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BY

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These Lectures, which were delivered at the College of Physicians in the spring of 1894, were minutely revised by the author and left by him completely ready for publication. Their revision was the last work of his life and was finished only a few days before his death. Nothing remained to be done by others but to see that the manuscript had been accurately followed by the printer.

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ITS FORMS.

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Mr. President, Fellows, and Gentlemen,—When I was honoured by the invitation of the Censors' Board to deliver the Lumleian Lectures, many doubts arose in my mind. None of these, however, had reference to the subject upon which to discourse. That point is, indeed, largely settled by the prevailing custom of selecting some topic relating to practical medicine on this occasion, but it is determined more imperatively in my case by the knowledge that for me there is but one way of fulfilling the allotted task. Could I but rehearse with tolerable fidelity some of the clinical experience of past years, especially could I do so in reference to some branch of practice to which chance has given me fuller access than falls to everyone, then, and then only, could I hope to interest an audience like this: and so I chose the subject of heart inflammation in children.

The study of heart disease in childhood, busy as it has been of late years, had hardly a place in the early observation of

such lesions. Seventy years ago—within the memory of one at least of the Fellows of this College[1]—when Laennec was investigating the physical signs of valvular disease, little mention was made of children. Fifty years ago, when Rilliet and Barthez wrote what was long the best known book about children, the part devoted to heart disease was meagre and halting; and pericarditis, which we now know to be especially common in early life, was spoken of as of rare occurrence and obscure signs. And coming to later times and to our own country, it is curious to note how the great workers in cardiac pathology—Stokes, Latham, Watson and Walshe—almost ignore little children.

The cause of this delayed study is obvious enough. The man was an easier subject than the child. The full-grown heart, moderate in pace, of decided rhythm, and well marked distinction of sounds and intervals, is the easiest to appreciate in health and to detect and define in disease. Moreover, chronic valve defects, both evident and enduring, occur most in adult life. Hence it was that Laennec and his contemporaries concerned themselves most with the physical signs and symptoms of gross structural lesions. The very beginning of heart disease, the active stage of its inflammation as it occurs in young subjects, was imperfectly known to these early observers. It was, in fact, largely pieced out by the bold, and, as we now know, the unwarranted assumption that the physicial signs of chronic

[1] Dr. Bisset Hawkins was admitted to the fellowship of the College in 1826, the year that Laennec published the second edition of his great work on Auscultation, wherein he replies at length to the attacks of his contemporary Broussais. Our Senior Fellow had thus reached the highest grade of his profession at the time when auscultation was being hotly discussed and its author in the thick of that short-lived obloquy which befalls most inventors. He was censor in 1830, when the new teaching was still imperfectly known and but partially accepted, and delivered the Lumleian Lectures sixty years ago (in 1835), a year before the publication of Laennec's last treatise upon Auscultation in conjunction with Andral.

heart disease might be made to apply with some modification to that which is recent.

It is now frankly admitted that the early diagnosis of heart inflammation, at whatever age, is among the most difficult problems of practical medicine, and that it depends largely upon the presence or absence of pericardial friction sound, a sign of which the significance was for long altogether missed. Laennec, indeed, was not ignorant of the existence of such a sound. He both heard and described it; but for reasons not hard to understand, and which, as I shall show, have a certain validity at the present time, he missed its meaning. "For some time," said he, "I thought that this noise, like the cry of the leather of a new saddle under the rider, might be a sign of pericarditis, but I have since convinced myself that it is nothing of the sort."[1] And, thereupon, when in actual touch of the truth, the great pioneer of auscultation goes astray upon certain theories of sound producing gases, leaving it to others, and especially to three distinguished physicians of our own—Latham, Stokes and Watson—to recover the path and pursue it to the desired end.

It thus appears that of the two main factors of cardiac inflammation, the one, pericarditis, had to await analysis until a comparatively recent period, while the other, endocarditis, may be said to await it still. At the same time the conflicting opinions of to-day upon many questions of physical diagnosis, as, for instance, the manner of the heart's displacement in pericardial effusion, the meaning of doubled sounds, the proper signs of pericardial adhesion or the earliest indications of mitral stenosis, supply evidence enough that to this hour we are seeking more light.

My purpose in these lectures is first, to describe and classify

[1] "Traité de l'Auscultation Mediate," vol. ii., p. 446, 1826.

certain forms of heart inflammation, forms both insidious and perilous, which are chiefly to be observed among children; secondly, to consider the physical indications of such inflammation and their significance; and thirdly, upon a review of the main facts of the case, to discuss briefly some points referring to prognosis and to treatment.

