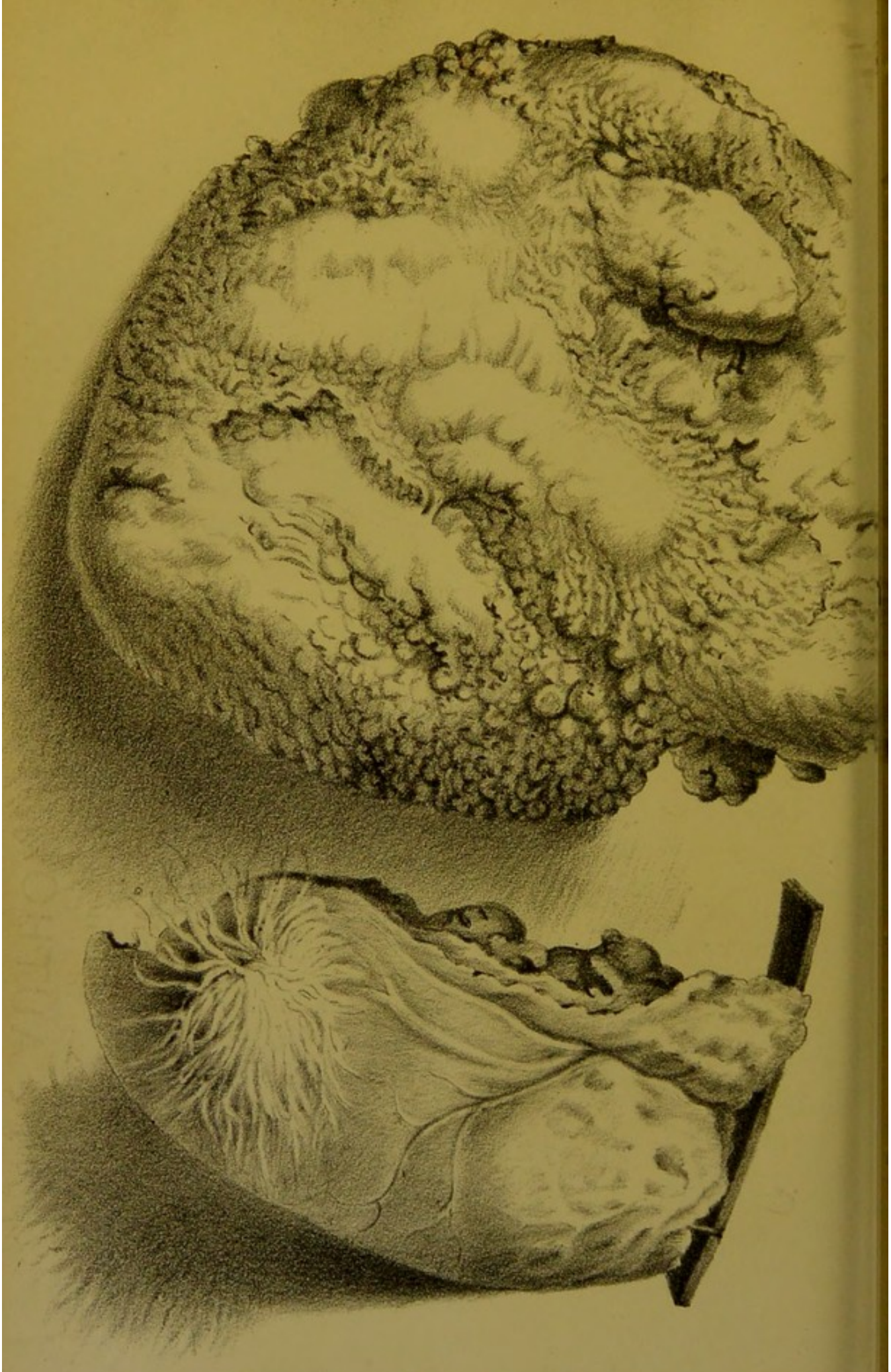
Clinical and pathological notes on pericarditis / by W.T. Gairdner.



Drawn from PRESERVED SPECIMENS

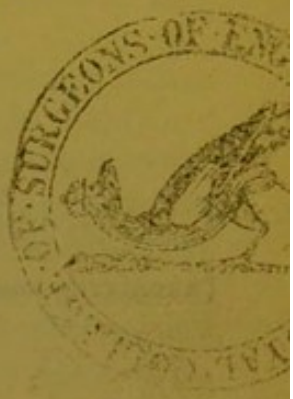
On stone by Neil Stewart.

W.H.M.ᶜ Farlane, Lithʳ Edinᵍʰ

CLINICAL AND PATHOLOGICAL NOTES

ON

PERICARDITIS.

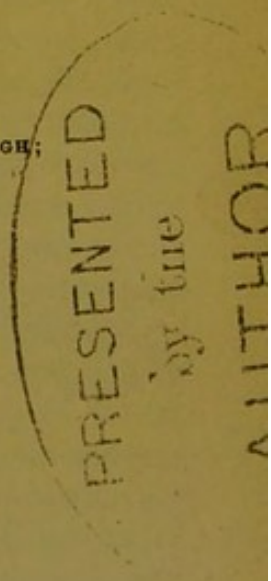


BY

W. T. GAIRDNER, M.D.,

F.R.C.P. EDIN.;

LECTURER ON PRACTICE OF PHYSIC, AND ON CLINICAL MEDICINE, EDINBURGH;
PHYSICIAN TO THE ROYAL INFIRMARY.



EDINBURGH: SUTHERLAND AND KNOX.

LONDON: SIMPKIN, MARSHALL, AND CO.

MDCCCLX.

may be present, when there is no pericarditis; 3d, It is not always easy to recognise friction-sound (as distinguished from other cardiac murmurs) even when it is present, and distinctly heard by the ear. Let me enlarge a little on each of these sources of fallacy.

1. *Friction-sound is not necessarily present in pericarditis.*—I do not mean by this to assert that cases of pericarditis often occur in which no murmur of any kind is present from first to last; but only, that for purposes of diagnosis a murmur is not to be counted on. I am not sure, indeed, that absolutely latent pericarditis (so far as friction-sound is concerned) may not occur;[1] at the same time, it is easier to believe, in a particular case, that we have missed the right moment—that the characteristic sound has been, or will be, there at some other time, than it is to suppose that the pericardial surfaces can have become rough and smooth again—that fluid and lymph effusion, and then adhesion, can take place—without any murmur at all. I am willing to suppose that the earliest period of pericardial effusion is not often, perhaps not ever, entirely devoid of friction-murmur. But what it is of practical importance to bring into strong relief is, that in the progress of pericarditis this most characteristic phenomenon may be absent for long periods in some cases, just as it may be present for long periods in others; that its presence and its absence bear no appreciable relation to the intensity of the disease; and that, therefore, you cannot practically make the diagnosis of pericarditis depend on the presence of friction-sound, nor can you gauge the pressure and extension of the disease by the amount and character of the murmur. This is surely a point of very great importance; and if I am correct in supposing that its importance is not fully brought out in the majority of works in the hands of our students and practitioners, it may be worth while to dwell upon it a little longer.

I think we may partially, but certainly not entirely, explain the singular diversities that occur in pericarditis as to the manifestation of friction-murmur. It is sufficiently obvious, and is indeed known to most auscultators, that the amount of fluid effusion has much to do with this. Once fairly established, the pericardial friction-murmur is less easily suppressed by effusion than the pleural; but only *less* easily. In the pericardium also, a really considerable effusion of fluid generally first muffles, then renders barely audible, and finally removes, the sound;[2] the friction-sound becoming indistinct as the

[1] Dr Mayne has published one, if not two cases, where this may reasonably be suspected. I have seen several in which the murmur was so very slight, that the total absence of it in other cases is not, to me, at all improbable. For Dr Mayne's paper, see *Dublin Journal of Medical Science*, vol. 7.

[2] It is curious, and strongly indicative of the insecurity of our knowledge of pericarditis, that two such excellent authorities as Dr Latham and Dr Walshe should be diametrically opposed to each other on this point. Dr Latham has seen "a few instances" only in which the murmur disappeared during the stage of effusion; the *general* fact being "that the murmur which is produced by lymph deposited upon the surfaces of the membrane, is neither abated, nor

heart's sounds are gradually extinguished, and, like the heart's sounds, continuing audible longest, and being recovered soonest, towards the base. But this is not nearly the whole explanation. For, on the one hand, I have heard friction-sound when there must have been pints of fluid in the pericardium; on the other hand, I have listened for it, and *not* heard it, when there were not very many ounces. The latter case has occurred to me so frequently, that I have no doubt it has also occurred to many other observers. I have observed several instances of it, so far as I can judge from merely clinical observation, within the last few months; and I shall presently give one very notable case, in which the fact was rendered quite certain by an examination after death. Of the persistence of friction-sound in the presence of very large effusion, one of the earliest cases of which I have notes, was a most prominent example. It occurred during the prevalence of scurvy in 1847, and was one of the very few well-marked examples I have seen of the form of disease called, by Laennec, hæmorrhagic pericarditis. The patient was under the care of Dr Andrew.

R. L., a railway labourer, was admitted into the Infirmary on March 22, 1847, after an illness of eight weeks. He had first cough, afterwards dyspnœa, and latterly palpitations with uneasy sensations in epigastrium. On admission, there were all the signs of grave pericarditis. Cardiac dulness was from 4½ to 5 inches across, and extended as high as the second intercostal space. There was very marked friction-sound, most distinct over the lower sternum, indistinct both at base and apex. On March 29th, the dulness on percussion was much the same, friction-sound diminished. On April 10th, dulness, which had somewhat diminished, was as great as on admission; friction-sound faint, but still appreciable. After this, dulness appears to have increased, and friction-sound is not noted; it continued, however, as I distinctly remember, to be audible within a week of the patient's death, which happened on 2d May 1847.

