a thrombosed superficial vein could be traced up the leg. The temperature was normal and never rose above 100° while the patient survived. The pulse was extremely weak and very irregular; respirations 46 per minute. There was more or less cyanosis. The area of cardiac dullness was much enlarged both in the right and left directions, and less so in the upward direction. Both heart sounds were weak though plainly audible and free from murmur. There was no friction sound. The impulse of the heart could not be determined. There were small bubbling sounds at the bases of the lungs with some impairment of resonance. The liver was enlarged, and its edge could be easily felt below the ribs in the mammary line. Laryngoscopic examination revealed oedema of the ary-epiglottidean folds. The patient died on October 31st, 1892.

Post-mortem Report.—No sign of external injury. Right ankle and right wrist much swollen. Thorax: Pericardium much distended with puriform fluid, and both layers, particularly the visceral one, coated with thick, soft flocculent lymph. Heart: Cavities, orifices, and valves normal. The jugular veins contain a distinct clot, and there is also a slight clot in the lower part of the right internal saphenous vein. Pleuræ: Both seat of acute pleurisy. Lungs: right intensely congested, but crepitant throughout; lower lobe presents several hæmorrhagic patches. Left lung in similar state. Larynx much congested, marked oedema of ary-epiglottidean folds. On making the median incision over the larynx a quantity of creamy pus flowed out from the cellular tissue of the right side of the neck. Abdomen: At the lower part of the ileum there were several hæmorrhagic patches. Liver considerably enlarged and intensely congested; no suppuration; many of the hepatic veins contained clot. Spleen enlarged, presented near the centre a patch of softened pulp, but no distinct foci of suppuration. Joints: Both right ankle and right wrist contained creamy pus; no erosion of cartilages or disease of bone. No ear disease.

The fact of the injury that seemed to be the origin of the septic condition not having broken the skin and the evidence of preceding ailing for three weeks are worth noting.

SECONDARY PERICARDITIS.

Then there is the form of pericarditis that is often met with in cases of granular disease of the kidney with enlarged heart. In them there is often no effusion although the friction sound may be well marked for days. Again, there is the pericarditis that often complicates acute pneumonia. A tuberculous pericarditis occasionally occurs with tuberculous lesions of other organs. I had a case I believed to be of this nature a few years ago. The only case of leprosy that has been in this infirmary for many years died of pericarditis. In scarlatina, pericarditis is not very rare, but the relationship between scarlatina and true rheumatism is not quite settled. Certain it is that joint affections that are not true rheumatism are frequent in the disease, but on the other hand, scarlatina seems to have the power of predisposing to an attack of true rheumatism. At least, one sees during convalescence a multiple arthritis that corresponds to true rheumatism with pericarditis and endocarditis during convalescence from scarlatina, the patient generally having had true rheumatism before. Beware of putting every multiple joint affection down to rheumatism, even if there be pericarditis or endocarditis present. Septicæmic processes are often accompanied by joint pains, as we have seen in the cases referred to to-day.

I must refer lastly to the most trivial forms of pericarditis to be met with—indeed, it is doubtful if they should be regarded as cases of pericarditis at all, but unquestionably there is some temporary nutritive disturbance in the pericardium that interferes with the smoothness of its opposed surfaces.

Here is a good example of the condition. A few years ago a patient of mine who had been under treatment for bronchitis and emphysema was about to be discharged from the infirmary convalescent. He was sitting by his bedside ready to go out when I was going round the ward. For some reason or another I placed my stethoscope over his heart, and to my astonishment found a very distinct double grazing friction sound. His discharge was of course cancelled, to his great indignation,

for he had neither pain nor fever, and felt quite well according to his standard of well-being. In a day or two the friction disappeared, without there ever having been any evidence of effusion, and the patient soon obtained his discharge, anything but grateful for the care that had been taken of him.

PHYSICAL SIGNS OF PERICARDITIS.

Inspection.

Fulness and obliteration of the intercostal depressions corresponding to the effusion and interference with the movement of the left side of the chest may be noticed on inspection. The apex beat has seemed to be elevated in large pericardial effusions, but one must be careful not to assume that any visible pulsation there may be must be the apex beat.

Palpation.