Now in physical respects, putting aside the clearer distinction of sounds and intervals presented by the adult, it would seem that children were the most promising subjects for the study of heart changes. In the case of the adult, there are many hindrances in the way of accurate observation—fatness, great muscular development, the intervention of emphysematous lungs. But in the child, with all these obstacles away, with but a thin wall of separation between the heart and your finger tips, and the organ almost within touch, its position, its shape and its acts can be easily felt and measured; all that is wanting is the actual sight of it. The heart of a little child, I say, at the first blush, would seem to be as simple in physical as it is in moral respects. But experience teaches otherwise. The child's heart holds as many secrets as the man's and is even more deceiving.

Heart inflammation in children, as is well known, occurs chiefly in connection with rheumatism, if young children be excluded almost wholly in that connection, as this table shows.

ONE HUNDRED CASES OF HEART DISEASE EXAMINED POST-MORTEM DURING ELEVEN YEARS.

Rheumatic (boys, 22; girls, 32)	54	100
Non-rheumatic (boys, 22; girls, 24)	46	

AGES OF THE RHEUMATIC.

Between 2 and 4 years	2	54
,, 4 and 6 years	4	
At 6 years old	6	
Between 6 and 12 years	42	

AGES OF THE NON-RHEUMATIC.

4 years old and under	32	46
Between 4 and 6 years	5	
Above 6 years	8	
Age omitted	1	

The babies, it will be seen, make the vast majority of the non-rheumatic; the older children the vast majority of the rheumatic. In most of these latter the rheumatism is ill-expressed in the way of arthritis, and sometimes it spares the joints altogether.

Rheumatic heart inflammation in children is almost always, so far as *post-mortem* evidence goes, both endocardial and pericardial, and in its later stages, when the pericardium has become adherent, it involves the myocardium also. For such reason, and to avoid the frequent repetition of so long a word as endo-pericarditis I speak of such inflammation as carditis.[1]

Non-rheumatic heart inflammation in children may be either endocardial or pericardial. Endocarditis by itself is a rare condition met with in very young children and in the subjects of chorea. Pericarditis by itself is found most in septic conditions, in tuberculosis and in pneumonia. All this is shown in the table.

[1] Pericarditis, I believe, never occurs to these young subjects without endocarditis, although the two inflammations may not be precisely concurrent. That endocarditis may occur without pericarditis I do not deny. It is probable, indeed, that in most instances of rheumatism it is the endocardium that suffers first. Still, in consideration of the fact that when rheumatic endocarditis occurs in childhood the pericardium is very nearly threatened, that when attacked that event is not necessarily indicated by either physical or general signs, and that there is little or no *post-mortem* evidence of the one inflammation occurring without the other in the circumstances we are considering, the term carditis is fully justified. In the early days of physical methods Corvisart and Bouillaud used the word as applying to the cardiac serous membranes and admitted that inflammation of the heart muscle alone was a condition unknown to them. Bouillaud, "Traité Clinique des Maladies du Cœur," i., p. 575, 1841.

PATHOLOGICAL RELATIONS OF 83 CASES OF HEART INFLAMMATION (PERI-ENDOCARDITIS [CARDITIS] PERICARDITIS, ENDOCARDITIS) EXAMINED POST-MORTEM.

I. Peri-endocarditis (carditis) ..	48	*All rheumatic* except one, a case of phthisis; in one other the rheumatism was obscure. In 11 the endocarditis was old.
II. Pericarditis without endocarditis ..	28	In the relations following :—
		Tubercle 8
		Empyema 8
		Pneumonia 4
		Septicæmia 3
		Diphtheria 2
		Meningitis 1
		Bronchitis 1
		Chronic arthritis 1
III. Endocarditis without pericarditis ..	7	In the relations following :—
		Old mitral disease 3
		Broncho-pneumonia; slight beading on tricuspid (a baby) 1
		Slight beading on mitral .. 1
		Pyæmia (newborn) 1
		Chorea 1

Empyema affecting the pericardium is found sometimes along with the ordinary pleural empyema, from which during life it is not easy to distinguish it; whilst when occurring alone, a purulent pericarditis hardly distending the pericardial sac, it is apt to be overlooked altogether. The pericarditis of pneumonia bears a near resemblance anatomically to the rheumatic, but the morbid sympathy is here not with the endocardium, but with the pleura. Neither as cardiac empyema nor in connection with pneumonia does non-rheumatic pericarditis show prominently; in both its physical signs are often too obscure for recognition.

