THE REMOTE PROGNOSIS OF PERICARDITIS

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THE immediate prognosis of pericarditis has been satisfactorily worked out. It is well known that the rheumatic cases, as a rule, terminate favourably, while those associated with renal disease commonly, though not invariably, end fatally. The remote prognosis, though it has from time to time received attention from clinical workers, appears still to be a matter of doubt. I was, therefore, led to undertake an inquiry into the after-history of a large number of patients who had suffered from pericarditis, to determine, if possible, the factors which influence the ultimate prognosis in any individual case.

PREVIOUS WORK.

It has long been recognised that considerable fluid effusions into the pericardial sac may be entirely absorbed,

but the possibility of the removal of fibrinous exudation without the formation of adhesions is still doubtful. It is certain that adhesions of greater or less extent occur in the majority of cases, and though there is no evidence that complete absorption does ever take place (1), the adhesions may be limited to a small area about the base of the heart, or to a single band between the parietal and visceral layers of the pericardium.

Adhesions being the common result of inflammation of the pericardium, it remained to determine how far they affected the comfort and well-being of the patient, and especially if they tended to shorten life. The earlier writers, Lancisi, Vieussens, and Morgagni (2), agreed that adherent pericardium was incompatible with long life. The same opinion was held by Corvisart (3). Laennec (4), on the other hand, was impressed with the fact that he had found in the post-mortem room universal adhesions in cases in which there was no evidence of cardio-vascular disturbance during life, and was led to conclude that such a termination of pericarditis was not unfavourable. Hope (5) followed the views of Morgagni and Corvisart, believing that, although for a time pericardial adhesions might not cause much inconvenience, the action of the heart in such cases was a "constant struggle," and that a fatal termination was inevitable. He believed that this occurred more rapidly in patients among the working classes.

Gairdner (6), in his classical paper upon the favourable terminations of pericarditis, pointed out that Hope's conclusions were too sweeping, and recognised that many patients, by the adoption of a "reduced scale of existence," might live in comfort for years.

Kennedy (7) analysed ninety cases of adherent pericardium in which there was no valvular disease, and found that hypertrophy and dilatation were a common, and atrophy an occasional result of pericardial adhesions. He concluded, therefore, that a most guarded prognosis should be given, and that in no case was it justifiable to

effect an insurance upon the life of the patient. Gairdner (8) wrote a strong criticism upon this paper, summing up his arguments thus:—"A certainly not small proportion of persons affected with pericarditis and its sequela of adhesion may survive the attack for many years, and may live in tolerable comfort, without the inconvenience attendant upon a dilated, hypertrophied, or atrophied heart."

Bauer (9) drew attention to the fact that in those cases in which the adhesions were the firmest, and the resistance to the movements of the heart presumably greatest, hypertrophy did not develop. This observation led many writers to put down hypertrophy in all cases of adherent pericardium to associated valvular disease (10), in spite of the fact that in Kennedy's ninety cases the valves were unaffected, and that Wilks (11) had shown that all the phenomena of failure of the heart from valvular disease may be produced by the influence of pericardial adhesions alone.

Sturges (12) and others, who have especially studied the cardiac diseases of children, have shown that the outlook in young subjects is decidedly unfavourable, inasmuch as the pericarditis is a part of a general carditis, the involvement of the myocardium being the most important factor. This opinion was supported by Dr. Lees and others in an interesting discussion at the Annual Meeting of the British Medical Association in 1898 (13).

In his monograph upon adherent pericardium (14), Dr. J. H. Broadbent states that "it is only in cases in which the heart is of normal size, and the pericardium is not adherent to the chest wall or to a large area of the diaphragm, that adherent pericardium gives rise to no serious consequences." Dr. Frederick Roberts (15) is inclined to agree with this, and believes that "there is a general tendency to make too light of the conditions remaining after acute pericarditis."

In a paper on the relation of heart disease to life assurance, Sir William Gairdner brought up the question

of the remote prognosis of pericarditis, and while recognising that pericardial adhesion is undoubtedly a lesion which would render a life insecure, Sir William is still in doubt whether acute pericarditis must necessarily end in adhesion (16).

A survey of the previous clinical work upon the subject leads, therefore, to the following conclusions :

- (1) Pericarditis commonly results in adhesions.
- (2) In some cases adherent pericardium is perfectly compatible with health.
- (3) The size of the heart appears to be an important point. If large, the outlook is unfavourable.
- (4) Hypertrophy does not appear to be due to the resistance imposed by the adhesions to the contractions of the heart.
- (5) Myocarditis and valvular lesions are of greater import than the condition of the pericardium.
- (6) Pericarditis in children is usually part of a general carditis, and the involvement of the myocardium in the inflammatory process is believed to be the essential feature in the rapid failure of the organ met with in the young.

RECENT PHYSIOLOGICAL WORK.

Recent physiological work has thrown much light upon the functions of the pericardium in animals, and the results of experiment appear to be so important that I will consider them before describing my own clinical work.

The pericardium consists of two essentially different parts, a serous sac enclosed in a tough fibrous bag. If a cannula be tied into the pericardium of a cadaver, and be then connected with a vessel of water raised to a height, the sac gradually dilates until the fibrous bag is tense. From this point onwards, even if the pressure is raised until it is equal to two atmospheres, no appreciable change takes place. Mr. Barnard (17) has shown that normally this inextensible sac prevents the heart dilating beyond a certain

point, and thus the mechanical disadvantages of dilated cavities and a thinned wall are prevented. For, in the first place, the heart will contain more blood when the fibrous pericardium is removed than when the organ is in the closed sac; and secondly, in conditions of extreme dilatation the heart actually "herniated" through an opening made in the pericardium, particularly in diastole. Pressure upon the abdomen by driving the blood from the great veins into the right heart produced the same result. Barnard further found that the extra blood was contained in the right heart, and that when the pericardium no longer supported the right ventricle, relative incompetence of the tricuspid valve occurred. Thus, in the light of these experiments, the pericardium may be considered to be to the heart what the adventitious coat is to an artery. Dr. Leonard Hill (18) has also shown some striking experiments which illustrate the same points. Animals with powerful abdominal muscles have a tough pericardium, whilst, on the other hand, those with weak abdominal muscles have a weak pericardium. He took for comparison the eel and the snake. The snake has a weak abdominal wall, and propels itself by muscles attached to its ribs, while the eel propels itself by its strong abdominal muscles. The pericardium of the snake is a weak bag, while the heart of the eel is contained in a very tough fibrous sac. If the eel be held up by its tail, blood will gravitate from the great abdominal sinuses into the heart, but no over-distension occurs if the pericardium be intact. If, however, the pericardium be open, the effect of gravity over-distends the heart, and the organ may be dilated to three times its normal size. Dr. Hill further showed that if it were not for the support of the pericardium, the muscular contractions of the abdominal wall would continually over-distend the heart. The weak pericardium of the snake was shown to afford a very poor resistance to the dilatation of the heart as the result of gravity.

If we apply what experiment has taught us occurs in animals to the conditions which obtain in man, it will be

seen that this protective support of the pericardium comes into play during muscular effort. All strong muscular contractions drive the blood from the veins of the limbs into the right heart, and especially in strains involving the fixation of the chest (such as lifting and carrying weights) the powerful contractions of the abdominal muscles drive the blood from the large venous sinuses in the abdomen into the right heart and distend it. Under normal conditions the fibrous pericardium prevents this dilatation from going beyond a certain point. If the glottis be open there is only this fibrous bag to prevent over-distension. If, on the other hand, the glottis be closed, as is usual in great muscular effort, the outflow of blood from the right heart is impeded, and this would directly aid dilatation if it were not for the fact that the closure of the glottis raises the intra-thoracic pressure.

I conclude, therefore, that (1) the pericardium prevents dilatation of the heart beyond a certain point; (2) this support is particularly of value in preventing dilatation of the right heart during muscular effort.

The conclusions thus reached render a reconsideration of the remote effects of pericarditis, and especially of adherent pericardium, necessary.

METHOD ADOPTED IN MY INQUIRY.

While Medical Registrar at the London Hospital I took 200 cases of acute pericarditis occurring during the years 1890 to 1897 inclusive, and endeavoured to trace them with the object of ascertaining the after-history, and I have been successful in following up 130.

Twenty-four of the patients traced had died before the inquiry was begun. In most the fatal event had occurred in a hospital or infirmary, and in the majority I have been able to obtain details of the post-mortem appearances. A few died at home, and I have carefully investigated the cause of death, and all cases of doubt have been excluded.

Of the 106 other cases I have personally examined 100, often at frequent intervals; the remaining six are either abroad or in the country, and in them I have had to rely upon their written account of their condition.

Of the 100 cases I have had under my own observation, nineteen are now dead, and most have died at the London Hospital or at the North-Eastern Hospital for Children. Wherever possible, I was present at the autopsy, and as in the other cases I have availed myself of the kindness of the registrars and resident medical officers of other institutions in obtaining details of the post-mortem appearances.

In all the cases I have endeavoured to obtain as full a history as possible, and as many of the patients have been at one time or another in different hospitals, I have had to trespass upon the kindness of the officials of these institutions, and would here gratefully acknowledge the assistance they have afforded me.

For the opportunity of following up so large a number of cases I have to tender my grateful thanks to the Staff of the London Hospital, and to my colleagues at the North-Eastern Hospital for Children, who kindly placed their cases at my disposal. Those patients who were well enough came up to see me at the London Hospital, while others were seen in the wards or at their homes.

In every case the features of the original attack were examined by reference to the original notes. The subsequent history was inquired into. The conditions at the time of examination were noted and recorded on clinical charts, and in many cases I have had the advantage of radiography in verifying the areas determined by percussion.