On examination after death, the pericardium was found to extend from right nipple to an inch to the left of left nipple, and as high as the first rib. It contained nearly three pints of turbid, bloody serum. Surface of heart coated with shaggy prominent masses of tolerably firm lymph, of spongy consistence and red colour. Some of these masses were nearly half an inch in thickness, and united by bands to the opposite layer of pericardium. Both layers of the

---

abolished, nor otherwise altered in character by the serum effused within its cavity."—*On Diseases of the Heart*, vol. i., p. 130. Dr Walshe makes (and I think more correctly) a statement on this subject which is pretty nearly the converse of Dr Latham's proposition:—"The friction-sound of the past stage may be either completely gone (*i.e.*, when there is fluid effusion); or heard in some spots about the great vessels; or pretty generally retained in the præcordial region,—but this is *very rare* even with eight ounces of fluid, and it is scarcely possible with more than ten."—*On the Diseases of the Lungs, Heart, and Aorta*, p. 590. Dr Walshe also mentions the persistence of a local friction-sound with a large amount of fluid in some cases; of which he gives an instance very similar to the one recorded below. Dr Hughes goes to the opposite extreme from Dr Latham, and is undoubtedly incorrect, when he says that "the intervention of fluid between the roughened surfaces of the pericardium prevents their attrition, and *most commonly* removes the rubbing sound."—*Clinical Introduction to Auscultation*, 1854, p. 275.

pericardium, and also the left pleura, which presented firm adhesions, were covered with scattered miliary granulations (tubercle?) in the midst of the fibrous deposit and lymph. No tubercle in lungs or other organs.

The precise amount of fluid effusion that existed in this case before the friction-sound was suppressed, can of course only be roughly estimated. But from the consideration that from the first there was dulness on percussion up to the second rib, and that the friction-sound was heard only a week before death, the course of the disease being protracted over three months, it seems probable that there was within half a pint of the quantity found on *post-mortem* examination, viz., three pints. The bands uniting the two surfaces no doubt contributed to maintain the friction-sound.

Another circumstance which greatly influences the production of friction-sound, is the amount and character of the exuded lymph. It needs no elaborate demonstration to show that a large amount of very rough lymph, adherent to both surfaces (as in the preceding case), will be more apt to produce a considerable rubbing-sound than a smaller quantity of lymph closely adapted to each surface of the membrane; or that lymph on the anterior surface will be more certain to produce audible friction-sound than a similar amount on the posterior surface of the heart. In the early stage of pericardial effusion it is probable, too, that a good deal of the lymph is unattached, or loosely attached, and that it is therefore shaken or churned about from one side to another. Hence, probably, the extremely rapid changes that take place in the character of the murmurs at this stage of the disease.[1] Sometimes both the solid and the fluid exudation is of a singularly adhesive quality; at other times, it is little more than a thin serum with floating films of fibrin. All these circumstances, no doubt, modify the sound in an endless variety of ways. But still it remains a remarkable fact, that acute and general pericarditis, with only moderate fluid effusion, should sometimes be attended by so very little sound, or by none at all. The following case is a very striking one of the entire disappearance of friction-murmur, after it had been once recognised, and that without any appreciable increase, nay, even with diminution, of the fluid effusion:—

W. I., æt. 46, was admitted into the Royal Infirmary, under my care, in the summer of 1854. He was considerably worn out by sickness, and had been complaining for some time of cough and dyspnœa, which obliged him to sit erect in bed. Soon after his admission the disease was recognised as pericardial and pleural effusion, and after carefully listening several times, a *single* friction-murmur was detected exactly over the fourth costal cartilage. This was heard by Professor Fleming, now of Birmingham, and by others who happened to visit the ward at the time, and to interest themselves in the progress of the case. None of us had any doubt of its existence, or of its being a friction-murmur; the situation, the character, the variations from day to day, and the coincident evidence of fluid effusion being regarded as conclusive. After being heard distinctly at several visits, however, the murmur disappeared; and although the patient continued several months under observation, it was never again heard. What was still more unexpected, was that the amount of effusion,

[1] See a marked instance of this in Latham, *op. cit.*, p. 126, foot of page.

as indicated by the dulness on percussion, instead of increasing, remained stationary, and finally diminished considerably before the patient's death, which happened in the beginning of November 1854.

The body was carefully examined. Both pleuræ were found coated with tubercular lymph, and the lungs contained tubercles. The pericardium contained not more than six ounces of milky serum. Both its surfaces were covered with a thick coating of lymph, which had evidently been very rough, but of which several of the anterior projections were polished and rubbed away on their surface till they were as smooth as pebbles. (See Fig. I.[1]) To the finger this lymph felt extremely dense, not at all spongy; and it was arranged in the form of ridges and furrows, rather than of villosities. It was least rubbed on the sides and back part of the heart; most so over the right ventricle.

Nothing can be more striking than the evidence afforded by this case of the uncertainty of the diagnosis from friction-sound. Looking back on all the facts of the case from the point of view of the *post-mortem* examination, I presume that shortly before the patient's admission the period of exudation had ceased. The pericardium was then somewhat distended with fluid, and coated all over with soft, rough, spongy lymph, which, from its bulk and uneven surface, must have grated and rubbed at every movement of the heart. But this lymph must have rapidly condensed into firm granulations, and the parts of these most exposed to attrition must have become polished and rubbed away, so that, although no further effusion of fluid took place, the opposed surfaces gradually receded from each other, presenting fewer and fewer points of contact; and just as this process had reached the point of complete separation of the surfaces, the vanishing point of friction-murmur, the patient was brought under our observation. The diminishing power of the heart's beat probably also contributed greatly to this result. Had the patient recovered, it is very certain that friction-sound must have re-appeared; but it probably would have been of a very different quality from that first observed.

2. But to proceed to the next proposition. *The presence of friction-sound is not necessarily a proof of the existence of pericarditis.*—This sound proves, indeed, that there is something abnormal in the state of the pericardium or adjacent parts; but it neither shows the existence of active inflammation there, nor of any active morbid process whatever. It requires no lengthened argument, nor any considerable details of observations, to make it apparent that a condition of the pericardium like that represented in Fig. II. must necessarily produce a very decided rubbing-sound. Such friction-sound will, moreover, be very permanent. It will be every way a most marked and typical example of the phenomenon: for the close approximation of the surfaces, the freedom of movement of the heart, the considerable degree of roughness, will all contribute

[1] *Explanation of the Plate*—The left hand drawing, Fig. 1, shows the granulations of fibrin, polished by continued friction, in the case of W. I. The right hand drawing, Fig. 2, shows a brush of organised fibrin projecting from the heart, such as must necessarily have caused a permanent friction murmur.

to its production; and the only natural termination of such a murmur would be in the absorption or polishing down of the lymph, neither of them processes likely to occur at all rapidly. I have repeatedly observed during life what I have believed to be permanent exocardial murmurs; and on several occasions, on which they had been accidentally observed during a fatal illness, I have had the opportunity of tracing them to their cause in such a loose floating piece of organised fibrous tissue as is represented in the plate. Sometimes they have been heard with the first sound only, sometimes with both sounds; occasionally they have strongly resembled endocardial murmurs. I need hardly say that they were unaccompanied by symptoms, or that the symptoms which accompanied them had a cause independent of the cause of the murmur. But imagine for a moment the confusion which might arise to diagnosis, were one of these rough villosities to exist in a person having a recent attack of acute rheumatism, or a somewhat hypertrophied or strongly acting heart. To the mere auscultator, who depends upon his ear, and not on his intelligence, such a case would almost certainly be interpreted as acute pericarditis; and to any one, however judicious and full of experience, it would be a startling, if not a perplexing case. Moreover, it is precisely in the rheumatically disposed that such chronic conditions are apt to occur.

It is commonly assumed by writers on pericarditis, that those curious and very common sequelæ of disease, variously called "milk-patches" and "white lymph-patches," are practically soundless, as well as devoid of symptoms. This is a very convenient doctrine, and leads to great simplification of the subject of cardiac murmurs; but I am quite sure that it is not always in accordance with fact. On the contrary, although the paucity of symptoms in these cases is a great obstacle to their recognition during life, I have again and again detected, quite accidentally, over the cardiac region, murmurs which to the best of my knowledge and belief could have no other probable origin than these white patches; and I have in a few instances traced these murmurs also to their cause by a *post-mortem* examination, which has shown both the existence of the white patches, and the non-existence of any other recognised cause of murmur. Let any one, moreover, observe how very frequently slight, short, and ill-defined murmurs, especially with the first sound of the heart, may be discovered in perfectly healthy persons about the left border of the sternum,[1] at the level of the third and fourth intercostal spaces, or lower;

[1] Some such murmurs were probably included among those described as "xiphisternal" by Dr F. J. Brown. I have often thought it probable also that some, at least, of the murmurs heard over the pulmonary artery by Dr Latham in phthisis may have been exocardial. But I would not be understood to insist upon this as a complete explanation of the phenomenon described by that distinguished physician; nor can I profess to understand fully the distinctive characters of the "chisel-sound," as described by Dr Brown. See the *Association Journal* for May 1856, pp. 384 and 409.

let him study these murmurs in various positions of the body, and in connection with every known test of their character; and then let him study equally closely the habitual position, relations, degree of roughness, and general characters of the white patches, and I believe he will come to my conclusion, that these two classes of facts are in some degree related to each other. I do not assert, because I do not believe, that all murmurs in this situation are exocardial; still less do I believe that all white patches may be detected by the presence of such murmurs; but that many white patches, which are passed over as insignificant, and believed to be soundless, are attended with a certain degree of murmur, I have no doubt whatever.

Another element in the diagnosis of pericarditis which is usually stated much too absolutely, is the cessation of the friction-sound on the occurrence of adhesion. While I fully admit that this is often the case (and perhaps it will hold good, as a general rule, that all *well-marked* friction-sound ceases with the formation of adhesions), I believe it is very far from true that adhesion of the pericardium necessarily leads to the absolute suppression of murmur. Loose adhesions, indeed, I have oftener than not found to be associated with a degree of murmur; a murmur very different, it is true, from the friction of recent acute pericarditis in its most characteristic stage; but quite sufficiently resembling friction-murmurs in general to be readily suggestive to some persons of a recent attack of pericarditis. And that such murmurs may persist for a long time I have equally good evidence. I have watched them, in fact, in cases of old pericarditis, for months together, and lost them only on losing sight of the patient.

I hardly think that any hospital physician of experience will refuse to bear me out in the observation just made with respect to lax adhesions. Possibly the following remark may not meet with so ready an assent; but it is, nevertheless, no doubtful matter with me that, under certain circumstances, the most close and dense adhesions possible are compatible with the existence of distinct friction-sound.[1] For a long time I did not suppose this to be so, and accepted the common doctrine; but the following case can hardly be explained without a modification of it. The facts are so curious and important, that I give them at greater length than the preceding cases.