It is safe to say, then, that the rheumatic heart inflammation of children when pericardial is always endocardial as well, and when endocardial is extremely likely with the recurrence

of rheumatism, if not in the first attack, to involve the pericardium also, an event which adds largely to the gravity of prognosis. Thus in the heart as elsewhere, as in catarrhal inflammation and tubercular infection, the differentiation of morbid processes is less marked in the child than in the adult, and a common sympathy more apparent.

But the rheumatic carditis of childhood—as I provisionally call it—differs from that of later life in other respects besides those already mentioned. Thus it is, or rather it seems to be, less intimately connected in time with the arthritic symptoms: these are habitually ill marked and sometimes altogether absent: its clinical course is more insidious and its immediate mortality far higher.

Such characters are perhaps best displayed by a rough classification into active, passive and chronic, terms perhaps not without objection, yet sufficient for their purpose of labelling.

Active carditis, rare in adults, has some special features in children, as I have now to show, and, owing to its sudden origin, rapid course and high mortality, claims our chief attention.

Passive carditis, that which is the usual form in the adult as an attendant on acute articular rheumatism—is similar to the first in physical signs, but without its symptoms and without its danger. It is free from cardiac pain or distress and, but for auscultation and facial aspect, might escape notice. It differs from the corresponding adult affection in two points: one that the attending joint pain is more limited and less severe; the other that the connection between it and the acute form is much closer than with the adult, the sudden transition from passive to acute or the converse being a quite common occurrence. Thus, passive carditis shows less in the child than in the adult because the rheumatism on

which it depends shows less, but it signifies much more, owing, so to speak, to its liabilities. It is, in fact, the rarity in children of what is sometimes called "frank" rheumatism—one of the many phrases by which we dictate to disease what it ought to be—their slighter pyrexia and fugitive joint pain removing them early from medical observation, that makes their danger. Apparent convalescence may be reached when cardiac inflammation is at its height. How much of evil actually ensues from the child returning to school or to play at this stage we know not, but of the commonness of such an occurrence there can be no doubt.

Chronic carditis affects the pericardium mainly or even wholly. It is almost always rheumatic, and is recognised by the changeful area of cardiac dulness as effusion ebbs and flows. This may be measured from day to day and from week to week by percussion. It is a condition not special to childhood, but the constant alternations of fluid—which eventually may disappear altogether—and the rarity of signs calling for paracentesis are its distinguishing features at that time of life.

Of these three varieties, active or acute carditis has for us now, for the reasons just mentioned, the chief interest. It happens in this wise. In the course of a rheumatic attack, or it may be following it, the heart affection is suddenly announced by restlessness, increased pallor, and a curious aspect of anxiety together with dyspnœa, perhaps delirium, and sometimes obstinate vomiting. Such symptoms suddenly transform a trivial illness into one of extreme gravity. Often there is heart pain, and tenderness over the heart region is highly characteristic. There may be but little rise of temperature and not much quickening of breath or pulse, but the act of breathing seems to give distress (sometimes, no doubt, from attending pleurisy), and there is that short expiratory grunt so often met with in the acute chest affections of children.

Along with these symptoms there may be a preference for the raised position, and some patients in their distress for breath will lean forwards, while sometimes the hands will be moved convulsively to the mouth and throat as if to drag away some material obstacle.

The posture, the pallor, the restlessness, the look of alarm and fitful delirium, coupled with dyspnœa due largely to enfeebled inspiratory effort—all these are symptoms so characteristic, that active carditis in a child may almost be detected at sight. Certainly when the chest is bared and the heart-region has been seen and felt, all doubt will disappear. When these symptoms rapidly progress to a fatal end, as they often do, it is sometimes dyspnœa, sometimes persistent vomiting, sometimes delirium, sometimes mere prostration that shows the most conspicuously. But the main source of the symptoms, however these may arrange themselves, is manifestly cardiac failure, failure too rapid to be explained by texture degeneration, and the more remarkable for being altogether absent in that passive form of carditis with which it is anatomically identical.

Without impugning the correctness of the common belief that rheumatism is the source of carditis, whether active or passive, we must guard against any rigid definition of that term, and even frankly recognise the difficulty of reaching any precise conception of its meaning. Its supposed modes or symptoms are so various that none of them can be quoted as invariable. Sometimes, for example, it is not articular, but limb pain that accompanies or precedes the heart inflammation; sometimes it is dyspnœa or cardiac pain, sometimes prostration merely. Again, in the recurrence of an attack which at the first was articular and, as we say, characteristically rheumatic, the bygone pyrexia will reappear; but in the fresh attack the heart will be actively concerned and the joints

escape altogether. To cover such variety we speak largely of the several "manifestations" of rheumatism, and of late years have added considerably to their number. But of the essential character of that morbid condition, or the order of relative frequency of its many asserted symptoms we know but little. So far as children are concerned, heart inflammation is of itself a far surer criterion of this ill-described morbid state than tender and painful joints.