As a result of this extensive experience I have been led to the conclusion that simple adhesion of the layers of the pericardium is of little moment, and that the dilatation of the fibrous pericardial sac, softened and sodden by inflammation, is of far greater importance. This dilatation is brought about from within. The heart

dilates and distends the yielding pericardium, which, while still inflamed, becomes adherent to the chest wall and diaphragm.

PERICARDIAL ADHESIONS.

It has long been a matter of surprise that the two layers of the pericardium may be completely adherent without any evidence, during life, of interference with the cardio-vascular mechanism. It is quite a common thing to meet with a universally adherent pericardium in the post-mortem room in a case in which the heart was supposed to be healthy. Probably the most remarkable instance of this condition is a heart in the museum of the University of Edinburgh. The pericardium is universally adherent, and there is so great a deposit of calcareous material that "except for about one cubic inch" over the apex the cardiac envelope is as "firm as the skull" (19). In this case there was no palpitation and no pain. It has also been observed that in the cases in which the adhesions are very firm there may be no hypertrophy of the heart (20). This I have verified in the post-mortem room on several occasions. The point is an important one, as it shows that the presence of tough adhesions does not induce hypertrophy by resisting the contractions of the heart.

In these cases I have found that the heart is not a large one, or, as I would rather put it, the pericardium is not dilated. There may, or may not, be adhesions between the chest wall and the diaphragm and the fibrous pericardium, but these are of little moment if the pericardium, and therefore the heart, are not much dilated.

On the other hand, there are cases in which the adhesions play an important part. The contracting bands of organised exudation may compress the great vessels, and especially the great veins. Wilks (21) first drew attention to these cases, and at a recent discussion at the

Medical Society of London (22) Sir William Broadbent and Dr. West mentioned instances in which the vena cava inferior had been completely occluded. I have not seen an instance of this compression by the contraction of adhesions, but in one case I saw the left auricular appendix so bound down that its cavity was almost obliterated. Grave disturbance of the vascular mechanism is inevitable under these circumstances.

The foregoing groups of cases of pericardial adhesions, however, form but a small proportion of the whole. In a large majority the pericardial sac is found, upon opening the chest, to occupy a large part of the thoracic cavity. The fibrous sac is adherent to the chest-wall and diaphragm, often over considerable areas. The two layers of the serous pericardium are firmly adherent to each other, and the cavities of the heart are dilated. The important point is to determine how this dilatation is brought about. It does not seem possible that with the heart in constant movement the cavities could be pulled open by contracting bands of organised exudation, and to say that adhesion takes place while the heart is dilated does not simplify the matter, for the dilated cavity must be in continual movement or the circulation would cease. As a matter of fact, it is not uncommon to find tags attached to both layers of the serous pericardium, and these tags are evidently fragments of adhesions broken by the constant movement of the heart; and at the apex, where the amplitude of movement is greatest, the adhesions may be pulled out into bands an inch or two in length.

The essential feature of these, the most important cases of adherent pericardium, is, as I shall shortly show, not the extent of the adhesions but the amount of dilatation. The whole difficulty of the problem is to determine how the dilatation is brought about.

Before leaving the consideration of pericardial adhesions mention must be made of the observations of Chevers (23), who pointed out that atrophy of the heart

was sometimes associated with adherent pericardium. Kennedy (24) found it in five of his ninety cases of adherent pericardium without valvular disease, and subsequent writers have supported his observations. The atrophy has been considered to be due to the strangulation of the heart by the adhesions. Sibson (25) does not give a single instance of atrophy of the heart in association with adherent pericardium, and in the records of the London Hospital for recent years I have failed to find one case. Atrophy of the heart appears to occur in wasting disease only.

Conclusions.

(1) In very rare cases adhesions may seriously interfere with the circulation by compressing the great veins.

(2) An orderly working of an undilated heart is quite compatible with very extensive pericardial adhesions.

(3) Adhesions of themselves do not produce dilatation of the heart.

(4) The essential point is not the presence of adhesions, but the amount of dilatation.

DILATATION OF THE PERICARDIUM.

We have already seen that the special function of the pericardium is to prevent over-distension of the heart during muscular effort. There is, however, no doubt that the sac will increase in size if the heart undergoes hypertrophy as the result of repeated strains, or of renal disease, or of a chronic valvular lesion. It is obvious that the sac which envelops a heart weighing 30 oz. must be larger than one which surrounds a normal organ.

My contention, however, is that when the pericardium is softened and sodden from acute inflammation, it does not oppose a limit to the dilatation of the heart, and that it is this loss of the normal support which is a marked feature of many cases of acute pericarditis, particularly in the young. In some of the patients I have watched,

the dilatation has been progressive, and manifest to the ordinary methods of examination from day to day. As the dilatation increases the tricuspid valve becomes incompetent, and pulsation of the liver and veins develops. In these cases I have found that the pericardium was dilated, that the heart and especially its right chambers, were dilated, but that the valvular lesions were insignificant. The acute inflammation had involved the fibrous and serous layers of the pericardium. There were loose adhesions between the outer surface of the sac and the chest-wall, and between the visceral and parietal parts of the serous coat, but there had been no time for the fibrous pericardium to become firmly adherent to the chest-wall and diaphragm. The dilatation of the softened sac was therefore progressive, there was no limit to the dilatation of the cavities of the heart, and compensation could not be established.

In the majority of the cases the process stops short of this. During the acute stage the dilatation of the pericardium goes on to a greater or less degree, and then the fibrous sac becomes adherent to the thoracic parietes. The adhesions become organised and ultimately are so firm and tough that a condition of permanently dilated adherent pericardium results. If the dilatation of the sac be extreme, and the sac become adherent, the heart may be dilated beyond the power of compensation. In the cases which I have personally observed, I have found that death occurs in from three to nine months after the original attack (see p. 435).

But, if the dilatation be not extreme, and adhesion take place, that is, if we have a condition of moderately dilated adherent pericardium, the thickened fibrous sac still acts as a support to the heart in muscular effort. But the conditions are abnormal, the heart is dilated and hypertrophied, and instead of being supported by a pericardium of normal size, it is in an unduly large envelope. During rest and freedom from muscular effort, there is no great call upon the heart, but with the resumption of

muscular activity, compensation which has been effected during the resting period, breaks down sooner or later. If the pericardium has not become sufficiently organised to resist the strains imposed upon it by the distension of the heart by muscular effort, coughing, &c., the softened sac may yield and the compensation established by the rest may break down at once. More often time has been allowed for the consolidation of the sac, and compensation is maintained temporarily and then breaks down. The failures may occur again and again, and compensation be restored, until finally, the failure is complete and death results. One of the patients I have had under observation for some time led a precarious existence for fourteen years with a moderately dilated adherent pericardium. Most cases, as I shall show later, succumb much sooner than this (see p. 424).

There still remain for consideration the cases in which there are no signs of failure of compensation, those cases, in fact, in which the post-mortem examination alone reveals disease of the cardiac envelope. The essential feature, as previous workers have pointed out (26), of the cases of adherent pericardium which do well, and present no symptoms, is the normal size of the heart. However great the amount of adhesion, there is no great increase in the size of the heart, if the supporting sac is undilated.

The pericardium may be acutely dilated with effusion to a remarkable extent. In Sir James Alderson's case, figured by Sibson (27), the effused fluid weighed three pounds and a quarter. But the plastic form of pericarditis, which is so common in children, also softens the pericardium. This I have been able to prove by injecting normal and inflamed pericardiums in the post-mortem room. The softening effect of inflammation upon fibrous tissue is more marked in children than in adults. Another point of importance is that pericarditis, particularly in children, is often latent, and sometimes recurrent. Last autumn I saw a boy of nine, who had been under medical treatment

for "influenza" for a fortnight. The parents were led to believe that the child was not very ill, but they had noticed that his mind wandered at night. I found a soft pericardial friction over the base of the heart, and a mitral systolic bruit. There had been very little complaint of pain in the chest, and articular symptoms were practically absent. It struck me at the time that but for the examination of the chest this boy might have been allowed to run about for some days longer with a softened pericardium, and that the sac must inevitably have dilated. I have no doubt that this is one way in which dilatation of the pericardium is brought about, but I think that the too early resumption of muscular effort after an acute attack, and before the fibrous sac has become consolidated, is the most potent cause of such a condition.

The movements of chorea deserve special consideration as a factor in dilating the pericardium, as the association of acute pericarditis with this disease is not infrequent. The movements are often very violent and of long duration, and they must tend seriously to strain a softened pericardium, especially as they are not accompanied by a rise in the intra-thoracic pressure which is brought about normally in great muscular efforts by closing the glottis. I came, therefore, to the following conclusions:

(1) A pericardium softened by inflammation offers little or no resistance to the dilatation of the heart, whereas the special function of the normal sac is to prevent over-distension of the organ.

(2) The dilatation of the pericardium may be progressive, and then compensation is never established.

(3) If the dilatation be extreme, compensation is impossible.

(4) The greater the dilatation, the more difficult it is for the heart to remain compensated.

(5) The formation of adhesions between the dilated fibrous pericardium and the chest wall and diaphragm constitutes a condition of permanently dilated adherent pericardium. Compensation may be brought about, but

as the conditions are abnormal there is always a tendency for it to fail.

(6) Dilatation of the pericardium without the formation of adhesions may bring about the same results.

(7) Extensive adhesions with the pericardium undilated do not interfere to any material extent with the orderly working of the heart.

THE EFFECT OF CONCOMITANT VALVULAR LESIONS.