M. R., a dressmaker, æt. 27, and in wonderfully good condition of body considering the length of her illness, but of sickly appearance, and feeling very weak, was admitted to the Royal Infirmary, September 27th, 1855. Her history was as follows:—Two years before admission she had a severe cold, with much cough and spitting of blood, which continued for a month. She also felt pain in the left side, and considerable difficulty of breathing. Under the care of Dr Robertson, in the Infirmary, she underwent the operation of tapping on the left

[1] Dr Walshe refers to a somewhat similar observation. "It has appeared to me that sound is sometimes generated in layers of firm false membrane, though so perfectly agglutinated together that attrition or separation of the opposed surfaces is physically impossible."—*Op. cit.*, p. 255.

side, for a collection of fluid, on two occasions. In four months she was dismissed, being much better; but slight cough still remained, and about a year afterwards all her symptoms returned. Two months before admission she observed her feet to swell, and she began to have precordial pain, palpitation, and a peculiar sense of stuffing in the chest, with occasional fainting fits and night sweats, under which symptons she re-applied for admission.

On examination of the chest all the signs were found of a contracted left lung with excavation; and of disease also of the right apex. But the most striking phenomenon in the case was a loud friction-murmur heard at the base of the heart, over the upper part of the right ventricle. The heart's action was regular; the impulse strong; the apex beat in the fifth interspace, and there was no marked increase of the cardiac dulness on percussion.

After this the patient suffered many attacks of sinking or fainting, bearing a strong resemblance to angina pectoris, though not devoid of hysterical character. Vomiting also became a distressing symptom, and towards the end of November she had an attack of spitting of blood, which lasted several days. The appetite at the same time fell off; she became thinner, lost her sleep, and was evidently much exhausted. The catamenia were suppressed for three months before death, which happened on December 14th, 1855.

During the whole course of this fatal illness, from September to December, the murmur over the heart maintained its position and its essential characters with remarkable constancy. It was loud and rough with the first sound, very short and indistinct with the second. I frequently demonstrated it to pupils as a good example of a permanent friction-sound, probably caused by roughness or by very *lax* adhesions of the pericardium. When the action of the heart was feeble and fluttering, it was usually indistinct, but otherwise it was never at all difficult to make out. It was quite limited to the region indicated, over the upper part of the right ventricle and the pulmonary artery.

The following are the details of the *post-mortem* examination, transcribed with a little condensation from the very full and accurate report by Dr Haldane, the pathologist to the Royal Infirmary.

External Appearances.—Limbs fat and rounded. Fully an inch of fat in the subcutaneous cellular tissue of the abdomen.

Thorax.—The heart contained a good deal of blood, nearly all fluid; there were a few small loose, undecolorized clots.

The pericardium was found to be universally adherent over the surface of the heart. The adhesions consisted of a thin layer of cellular tissue, pretty readily broken down, but apparently of some standing.

The valves of the heart were quite healthy (*i.e.*, free from deformity). The heart was enlarged; it weighed 13 oz. The left ventricle was a little dilated, its walls retaining fully their natural thickness. On the anterior flap of the mitral valve, and also in the commencement of the aorta, were one or two small, smooth patches of atheromatous matter. The muscular substance of the heart was pale, being of a reddish-fawn colour, and was softer and more friable than natural. There was a layer of fat, from a line to a line and a half or two lines in thickness, over the greater part of the surface of the heart. On microscopic examination the muscular substance of the heart was found to be in a very advanced state of fatty degeneration, the muscular striæ having entirely, or almost entirely, disappeared, and being replaced by minute highly refracting granules and globules.

The left lung was universally and very strongly adherent. In removing it, a cavity in its external and lateral portion was broken into. The pleura was foundvery much thickened; at the apex it was converted into a fibro-cartilaginous mass, more than an inch in thickness; over the rest of the lung it was from half a line to two lines thick. The lung was compressed and contracted; its greatest length was $5\frac{1}{2}$ inches, breadth $3\frac{3}{4}$ inches. The upper lobe was most compressed. This lobe contained two or three small cavities a little larger than horse-beans, and lined by distinct membrane. The lower lobe con-

tained a large oval cavity about three inches in length, bounded only by the thickened pleura, and crossed by altered bronchi and vessels. There was a very little old tubercular matter, in the form of small granulations scattered here and there through the pulmonary tissue, which, though crepitant, was somewhat collapsed. The lower lobe was to a great extent occupied by the large cavity; otherwise its tissue was similarly affected.

In the right lung there was found a cavity at the apex, and one or two smaller ones in the upper lobe, with a good deal of tubercular matter, partly in the form of grey granulations, partly of yellow and softening tubercle. The lung was universally adherent, the adhesions fibro-cartilaginous at the apex.

Abdomen.—The liver and kidneys contained an excess of fatty granules in their epithelium: other abdominal organs normal.

We have arrived, then, at the conclusion, that chronic roughness of the pericardium of any kind, and under some circumstances adhesions, even when close and universal, may cause a distinct friction-murmur. Now observe the practical effect of these facts upon the diagnosis of acute pericarditis. We cannot safely assume that pericarditis exists, when we have discovered a friction-murmur, single or double; and least of all can we make this assumption safely, in the case of a rheumatic subject. The less distinct the murmur, the greater is the caution required in pronouncing it to be a murmur from acute disease; because the probability is the greater that it may have been an old murmur overlooked till now. In second or third attacks of pericarditis, the disentangling of old from new murmurs is a matter of still greater difficulty, nay, it is often an impossibility. It is only, therefore, when the murmur *arises for the first time under observation*, or when it accurately coincides with the development of symptoms, or where it corroborates and explains the symptoms and the other physical signs already existing in such a manner as to leave no doubt of its nature, that we are justified in assuming that a friction-murmur over the heart is pathognomonic of acute pericarditis. I shall have occasion to glance at this subject again in a subsequent part of this paper.

3. But the difficulties of diagnosis that attend friction-murmurs are not yet exhausted. For *the distinction of exocardial from endocardial murmurs is not always easy, nor to be effected by the ear alone;* it is, on the contrary, a matter requiring a really refined and *intellectual* diagnosis; one, too, in regard to which I believe no physician need be ashamed to confess that he has been occasionally mistaken.

This reflection is, with me, no new one. More than ten years ago, I was startled by hearing in public the diagnosis of an alleged case of pericarditis called in question by a physician who had given great attention to the subject of auscultation. The impugned diagnosis was defended on the principle, that it was impossible for a physician of considerable experience in hospital practice not to have learned exactly what a friction-murmur was, and what it was not. And, no doubt, this view concurs with the teaching of many of our manuals, which lay down with much more precision the fact of the distinction, than the mode in which the diagnosis is to be made; leaving

it to be inferred that the difference between the sounds is one which the ear can readily appreciate,—a mere *difference of sound*, in short; which may be heard, but cannot be talked about or reasoned upon. And such, I suspect, is still the way of thinking of many who take their impressions upon this subject from authority, without having had ample opportunities of correcting them at the bedside.

At the time the incident above-mentioned occurred, I was not without a misgiving, that the physician whose diagnosis was attacked in this case might possibly have failed to do justice to his own opinion, by not submitting it to a fuller analysis, and thereby being prepared to meet criticism on a different ground. This feeling has since been confirmed by numerous personal observations, and by the progress of clinical and pathological knowledge. Indeed, it is impossible, in the face of the carefully recorded experience of Dr John Taylor[1] and others, to deny the serious fallacies attaching to a mere *aural* diagnosis of pericarditis, as founded on the recognition of friction-murmurs by their acoustic characters. That these murmurs may be securely so recognised in some cases is, indeed, true; but in very many instances this is not so; and I have very little doubt that, in even the majority of cases where friction-sound is recognised, it is known to be such by the circumstances in which it occurs, rather than by the mere character of the sound itself. In other words, the recognition of the friction-sound, usually placed as the first step in the diagnosis of pericarditis, is often, in fact, the last; we recognise the sound as friction, because it accompanies the other elements of a diagnosis of pericardial exudation, instead of presuming pericardial exudation to exist, simply on the ground that we hear a friction-murmur. Let all who are disposed to think otherwise remember these facts:—1st, That the finest and most remarkably endowed ear in Europe, that of Laennec himself, must have again and again listened to the pericardial friction-murmur with all the advantage derived from the knowledge, personally worked out, of its pleural correlative—but that Laennec knew no distinction between the *bruit de soufflet* and the rubbing-sound in the pericardium; 2dly, that Dr Latham, who discovered the relation of the bellows-murmur to acute rheumatism in 1826, tells us that he observed and noted it for years, under the impression that it was produced in the pericardium, and without accurately distinguishing from it the less frequent and more characteristic murmur of attrition;[2] 3d, that Dr Stokes, who was, unquestionably, the first physician in this country to make the distinction in question (though Bouillaud and Watson arrived at the same discovery independently), is even now most careful to avoid an un-

[1] See the admirable abstract of Dr Taylor's papers, in the *British and Foreign Medical Review*, vol. xxiv., p. 549.

[2] "I am now fully aware that, for a series of years, half the cases at least which I regarded as inflammation of the pericardium were in fact inflammation of the internal lining."—*Latham, op cit.*, vol. i., p. 123.

due reliance on the mere "acoustic character of the sound," which he regards as only one out of many means of diagnosis;[1] 4th, that Skoda, the greatest living auscultator of Germany, expressly declares that he knows no sign by which, apart from a consideration of their rhythm, friction-sounds can be distinguished from endocardial murmurs.[2] These circumstances are surely calculated to make us distrust the extreme facility with which this important subject is got over in many of our elementary works.

But it is not my purpose to enlarge on this general statement of a difficulty. My object is rather to illustrate, by one or two examples (for the complete discussion of the subject would be ample material for an entire paper), the circumstances under which the difficulty is most apt to occur.

It has occurred to me frequently to observe cases in which murmurs *heard exclusively at the apex of the heart* were probably exocardial, while presenting most of the characters of the mitral murmur. In the following case this difficulty presented itself in a well-marked form, and led me into error for a time, although the error was soon corrected.