In no small proportion of cases of carditis, wasting and sweating are the main premonitory symptoms, while the history of joint pain with tenderness is extremely doubtful. Even in those instances, the majority, no doubt, where there is arthritis with the associations commonly called rheumatic, it is noticeable how soon the joint pains recede and the state of the heart comes to occupy the whole attention of the physician, whether from the changeful character of its physical signs, or else owing to the sudden appearance of those urgent symptoms of heart-failure of which I have just spoken. The mortality of acute carditis in childhood is considerable, but it is variously computed. In cases got together with a view to showing characteristic symptoms, the death-rate is likely to be higher than when the object of the collection is to commend some particular method of treatment. There is material enough, however, in hospital records—even in those of the Children's Hospital alone, now covering over forty years—to furnish reliable statistics upon this subject. The difficulty is in classification.

A single example will serve to bring before you the special characters of this active form of carditis in children, and the many points in which it differs from the corresponding affection in adults :

A girl, aged 7 years and 10 months, anæmic and ill-nourished, was admitted to the Children's Hospital on April 28, 1893,

with a history of six weeks' pains in the limbs, and for the last three days swollen and painful joints. These symptoms had attracted but little notice in the family until twelve days before admission, when the child complained of pain at the heart, was seen to be short of breath, and had to be propped up in bed. The girl had never had rheumatism before, and was not of rheumatic family.

On admission, respiration was 36, pulse 120, regular both in force and frequency; temperature 99°. There was marked pulsation in the vessels of the neck, but no venous enlargement elsewhere. The area of cardiac dulness was not increased. A systolic thrill could be felt at the apex, and a loud blowing systolic murmur heard at the same place and conducted round to the back. Inside the nipple and a little above it the second sound was inconstantly reduplicated. For the following three or four days no particular change occurred. The child soon ceased to have pain. The area of heart dulness did not alter, nor did the heart sounds. The child was quiet, restful, but lethargic. Anæmia was more extreme.

On the fourteenth day of residence a marked change occurred. It began with an attack of nose-bleeding followed by a sleepless night. The child complained of headache; temperature rose to 102°, although there was no return whatever of joint pain. The heart was now tumultuous in action, its impulse forcible and diffuse, apex apparently in fifth space; the murmur had become of harsher pitch, so-called "musical" in character, and audible a finger's breadth outside nipple line, where it abruptly ceased. Within the nipple a very slight diastolic sound was heard, the commencement, as it seemed, of distinct friction. The urine was now found to contain a trace of albumen and some blood.

Hereupon (we have now reached the thirty-first day from the first heart pains and dyspnœa) the most characteristic

signs of active carditis appeared; that is to say, the child, though free from actual pain, and without marked dyspnœa, was anxious and restless, the little sleep she got being disturbed by muttering and painful visions. A friction sound (to and fro) was now audible, its maximum intensity at the ensiform cartilage, but distinctly heard as high as the second costal cartilage. The pulse, 140, was still regular; temperature 100° to 101°. The area of cardiac dulness now for the first time began to enlarge, transgressing the left border of the sternum to the right and a finger's breadth outside the left nipple to the left, the upper border reaching the third space. It continued to enlarge somewhat for a while, but presently receded, and at death was hardly larger than on admission.

Without staying to record daily changes—as in friction rub and thrill, varying character of apex murmur, the heart's action now more now less forcible and excited, while the veins of the neck began to show marked reflux, the patient being restless, moody, without continued sleep, free of pain but tender on percussion over the cardiac area—we come to May 31, the forty-second day from the first heart symptoms and within five of death. It was on this day that vomiting set in, and it persisted. The area of cardiac dulness rapidly decreased; the rub was but indistinctly heard. There was increased restlessness, difficult groaning, yet not very rapid breathing, intense anæmia, constant sickness, *pericardial effusion rapidly diminishing the while.* The regurgitation in the neck veins increased, and now first, a few days before death, œdema of the legs and signs of pulmonary œdema were noticed. So the story closes June 5. At the latest stage considerable engorgement of the lungs perhaps accelerated death by a little.