We have already seen that the whole sequence of events associated with failure of compensation may be produced by pericardial adhesions alone. It is, however, more common to find that the valves are affected at the same time as the pericardium. The mitral orifice is the most often involved, but the valvular lesion is frequently of secondary importance; and we know that such a lesion, even when it has begun in childhood, may be, and commonly is, perfectly compensated for years. It is now generally recognised that some of the cases of mitral stenosis met with in adolescents and adults are the result of endocarditis occurring in early life. It is years—often many years—before the compensation breaks down. On the other hand, the characteristic feature of the cases associated with pericarditis is the rapid failure of the heart. It must, nevertheless, be recognised that the association of valvular disease with dilatation and adhesion of the pericardium increases the gravity of the case, since the valvular lesion introduces another factor tending to dilatation of the cavities and impairment of compensation. If the pericardium be undilated, valvular disease does not appear to be of so much importance. One man of twenty-seven who had well-compensated mitral and aortic disease when attacked with pericarditis, presented no more hypertrophy when I saw him six years later than he had before the attack of pericarditis.

Conclusion.

The association of valvular disease with pericardial lesions, especially with dilatation and adhesion of the pericardium, enhances the difficulties of compensation.

MYOCARDITIS.

The rapid dilatation of the heart which is so frequently seen in the rheumatic heart disease of children has been considered, as I have already shown, to be due to a general carditis. The endocardial affection being of minor importance, and the condition of the pericardium as regards dilatation not having been considered, attention has been directed to the myocardium. So far as pathological observations go, there does not appear to me to be sufficient evidence to warrant the contention that the dilatation is entirely due to myocardial degeneration. Dr. Poynton analysed the post-mortem records of the Children's Hospital, Great Ormond Street, and found that out of 150 cases in which death was due to heart disease, myocardial changes were noted in 23. In 13 the muscle was described as being soft and pale; in 4 it was fatty; and in 6 it was tough and fibroid. In the records of 62 post-mortem examinations made during recent years at the London Hospital on cases at all ages in which there was evidence of old pericarditis, I have found that there was fibrosis or other marked evidence of myocarditis in six instances only.

It appeared to me to be more profitable to examine carefully the myocardium of patients dying with acute dilatation of the heart, with pericarditis. I have cut sections of three—two specimens from children and one from the heart of a lad of eighteen. In each I have failed to find evidence of myocarditis, except in the layer of muscle immediately below the serous coat. As a precaution, I have sent specimens to be examined by independent pathologists, who have confirmed my own observations.

As we have already seen, the essential feature of the cases of so-called "pan-carditis" is the marked dilatation of the heart, and the consequent failure of compensation. By analogy with other toxic processes—*e. g.* diphtheria and enteric fever—it is urged that the rheumatic poison particularly affects the heart muscle, and that fatty change in this part of the organ is the cause of the dilatation. It is even urged that the toxine has a particular affinity for the fibrous valvular ring. Fatty change in the myocardium is undoubtedly injurious, but does it cause extreme dilatation of the heart and failure of compensation? Pernicious anæmia is a toxic process in which fatty degeneration of the heart is found in a marked degree; but even when it is extreme the heart may act quite regularly. Again, we do not meet with the phenomena of dilatation and failure of compensation in typhoid fever and diphtheria; and these are both toxic processes, in which myocardial degeneration is common. The typhoid heart is, as Dr. Sansom (28) has shown, a small heart. Since the publication of his paper, the results which he obtained by percussion have been completely borne out by a series of radiographs taken at the London Hospital.

Balint (29) has recently shown that in animals the removal of even two cusps of the aortic valve, and the production of fatty degeneration of the heart muscle by the injection of phosphorus oil, does not cause failure of compensation.

Local dilatation of a cardiac cavity—that is, cardiac aneurysm—can be brought about during the systole of the cavity provided that there is local weakness of the heart wall. But general dilatation of a cavity cannot take place during its systole. If the muscle is weak, its contraction must be proportionately weak; and if the resistance cannot be overcome, the cavity simply does not empty. During the diastole the cavity immediately behind, provided that its muscle can overcome the resistance, throws its blood into the unemptied chamber; and if

the heart wall is softened by inflammation or fatty changes the cavity will dilate. Strong contractions of the limbs and abdominal muscles by driving venous blood into the right heart will act powerfully in the same direction. But it must be remembered that if the pericardium is intact—that is to say, inextensible and undilated—the heart muscle, however soft it may be, cannot dilate beyond what it probably does during any violent exercise.

If, however, the pericardium is dilated and the heart muscle softened by some degenerative process, moderate muscular efforts may produce a greater dilatation than the normal, but only so far as the dilated pericardium allows. Again, let us suppose that the pericardium is moderately dilated, and that the heart within it is hypertrophied and somewhat dilated, but the compensation is so adjusted that the cavities empty themselves, and there is no strain upon the pericardium. In such a case any condition of degeneration of the heart muscle may so weaken the walls that the cavities cannot empty, and compensation fails.

Conclusions.

1. The layer of muscle immediately below the pericardium is usually inflamed in pericarditis.

2. General myocarditis is rare.

3. Extensive fatty changes in the muscle of the heart are found in conditions in which dilatation and failure of compensation are unknown.

4. Experiment bears out clinical experience upon that point.

5. Dilatation of a softened cavity can only take place during diastole.

6. The dilatation is limited by the amount of dilatation of the pericardium.

7. Fatty degeneration of the myocardium is often the result, rather than the cause, of failure of compensation.

§ *The question of hypertrophy.*—Hope (30) believed that hypertrophy of the heart was an invariable result of pericarditis. Fagge (10), on the other hand, thought that hypertrophy was always due to concomitant valvular disease, previous or concurrent. Kennedy showed (7), however, that hypertrophy was present in 56 per cent. of cases of adherent pericardium uncomplicated with valvular disease, while Gairdner made it only 33 per cent. (8). It has been held that the hypertrophy of the heart secondary to pericardial adhesions is not real hypertrophy at all, but a thickening of the organ by “changes mostly fibroid,” secondary to an interstitial myocarditis (31). On the grounds already stated I cannot agree with this view. In one of my cases the heart weighed $31\frac{1}{2}$ oz., and there were no valvular lesions at all. The muscle was an excellent example of genuine hypertrophy.

If the heart dilates, hypertrophy must follow if the patient lives long enough, and if a softened pericardium permits greater dilatation than any other condition, we should expect to find the heaviest hearts in patients who had suffered from pericarditis. This, as I shall shortly show, is the case. Hypertrophy may occur very rapidly. Quite recently I made a post-mortem examination of the body of a boy aged eleven. He had been ill for three weeks with rheumatic pericarditis and chorea. The heart weighed 12 oz., double the normal weight at that age. There had been no previous illness, so that there is every probability that the increase of weight was brought about in three weeks. Dr. Goodhart (32) described a case the history of which suggested that the heart grew to a weight of 19 oz. in three or four weeks as the result of an attack of pericarditis.

From the post-mortem records of the London Hospital for recent years I have collected a consecutive series of sixty-two hearts affected with valvular disease, in which there was evidence of old pericarditis. In only four of these, *i. e.* about 6 per cent., was the weight of the heart less than 10 oz.

In the appended table the weights of these hearts are compared with the weights of a consecutive series of hearts affected with valvular disease, but with no evidence of antecedent disease of the pericardium.

TABLE I.

Weights.	Valvular disease.	
	With pericarditis.	No pericarditis.
Over 40 oz.	3	0
30—40 oz.	5	1
25—30 oz.	7	5
20—25 oz.	7	6
15—20 oz.	22	28
10—15 oz.	14	18
Under 10 oz.	4	4
	—	—
	62	62

It will be seen that the effect of valvular disease, as might be expected, is to increase the weight of the heart. Pericardial complications tend still further to hypertrophy. Sibson's tables give similar results (33).

The weights of the four heaviest hearts and the associated conditions are appended.

1. 47 oz. Male aged 20. Universal adhesions. Mitral and aortic disease. Pericarditis aged thirteen.

2. 45 oz. Male aged 25. Universal adhesions. Mitral slightly stenosed. Aortic incompetent. Pericarditis aged seventeen.

3. 40 oz. Male aged 18. Universal adhesions. Mitral slightly stenosed. Aortic incompetent. Pericarditis aged six.

4. 38 oz. Female aged 51. Universal adhesions. Mitral and tricuspid incompetent. Aortic unaffected. Several attacks of rheumatism, the first aged fifteen.

The involvement of the aortic orifice in three cases no doubt played a considerable part in producing the

¹ All cases of valvular disease, rheumatic or other, are included.

enormous hypertrophy. It does not explain it all, as I have been unable to find a single case in which such an extreme condition was the result of valvular disease alone. In the following case there was exceptional hypertrophy and no valvular disease.

Weight $31\frac{1}{2}$ oz. Male aged 21. Universal adhesions. Valves unaffected. Pericarditis aged sixteen.

A remarkable point is the enormous hypertrophy met with in some of the young patients. The following instances illustrate this.

1. Weight 32 oz. Female aged 16. Universal adhesions. Mitral admitted the tips of two fingers. Tricuspid and aortic incompetent. Pericarditis aged eleven.

2. Weight 28 oz. Female aged 17. Universal adhesions. Mitral stenosis. Aortic healthy. First attack aged thirteen.

3. Weight 27 oz. Female aged 17. Universal adhesions. Mitral admitted two fingers to second joint. Other valves healthy. Myocarditis with recent hæmorrhagic patch in wall of right ventricle. Pericarditis aged one, five, and seventeen.

4. Weight $22\frac{1}{2}$ oz. Female aged 12. Universal adhesions. Mitral and tricuspid incompetent. Pericarditis aged eight.