In a case of rheumatic fever, especially a first attack, one should always take pains, however, to determine the situation of the apex beat by palpation and inspection, although there be no indication of heart implication at the time, so that if pericardial effusion were to occur, any change in the position of the impulse might be noted. Often one fails to detect any cardiac impulse when there is effusion, and the inference then is that interference with impulse is not simply the result of the effusion intervening between the heart and chest wall, or mechanically embarrassing the heart, but more especially of the heart muscle being debilitated by the inflammation surrounding it. Sometimes a friction rub is easily felt on placing the hand over the cardiac region, but this usually occurs in cases of some standing, and when it occurs the effusion is usually not great. It will naturally be more likely to occur in cases of hypertrophied heart, as in Bright's disease (granular kidney), and in such cases the effusion is often scanty or absent.

Percussion.

Percussion is a very much more important method of examination in pericarditis than either inspection or palpation. When the pericardial sac is distended, the dull area which represents the heart in health becomes much enlarged, and the increase takes place first of all upwards. The fluid would seem to collect about the large vessels, and the upper part of the sac to be the part most prone to yield. As the effusion collects, the right and left boundaries of the heart dullness, however, soon extend laterally below, so that the whole dull area assumes a more or less pyramidal form, the base of the pyramid being below. If you use the method of percussion which I employ, these changes will be readily discerned. Instead of extending to the third cartilage, 1 inch to the left of the sternum, the precordial dullness will be found to reach to the second cartilage, or even above it. To the right of the sternum, again, above the level of the liver, considerable dullness will be found, and to the left of the sternum the lowest part of the left boundary of the dull area will be found situated at a very much greater distance from the middle line than under normal circumstances. Now the rule is that extension of the cardiac dullness to the right indicates enlargement of the right auricle, and to the left enlargement of the left ventricle. It is extension in the upward direction that is characteristic of pericardial effusion; but when such extension is found we must be careful to map out the whole area of dullness, and to note if there is extension laterally below. There is another condition which occasions extension of cardiac dullness in the upward direction, although practically it very seldom causes any confusion in diagnosis. This other condition, I have already said, is enlargement of the infundibulum of the right ventricle, from which you will remember the pulmonary artery arises, and at the extremity of which the pulmonary valves are situated. Although a part of the right side of the heart, bear in mind that the infundibulum or conus arteriosus, as it is often called, if dilated, will extend to the left of the sternum above the third costal cartilage in the second space, where not infrequently it occasions a visible impulse.

Cases in which dilatation of the infundibulum is so great as to give rise to extension of cardiac dullness above the third left cartilage are necessarily chronic, and have usually long before developed symptoms of disturbed circulation—dyspnoea, dropsy, and enlarged liver; while pericarditis is an

acute disease in the majority of instances. In cases of enlarged infundibulum one usually feels the thud of the closure of the pulmonary semilunar valves easily as they lie superficially, and are closed too forcibly. The general rule then is that if you find extension of the cardiac dullness in the upward direction suspect pericardial effusion, and if such effusion be at all abundant you will find the shape assumed by the dull area as a whole to be more or less pyramidal, the apex of the pyramid above, the base below.

When an enormous quantity of effusion is present the dullness may encroach considerably upon the area of the left lung, and there is a possibility of the effusion being mistaken for a pleuritic one. If care is taken to examine the back this can hardly happen. Mention of this possible confusion in diagnosis reminds me to call your attention to certain pulmonary physical signs which are occasionally present at the base of the left lung behind the pericarditis, and which may simulate the signs of pneumonia. They are a change in the percussion resonance characterised by a diminution in the volume of the sound, with sometimes an approach to tympanic quality, and even the presence of a few crackling sounds like crepitation with inspiration. In some cases even bronchial breathing is heard. These signs appear to be the result of encroachment of the effusion on the base of the left lung, and are therefore naturally limited to cases accompanied by a very considerable amount of effusion. I mention these signs simply to put you on your guard against being led to a diagnosis of pneumonia by them. True pneumonia is often associated with pericarditis, but the pneumonia is generally the primary disease, and affects, as a rule, the left lung. In simple pericardial effusion, however extensive, although the resonance of the left lung at the base may be interfered with, there is not likely to be absolute dullness behind, and though the pericardial dullness may approach the posterior axillary line, it will hardly pass beyond it.

Of course, given pericardial effusion, there is no reason why there should not also be in certain cases left pleuritic effusion, when the dullness at the left base would be absolute, as happened in the case of our patient recently in the wards, and in his case it would seem to have been the pleurisy rather than the pericarditis that occasioned the pain on the left side on November 9th.

Auscultation.