The whole duration of the carditis was forty-seven days; the more active symptoms occupied thirty-six days, and the

most urgent of them (incessant vomiting) lasted less than a week. Rub was indistinctly audible five days before death, and during that period the signs of pericardial effusion disappeared.

Of the *post-mortem* examination it will suffice to say that the heart weighed 9¼ ounces and showed both dilatation and hypertrophy; the latter as regards right auricle and ventricle. The mitral valve admitted three fingers and at its extreme edge were some minute vegetations. The other valves were normal. The pericardium was universally adherent. No fluid remained, and the surfaces were separated with difficulty.

The salient points for notice are these:—

(1) The child was young and probably of sound heart before the fatal attack, since she had never had rheumatism and was not of rheumatic family.

(2) The symptoms were mainly cardiac, and the well-marked physical signs probably less than a fortnight old on the child's admission.

(3) Characteristic nervous symptoms—restlessness, insomnia, delirium—appeared suddenly a month from the beginning of illness and concurrently with pericardial friction rub.

(4) In the progress of the case the physical signs indicated gradual cardiac failure, and, for a while, some pericardial effusion.

(5) *Post-mortem*, firm and universal pericardial adhesions were found, notwithstanding that pericardial friction had been audible shortly before death.

Time will not admit of separate mention, even in this brief form, of other examples. They have a strong family likeness, but the end does not always come in the same way. As I have said, prostration is sometimes the main feature, the patient lying for days apathetic and but partially conscious. I have had two such cases within the last year, one fatal and one

recovering. In the latter, although the boy, aged 9, ultimately recovered sufficiently to leave the hospital, prostration was so extreme that for some days the motions were passed into the bed.

Within the last three years I can recall sixteen examples of active carditis, of whom some particulars are given in this table.

CASES OF ACUTE CARDITIS.

Boys	6	.. 16
Girls	10	
The three youngest	4 years 2 months (died).	
	4 years 8 months (died).	
	5 years (recovered).	
The three oldest	10 years 5 months (died).	
	10 years 8 months (recovered).	
	11 years (died).	

DURATION OF ACUTE SYMPTOMS.

The two longest, 9 days and 8 days respectively; shortest, 1 day.
One patient showed prostration during three weeks preceding death.

Recovered	4	Boys	1
		Girls	3
Died	12	Boys	3
		Girls	9
Previous attacks of rheumatism in			12
,, ,, acute carditis in			2
Subcutaneous nodules in			4

As will be seen, the patients are between 4 years and 11. The girls much out-number the boys, and only four of the sixteen recovered. All but four were supposed to have had previous rheumatism. The pericardium was adherent in all the fatal cases. In only one was there extensive valve disease. In most the endocardial disease was limited to small, recent granulations on the mitral valve.

It is not easy to summarise the symptoms of these sixteen acute cases. It may suffice to say that in only one were joint

pains prominent and long continued. In one, pain affected the limb muscles, but not the joints; in two, slight rheumatic pains were replaced by prostration, the while, the physical signs indicated pericarditis: four were admitted for cardiac pain and dyspnœa, rheumatism in its articular form being a thing of the past; in three, wasting and sweating were the most prominent symptoms; in one, a condition of drowsiness and apathy had followed epigastric pain and dyspnœa, no obviously rheumatic symptom having been observed for the past three months.

Passive carditis exhibits precisely the same physical signs as the active form, excepting perhaps that exocardial sounds are less frequent and less obvious. But there is no similar response on the part of the system; especially no nervous response. Heart inflammation may indeed be suspected in children as in adults owing to facial change, or some hurry of respiration in the course of articular rheumatism. It can only be made sure of by physical search. Yet so nearly related are these two forms of carditis, the active and the passive, that at any moment, and with the shortest warning (and herein is the child's peculiarity), the one may be exchanged for the other. Thus, a rheumatic carditis, almost latent at starting, may at any part of its course display delirium, prostration and heart failure; or otherwise a passing restlessness or slight delirium concurring with the earliest physical signs of carditis may threaten an acute attack, but presently calm down without further development. It is true that pericardial friction seems sometimes to give the signal for active symptoms, but this is far from being always the case, and friction sound will sometimes coincide with marked general improvement.

Chronic carditis, my third division, is a less accurate description than either of the other two because the endocardial element is commonly ill expressed. The main feature of the

affection is pericardial effusion, whose variations may be measured day by day over a long period. There are no active symptoms and little change of any kind except in physical signs, of which I shall speak presently.

In fatal cases of carditis, without or with but moderate effusion, it is the rule, as I have elsewhere shown, to find adhesion, and the exception to find the heart free. At the same time it is the rule to hear exocardial rubbing to within a short time of death. Thus diagnosis upon this point derived from physical signs is very precarious, as this table shows, and as likely to be wrong as to be right.