In the four cases in which the weight of the heart was not above normal the conditions were as follows:

1. Weight $9\frac{1}{2}$ oz. Male aged 20. Pericardium universally adherent. No valvular disease. Emphyema. No rheumatism. Ill a year.

2. Weight 9 oz. Male aged 72. Slight adhesions very old. Mitral and aortic valves slightly thickened. Rheumatic fever aged six.

3. Weight $8\frac{1}{2}$ oz. Male aged 30. Universal adhesions. Mitral stenosed. Cancer of the sigmoid. Acute rheumatism aged sixteen, nineteen, and twenty-six.

4. Weight 8 oz. Male aged 34. Universal adhesions. Valves unaffected. Phthisis, syphilis, lardaceous liver, &c. No rheumatism.

THE EFFECT OF AGE AND SEX UPON THE PROGNOSIS OF PERICARDITIS.

In order properly to understand the effect of age and sex upon the prognosis of pericarditis, it is necessary to ascertain the relative frequency of the disease at different ages in the two sexes. The ages at which the original attack occurred in the 130 cases which I have had under consideration, are set forth in the appended table. I have made five groups.

1. Young children, five years and under.
2. School age, six to fifteen.
3. Years of early labour (these are all hospital cases). sixteen to twenty-five.
4. Mature cases, twenty-six to thirty-five.
5. „ „ over thirty-five.

TABLE II.

Age at first attack of pericarditis.	Males.	Females.
5 years and under . . .	4	6
6—15 . . .	27	45
16—25 . . .	20	11
26—35 . . .	8	1
Over 35 . . .	7	1
	<hr/>	<hr/>
	66	64

It will now be most convenient to classify these cases according to compensation.

1. Those cases in which compensation was never established. These are the cases in which the pericardial dilatation is extreme.

2. Those in which compensation was established at least once, but which tended to break down, many of the patients coming under treatment again and again. Moderately dilated pericardium.

3. The cases in which compensation remains good. Undilated pericardium.

TABLE III.

MALES.

Age at first attack of pericarditis.	Never compensated.	Precarious compensation.	Good compensation.	Total.
5 and under ...	2 ...	2 ...	0 =	4
6—15 ...	3 ...	22 ...	2 =	27
16—25 ...	2 ...	10 ...	8 =	20
26—35 ...	0 ...	0 ...	8 =	8
Over 35 ...	0 ...	2 ...	5 =	7
	<hr/> 7	<hr/> 36	<hr/> 23	<hr/> 66

FEMALES.

5 and under...	3 ...	3 ...	0 =	6
6—15 ...	3 ...	40 ...	2 =	45
16—25 ...	0 ...	6 ...	5 =	11
26—35 ...	0 ...	0 ...	1 =	1
Over 35 ...	0 ...	1 ...	0 =	1
	<hr/> 6	<hr/> 50	<hr/> 8	<hr/> 64

The cases in which compensation was never established lived from three to nine months after the original attack of pericarditis.

The precarious cases present all varieties; in some compensation was established once only, in others the heart failed, and recovered again and again. In some cases the failures were spread over as many as eight years (one case, already referred to, went on for fourteen years with repeated slight failures, but I have not included it here as I am only dealing with the patients who have had pericarditis between 1890 and 1897).

It will be noticed that the female cases more commonly present the phenomena of precarious compensation than the males, and especially between the ages six to fifteen.

If all my cases had been watched for as long as five years it would have been an easy matter to have calculated an average mortality for this group, but it will be readily understood that the majority of the precarious

cases which I have been able to personally observe have had the original attack during the last three years, and are still living. I am afraid that the percentage death-rate which follows is based upon too few cases to be of great value.

Of nine males who had pericarditis between the ages six to fifteen, and in whom compensation was precarious, the average length of life after the attack was three and one third years.

Of sixteen females, in whom the attack occurred between the same ages, the average length of life was two and one-fifth years.

At the other ages there are still fewer deaths, but so far as they go they give for the males an average length of life after the attack of three and a half years, and for the females an average of three years. But there are only eight deaths in all.

Between the ages of twenty-six to thirty-five there were three deaths due to intercurrent disease, viz. diabetes, phthisis, and hæmorrhage from gastric ulcer. One female, aged forty, died apparently from the cardiac condition. The fatal event occurred suddenly at home, and the evidence at the coroner's inquest gave heart disease as the cause.

The following tables are, I think, of greater value in elucidating some of the factors which are to be considered in the prognosis in respect of age and sex. I have tabulated the condition of the pericardium in 200 consecutive autopsies on 100 male and 100 female patients dying in the London Hospital from heart disease. They are taken consecutively from December 31st, 1897, back to 1894, and, of course, include many of the fatal cases of pericarditis which I have traced. All cases in which the valves were diseased as the result of rheumatism, chorea, ulcerative endocarditis, renal disease, or atheroma are included. The cases are arranged in quinquennial periods, and each case is represented in the columns by the age at death.

TABLE IV.—200 consecutive cases (100 males and 100 females) dying in the London Hospital from heart disease, collected from the post-mortem registers, 1894 to 1897, inclusive.

MALES.

Quinquennium.	No pericardial complications recorded. Age at death.	Acute pericarditis. Age at death.	Old pericarditis, adhesions, etc. ¹ Age at death.
1—5	—	—	5
6—10	8	6, 9	8, 8, 7, 7, 9
11—15	15, 14	12	13, 14
16—20	16, 19, 16	17, 19, 18	20, 16, 18, 19
21—25	22, 25, 25	22, 23, 21	25, 22, 21, 25, 23
26—30	27, 27, 30, 30	—	27, 28
31—35	31, 32, 32, 33, 35, 35, 33	34, 33	31, 35
36—40	40, 39, 39, 36, 38, 37, 38, 38	37	40, 36, 38
41—45	43, 42, 41, 43, 43, 44, 41, 45, 44	42	44
46—50	46, 49, 50, 50	49	50
51—55	53, 53, 52, 52, 51	—	—
56—60	56, 60, 58, 60, 59, 56, 56, 58, 57	—	57
61—65	62, 63, 61	—	—
65—70	—	—	—
71—75	—	—	72
76—80	—	—	—

FEMALES.

1—5	5	—	—
6—10	9, 7, 9	9	9
11—15	12, 13, 11, 12	—	14, 15, 15, 13, 13, 15, 15, 14
16—20	17, 18, 17, 19	19	19, 20, 17, 19
21—25	21, 22, 24, 24, 21, 24, 24	24	23, 21, 25
26—30	30, 29, 30	29, 30	27, 30
31—35	32, 33, 34, 33, 35, 35, 35, 32	—	32
36—40	39, 37, 40, 36, 36, 39	—	38
41—45	41, 42, 42, 45, 44, 42, 41, 41	44	44
46—50	50, 49, 50, 47, 49, 48, 49, 50, 49, 47	49	—
51—55	51, 54, 55, 54, 52, 51	52, 52	51, 51, 54
56—60	57	60	—
61—65	61, 63, 63	—	—
66—70	68	—	—
71—75	—	—	—
76—80	77	—	—

¹ White or milk patches are excluded.

In the first column are the cases in which the pericardium was healthy. In the second are the cases in which there was evidence of acute pericarditis at the post-mortem examination. In the third those in which there was evidence of old pericarditis, such as local or general adhesions. Simple white or milk patches are excluded.

By this arrangement the special ages at which pericardial adhesions are found in the post-mortem room are seen at a glance. It will be observed that patients with valvular disease uncomplicated with pericarditis die at a much later age than those who have suffered from pericarditis.

Coming now to an analysis of the cases in which there was evidence of old pericarditis, a marked difference is seen in the two sexes, a difference which is not accounted for by the average incidence of the acute disease. In part this appears to be due to the difference in the rapidity of the increase in the body-weight in boys and girls. From five to nine a boy and a girl grow at nearly the same rate. But between ten and fourteen the boy's body-weight increases much less quickly than does that of the girl. The girl reaches her maximum annual increase in the eleventh, twelfth, and thirteenth years. The boy has a minimum annual increase in the tenth and eleventh years, but after that steadily progresses till he reaches sixteen, when there is the maximal annual increase. It will be seen from my tables that between seven and nine several boys died, but that after that there are very few deaths until just after the period of maximal annual increase of body-weight, and the rapid growth together with early years of active labour strain a heart hampered with the results of pericarditis and it is very prone to fail.

DIAGRAM comparing the annual increase of body-weight in school boys and girls between five and seventeen years of age. The vertical height in the columns represents the annual increase in pounds (constructed from Dr. Bowditch's tables). The male line is marked thus..... the female—.

In girls the mortality is excessive between thirteen and fifteen, and is quite out of proportion to the number of cases, which itself is high. The most obvious explanation is the incidence of puberty, but the incidence of puberty is not accompanied by an increased death-rate in other affections. The essential feature is doubtless the rapid increase of the body-weight during this period.¹

As these are hospital cases the influence of work may come in, for after leaving school some of the house-work involving lifting and carrying often falls upon these young girls. Later, between seventeen and thirty, the influences of hard work, pregnancy, and partu-

¹ I am indebted to Dr. Francis Warner for calling my attention to the importance of considering the body-weight, and for references to Dr. Bowditch's statistics (34), from which the annexed diagram has been constructed.

rition are shown. It will be noticed that scattered through adult life and even in the senile cases evidence of old pericarditis is met with. Some of these are, no doubt, the well-compensated cases to which I have already referred. The male patient dying at the age of seventy-two had had rheumatic fever at the early age of six. I do not, however, think that we can assume that all of these patients suffered from pericarditis in early youth. In some the affection appeared to be due to extension from pleuro-pneumonia. In a few the adhesions found were probably the result of subacute attacks of pericarditis occurring in the course of renal disease. In three instances, in which there was no history of rheumatism, there were adhesions about the liver and spleen and evidence of syphilis, and I have been led to question whether some of these may not have been of syphilitic origin.