H., a tailor, rather slender and delicate looking, was first brought under my notice in the autumn of 1852. He was sent into the Royal Infirmary by Dr J. D. Gillespie, whom he had consulted at the New Town Dispensary the preceding day. He was found to be labouring under symptoms of old-standing cardiac disease—viz., palpitation, dyspnœa, oppression. The action of the heart was hurried and preternaturally strong. There was an obscure thrill communicated to the precordial region, and, on auscultation, a pretty distinct though not very prolonged murmur was heard at the apex, interposed between the second and the first sound of the heart. (I regarded the murmur as diastolic, *i. e.*, following the second sound; but, having frequently observed it since that time with more care as to its exact rhythm, I now believe it to have been then, as it certainly was afterwards for long periods together, a murmur slightly anticipating the first sound—*i. e.*, a presystolic rather than a diastolic murmur.) There was no dropsy, and no lividity. The first sound was muffled, the second distinct, and, I think, preternaturally strong.

The first impression I formed of the case, was that it was one of mitral obstructive disease, with diastolic murmur; the history of an old cardiac affection having, of course, contributed to puzzle the diagnosis. On meeting Dr Gillespie, however, the next day, I was surprised to hear that he had regarded it as a case of pericarditis; and a little further consideration and observation of the case itself, convinced me not only that I was wrong in the first instance, but that I had been misled by the occurrence of the murmur at the apex into what might be called a prejudiced appreciation of its acoustic character, which was by no means so distinctly endocardial as I had at first supposed it

---

[1] *Diseases of Heart and Aorta*, p. 33. It is right, however, to remark that Dr Stokes considers the absolute difficulty of diagnosis, as between pericarditis and endocarditis, to have been over-estimated (probably referring to Skoda's views, which see below).

[2] "I believe I have found by experience, that a friction-sound in the pericardium may simulate every kind of murmur which can arise within the heart, with the exception of the musical murmur; and, on the other hand, that every variety of the pericardial friction-sound may be produced in the interior of the heart"—Skoda, *Abhandlung über Perkussion und Auskultation*, Wien, 1850, p. 206.

to be. In fact, a few days observation of the changes taking place in the murmur converted my ear, as it were ; and it became plain, that the case was one of chronic pericarditis, with little, if any, fluid effusion, and a murmur which varied a good deal both in its character and limits. When Dr Gillespie heard it a second time, he assured me that it was quite different in character from what it was on the visit of the patient to the dispensary ; and, during a very few days after this, it underwent further alterations.

Ultimately, this man made an imperfect and slow recovery ; and I have no doubt whatever that extensive adhesions were formed. The action of the heart continued long irregular, and generally inordinate ; there was often that peculiar "jogging action" alluded to by Dr Hope, and which, though certainly not peculiar to adhesion, is often found in connection with it. Moreover, the movements of the organ were accompanied by retraction in several intercostal spaces ; the sounds at the base were unusually distinct ; and both action and sounds were violent out of all proportion to the strength of the pulse at the wrist. Notwithstanding these symptoms, he had no dropsy, and no serious pulmonary complication ; and he had confidence enough in his own life to marry within a very few months after he left the infirmary. I have seen him since repeatedly, at long intervals, the last occasion being about eight or nine months ago. There was then considerable hypertrophy, and all the symptoms above mentioned, but still no dropsy, nor any of the usual concomitants of valvular disease. And there was, on every occasion on which I have examined his chest, *a very peculiar rasping murmur, slightly anticipating the first sound at the apex*, but not either masking the sound, or continued into the interval between the first and second sound.

In the acute or sub-acute stages of pericarditis, attended with effusion of serum, the fact of that effusion, as ascertained by percussion and by the modification which it causes both of the sounds and of the murmurs, becomes a valuable aid to the diagnosis ; for murmurs which exist simultaneously with evidences of fluid in the pericardium, and with symptoms of acute disease, may generally be safely presumed to be exocardial, at least in part. But when fluid effusion is not present, and when the symptoms are, as in the preceding case, more those of old-standing than of acute disease, the difficulty is greatly increased of forming a correct diagnosis ; for in such circumstances there is a probability that valvular deformity may concur with a pericardial affection to create murmur. This probability depends not only on the well-known tendency of the outer and inner surface of the heart to become simultaneously affected in the course of acute rheumatism, but on the presumption, which I have reason to believe supported by good evidence, that adherent pericardium, by its effect on the movements of the heart, may sometimes lead to a secondary endocardial murmur. I base this statement (which I admit requires further corroboration) chiefly on the observation of one very interesting and protracted case of adherent pericardium, which was for many years under my medical care before it terminated fatally last summer, and which I abstain from inserting here at length, only because it contains data too complicated for consideration in connection with the present paper. The main facts connected with the occurrence of murmur may be very shortly stated. The usual state of the heart's action was that of considerable excitement, with a marked degree of "jogging"

movement, and retraction of the parietes at several intercostal spaces. The history was a long and complicated one, even when I first saw the case, but there had been traces of early rheumatism. Ordinarily there was no murmur, or at most a little roughness or reduplication of the sounds; but on various occasions during the course of a long illness I detected transitory murmurs, which appeared to me to be blowing murmurs, both in the region of the apex, and between the apex and sternum, always with the first sound of the heart. What was the real nature of these murmurs? On this subject I had many doubts during the life of the patient; and, as it happens, these doubts were not resolved by the *post-mortem* examination. The heart was found somewhat dilated and hypertrophied; but I have often seen more, both of dilatation and hypertrophy, without any murmur. The valves were perfectly free from deformity, and the endocardium had apparently never been the subject of any disease leaving even a trace behind. Nor were there any of the usual symptoms of regurgitation at the mitral orifice; no dropsy, no hæmoptysis, nor indeed any marked pulmonary symptoms, except such as were accounted for by an old pleuritic attack, which had to a great extent disabled the left lung. Were, then, these murmurs, though of well-marked blowing type to my ear, after all exocardial? The adhesions were universal, and very firm; but in the case of M. R., formerly related, we have seen that this does not necessarily exclude the possibility of murmur. Against this idea, therefore, I have only to oppose the strong impression of the ear (*valeat quantum*) that the sound was not at all of the rubbing type, but decidedly of blowing character. The reader already knows how little I am disposed to place absolute reliance on such an impression; but in the present instance I incline, on the whole, to think it was correct, and that these transitory murmurs indicated something quite different from the permanent condition of adherent pericardium; perhaps an occasional slight degree of regurgitation through the auriculo-ventricular orifices. Be this as it may, the fact of the occurrence of an apex-murmur in connection with old pericarditis, and without any positive valvular deformity or endocardial disease, is plainly an extremely important one in its bearing upon diagnosis; for such a murmur would be almost certain, unless it possessed very strongly the character of friction, to mislead the ear into the belief of disease of the mitral valve. I have seen several cases in which this difficulty of diagnosis has arisen, and more than one in which, after every care had been employed, the true solution of the murmur remained, after all, doubtful.

The following case is valuable, as showing that a murmur, regarded as endocardial during nearly a whole month of observations by a number of educated ears, may nevertheless have had an exocardial origin:—

A. P., æt. 45, a widow, acting as a night-nurse in the Royal Infirmary, came under my care on the 25th of February 1853, labouring under febrile oppression,

with a tendency to coma, and a degree of anasarca of the face and limbs. The respiration was slow, and forced; the voice husky; the tongue dry; the pulse 100, and small. The epiglottis was found swollen, but there was no laryngeal stridor. The cardiac dulness was increased in extent, and there was an obscure murmur. When the urine was produced, it was found to be (after brisk medicinal action on the bowels) not scanty, of specific gravity 1010, highly albuminous, containing abundant epithelial elements (doubtfully renal), and a few blood discs. Notwithstanding the very active employment of purgatives, the coma deepened; the face became slightly erythematous; and the patient died on the 2d of March, five days after she came under my care. During this short period, the signs connected with the heart were the subject of very frequent observation; and, owing to circumstances presently to be stated, the *post-mortem* examination excited great interest in the hospital at the time. Without dwelling on the facts relating to the general symptoms or *post-mortem* appearances, which were in all respects those of Bright's disease terminating in uræmia, I will extract from the notes made at the time the successive observations relating to the heart's sounds and their explanation.

1. *On admission.*—"Cardiac dulness increased in extent, but not well defined; at the apex of heart there is an obscure murmur; at other points there are variable traces of murmur, strongly resembling friction, especially at the base, when she is sitting upright."

2. *Day after admission.*—"The cardiac murmur much more distinct; it has now a distinct friction character, being superficial and rough. The extent of cardiac dulness is diminished as compared with the day of admission."

3. *Two days after admission.*—"Friction-sound well marked; percussion-dulness of heart still diminishing, not much greater than normal."

4. *Three days after admission.*—"The cardiac dulness as before. Friction-sound much more distinct and very harsh."

5. *For days after admission.*—"The friction-sound, though well marked, not so distinct; there is a murmur attending the first sound of the heart which appears much deeper than the other, and *is thought by some to be endocardial.*"

6. *Same evening as* (5).—"The cardiac dulness not perceptibly increased; line of demarcation well defined on the right side. The impulse at apex very strong, and somewhat diffused. On auscultation there is no murmur whatever to be detected, though listened for with great care (J. K.). Pulse strong, about 90 per minute."

Death occurred at five o'clock next morning.

The last of these observations (6) is noted upon the authority of a gentleman, then acting as one of my assistants in the wards, and now well known to many persons as one of the naturalists engaged in Dr Livingstone's expedition. The rest of the notes were made at my special request, in such a manner as accurately to describe the impressions conveyed to those who observed the case along with me in regard to a murmur which excited great attention at the time.

The cause of the qualified opinion introduced into the note (5) was as follows:—It was ascertained that the patient had been under treatment in the hospital during almost the entire month of January preceding her admission to my ward, under the care of another physician; and that a murmur then existing had been regarded as exclusively endocardial; the idea of a pericardial friction-sound having never occurred to any one, although the case was repeatedly examined, in the presence of many clinical students and junior practitioners; and although some doubts were entertained whether the mitral valve, or the aortic, or both, were affected. I was informed of these facts by some of the clinical clerks previously concerned in the case, and immediately invited them to re-examine it along with my own assistants. The result of this examination was the note (5); it was generally agreed that a friction-sound was probably audible, but those who had previously heard a blowing murmur

thought that it could still be detected. I myself continued to believe the sounds heard to be friction only.