PERICARDIAL ADHESION IN RELATION TO FRICTION SOUNDS.

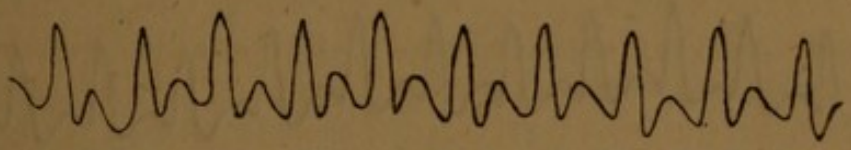
A.	Characteristic friction sounds heard shortly before death in 29:	
	Pericardium adherent "universally," "generally," "entirely," "everywhere," "universally and firmly," "extensively" in	15
	Pericardium "adherent and also to chest wall," "partly and recently adherent," "a few adhesions" in	5
	Pericardium not adherent, but there exists "pericarditis," or "roughened pericardium," or "shaggy cords" in	5
	No adhesions mentioned (probably none existed) in	4
B.	No friction sounds audible in 17:	
	Pericardium "adherent throughout" in..	6
	Pericardium "adherent at apex" and "partially adherent" in	3
	Pericardium contains "excess of fluid" in	3
	Pericardium soft buttery lymph in	1
	Recent flakes of lymph in	4

Physical signs misleading in 22.
„ „ informing in 24.

The phenomena of pericardial adhesion, whether old or new, are beset with difficulty. And as regards the recent cases, it is to be considered that *post-mortem* inspection does not always reveal the ways of life. It may even confuse them, and much caution is needed in applying to patients that recover what we see to happen to those that die. The act of dying itself has to be considered as a differentiating agent. As death approaches

the heart movements become more and more feeble, and pericardial adhesion is directly favoured up to the moment that the heart is still. But we can well suppose that, in the event of recovery, returning cardiac energy may suffice, if too late to prevent the adhesion due to the period of syncope, yet to break down these young and feeble bonds and set the heart free again. Adherent pericardium in that case would be a less frequent event of rheumatic carditis on the whole than *post-mortem* records would lead us to suppose.

In some instances of carditis, *post-mortem* examination discloses unusual conditions which may have influenced mechanically the mode and the distress of dying. Thus, in a



Henry Drinkwater, aged 9.

December 20.—A.C.D.[1] increasing. Loud friction. Patient pale and prostrate.

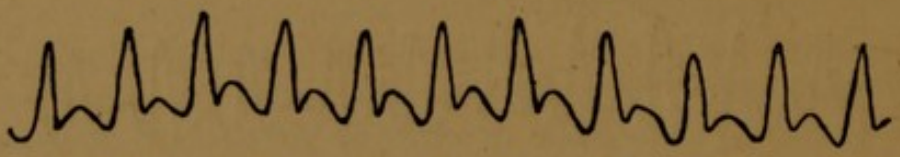
girl under 5 years old, who just before death became very blue and had a sort of anginal attack, the appendices of both auricles were pretty firmly adherent to the adjacent portions of the heart. In another case a girl of 4 had sudden and fatal collapse. *Post-mortem*, besides pericarditis a continuous decolorised clot extended from right auricle to ventricle through the tricuspid valve.

The pulse in active carditis is small and of low tension. Pulse tracings are less reliable in children than in adults, but in some cases there is marked dicrotism with some unevenness. Dicrotism, however, is no accurate measure of the patient's condition or progress. In the case here represented there was

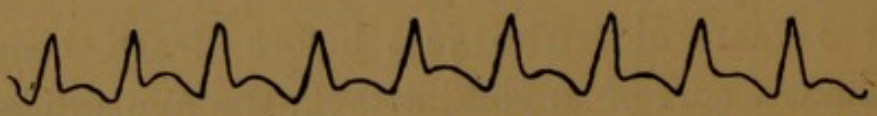
[1] A.C.D.=area of cardiac dulness.

but little difference in this respect between the tracings on a day when the child was at his worst, almost unconscious, and nine days later, when, although still prostrate, he was plainly on the way to recovery.