It will be noticed that the later deaths generally occur in the period of decay. The pericardial adhesions, if of early date, gave no trouble, and death was due to inter-current disease.

Conclusions.

1. In young subjects death occurs at different periods in the two sexes. In the females a pericardial lesion occurring in early childhood commonly leads to a fatal issue during or immediately after the period of most rapid increase of the body-weight, that is between the twelfth and fifteenth years. In boys there is a period of comparative immunity until the end of the years of rapid growth, that is until after sixteen.

2. Heavy labour, and in the females pregnancy and parturition, tend to cause failure of a heart hampered with the results of pericarditis.

3. The consideration of these influences points to some mechanical interference with the cardio-vascular mechanism, since it depends rather upon the growth of the body

and the muscular power exerted, than upon any other vital condition.

4. A certain proportion of the cases live until the period of decay, and these are, no doubt, the cases of undilated pericardium which have been already considered.

CLINICAL OBSERVATIONS.

Clinically, as I have already indicated, the cases fall into three great groups :

1. Those in which compensation is well established.
2. Those in which compensation easily breaks down.
3. Those in which compensation is never established.

1. *Well-compensated cases.*—Thirty-one out of my 130 cases come into this group. I have had frequent opportunities for observing nearly all of these patients. The youngest had the acute attack of pericarditis when fourteen years old. A reference to Table III will show that the male cases preponderate, and that adult males who suffer from acute pericarditis almost invariably do well. A careful survey of the histories has convinced me that any condition which necessitates a prolonged confinement to bed tends to a favourable issue ; that is, time is required to render compensation secure, or as I would prefer to put it, resumption of the round of daily exertion should not be undertaken until the risk of dilatation of a softened pericardium is past. One of the worst cases, a man of twenty-one, had articular rheumatism, endocarditis, pericarditis, and chorea. There were many subcutaneous nodules, and after the acute symptoms had subsided a series of relapses of the articular pains took place. The nature and multiplicity of the lesions kept this patient confined to bed for four months. I saw him over two years later ; he was perfectly well, and had had no symptoms. There was a systolic bruit at the apex, and that was all. A similar favourable result has been observed in several other cases, in which, for one reason or other, a long confinement to bed was necessary.

One important fact has struck me in the physical examination of all these cases: there is no great increase of the precordial dulness. Definite exocardial sounds were heard in five out of the thirty-one cases examined. As a rule they were present at the base of the heart. One lad of seventeen, who is engaged upon a sailing barge, complained of pain when he had to pull upon his ropes. He had a well-marked exocardial sound at the base, and located the pain in that region. In the other cases there was no complaint of dyspnœa or of pain, and in no single instance, even with the history before me, did I find evidence of old pericarditis beyond the sounds mentioned above. It is certain that some of these patients had pericardial adhesions, but I was unable to find definite evidence of them. As a matter of fact, these are the cases in which a diagnosis of pericardial adhesions is of little moment. The pericardium is undilated, the heart is undilated, and the adhesions give no trouble of themselves, although they would probably handicap the patient in the event of an acute illness supervening.

2. *Cases in which compensation easily breaks down.*—Eighty-six of the 130 cases come into this group. They are of all gradations, from the patient who has been in and out of hospital for years to one in whom compensation has been established but once.

The younger patients, and especially the young females, form the bulk of the cases, and a few of the patients in whom the original attack occurred after thirty-five must be included. (See Table III.)

Where there are so many gradations the signs and symptoms vary very much. In some cases it would be impossible in the absence of a history to arrive at a correct diagnosis, but if the condition be borne in mind I am certain that dilated adherent pericardium can be diagnosed with precision in not a few cases. The history is, of course, of great assistance, and if the case has been watched throughout there is very little risk in overlooking

it. It will be remembered that Sturges (12) found evidence of pericarditis in 94 per cent. of the children who die from heart disease, and a history of rheumatic heart disease in early youth, especially if followed by frequent failures of compensation, should at once lead to a careful search for evidence of adherent pericardium.

Symptoms and physical signs.—The aspect of the patient will vary somewhat with the age at which the acute attack of pericarditis occurred. If it was early there is often a peculiarly youthful appearance. Puberty is delayed, and in several instances I have seen lads and girls of seventeen looking no more than thirteen. The face is pale, or there may be a “mitral” flush upon the cheeks. In prolonged cases the fingers and toes are clubbed. There is emaciation and dyspnœa on exertion. Many, even of the less acute cases, cannot lie down at night. During the period of failure orthopnœa is a constant symptom. The presence of dropsy depends upon the condition of the compensation at the time, but it is remarkable how little dropsy there is in some of the young children. In them the great stress appears to fall upon the liver, which is greatly distended and tender.

Pain is a very prominent feature. The precordium is the chief seat, but it may radiate down the inner side of either arm and to the back. In several cases I found that it was referred to the left shoulder. These pains are the referred pains of the dilated heart, and are frequently accompanied with cutaneous hyperæsthesia. Both the pain and the hyperæsthesia are essentially different from the pain and tenderness of acute pericarditis, as Dr. Henry Head (35) has pointed out. They are evoked by lightly picking up the skin, and are diminished by firm pressure, while the pain in acute pericarditis is increased by pressure and by percussion. One of my own patients, a boy of eight, has shown these phenomena several times when he has come up for treatment. These referred pains involve the cardiac area pointed out by Dr. Head. They are essentially transitory, and often last but for a few

hours. In some cases the pain has a remarkably wide distribution. One case under my care had a band of hyperæsthesia extending from the nipples to the level of the umbilicus, and from the angles of the scapulæ to the loins. It will be understood that I do not regard these pains as in any way pathognomonic of dilated pericardium, but in that condition great dilatation of the heart is the rule, and, as Dr. Head has pointed out, a constantly dilated hollow viscus, which from time to time is subject to further distension, presents just the conditions which are likely to cause referred pains.

From time to time the cardiac distress becomes acute, and cyanosis and grave dyspnœa are manifest. Sometimes the attacks may be warded off by timely rest. One patient I have traced tells me that she has to spend about one day a week in bed. Even when the pericardium is very dilated, it is remarkable with what rapidity a few days' rest restores the easy working of the heart. One child of seven, in whom the cardiac dulness reaches from nipple to nipple, has been an in-patient in the London Hospital eight times in eighteen months. At last compensation cannot be restored, and death follows.

In young subjects the chest wall is bulged on the left side. The area of visible pulsation is often very extensive. I have seen it as high as the second left inter-space and to the right of the sternum. The impulse often has a curiously rippling character in the intercostal spaces just above the apex. In many cases the action of the heart is obviously laboured. Retraction exactly at the apex during the systole is undoubtedly a valuable sign of adherent pericardium, but is by no means always present. In three instances I have seen systolic retraction of the lower intercostal spaces, pointed out by Dr. J. H. Broadbent as a characteristic sign of pericardial adhesions. Occasionally the epigastrium is retracted at each systole, but I have only observed it in two of my cases, and I have not seen the lower end of the sternum pulled in. On palpation a diastolic shock may be

frequently felt in the front of the chest, but in my experience very rarely posteriorly. The hypertrophied right ventricle is often felt pulsating in the subcostal angle.

On percussion the area of cardiac dulness is always increased, and corresponds with the dilated pericardium. The dulness may extend from nipple to nipple, and on the left side as far as the anterior axillary line. I have been struck with the fact that the margin of the dulness is often particularly well marked, especially along the left upper border. If it extends above the third rib, and if the dull area in the second left interspace is not altered by a deep inspiration, there is good ground for suspecting antecedent pericarditis, provided that retraction of the upper lobe of the left lung as the result of pleurisy or pulmonary disease is excluded.

I have found radiography of great assistance in verifying the results of percussion. In a few instances it has appeared to me that the fixation of the dilated cavities by adhesions has led to a more definite margin, especially along the left upper border. Examination with the fluorescent screen has been of value, but until there is greater steadiness in the illumination the difficulties are very great.

Auscultation does not give much assistance. A systolic bruit is almost always present. It is frequently heard over a very large area in front, and for some reason appears to be particularly well conducted to the back, being often heard at the angles of both scapulæ. It usually entirely replaces the first sound. A rough pre-systolic murmur is often present, even when there is no stenosis of the mitral orifice; and this fact, together with the frequent absence of the second sound at the apex, often leads to the diagnosis of mitral obstruction, although the sharp first sound characteristic of the latter condition is not heard. In one case which I observed there was such a murmur, and at the autopsy the mitral orifice was found to admit four fingers. Sometimes this bruit, which

must be of exocardial origin, is unaffected by pressure and by respiration. I am inclined to exclude mitral stenosis in very young children, even when a rough presystolic bruit is heard at the apex, because contraction of the mitral orifice is rare in the very young.

Exocardial sounds at the base of the heart may also lead to grave errors in diagnosis. In one case which I saw in 1897, although pericardial adhesions were diagnosed, the double bruit heard at the base was thought to be due to aortic stenosis and regurgitation. The heart was very large, and the hypertrophy and dilatation were considered to be mainly due to the valvular lesion. At the autopsy the pericardium was found to be universally adherent. It was hugely dilated, and the heart weighed thirty-one and a half ounces, but the valves were quite healthy. In several other cases I have found that aortic lesions have been diagnosed on account of the presence of exocardial sounds at the base of the heart.