Under these circumstances, the *post-mortem* examination, which took place in the theatre of the hospital on March 3d, excited more than ordinary interest. A considerable concourse of students and of clinical clerks was attracted to the theatre; and the following facts were ascertained :—

"The pericardium contained from two to three ounces of rather turbid yellowish fluid, containing some shreds of fibrin. On both surfaces of the pericardium a thin and partially distributed layer of fibrin, generally about one-eighth of an inch in thickness, easily torn, and easily separable from the surfaces of the membrane. No congestion of the vessels of the pericardium. Over the right ventricle towards the base, and on both ventricles towards the apex of the heart, firm patches of opaque, pearly-white false membrane, presenting a shreddy and villous aspect in the case of the patch nearest the apex. These patches were separable without much difficulty from the subjacent membrane, but were evidently of different date and consistence from the softer deposits of lymph above mentioned. Heart generally enlarged and hypertrophied to a moderate extent. Left ventricle chiefly affected. Slight atheroma on mitral and aortic valves."

It is only necessary to state, further, that no doubt was entertained among those present at the *post-mortem* examination, that the murmur heard at the earlier date, and noted at the time as "a blowing murmur, heard with the first sound loudest at the apex," was in reality due to the roughness caused by the lymph-patch above described, and which, in its degree of firmness and organisation, corresponded in all points with the theory of an attack of pericarditis of a few months' standing. Further, the symptoms of dry cough, nausea, and vomiting, which the patient had experienced during November, added to pain in the chest anteriorly, and great dyspnœa, which she complained of in January, agree with the idea of an attack of pericarditis; although these symptoms, no doubt, were in part owing to the bronchitis which existed on her first admission, and to the renal disease.

In conclusion, I would place on record, very shortly, some of the more important observations which remain to be made on this subject.

We have seen that a friction-murmur may readily be mistaken for a mitral valvular murmur, and *vice-versâ;* the conditions being, that it is limited to the apex-region, or loudest there.

A friction-murmur is still more apt to be mistaken for a murmur of tricuspid regurgitation; because such murmurs (and they are not at all uncommon) are usually heard distinctly over the right ventricle, which is precisely the most ordinary seat of friction-sound. The purity or roughness of the second sound, and the presence or absence of pulsation in the veins of the neck, may assist in determining the character of the murmur; but negative signs must not be trusted to. Tricuspid murmurs have, moreover, in a very peculiar degree, the "superficial" character considered by many authorities so characteristic of friction.

A somewhat similar remark applies to pulmonic arterial murmurs. They exactly correspond in situation with one of the commonest sites of the friction-sound; they may coincide with both sounds of the heart; and, on the other hand, friction-murmur may be heard only with the first sound. The character of both is intensely superficial. As the true pulmonic murmur is very rare, it is generally safer, unless

there be strong evidence to the contrary, to presume that a murmur in this position is exocardial, than to suppose it endocardial and of organic origin.

I was on one occasion, however, greatly deceived by the application of this principle. In a case of incipient phthisis with distressing palpitation and strong heaving action of heart, and with an ill-defined murmur, or rather roughness, with the first sound over the third left costal cartilage, I believed chronic or sub-acute pericarditis to have existed and to have left partial adhesions. On the death of the patient some time afterwards, it was found that the only abnormal condition of the heart itself was an enormously large and open foramen ovale, which had caused so little of lividity or cardiac disorder, that the physician who saw the patient in his fatal illness had no suspicion, I believe, of any serious disorder in the heart at all. *Quære*—Was the murmur in this instance caused by the malformation, or was it an instance of Dr Latham's pulmonic murmur in phthisis?

Aneurismal murmurs may not unfrequently be mistaken for pericardial friction-sound, and *vice-versâ;* especially when the aneurism is in the ascending aorta. The risk of error will be all the greater, if the aneurism be attended, as it often is in these cases, with marked disturbance of the circulation, and pain referrible to the heart. It is useful to recollect, in such cases, that pericarditis is very much more frequent than aneurism in persons under 35 years; and this rule applies with all the greater force in proportion as the patient is younger. But aneurism may occur in a young subject; and further, aneurism of the ascending aorta may become the cause of pericarditis. I have seen several instances of this complication, and have recorded one in the case of a young girl, in the *Edinburgh Medical Journal* for July 1856, p. 87.

Aortic valvular murmurs are less liable than any others within the heart and great vessels, to be mistaken for exocardial murmurs; although their frequent correspondence with both sounds of the heart, and their rough grating character in some instances, gives them a general resemblance to the friction-sound, greater than that of any other endocardial murmur. An aortic murmur which accompanies the second sound can never be mistaken for friction-sound, if it be distinctly prolonged into the neck with a loudness in any degree proportionate to its loudness over the heart. But, in the case of a murmur with the first sound, this is not a safe criterion; for a functional murmur in the vessels of the neck may often accompany a pericardial friction-sound, and may be even louder than the friction-sound itself. It is better to observe accurately the limits and direction of propagation of the sound within the bounds of the thorax, as functional murmurs in this situation are of less frequent occurrence, and are less easily produced by the pressure of the stethoscope.

In many cases, therefore, where a diagnosis rests in any degree upon the presumed presence of friction-sound, the greatest care is neces-

sary to avoid fallacy; and although Skoda's observations on this subject may, as Dr Stokes appears to think, bear a little too strongly, on the whole, in the direction of exaggeration of the practical difficulties of the diagnosis, they are unquestionably well founded in detail, and should be known to every practitioner. Their only effect on the mind of an observant physician will be to prevent his committing himself to an opinion on insufficient grounds. For myself, I can say, that I hardly ever arrive at the conclusion of determining a friction-murmur to be such, without having first applied to it, by a sort of exhaustive analysis, the testing characters of every other well-known cardiac murmur.[1] And, by exercising this degree of caution, I feel assured that I have been saved from important errors.

In the next part of this paper, I shall deduce some important consequences, as regards the frequency, progress, and treatment of pericarditis, from the preceding remarks, and from renewed observations.

II. *Frequency of Pericarditis and its Results, as indicated by Examination after Death.*

THE uncertainty which we have seen to be inherent in the diagnosis of pericarditis during life makes it impossible to form a trustworthy idea of its frequency, either absolutely, or as a morbid state requiring the assistance of the physician. Besides, almost all the detailed researches existing on this subject relate exclusively to rheumatic pericarditis, which, though perhaps the most frequent form in practice, taking mild and severe cases together, is by no means to be considered as forming a guide to the characters of the disease as a whole. No one has yet attempted (so far as I know) to estimate from clinical data alone the frequency and results of pericarditis, in all its forms, and over a field of practice quite general in its character.[2] Indeed, it is evident that the difficulty of procuring sufficiently accurate data for such an attempt, is almost insuperable. We must, therefore, fall back on the pathological records of large hospitals, or of unselected aggregates of cases, for such data as may throw light upon this question.

Louis (Mémoire sur la Péricardite[3]) was the first who made an approach to a correct use of the anatomical data available for this purpose. Most of the preceding inquirers had failed to appreciate rightly the important information given by morbid anatomy, as to

[1] This is the more necessary, as none of the absolute diagnostic tests proposed for these murmurs can be relied upon; and even that which Skoda indicates as the most worthy of confidence,—the non-correspondence of the murmurs, in point of time, with the sounds of the heart,—is only applicable, as he states, to prolonged murmurs; and not to all even of these.

[2] Dr John Taylor, indeed, has recorded his whole experience of "severe" pericarditis to have been about 1 in every 80 cases in University College Hospital. But the extent and character of the field from which that experience was drawn is not detailed, and it is very probable that his cases were, more or less, *selected* for purposes of clinical inquiry. Besides, the statement itself presents elements of vagueness which make it useless in a numerical point of view.

[3] *Mémoires ou Recherches Anatomico-Pathologiques*, etc., 1826.

the recoveries from pericarditis; and almost all of them had stated the facts, even of the recently fatal cases, in too isolated a form to be of much service in a numerical point of view. Nevertheless, Louis collected from various authors 1263 cases in which the state of the heart, as found on dissection, was recorded. Among these he found that 36, or 1 in 35, were cases of recent pericarditis; while 70, or 1 in 18, were instances of adherent pericardium, which Louis regards as indicating the cure of a pre-existent inflammation. He remarks, however, with truth, that the average as to both these results must be affected by the circumstance, that cases in which the heart was carefully examined are necessarily procured in preponderating numbers from works on cardiac pathology, in which, of course, pericarditis and its results may be expected to be unduly frequent. In other words, the cases are virtually a select class.

To get rid of this source of error, Louis gives the results of a further series of 443 unselected cases examined by himself. Among these cases Louis found 7 cases of recent pericarditis, or 1 in 63; and 11 of adhesions, or 1 in 40. From which he concludes that, in about 1 in 23 of the subjects examined by him, there either existed at the time of death, or had been present at a former period, a well-marked attack of pericarditis.

These results are not easily brought into harmony with those of other and more recent researches. Generally speaking, it appears from the latter, either that pericarditis and its results were more rare in Louis' field of experience than they are in other hospitals; or, what is perhaps more probable, that from only noting the more serious, and disregarding the slighter cases, Louis underrated the frequency of pericarditis in his own experience. This last conclusion seems to be strongly borne out by the fact, that in 4 out of the 7 cases of recent pericarditis Louis records details as to the quantity of fluid in the pericardium, and it was in two cases upwards of a pint, and in one eight ounces,—being in one case only indicated as "un peu de sérosité;" whereas it is a fact well known to morbid anatomists, that very many cases of this latter degree of pericardial inflammation usually occur for one in which the effusion is so large as it was in the rest of Louis' cases.

Among the more modern observers, Dr Taylor[1] found recent pericarditis in 16 out of 355 fatal cases in University College Hospital, or 1 in 22 cases. Mr Wilkinson King,[2] in 665 fatal cases occurring in Guy's Hospital, found 20 of "acute, recent, and fatal, or rather final pericarditis, or 1 in 33¼; while Dr Thomas K. Chambers,[3] in ten years' experience of St George's Hospital, found, among 2161 fatal cases, 135 of recent inflammation, *i. e.*, pus, or soft fibrin in the pericardium. This last observer thus raises the proportion of cases of acute pericarditis among the fatal cases of an hospital, from 1 in 63 (as stated by Louis) to 1 in 16.

[1] *Lancet*, 1845-46 Vol. ii. for 1845, p. 13. [2] *Lancet*, November 29, 1845.
[3] *British and Foreign Med.-Chir. Review*, vol. xii., p. 493.