The body heat in acute rheumatic carditis is seldom much raised, and its variations are difficult to interpret. The comparison of a number of temperature charts conveys no notion whatever of their relative severity, nor does it even enable us to separate the active from the passive form. So long as the articular pains remain, we have the irregular trace of acute rheumatism. Later on, when in most instances the heart has begun to claim the chief attention, there is usually a fall in



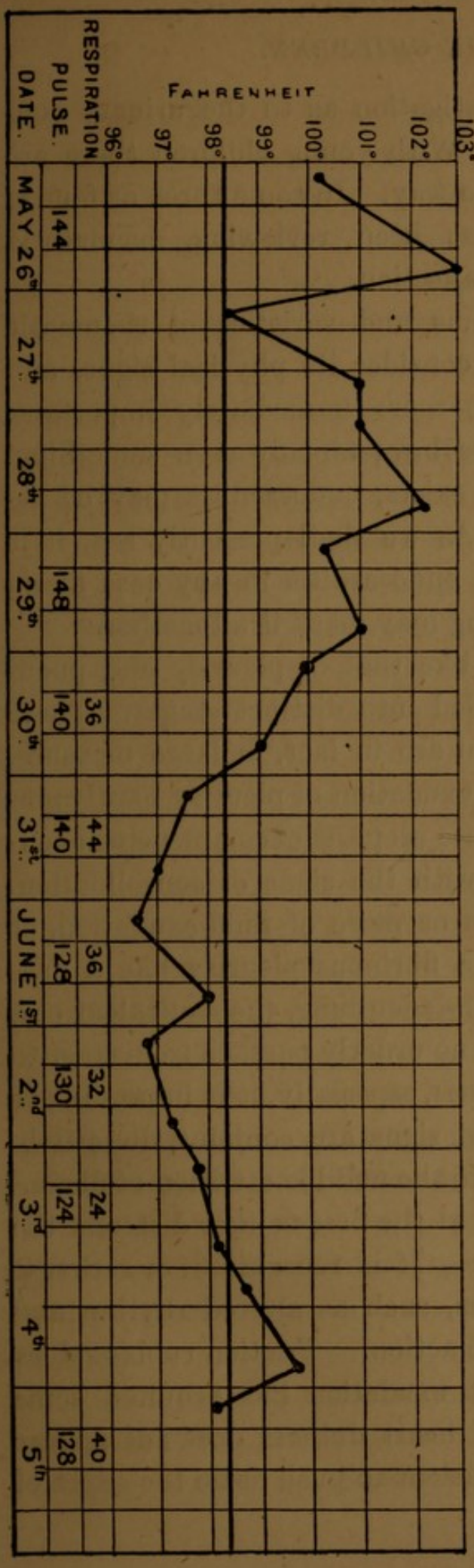
January 1.—A.C.D. much decreased, pulse 32. Still markedly dicrotic. Patient slowly mending.



January 9.—Slowly recovering. A.C.D. fixed, and as on January 1.

temperature to normal or a little above or below normal, the only constant feature being irregularity from day to day. Temperature is not to be disregarded, however. Should pyrexia persist when the joint pains have subsided, without being otherwise accounted for, it must be taken for a sign that the rheumatic state, so to speak, is still present, and the heart still threatened.

You see by this chart, which represents the last days of the patient whose case has been related, how little temperature corresponds with the other symptoms.



Cardiac dulness increases; very rapid action, friction less loud.

Marked venous regurgitation, friction less audible, vomiting.

Vomiting prominent symptom, acute cardiac dulness again decreases, regurgitation in neck, friction feebler.

Heart dulness much diminished, friction more distinct, constant vomiting, fed by enemata.

Sinking, sickness and rub the same, lung œdema and some of feet.

Very restless, vomiting frequently, cardiac dulness the same, no rub audible.

Vomiting ceased and child could be fed shortly before death, heart dulness not increased, no distinct friction sound.

I have made no exact investigation as to the urinary condition in rheumatic carditis. With young children there are some obvious difficulties in the way. In some three or four of the sixteen cases I have just been reviewing, hæmaturia occurred at the height of the affection.

From the general symptoms and varieties of rheumatic carditis we may proceed to consider its physical signs, and especially the exocardial, the more immediately important, and of whose physical basis we have already seen something.

Where recent pericarditis exists, endocarditis may be assumed to be present also. As we shall presently see, it is difficult enough to appreciate endocarditis in any case at an early stage; exocardial rubbing may mask it altogether.

The course of pericarditis, like that of pleurisy and pneumonia, is conventionally divided into distinct stages. These are described as successive, but are in fact, in large measure, concurrent. Thus the plastic exudation of pleurisy hardly precedes fluid effusion. The minute crepitus of commencing pneumonia is heard side by side with the signs of consolidation. There may be more of plastic or more of fluid exudation, or either the one or the other, a fibrinous pleurisy like a misplaced pneumonia, or a serum-laden lung, the equivalent of a pleural effusion. But there is no orderly passage from stage to stage. In the case of pericarditis, especially, it is important to notice that its earliest physical signs are conjoint, due partly to fluid distension and partly to the audible attrition of fibrin.