During the period of failure the heart is always rapid and often irregular. One case was admitted to the hospital suffering from tachycardia. There were no bruits, and at the autopsy a dilated heart with adherent pericardium was found. The *pulsus paradoxus* has been shown to be by no means pathognomonic of adherent pericardium, and it is in my experience rare. The surface veins are commonly dilated, and those of the neck often show the phenomenon of diastolic emptying, frequently miscalled pulsating veins. The liver is distended and tender, and, as I have already remarked, the distension is greater in the young cases. When the dilatation of the right heart has led to tricuspid regurgitation there is pulsation of the liver and of the cervical veins. In those cases in which the inferior vena cava is compressed the ascites and oedema are sometimes indistinguishable from the conditions found in cirrhosis of the liver.

Many of the younger patients have a particularly good appetite and are free from gastric troubles for a long time, but ultimately the chronic congestion of the stomach

induces pain after food, flatulence, and vomiting. Towards the end vomiting is persistent, but it does not then appear to be altogether due to the condition of the stomach, but rather resembles that found in association with renal disease or toxæmia. In any case, severe vomiting is a grave symptom. Back pressure upon the kidneys naturally results in a diminished flow of urine with the presence of albumen.

Sleeplessness is a very troublesome symptom. In the periods of cardiac failure the patient cannot sleep in the horizontal position; and I have found that many patients, even in the intervals, prefer to be propped up in bed. The older patients often complain of dreams which cause them to awake frightened. Occasionally there is great mental excitement with hallucinations, particularly of sight and hearing. The pain in some of the younger patients causes grave nervous symptoms. In one infant, aged fifteen months, the constant cry suggested meningitis, and the case was sent to me as probably suffering from that disease. In one case death was preceded by twitchings of the right arm, leg, and face. There was no evidence of uræmia, and at the autopsy there were no obvious signs of disease of the brain. It is not at all uncommon for the nervous symptoms to so mask the condition, that attention is directed to the brain and not to the pericardium and heart.

Manner of death.—Some of the patients died quite suddenly, the heart being arrested in a condition of asystole. In most cases the body becomes more and more water-logged, and the failure is gradual. As I have already said, death may be ushered in with fits, which are not of uræmic origin. In one case, however, the condition of the kidneys appeared to be the determining factor in the production of puerperal eclampsia, the patient dying from uræmic convulsions soon after the birth of her first child.

3. *Cases in which compensation is never established.*—Here the failure is progressive from the first. The

orthopnoea of the acute stage is persistent. Vomiting is early, and the nervous symptoms are marked. I have seen the precordial dulness steadily increase, and in one case it extended from the right nipple far into the left axilla. The dilatation in some instances has been so extreme that the heart has been punctured, under the impression that the enormous area of dulness could only be produced by a large pericardial effusion. The essential point is that it could only occur in a dilated pericardium. The heart becomes more and more feeble, and there is tricuspid regurgitation. The evidence of back pressure becomes more and more marked, and death occurs in from three to nine months after the acute attack.

FINAL CONCLUSIONS.

The remote prognosis of pericarditis depends in the main upon the amount of dilatation of the pericardium. The dilatation is due to the softening of the sac by inflammation. It may occur at once before the patient has recovered from the acute illness. It may be brought on by too early resumption of work after the acute attack, or it may be due to the patient keeping about while there is latent inflammation of the sac.

The dilatation may be progressive, and then compensation can never be established, but a similar result occurs if the primary dilatation be excessive.

The adhesion of a dilated pericardium to the chest-wall, while it renders the condition of dilatation permanent, may be of advantage when the adhesions become consolidated, for the thick sac acts as a support to the cardiac cavities. If the dilatation of the sac is not very great, the support thus afforded in some measure replaces that of the normal pericardium; but as the cardiac envelope is dilated beyond its normal dimensions, so also is the heart. In these abnormal conditions there is a great tendency for the compensation to be impaired. Such

cases may live for years after the original attack; but they are subject to frequent breakdown, and are in a precarious state. If the pericardium is undilated there may be universal adhesions, but these are of no moment.

As regards age and sex the young cases show a greater tendency to dilatation of the pericardium, and therefore do worse than the older patients. The period during which there is rapid increase of the body weight is one which is especially trying to a heart working in a dilated pericardium. In girls who have suffered from pericarditis in childhood, and in whom compensation has been more or less established, the failure is almost certain to take place between the thirteenth and fifteenth years. In women the years of heavy work, pregnancy, and parturition are those in which failure of the heart is prone to occur.

In boys the failure occurs later than in girls—in the sixteenth and seventeenth years, or in the years of early labour. In adult males the prognosis is good, but heavy work, especially soon after an acute attack, leads to dilatation of the softened sac, and consequent failure of compensation.

REFERENCES.

1. WEST.—Discussion at the Medical Society. *Lancet*, January 15th, 1898.
2. MORGAGNI.—*De sedibus et causis morb.*, tom. ii, lib. ii, ep. 17—24.
3. CORVISART.—*Maladies du cœur*, ch. i, art. 2.
4. LAENNEC.—*De l'auscultation médiate*, part 4, sec. 2, ch. xx.
5. HOPE.—*Diseases of the Heart*, 4th edit., pp. 181—2.
6. GAIRDNER, W. T.—*Edin. Monthly Journ.*, 1851, vol. i, pp. 103 seqq.
7. KENNEDY.—*Edin. Med. Journ.*, 1858, vol. iii, p. 987.

8. GAIRDNER, W. T.—Edin. Med. Journ., 1858, vol. iii, pp. 1119 seqq.
9. BAUER.—Ziemssen's Cycloped., vol. viii, pp. 638 seqq.
10. FAGGE.—Principles and Practice of Medicine, 1st ed., vol. ii, p. 43.
11. WILKS.—Guy's Hosp. Reports, 1872, vol. xiv, p. 196.
12. STURGES, O.—Lumleian Lectures. Heart Inflammation in Children, 1894.
13. Brit. Med. Journ., 1898, vol. ii, pp. 1120 seqq.
14. BROADBENT, J. H.—Adherent Pericardium, 1894.
15. ROBERTS, F.—Allbutt's System, vol. v, p. 773.
16. GAIRDNER, Sir Wm.—Brit. Med. Journ., vol. ii, p. 759, 1898.
17. BARNARD.—The Functions of the Pericardium, Physiological Society, 1898.
18. HILL, L.—Physiological Congress, Cambridge, 1898.
19. BURNS.—Observations on Diseases of the Heart, p. 151.
20. BAUER.—Ziemssen's Cycloped., vol. viii, p. 638.
21. WILKS.—Guy's Hosp. Reports, 1872.
22. Discussion on Adherent Pericarditis, Medical Society, January, 1898.
23. CHEVERS.—Guy's Hosp. Reports, vol. vii.
24. KENNEDY.—Edin. Med. Journ., 1858.
25. SIBSON.—Reynolds' System, vol. iv, p. 421.
26. BROADBENT, J. H.—Adherent Pericardium, 1894.
27. SIBSON.—Reynolds' System, vol. iv.
28. SANSOM, A. E.—Clinical Journ., 1898, December 21st.
29. BALINT.—Deutsch. med. Woch., January 6th and 13th, 1898.
30. HOPE.—Diseases of the Heart, 1839, p. 142.
31. DOUGLAS-POWELL, Sir R.—Allbutt's System, art. "Myocarditis."
32. GOODHART.—Path. Trans., vol. xx.

33. SIBSON.—Reynolds' System, vol. iv, p. 448.
34. BOWDITCH.—The Growth of Children, Boston, 1891.
35. HEAD, H.—Brain, parts lxxiv and lxxv, pp. 158, 159.

TABLE OF CASES.

TABLES OF CASES.
Cases in which Compensation was never established.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Date of death.	Remarks.
1	Harry P.	15 mos.	June, 1896	?	Sept. 21, 1896	Universal adhesions; heart large; valves unaffected.
2	Florence C.	4	Christmas, 1890	Rheumatism	June 29, 1891	Universal adhesions; heart 5 oz.; mitral slightly dilated.
3	Emily L.	5	June 3, 1891	"	March 31, 1892	Mitral slightly stenosed; recent endocarditis; universal adhesions; heart 10 oz.
4	Sarah Ann R.	5	Oct. 27, 1893	"	June 10, 1894	In hospital practically till death. M.S., bruit. No P.M.
5	Sidney D.	5	Early in 1896	"	June 1, 1896	Universal adhesions; valves healthy.
6	Michael P.	6	April, 1898	"	June 27, 1898	Huge dilatation; M.S. No P.M.
7	Chas. S.	8	Sept. 29, 1897	Chorea	Feb. 11, 1898	Universal adhesions; heart large; mitral two fingers; small vegetations on aortic.
8	Arthur M.	9	Dec. 11, 1895	Rheumatism	March 22, 1896	Universal adhesion; recent vegetations on mitral, but no stenosis; heart large.
9	Eliza H.	12	April 26, 1894	"	Jan. 26, 1895	Universal adhesions; mitral stenosis; recent vegetations on mitral and aortic; heart 22 oz.
10	Rose C.	12	Aug. 7, 1894	"	Sept. 30, 1894	Removed by friends and died at home. No P.M.
11	Lizzie B.	14	Dec., 1896	"	June 30, 1897	Universal adhesions; recent vegetations on mitral and aortic; no stenosis; heart 19 oz.
12	Alfred S.	17	Sept., 1894	"	March 4, 1895	Universal adhesions; mitral two fingers; aortic slightly incompetent; heart 19½ oz.
13	Thos. T.	21	June 26, 1895	"	July 6, 1896	Complicated with ulcerative endocarditis; universal adhesions.