Again, in regard to adhesions, Mr Wilkinson King found these in about 1 out of 23 cases; Dr Taylor, in 1 out of 16; and Dr Chambers, in 1 out of 25 cases. This, however, includes both universal and partial adhesions. Universal adhesions were found by Mr King only in 1 out of 31½ cases; and by Dr Chambers, in 1 out of 42 cases. This latter result, it will be observed, is not very different from that of Louis, who may therefore possibly be conceived to have overlooked the greater number of partial adhesions in his summary of experience.

Not being altogether satisfied with these results of previous investigations, as to the frequency of pericarditis in its different stages and forms, I determined to make a more particular analysis of some portions of my own hospital experience, on which I thought I could rely with confidence for the solution of some important questions bearing on this subject. My object was, however, not merely to seek a new basis for observations similar in kind to those of Louis, but to assist, if possible, in defining more accurately than hitherto the significance of the clinical phenomena usually considered distinctive of pericarditis.

I have already pointed out that the presence of friction-sound is not necessarily a proof of the existence of pericarditis, at least in the acute and general form.[1] But as any local roughness in the membrane will produce friction-sound, and as friction-sound is apt to be considered as indicating pericarditis, often without regard to its mode of origin and concomitant phenomena, I thought it desirable to keep in view all the lesions of the pericardium which might be conceived, at any period of their formation, to have given rise to such local roughness as could generate murmur. I do not care to enter too closely, for the present, into the pathological questions connected with some of these lesions. They may, or they may not, be entitled to the name of inflammation, as being similar in their mode of origin to the more decided sequelæ of acute disease. This question has been ably discussed by Mr Paget and others. But for practical purposes, all alterations of the pericardium which were or had possibly been capable of causing murmur, fell within the scope of my inquiry; and all such alterations must be regarded as coming within the clinical definition of pericarditis and its results, so long as friction-sound forms an important part of that definition.

I therefore proceed to inquire into the relative frequency,—

1*st*, Of pericardial adhesions;

2*d*, Of recent lymph or pus in the pericardium;

3*d*, Of chronic thickening of the pericardium, local or general, having the characters of a *possible* product of former inflammation;

[1] The lithographic plate by which this and other propositions in the former paper were intended to be illustrated, is furnished with the present article. I have to apologise to the reader for the delay; but the most repeated requests on my part have failed to get the drawing out of the artist's hands till a few weeks ago.

And I shall follow this numerical inquiry with some observations upon the relation of these different forms of lesion to one another, in certain cases,—reserving for another paper the more strictly clinical deductions from the inquiry.

To begin, then, with the crude facts, as taken from the different indices constructed (without any special view to this inquiry) for certain parts of the register of dissections, kept while I held the office of Pathologist to the Infirmary:—I have grouped the cases analysed into three series, because the amount of care bestowed in the recording of details was different in each series, and these differences are, in fact, the source of much of the instruction to be gained from the whole.

I. In a small series of 84 miscellaneous cases, observed with the most minute care to omit no single pathological phenomenon in a great number of organs, and recorded in such a way as to give the exact numerical frequency of the phenomena, I found,—

7 cases of pericardial adhesions—or, 1 in 12;

5 cases of recent inflammation (*i.e.*, lymph or pus in pericardium) —or, 1 in 17;

28 cases of chronic thickening, mostly of what are called white and smooth "lymph-patches"—or, 1 in 3.

II. In a larger series of 230 miscellaneous cases recorded, not with equally minute care as to numerical results, but still with fair and trustworthy accuracy (though without special reference to the present inquiry), I found,—

17 cases of adhesions—or, 1 in 14;

15 cases of recent inflammation—or, 1 in 15.

In this series I do not venture to appreciate the number of white lymph-patches, as it is quite certain that they must often have been passed over without being noted.

III. Finally, among 500 cases of all kinds, observed over a longer period, and not with so much accuracy as either of the other series, but in almost all of which the heart was examined, I found the frequency of adherent pericardium, *disregarding adhesions of no clinical importance*, to be 1 in 33—*i.e.*, 15 cases in all. These cases are recorded in greater or less detail in the *Monthly Journal* for Feb. 1851.[1] It is only necessary to mention here, that adhesions were regarded as of clinical importance, only when they were so placed as to involve the possibility of affecting seriously the movements of the heart. In the opposite circumstances they were disregarded.

If now, keeping in view the numerical details above stated, we assume for a moment that adhesions are the indication of a recovery from pericarditis, and that recent lymph and pus show forth a fatal

[1] On the Favourable Terminations of Pericarditis, etc.

pericarditis, we shall arrive at the result, that among the patients of a general hospital who die, from 6 to 8 per cent. die of pericarditis, while a proportion, varying from 3 to 8 per cent., have had the disease at a previous period, and recovered from it. But both these propositions, though in a certain sense true, require to be scanned much more closely, and will have to undergo considerable modification.

For, in the first place, in hardly any of the 6 or 8 per cent. of cases of recent exudation within the pericardium, can that exudation be regarded as the principal cause of death. In the very great majority the fluid effusion did not exceed an ounce or two; in a great many it would have had to be measured by drachms; in only a few was it more than 6 ounces. Yet in all the cases thus numbered, the exudation had the characters commonly ascribed to acute inflammation; only a few of them being complicated by dropsical or tubercular exudations in the pericardium itself. On the other hand, it appears from a detailed scrutiny, that the other organs, and especially the lungs and pleuræ, were almost invariably involved in the fatal disease in these cases; in several there was empyema, of much greater extent and intensity than the pericarditis; in a very great proportion there was pneumonia; in some tubercle, in some gangrene; some had been the subjects of severe operations, and others of severe acute or chronic primary diseases. In short, in all, except a very small proportion, the pericarditis was quite evidently a secondary and subordinate lesion.

These facts qualify, to a large extent, the numerical statements above indicated, as to the apparent frequency with which pericarditis leads to death. But the proportion of cases, as stated above, in which recovery may be presumed to have followed pericarditis in a mixed series of *post-mortem* examinations, must be considered as subject to equal modifications. For, from a further minute scrutiny of the cases of adhesion, it appears that in a large proportion the adhesions were of extremely insignificant amount, and hardly of a character from which acute inflammation can be inferred as a matter of certainty.

If we take, for instance, the difference between 1 in 33 and 1 in 12 (in the largest and the smallest series), we shall have pretty nearly the measure of the difference between the proportion of adhesions which were regarded by me, at the time and according to the record, as being of clinical importance, and those which were not so regarded; in other words, the proportion of adhesions not clinically important, probably amounts to the difference between 3 per cent. and $8\frac{1}{3}$ per cent. of all the cases examined. In 5 per cent., then, or 1 in 20, of miscellaneous fatal cases of the same class as those I examined, it may be presumed that there will be found adhesions of a character so slight, or so disposed, as not to be of clinical importance, and not to indicate with certainty a general pericarditis; while in only 3 per cent., in all probability, will there be found adhesions more generally distributed, or more inconveni-

ently placed. Moreover, among these last cases there will be a certain proportion in which the adhesions, though inconveniently placed, are not general. In the cases referred to, this proportion was about a third; and thus the proportion of cases of *general* pericarditis ending in *universal* adhesion is to be cut down, according to my records, to 10 out of 500 miscellaneous cases, or 1 in 50 = 2 per cent.

But now there arises a new question. We may fairly ask if *general* pericarditis does not occur without leading to adhesion as a necessary consequence. The discussion of this question will still further modify our position.

The compatibility of acute and general pericarditis with a termination short of adhesion, has been maintained by Dr Kirkes,[1] in opposition to the opinion of Dr Latham, and of the majority of modern authorities. The evidence founded on is the extreme frequency of white patches and thickenings on the pericardium, apart from considerable adhesion, and the relation established by the researches of Mr Paget[2] between some of these lymph-patches and the inflammatory exudations. Dr Kirkes thinks that the ordinary termination of acute and general pericarditis is in re-absorption of the exudation, with more or less complete restoration of the translucency and smoothness of the membrane; and that, to whatever extent this healing process remains imperfect, white patches are left as its permanent memorial. Adhesion he regards as quite an exceptional termination; general and firm adhesion as "an extremely rare occurrence," compared with the frequency of pericarditis.

There is something to be said for this opinion, if it be merely to the effect that all pathological processes in the pericardium do not necessarily terminate in adhesion. But everything depends upon what is meant by the term acute and general pericarditis. Like all other parts of the body, the pericardium is subject to slighter and graver degrees of inflammation. That the slighter forms may end in the manner supposed by Dr Kirkes is fully admitted by Dr Latham, and is in accordance with the general opinion; that the severer forms sometimes end in adhesion, could not be denied by Dr Kirkes. The question is altogether one of degree, and depends on the latitude given to the idea of acute inflammation.

My own experience is not decisive as to the question, whether acute and general pericarditis is susceptible of repair without adhesion. I have never yet seen a case in which *acute pericarditis, attended with considerable effusion*, had been cured, and in which (the patient dying from some other cause) the absence of adhesions has been demonstrated by a *post-mortem* examination. Yet such cases ought to occur, not very rarely, if the healing of acute and general pericarditis without adhesion were the ordinary rule, and healing by

[1] *Medical Gazette*, 1849. [2] *Medico-Chirurg. Trans.*, vol. xxiii.

adhesion the exception. Again, I have never seen a case of acute and general pericarditis fatal in the period of commencing repair—viz., when the fluid effusion had disappeared, leaving only the thickened pericardium and the lymph on the opposite surfaces—without observing the commencement of a process of adhesion. Dr Kirkes, indeed, considers this cohesion of recent lymph to be a different process from the formation of permanent adhesions; but he advances no proof that the opposing surfaces, once welded together by soft lymph, ever separate again. Nor can I imagine on what ground such an opinion can be sustained. It is to me very plain that cohesion of soft lymph is, in general, the first stage of permanent adhesion; and it is certain that when soft lymph exists in considerable amount on both surfaces, and the fluid effusion has been absorbed, the cohesion of the lymph-covered surfaces is the rule, and not the exception.

On the whole, then, I am disposed to concur in the main with Dr Latham in believing that a general and copious deposit of inflammatory products on the pericardial surfaces is rarely, if ever, followed by repair so complete as Dr Kirkes would have us believe. But this must not shut our eyes to the immense importance of the fact, that deposits of lymph do occur very frequently on the pericardium without being followed by adhesion. The significance of these local deposits, which occur in upwards of 30 per cent. (50 per cent. according to Mr Paget) of all hospital patients who die, is a subject of great interest to the practical physician as well as the pathologist. I must for the present, however, leave the prosecution of this part of the inquiry with the remark, that these local exudations, whatever their cause, may be expected to play some part in the physical signs, if not in the symptomatic history, of what is commonly called Pericarditis.