Auscultation in most cases of pericardial effusion reveals to us the presence of a friction rub. This is usually double, to and fro, and varies greatly as regards quality of the sound, being sometimes fine and grazing, at other times very harsh and rough. When friction sound is fully developed it completely covers the heart sounds or murmurs that may be present. It gives an unmistakable suggestion of its cause, conveying a distinct impression of the rubbing of two surfaces together. Moreover, it is generally increased by pressure with the stethoscope, and is little conducted beyond the pericardial region, although there are exceptions to this rule. If in the undeveloped or receding stage of pericarditis one or other or both the cardiac sounds are heard with a friction rub or graze, such friction bears no definite relation to the cardiac sounds, but seems to occur independently of them. This is, as you know, very different from that which is the case with regard to endocardial murmurs, for they bear definite relationship to the sounds they accompany. An endocardial murmur runs off from or runs up to a cardiac sound.¹ A friction sound is generally fairly uniform and continuous in its course, but at times it is more or less interrupted, so that a triple rhythm may seem to be assumed. Sir George Johnson believed that he had recognised a triple rhythm due to the contraction of the auricle producing a rub, as well as the systole and diastole of the ventricles. However, I doubt if this was the explanation of the triple rhythm. We must remember that the systolic and diastolic cardiac sounds fall very far short of representing in duration the systolic and diastolic movements of the heart. For instance, in mitral regurgitation it is common to hear the first sound followed by a systolic murmur, the murmur showing that the contraction of the ventricle is still proceeding after the sound has ceased. We may consider that friction may be heard at any time of cardiac movement, and when we reflect that practically as soon as systole is over diastole begins, it is no wonder that in most cases a friction rub is an almost continuous to-and-fro sound.

As already stated, friction is usually first detected at the base of the heart, in the vicinity of the great vessels, for it is here probably that the greatest amount of movement takes place; otherwise the site of the maximum intensity of friction sound varies infinitely, not only in different cases but in the same case at different times of its course. In the small triangular area where the heart is uncovered by lung friction is apt to be specially distinct. No amount of effusion will necessarily annul friction sound, as the heart has free movement.

Pleuritic friction sound occasionally takes on cardiac rhythm over the heart; but when the stethoscope is moved off the lung to the small triangular area in which the heart is exposed it is usually no longer heard, and the heart sounds are pure.

Usually friction sound gives to the observer an unmistakable impression of the mode of its production, and this is true even of the fine grazing varieties of the sound. But there are two endocardial murmurs that together may occasionally be mistaken in a hurry for friction sound or *vice versa*. I refer to a double aortic murmur unaccompanied by any cardiac sound in the technical sense of that word. But the history of the case, the arterial condition, and the other physical signs of aortic incompetence will always prevent error, unless the two pathological states—aortic incompetence and pericarditis—should coexist.

NOTE.

¹ A very rare variety of diastolic murmur in mitral stenosis is the only exception to this rule that I am aware of.

REMARKS ON THE PURE-AIR TREATMENT OF PHTHISIS AT HOME.

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DURING the last few months many papers have been written giving high praise to the sanatorium treatment of phthisis, and, in my opinion, these laudatory notices have been fully justified by the results. These institutions for the "open-air" treatment have, in truth, many advantages. They are expressly adapted for their purpose; with all the needful appliances, shelters, galleries, or verandahs, and their windows open fully to the ceiling of the rooms. Most of them are newly-erected upon healthy sites, on dry, pure, and porous subsoils; and they are appointed and furnished with a view to the avoidance of dust; hence they have a minimum of microbic impurity in the air. Special attention is paid to the food of the patients; and systematic supervision is kept up by both nurses and doctors. The example of the inmates, and their general well-doing, makes it easy to carry out the otherwise unpleasant discipline, and, in most of these places there is an air of general contentment. All these circumstances renders the treatment in sanatoria more perfect than it can be in most private houses.

But there are many persons who cannot or will not be treated in sanatoria. There are fathers and mothers whose cure would be greatly interfered with if they were separated from their children; sons and daughters who are unwilling to leave their parents; delicate persons who shrink from the public life of a sanatorium, or even of a nursing home. There are also large numbers of persons who, having been "greatly improved" by a residence of from three to six months in sanatoria are yet susceptible to reinfection, and who must, for many months, continue to pursue the open-air treatment at home. Add to these the many patients who, owing to the present deficient accommodation, are unable, for many months, to obtain an entrance to any well-equipped institution; all these persons need efficient open-air treatment, and most of them greatly desire that it should be carried out at home.

Under certain stringent conditions, I think that it is quite possible to do this, and it may be of some service to point out in what ways it may be accomplished.

CLIMATE.

Sanatoria are for the most part placed in carefully sheltered situations, yet fully exposed to the air and sunshine. Our