Cases in which Compensation is easily broken.
MALES.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
14	Charles K.	8	1890	Rheumatism	Seen May, 1899	Always ailing. Huge heart. Adhesions diagnosed. (See note at end of tables.)
15	David S.	13	1890	"	Seen Sept. 31, 1898	Always under treatment. Several times warded. A.S., A.D., M.S.
16	Sidney R.	10	Jan. 23, 1890	Rheumatism, chorea	Died, L.H., May 10, 1891	Nodules. Readmitted with fresh pericarditis. P.M.—Adhesions; valves little affected.
17	Albert D.	12	1891	Rheumatism	Died, L.H., April 5, 1898	Several times in hospital. Double bruits at base (1898). P.M.—Adhesions; heart 15½ oz.; mitral affected; aortic free.
18	Morris R.	13	Dec. 16, 1891	"	Died, L.H., May 21, 1897	Several times in hospital. Enormous heart, thought to be effusion; A.S., A.D., M.S. P.M.—Adhesions; heart 47 oz.; aortic and mitral affected.
19	John D.	16	1892	"	Died, L.H., July 28, 1897	Often under treatment. Double bruits at base. P.M.—Adhesions; heart 31½ oz.; valves healthy.
20	Charles L.	7	1893	Chorea, rheumatism	Seen July 5, 1898	Cannot play because of pain at heart; A.C.D. not much enlarged; M.P.S.
21	Robert C.	11	1893	Chorea	Died, L.H., July 7, 1896	Several times in hospital. P.M.—Adhesions; large heart; mitral stenosis.
22	Alexander C.	17	1893	Rheumatism	Died, L.H., Jan. 29, 1897	Always ailing. P.M.—Adhesions; mitral incompetent.
23	Samuel G.	10	May 11, 1893	"	Seen June 11, 1898	Well nourished; slightly cyanosed; A.C.D. increased; M.S.
24	George S.	10	June 30, 1893	"	Seen July 5, 1898	No school; several times laid up; A.C.D. large; A.S., A.D., M.S.
25	Robert S.	18	Sept. 9, 1893	"	Seen June 25, 1898	Anæmic; A.C.D. not much increased; M.S. and exocardial; veins distended.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
26	William L.	4	1894	Rheumatism	Seen July, 1898	A.C.D. much increased; M.S.; signs of adhesions; pulsating veins, &c.; nodules.
27	Arthur R.	5	1894	"	Died, L.H., Nov. 2, 1896	A.C.D. large; M.S.; nodules. P.M.—Adhesions; mitral slight stenosis; heart 13 oz.
28	Chas. D.	6	Feb. 2, 1894	"	Died, L.H., Nov. 2, 1896	Large heart; M.S., M.P.S.; nodules. P.M.—Adhesions; mitral stenosis.
29	Chas. S.	13	June 2, 1894	"	Seen July 5, 1898	Ill developed; anæmia; large heart; exocardial at base; M.S.
30	Thomas P.	12	Sept. 4, 1894	"	Seen June 11, 1898	Anæmic; wasted; no school. Large heart; adhesions diagnosed.
31	Albert B.	17	Sept. 26, 1894	"	Died at home, Dec. 26, 1898	Often in hospital. Adhesions diagnosed. No P.M.
32	Herbert J.	10	Oct. 9, 1894	"	Seen June 22, 1898	Wizened, small boy; looks younger. Adhesions probable.
33	Gershon L.	10	Feb. 10, 1895	"	Died, L.H., Sept. 28, 1898	Often under treatment. Large heart; M.S., bruit. No P.M.
34	Edward S.	15	April 9, 1895	"	Seen July 5, 1898	Cannot work. In and out of hospital and infirmary. Large heart; M.S.
35	Henry H.	17	June 13, 1895	"	Seen Nov. 9, 1898	Anæmic; large heart; A.S., A.D., M.S. Attends L.H. out-patient department.
36	William C.	37	Sept. 11, 1895	Extension	Seen June 18, 1898	"Short-winded," and not the man he was. A.C.D. large; M.S.
37	John C.	11	S pt. 30, 1895	Rheumatism	Died, L.H., Jan. 8, 1897	Adhesions diagnosed. Confirmed P.M. Mitral stenosed; large heart.
38	Arthur C.	16	Nov. 16, 1895	"	Seen often, 1897-98	Adhesions diagnosed. Frequently in hospital.
39	Albert B.	17	Nov. 19, 1895	"	Sister seen, June, 1898	Has had to give up work. Lives in country. Edema of feet from time to time.
40	Clifton W.	8	Feb., 1896	"	Seen March, 1899	No school. Always under treatment. Adhesions diagnosed. Final failure appears imminent.
41	Fred. S.	15	Aug. 7, 1896	"	Seen June 11, 1898	A clerk; very anæmic; wasted; dyspnoea; cardiac area large; M.S.
42	William S.	46	Oct. 17, 1896	"	? Seen June 13, 1898	Much dyspnoea; heart irregular; large dulness; no bruit; distended veins.

43	William C.	6	Nov. 1, 1896	"	Died, L.H., July 26, 1897	Recurrent pericarditis. P.M.—Adhesions; mitral thick- ened; not stenosed.
44	William E.	22	Dec. 10, 1896	"	Seen Aug. 9, 1898	Large heart; A.S., A.D., M.S.; adhesions probable. Pain with heavy work.
45	Wm. H. B.	11	Jan. 4, 1897	"	Seen June 13, 1898	Thin, anæmic; heart area large; M.S.
46	Fred. Y.	23	Jan. 4, 1897	"	Seen May 22, 1898	Pain ever since. Anæmic; M.S., M.P.S.; double ? exo- cardial sounds at base.
47	Chas. S.	11	Feb., 1897, Great Ormond Street	"	Seen June 13, 1898	In hospital twice since. Nodules; large heart; adhe- sions probable.
48	Thomas W.	40	June 29, 1897	?	Seen June 13, 1898	Cannot work so well; dyspnoea; heart area large; M.S.
49	George D.	9	July 23, 1897	Rheumatism	Seen June 13, 1898	Small, weak, and anæmic; dyspnoea; large heart; M.P., M.P.S.
50	William C.	21	Sept. 20, 1897	"	Seen Nov. 10, 1898	Huge heart; adhesions diagnosed; A.S., A.D., M.S.
FEMALES.						
51	Ellen C.	8	Jan., 1890	"	Seen Oct. 30, 1898	Anæmic; emaciated; adhesions; several times in hos- pital.
52	Catherine T.	13	July 1, 1890	"	Died, L.H., Aug. 7, 1894	Huge heart; several times in hospital; no P.M.
53	Alice W.	14	Oct. 23, 1890	"	Died, L.H., June 24, 1892	Huge heart; never well after attack. P.M.—Adhesions; heart 32 oz.; mitral stenosis.
54	E. A.	14	1890	"	Died, L.H., Sept. 8, 1895	Ill off and on; A.C.D. large. P.M.—Adhesions; heart 27 oz.; mitral incompetent.
55	Selina M.	13	Dec. 20, 1891	"	Died, L.H., Sept. 27, 1894	Several times in hospital. P.M.—Adhesions; large heart.
56	Frances S.	9	Jan. 6, 1892	"	Seen May, 1898; heard from Oct., 1898	Ill off and on ever since; small, anæmic; too ill to com- up for examination. ? Since dead.
57	Kate W.	10	Jan. 28, 1892	"	Died at home, 1894	Several times in hospital; failing compensation.
58	Caroline D.	13	July 3, 1892	"	Died, L.H., Jan. 6, 1894	Several times in hospital; M.S., M.P. P.M.—Adhe- sions; heart 26 oz.; mitral 3 fingers; tricuspid dilated.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
59	Sarah B.	7	Oct. 3, 1892	Rheumatism	Died at home, Dec. 30, 1896	Never well since; has been at several hospitals.
60	Amy B.	15	1893	"	Died, L.H., Jan. 21, 1895	Several times in hospital; fresh pericarditis before death. P.M.—Adhesions; heart 19 oz.; valves normal.
61	Mary Ann I.	16	Feb. 1, 1893	"	Died, L.H., Aug. 22, 1898	In hospital in 1896; married 1897; 6 months pregnant on admission. Death from uræmia. P.M.—Adhesions local; dilated pericardium.
62	Maud M.	10	Aug. 9, 1893	"	Seen June 7, 1898	In hospital frequently; several subacute attacks.
63	Mary D.	12	Sept. 6, 1893	"	Died, L.H., Oct. 15, 1895	Double mitral and aortic bruits. P.M.—Adhesions; heart 17½ oz.; mitral incompetence; aortic free.
64	Elizabeth F.	11	Sept. 12, 1893	"	Seen July 1, 1898	A.C.D. not much increased; M.S.; anæmia; pain; cedema of legs.
65	Sarah A. K.	5	Oct. 27, 1893	"	Died, L.H., June 5, 1894	Went out compensated; returned in a week and rapidly failed. No P.M.
66	Elizabeth D.	14	1893	Scarlet fever	Seen June 11, 1898	Frequently in hospital; A.C.D. large.
67	Sarah B.	12	Feb. 20, 1894, E.L.C.H.	Rheumatism	Died, L.H., April 17, 1897	M.S., M.P.S., bruits. P.M.—Adhesions; heart 19½ oz.; mitral admitted 4 fingers.
68	Jessie K.	23	Mar. 15, 1894	"	Died, L.H., Sept. 8, 1897	Several times in hospital. P.M.—Adhesions; heart 32 oz.; mitral stenosis; large patent foramen ovale.
69	Annie S.	11	Mar. 21, 1894	"	Seen June 20, 1893	Has been laid up several times; dyspnoea and cedema of legs.
70	Alice Q.	16	May 10, 1894	"	Died, L.H., Aug. 29, 1895	Several times in hospital. P.M.—Adhesions; heart 27 oz.; myocarditis; mitral 3 fingers.
71	Eliza H.	8	June 29, 1894	"	Died, L.H., June 9, 1895	Several times in hospital; adhesions; heart 12½ oz.; mitral and aortic disease.
72	Ellen P.	14	July 4, 1894	?	Seen Oct. 5, 1898	Heart pulled over to right; retraction, &c.; poorly developed; often in hospital.
73	Rose H.	8	Sept. 18, 1894	Rheumatism	Seen June 20, 1898	Wasted; irregular at school; adhesions diagnosed.
74	Grace D.	8	Oct. 24, 1894	"	Seen Aug. 7, 1898	Dyspnoea; pain; large heart; adhesions probable.
75	Florence H.	13	June, 1895	?	Seen June 24, 1898	Often ill; at several hospitals; adhesions diagnosed.