From this inquiry, then, it results,—

1. That general and severe pericarditis—*i.e.*, pericarditis attended with copious deposit of fibrin on every part of the membrane—commonly ends in adhesion.

2. That local exudation from mild pericarditis, and from the slighter forms of disease of the pericardium, may end in the production of local lymph-patches, or in local adhesions of greater or less extent (as described by Mr Paget).

3. That pericarditis, ending in *considerable* adhesion, occurs at one period or other of life, in from 2 to 3 per cent. of the patients that form the hospital population of Edinburgh, and that die in hospital.

4. That *less considerable* adhesions (not clinically important) occur in about 5 per cent. more (making altogether adhesions present in about 8 per cent. of the hospital population that die from all causes).

5. That lymph-patches, chiefly on the surface of the right ven-

tricle, or mere threads of adhesion at the extreme base (indicating the previous occurrence of morbid processes of a more local kind and of lesser intensity), occur in not less than a *third* of all the patients who die in Edinburgh Royal Infirmary.

6. That acute pericarditis, in actual progress, occurs in about 6 per cent. of the fatal cases; but that in very many of these cases it is slight, and in almost all of them subordinate to other grave constitutional or local diseases; so that primary and uncomplicated fatal pericarditis is a disease of exceedingly small mortality.[1]

7. That the healing or repair of pericarditis by adhesion, or by lymph-patches, must be regarded as a greatly more frequent event than its fatal issue; and that the formation of lymph-patches, as the result of slight and local irritation, is one of the commonest of morbid affections.

III. *Prognosis and Treatment of Pericarditis.*

In two previous sections of these notes, I have endeavoured to bring both clinical and anatomical observations to bear on some of the more obscure points of the natural history of pericarditis. In the present communication, I propose to investigate some of the practical questions arising out of the preceding details.

What is pericarditis, considered as a subject for treatment? How is the clinical idea of this disease to be defined and limited, as compared with the pathological idea of it? What are the circumstances that determine the necessity for this or that particular course of treatment in the individual case? These questions must, I think, have often occurred to most modern physicians who have thoughtfully studied the course of pathological research; they emerge, too, very directly and immediately from the inquiry to which this paper is a sequel.

That a friction-murmur, *per se*, cannot be taken as proof of pericarditis requiring active treatment, is very clear from the preceding observations. For a friction-murmur may be present, and may continue an indefinite length of time, when there is no pericarditis properly so called, but only a roughening of the pericardium, the result of a former morbid process; and further, pathological anatomy shows, that in a considerable proportion of cases, a condition of the exterior of the heart, which in all probability must have led to exocardial murmurs of some kind, has actually been present, and has passed away, if not without symptoms, at least without the least trace of a history of acute pericarditis. Whatever be the pathology of the white lymph-patches and their allied fibrinous deposits in the pericardium, it is impossible to attribute to most of them the history of an acute

[1] See, on this subject, Dr Chambers' analysis of 135 cases, only two of which appear to have been uncomplicated by either constitutional or local disease. *British and Foreign Med.-Chir. Review*, loc. cit.

inflammation, and it is equally impossible to deny them the power of producing a murmur. I am satisfied, in fact, on grounds previously stated (p. 8), that they do produce murmurs in certain stages of their progress; and that these murmurs must necessarily resemble, to a considerable extent, the friction-murmurs of pericarditis.

Friction-murmur, then, is only a sign that the pericardium is roughened; not a sign that it is inflamed or actively diseased. In other words, friction-murmur, though always a sign of disease, past or present, is one to be carefully scrutinized and judged by other signs and symptoms, before pronouncing it an indication of pericarditis requiring active treatment, or indeed any treatment.

Owing to the great difficulty, already pointed out in my first paper, of judging with certainty of the presence of friction-murmur in cases in which it is slight and ill-defined, I feel that it would be quite impossible to attempt any numerical estimate of the cases in which it has occurred to me to observe friction-murmurs, either *of insignificant clinical value*, or only thus far significant, that they require to be watched and studied, as the indications of a *tendency* to acute disease. I can only say, in general terms, that such cases are of considerable frequency, and would probably be discovered more frequently than they are if the state of the heart were carefully scrutinized by auscultation in every case. The stethoscope has in this particular, as in many others, operated upon the course of physical diagnosis somewhat after the fashion of the Romish confessional in moral diagnosis: it has brought many "secret sins" to light, and in the affairs of the heart has perhaps tended rather to exaggerate the importance of small aberrations than to furnish any new principle of treatment; possibly, indeed, it has in some hands proved to be even a dangerous instrument, by bringing strong remedies to bear, with injudicious rigour, upon comparatively insignificant diseases. Fortunately, the general diffusion of stethoscopic skill has, in this country at least, been closely accompanied by a bias towards the more sober and guarded use of heroic treatment, and hence this evil consequence of minute diagnosis has been gradually corrected. But I can recall many instances of patients frightened very unnecessarily by the observation of "something wrong" about the heart; and it is impossible to doubt that active treatment of one kind or other has often been applied to cases of supposed acute pericarditis upon indications which by no means justified it. Of course, the opposite error is also possible; but I think it will be generally admitted that the principles advocated in several of our standard works on the physical diagnosis and treatment of pericarditis expose us far more to the error of too meddlesome, than of too inert, practice.

There is one disease so notoriously the cause of pericarditis in a large proportion of instances, that few cases of it now pass under the notice of physicians without careful and frequent stethoscopic

observations of the heart. "Every prudent physician, I presume," says Dr Latham, "searches after it (pericarditis), day by day, with his ear in all cases of acute rheumatism."[1] This principle of daily stethoscopic observation is fully adopted by several other authorities; and Dr Stokes goes much further than to watch for the first development of friction-sound in pericarditis, inasmuch as he says "it cannot be too strongly impressed upon the mind of the practitioner that, valuable as the discovery of the signs of an inflamed pericardium may be, it is not for these alone that he is to look, but rather for the indications of excitement of the heart, whether attended or not by the signs of exocardial or endocardial disease." And he proceeds to remark that these indications may demand local depletion, "even though no friction-sound or valvular murmur whatever be present."[2] Dr Walshe gives instructions tending in the same direction, though not so precise in detail; he thinks, however, that "before the occurrence of friction-sound there is no certainty in the diagnosis."[3] And even Dr Hope (with less of special reference to acute rheumatism) recommends the hand to be daily placed on the precordial region in every severe inflammatory or febrile affection, with a view to the discovery of any excited action which might lead to the discovery of pericarditis by auscultation.

Such precepts are of unquestionable importance, for good or for evil; they are, moreover, well founded in fact and careful observation. In all that Dr Stokes, in particular, says about the very early diagnosis of pericardial irritation before the period of friction-murmur, I most entirely concur. But this refinement of diagnosis must be accompanied with great caution, and an almost indefinable practical instinct, or tact, in the application and withholding of remedies, to make it either safe or profitable to the patient. Dr Stokes, indeed, well remarks elsewhere,—and it is the absolutely indispensable corollary of his instructions, as above given,—that "the boldness of treatment often betrays the timidity of the practitioner; he is terrified at discovering the disease, and his mind is more occupied with its name than its nature or actual condition." It is to the great importance of a practice founded on the vital manifestations, rather than on the physical signs exclusively, or even chiefly, that I wish now to remark. I am well aware of the cautions given by Dr Stokes on this subject, which make his chapters on Pericarditis so full of the best kind of instruction for the practical physician; and it is no less a duty than a pleasure to refer, also, to the excellent article of Dr Sibson, in the *British and Foreign Medico-Chirurgical Review* for July 1854, as embodying much sound and well-considered doctrine with regard to the prognosis and treatment of pericarditis, particularly in its rheumatic variety.

During upwards of six years of almost constant attendance in the

[1] *Diseases of the Heart*, vol. i., p. 139.
[2] *Diseases of the Heart and the Aorta*, p. 93.
[3] *Diseases of the Lungs, Heart, and Aorta.* Second Edition, § 1197.

Edinburgh Royal Infirmary as physician, I have of course had under my care a great many instances of pericardial affections arising in the course of rheumatism, and a certain number also concurring with other constitutional or local diseases. Purely idiopathic pericarditis I have rarely, if ever, witnessed; and I am much inclined to believe that (as morbid anatomy teaches) it very rarely occurs as a severe or clinically important form of disease. Pericarditis from direct injury, or from purulent infection after injuries; pericarditis following erysipelas, or pleuro-pneumonia; chronic or subacute pericarditis in connection with unhealthy suppurations in various parts of the body; with epidemic fevers, especially small-pox and scarlatina; and, finally, pericarditis associated with Bright's disease,—have all fallen more or less frequently within my observation, and in many of these last-mentioned forms the issue has been fatal. But of *rheumatic pericarditis*, and of *pericarditis without previous disease*, acute or chronic, *I have not had under my own personal care a single fatal case.*[1]

I am particular in stating this fact, not for the purpose of making invidious comparisons, but that the truth, as regards the prognosis of rheumatic pericarditis, and the results of carefully adjusted and cautious treatment, regulated mainly by symptoms and vital manifestations, may be fairly brought into view. It is now a pretty well established fact that rheumatic pericarditis, whatever may be its ultimate bad effects as predisposing to hyper-

[1] Two or three facts require to be mentioned, not as qualifying this observation, but as removing from it the suspicion of latitude and vagueness. Very lately a case occurred to me of pericarditis, in a patient (E. J.) sent in from the jail as labouring under acute rheumatism of fourteen days' standing. There was no swelling of the joints, but very acute pains in the limbs and back. The pericardial effusion, which was considerable, rapidly subsided under a few leeches, with continued warm fomentations. The patient showed, nevertheless, a degree of prostration very unusual in rheumatism,—symptoms, in fact, more resembling pyæmia. After some time an abscess formed beneath the right sterno-clavicular articulation, and this was followed by a host of abscesses in almost every part of the body, *but in no instance primarily in the joints*, although the shoulder-joint and the sterno-clavicular were ultimately laid bare, the abscesses in their neighbourhood having quite destroyed the ligaments,—leaving, however, the articular surfaces comparatively unaffected. The patient died. About two years ago, three cases of rheumatic pericarditis, two of which were pretty severe, occurred to me at once. One of these, a girl from Larbert, passed through the disease, and recovered. She was retained under observation a considerable time, and was then dismissed, having been able to walk about freely for several weeks, and almost every trace of abnormal sound, as well as of abnormal dull percussion, having disappeared. She went home in good health, but took ill within a fortnight, and died. I did not hear of this till some time afterwards, and I heard of it with great regret, and not without suspicion of a relapse. On writing, however, to Dr Cuthill of Denny, I found that no doubt had been entertained that the cause of her death was *typhus fever;* that the disease was characterized by the usual symptoms; and that there was no complaint or suspicion of anything wrong with the heart during the whole period of the illness. I have no doubt that the diagnosis was correct, for there was reason to fear that the disease might have been contracted in the Royal Infirmary at the time.

trophy, or atrophy, or other chronic disease of the heart, is not directly fatal in a large proportion of cases; and it is therefore extremely probable, if not certain, that the violently perturbative practice of Bouillaud and others was the cause of many more deaths than were properly due to the disease itself, or than have usually been seen under a milder and more cautious system. I must add, that I have seen reason to suppose that rheumatic heart affections generally may be somewhat less common, and therefore probably milder in degree, in Edinburgh than in London, or at least in the great hospitals of the West End; and perhaps, like pneumonia, they may have been milder of late years. Still, I think I am entitled to add a strong and personal testimony in favour of the rapidly accumulating evidence, that rheumatic pericarditis, cautiously treated and not too much interfered with by special practice, is the very opposite of what it was supposed to be by Bouillaud, Hope, Graves, and even Latham; and that it does not require, as a general rule, violent remedies tc obviate its tendency to death, but is, on the contrary, easily relieved by mild and almost purely palliative measures, superadded to the general treatment of the rheumatic affection.

Let me endeavour to indicate, shortly, what has been my usual course of proceeding in dealing with such cases. In every case of rheumatic disease, whether acute or chronic, I have made, as a rule, at the commencement of treatment, at least one or two very complete investigations of the cardiac sounds and impulse, to be used as testing observations for the future. I have also, at the same time, carefully investigated the habitual and the existing state of the cardiac function, and the history of any uneasy sensations or functional phenomena indicating disease, which may have been present at a former period. This done, and the heart being found free from suspicion, I have in future observations dwelt as lightly as possible upon the local examination of the heart, merely assuring myself, from time to time, that there was no very material change requiring more systematic attention.

If a murmur has been present on admission, it has not been too hastily assumed to be a new morbid process, unless accompanied by pain, or by the signs of effusion, or by marked tenderness on pressure, either over the costal cartilages or in the epigastrium. I have not thought it too much to wait for twenty-four or forty-eight hours, before commencing treatment, for corroborative evidence of the existence of true pericarditis in a doubtful case. Of course, careful watching, and examination at least twice a-day, have been the rule in all suspicious cases.

But in many cases of murmur, even when decidedly originating under observation, it has been my practice to look for something more than murmur as an indication for commencing the treatment addressed to an acute pericarditis; and this for two reasons. The alarm created by the announcement that the heart is wrong, is, in susceptible subjects, a serious objection to making this announce-

ment on slight grounds. Having, accordingly, the clear conviction, for reasons which have been already submitted, that many friction-murmurs, unaccompanied by signs of effusion, or by cardiac symptoms, may be safely neglected; and being further of opinion that, as a general rule, the treatment of the rheumatic condition of the system is the best treatment also for the pericarditis, I have been slow to give effect to anticipations of evil founded on the presence of a mere murmur, particularly if slight and narrowly circumscribed in locality. The immense majority of such murmurs are found to present themselves over a part of the right ventricle between the third and the fifth costal cartilage; and I have rarely found them, except when accompanied by tenderness on pretty firm pressure, or by signs of effusion, or by marked excitement of the heart's action, or by a short dry cough, or by cardiac oppression and angina, give any cause for permanent uneasiness.

It is consistent with my observation, though opposed to the statements of several authorities, that one or other of the above-mentioned symptoms is rarely wanting when signs of effusion, even to a limited extent, are superadded to those of murmur. In fact, I more and more tend to disbelieve in *really acute pericarditis, apart from vital phenomena or symptoms;* although many cases called pericarditis may have been insidiously developed, and have escaped attention till a late period, either from inattention, or from their not being really acute in the sense of demanding treatment. Among the symptoms mentioned, the dry short cough is perhaps the one most likely to attract attention, and should always lead to the suspicion of pericarditis, when not explained by the state of the lung. Moreover, the symptoms mentioned are occasionally developed as the earliest phenomena of the disease, being succeeded by the murmur within two or three days, as has been shown by Dr Mayne, of Dublin, and by Dr Stokes.

With regard to the treatment of pericarditis actually pursued by me in hospital practice, I shall keep in view chiefly the rheumatic form, inasmuch as most of the others present little room for remarks tending to any satisfactory result.

In saying that rheumatic pericarditis will usually end favourably under various methods of treatment, I am very far from wishing to depreciate the value of medical practice in this disease. No doubt, the field within which our operations are to be restricted is more limited than it has been supposed to be by those who think that a particular method is a *specific* against inflammations in general, or pericardial inflammation in particular. But I trust that few educated members of the medical profession will think the worse of any treatment, because it does not claim the character of a specific method. What I argue is, not that treatment is of no use, but that the general and constitutional treatment applicable to rheumatism should overrule the means addressed to the local disease, except within the very narrow limits which I shall presently point out.

I find it impossible to arrange the facts of my experience in this matter in the statistical form. For, in the first place, I do not know how many cases of really acute pericarditis I have treated; and in the present state of our knowledge, as indicated in a preceding part of this paper, it may safely be said, that every attempt to number such cases must involve elements of most serious fallacy. But, in the second place, I do not know how many of the cases I have treated have got well in the end, and looking to the remote consequences; some of them, for instance, may have recovered for the time with more or less of adhesion of the pericardium. Of this only I am certain, that, as physician to the Royal Infirmary, I have not had under my care a case of rheumatic pericarditis fatal during the acute period of the attack.

Let me try to turn this fact to its proper use, as regards what may be called the heroic methods, bleeding and mercurial salivation.

I believe that the profuse employment of blood-letting in inflammations has long been diminishing, and that in Edinburgh, at least, it is pretty nearly extinct. I shall not, therefore, occupy space in demonstrating that that is a wrong thing which, to the best of my knowledge, very few indeed are in the habit of doing. For many years past, I have not heard that any physician in Edinburgh has used a lancet in rheumatic pericarditis. At all events, I am very sure that the lancet is used most sparingly by the profession in general, and has been so for many years past.[1]

But I am not so sure about mercury. Undoubtedly the use of this treacherous mineral is now marked by very great caution; and we almost never hear of those bad consequences which are the direct result of excessive mercurial action. But is its use, as some even now use it, expedient or necessary? And in rheumatic pericarditis in particular, would patients recover better, or worse, were no mercury exhibited? Without altogether holding the question as decided, I am strongly inclined to answer both of these questions in a sense unfavourable to the use of mercury. My own use of this much vaunted and much abused remedy has been rather experimental than founded on conviction.

The reserve with which I have used a remedy which has so much testimony in its favour, may appear to require some explanation. The truth is, that, as a student, it was my fortune to serve in the hospital under a very bold mercurialist—a man of the most humane character, and of the most entire conscientiousness, who proved his sincerity in regard to this subject by undergoing, in his own person, three distinct salivations in the course of his fatal illness. Under this gentleman's directions I learned much that was valuable; and,

[1] Since the statements in this paragraph were published (after being read in the Medico-Chirurgical Society), I have learned that my colleague, Dr Warburton Begbie, has on three occasions employed venesection in pericarditis with a favourable result. I am not the less convinced that in his hands, as in others, the lancet has been "used most sparingly," as stated in the text.

among the rest, something in regard to the bad effects of mercury in rheumatic pericarditis. But I have never succeeded in learning anything as to its good effects, though on many occasions afterwards I have administered it with such caution as my knowledge of it inspired. It may be said that I have not done justice to the remedy. In one sense this is true; for I have very rarely given it, except after other remedies. But surely, with a remedy of the power ascribed to this one, and specially regarded as promoting the absorption of exudation, it is no real injustice to call it into operation only in cases of a certain degree of severity, and to watch with care its influence upon cases that have in some degree resisted other treatment.

But whether I have done justice to mercury or not in my personal trials of it, I believe I have used it so as to do justice to nature, which is, after all, perhaps a better thing. For, assuredly, if I had adopted the plan of giving mercury instantly, in every case where a slight roughness existed in connection with the first sound of the heart, I should have failed to observe that the immense majority of these cases never went beyond a slight roughness, or, at most, a slight but decided friction-sound; that of those which went beyond this, and were accompanied by a degree of effusion, a large proportion had only moderate effusion; and that, even when considerable effusion was present, a good cure was still possible without mercury.

I will conclude by mentioning, as nearly as possible, what has been the usual course of treatment adopted in the cases here referred to, in so far as it has differed from that of acute rheumatism, or of the other primary disease. I trust I have not acted under any narrow or bigoted feeling of opposition to established doctrines, any more than of empirical devotion to single remedies.

In the very beginning of some cases of pericarditis, where the pain was very marked, and especially where it had strongly the characters of angina, leeches have not unfrequently been applied in moderate numbers. From four to six leeches so applied, and followed by fomentations, have very commonly relieved the pain, and been followed rapidly by improvement. Where relief occurred, but was not complete, the application has sometimes been repeated. More commonly, one application has been all that I have thought requisite; and this only when strength and condition permitted, and when the symptoms had a certain degree of urgency. General blood-letting has not once been practised.

Fomentations, sometimes plain and sometimes medicated with opium, friction with camphorated and ioduretted liniments, and in obstinate cases the use of blisters, have been the chief local remedies besides leeches.

To conclude, I believe the principles of the safe treatment of pericarditis to be as follows:—1. To make large allowance for the insignificant and spontaneously healing class of cases revealed more by physical signs than by symptoms, and to regard them as demanding

little active treatment; 2d. To consider rheumatic pericarditis in general as a disease susceptible, to a great extent, of cure under mild palliative local remedies and fitting constitutional treatment; 3d. To hold the constitutional treatment as subordinate to that of the disease with which the pericarditis is associated.

MURRAY AND GIBB, PRINTERS, EDINBURGH.