76	Ruth H.	13	1895	Rheumatism	Seen often Aug., Sept., 1898	Anæmic; wasted; large heart; double exocardial mur- mur at base.
77	Any F.	10	Sept. 25, 1895	"	Seen July 29, 1898	Frequently under treatment; large heart; exocardial at base; M.S. and doubtful M.P.S.
78	Ellen H.	6	Dec. 5, 1895	Chorea	Seen June 22, 1898	Only 5 weeks at school in 3 years; pain; wasting; large heart.
79	Phæbe H.	7	Dec. 5, 1895	Rheumatism	Died, St. Bart's Hosp., Sept., 1896	Adhesions diagnosed, confirmed by P.M. at St. Bartholo- mew's Hospital.
80	Florence D.	9	1896	"	Seen Oct., 1898	Never well since; large heart; adhesions probable.
81	Violet H.	10	1896	"	Seen July 10, 1898	Much dyspnoea and palpitation; large heart; M.S., M.P.S.; exocardial sounds.
82	Rose Le B.	20	April 8, 1896	Pneumonia	Seen July 10, 1898	Pain, dyspnoea, &c.; large heart; no bruit.
83	Rachel C.	7	June 18, 1896	Rheumatism	Seen often, 1898	In hospital 8 times in 18 months; large heart; adhe- sions.
84	Ellen T.	40	June 18, 1896	"	Died at home, Sept., 1897	The attack was probably acute on old trouble. Died suddenly at home. Inquest: heart disease.
85	Eliza T.	18	July 29, 1896	"	Seen June 18, 1898	No work; anæmia; dyspnoea; large heart; trouble chiefly aortic.
86	Maud B.	9	Aug. 9, 1896	Chorea	Seen June 18, 1898	Always ill; often in infirmary and hospital; large heart; adhesions diagnosed.
87	Clara C.	10	Aug. 19, 1896	Chorea; rheumatism	Died, L.H., April 12, 1898	Never well after; large heart: no P.M.
88	Ellen R.	4	Aug. 22, 1896	Rheumatism?	Seen June 13, 1898	Edema from time to time; purpura; large heart; M.S.
89	Theresa M.	5	Oct. 11, 1896	Rheumatism	Seen Mar. 13, 1898	Often in hospital; very anæmic; large heart; adhesions probable.
90	Ellen C.	10	Nov. 13, 1896	"	Seen Oct. 24, 1898	In hospital since with broken compensation; large heart; adhesions probable.
91	Caroline S.	8	Dec. 12, 1896	"	Seen June 14, 1898	Very little school; in-patient since; large heart; adhe- sions probable.
92	Annie C.	14	Feb. 21, 1897	Chorea	Seen June 14, 1898	Anæmia; dyspnoea; heart area not much increased; M.S.
93	Priscilla E.	11	Feb. 24, 1897	"	Seen May, 1898	In hospital, 1898, with broken compensation.
94	Eliza B.	12	April 27, 1897	Rheumatism	Seen Oct. 10, 1898	Many times in hospital since; heart area very large; M.S.

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
95	Annie B.	10	June 5, 1897	Rheumatism	Seen June 13, 1898	In hospital since; pain; large heart area; M.S.
96	Mary A. G.	17	June 29, 1897	"	? Seen July, 1898	Admitted with great failure of compensation, May, 1898.
97	Alice G.	14	July 23, 1898	Rheumatism	Seen July 14, 1898	Compensation once broken since; heart area increased; M.S.
98	Eliza M.	8	Aug. 30, 1897	Chorea	Seen Oct. 13, 1898	Always under treatment as in- or out-patient.
99	Gertrude G.	8	Nov. 15, 1897	"	Seen Nov., 1898	No school; large heart; adhesions diagnosed.
100	Annie W.	8	Nov. 23, 1897	"	Died at home, Nov. 14, 1898	In hospital twice with broken compensation; no school; adhesions diagnosed; no P.M.

Cases where Compensation remains perfect.

MALES.

101	Walter H.	26	May 3, 1891	Rheumatism	Oct. 6, 1898	Lives in America; good health; information from brother.
102	William M.	19	Aug. 22, 1892	"	Sept. 17, 1898	Light work; no symptoms.
103	William W.	21	Nov. 6, 1892	"	"	Engineer; rather anæmic; no symptoms.
104	Charles A.	27	Nov. 16, 1892	"	Sept. 19, 1898	Builder's labourer; old aortic disease.
105	Charles T.	31	April 28, 1893	?	Aug. 10, 1898	Carman; no pain, but cannot carry very heavy weights.
106	Henry P.	23	Sept. 6, 1893	Rheumatism	Sept. 17, 1898	Waterside labourer; no symptoms.
107	George L.	14	Sept. 4, 1894	"	Aug. 18, 1898	Sailing barge mate; curious attacks of pain.
108	John A.	36	Sept. 2, 1894	"	June 22, 1898	Beer bottler; no symptoms.

109	Edward A.	38	Oct. 22, 1894	" syphilis	June 20, 1898	Light work; no trouble except flatulent dyspepsia; A.C.D. increased,
110	Henry B.	18	May 30, 1895	Rheumatism	July 5, 1898	Grocer; cycles; no symptoms; signs of mitral stenosis.
111	William W.	27	March 26, 1896	"	June 13, 1898	Brewer's labourer; no symptoms; soft M.S.
112	Arthur S.	33	Sept. 1, 1896	Renal	"	Night coffee-stall keeper; slight dyspnoea.
113	David H.	32	Sept. 11, 1896	Rheumatism	"	Cook; no symptoms.
114	Samuel T.	20	Sept. 21, 1896	"	June 18, 1898	Shipwright; occasional pain at night.
115	Herbert B.	21	Oct. 10, 1896	" chorea	Oct. 15, 1898	Railway work (G.E.R.); slight pain; initial attack very severe.
116	Woolf, H.	15	Oct. 30, 1896	Rheumatism	June 13, 1898	Bootmaker; occasional epistaxis; M.S.
117	Fred. R.	27	Nov. 7, 1896	Pleurisy	June 11, 1898	Glass-blower; no symptoms; exocardial sounds.
118	Alfred C.	16	March 13, 1897	Rheumatism	June 13, 1898	Works at lathe; occasional pain.
119	Samuel C.	19	April 10, 1897	"	June 5, 1898	One attack of pain since.
FEMALES.						
120	Sarah D.	35	Feb. 7, 1890	Rheumatism	Oct. 10, 1898	Well nourished; slight dyspnoea; M.S.; dyspepsia.
121	Alice M.	19	Nov. 12, 1890	"	Oct. 4, 1898	Patient lying-in with third child; husband seen; general health good.
122	Ellen L.	14	Oct. 10, 1894	"	July 21, 1898	In service; light place; no symptoms.
123	Annie T.	18	Oct. 10, 1895	"	June, 1898	No symptoms; M.S.
124	Frances P.	14	April 21, 1895	"	June 17, 1898	Tailoress; no symptoms; M.S.
125	Ellen M.	20	Aug. 10, 1896	" ?	June 15, 1898	Letter from; "quite well."

No.	Name.	Age.	Date of pericarditis.	Ætiology.	Latest information.	Remarks.
126	Elizabeth B.	16	Nov. 12, 1896	Rheumatism	June 13, 1898	No symptoms; exocardial at base.
127	Annie C.	22	Dec. 11, 1896	"	June 22, 1898	Married since; lives at Bristol; letter "quite well."
<i>Three Cases in which death was primarily due to other causes.</i>						
128	Henry C.	28	June, 1898, Windsor Infirmary	Rheumatism	Died L.H., Nov. 25, 1895	Graves' disease and diabetes; death from coma; P.M. adhesions; heart 13 oz.; valves normal.
129	John B.	43	Christmas, 1894	?	Died L.H., May 16, 1896	Admitted with tachycardia; heart dilated; death from hæmatemesis; P.M. adhesions; heart 14 oz.; valves normal; gastric ulcer.
130	Fred. M.	37	March 31, 1896	Tubercle	Died at home, Sept., 1897	Tubercular pleurisy and pericarditis; later in hospital with phthisis, from which he died.

In these tables the following contractions are used:

A.C.D. = Area of cardiac dulness.

M.S. = Mitral systolic bruit.

M.P.S. = Mitral presystolic bruit.

A.S. = Aortic systolic bruit.

A.D. = Aortic diastolic bruit.

L.H. = London Hospital.

E.L.C.H. = East London Hospital for Children.

The dates given depend, except where otherwise stated, upon the Registers of the London Hospital and North-eastern Hospital for Children, or upon the writer's own investigations.

Note.—Case 14. This patient has since died. The pericardium was dilated and adherent. The heart weighed 36 oz. The valvular lesions were slight.