Pericardial effusion, indeed, at the first neither distends the sac nor causes audible rubbing. It is recognised, or rather it is suspected, from fallible signs, such as altered rhythm and quickened, tumultuous cardiac action. Friction rubbing does not appear until the earliest exudation has acquired some consistency; enlarged area of heart dulness does not appear until the fluid collection is sufficient to push aside the edges of

the lungs. But the two signs, friction and extended cardiac dulness, appear together, not at the first blush of pericarditis, as Sibson's phrase is, but a day or two later.

And observe that from this point, from the earliest physical evidence of pericarditis to the very end of it, the progress of the inflammation is measured, not by the rub, which is eminently fallacious, but by the daily changes in the area of cardiac dulness. In the acute cases the increase of fluid is usually rapid, but inconsiderable, and reaches its term within three or four days. Then the tide turns, the ebb being slower than the flow. In the chronic cases, on the other hand, as has been mentioned, the changes of area are frequent and considerable, while the alternations of fluid, now more, now less, now again more, may occupy much time.

Pericarditis is thus analogous to pleurisy, both in the signs that discover it and the mode of its measurement. But the effect of the fluid pressure is quite different in the two cases. In pleurisy the yielding lung is compressed into smaller and smaller space as effusion increases. In pericarditis, as we shall presently see, the effect of pressure upon the heart is not always the same. Probably the commonest result is that the organ is displaced upwards into the narrower portion of the thorax, where it presses against the sternal and costal cartilages, and friction is thereby made more loud and grating. But though both the area and the intensity of friction are apt to be greatest when effusion is most, this is not always so. Sometimes the fluid shows in front of the heart, separating the ventricles from the anterior pericardium, and so abolishing or lessening exocardial rub. The layer of fluid thus interposed is sometimes so thin that firm pressure with the stethoscope will suffice to bring the pericardial surfaces together, and for the moment restore the friction sound. This dislocation of the heart, of course, does not apply to the great

vessels nor to the upper part of the right ventricle, that is to say, from the first to the third spaces. It is in this situation, therefore, that friction is the least variable. I shall have to recur to this point when speaking of the physical signs of effusion.

It is within the experience of every clinical student that pericardial friction is a fitful sound, coming and going with absolute suddenness. It may be so fugitive that on the same day, even within the same hour, one will chance to hear it and not another. Moreover, as I have shown, exocardial sound is not inconsistent with extensive adhesion.

Thus the hazard of friction rub as a physical sign is both early and late, early because at first it is only by happy chance that we hit upon it; and late because exocardial sound, rub-like, may be still heard when actual attrition is rendered impossible by firm adhesion. And it may be said further of this sound, as of other things in nature, that the noise it makes is not proportionate to its importance. The slightest scratching—often undiscovered and yet more often misinterpreted—may be the prelude to severe, perhaps, fatal carditis, while, on the other hand, loud and extensive rubbing sound may become audible when effusion is subsiding and the improved aspect of the patient clearly indicates that the danger has passed.

There are certain attending signs in recent pericarditis which, in these circumstances, are by no means superfluous. Observe especially the precordial tenderness which accompanies the rub in acute cases, as well as the change that the sound undergoes—or rather, the changes—with varying pressure of the stethoscope, and sometimes, also, with altered position of the body and with time.

Some morbid sounds of the heart are apt to be imputed when not actually present, notably (as I shall presently try to show)

presystolic murmur. There are others often present, yet overlooked or misunderstood. Of these latter exocardial sounds of many varieties, rubbing, creaking, scratching and squeaking are the chief. Even the true friction sound, indicative of the pericardial surfaces rubbing the one against the other, may escape recognition for such reasons as these:

At its beginning and at its end it is often murmur-like.

It is often fitful.

It may need varying degrees of pressure to elicit it.

It must be admitted that the physical conditions necessary to produce friction, or, rather, sound that is friction-like, are, as yet, imperfectly known. It will be unheard when all its conditions are present; it will be present when there seems nothing to make it.

Friction, or apparent friction, in the case of extensive and firm adhesion is not uncommon, as I have shown. And in rare instances, what is taken for friction by expert observers is found, after death, to concur with a smooth and apparently healthy pericardial surface. Thus, a very anæmic girl, aged 12, under my care, August 26, 1893, had a double rub at the junction of the fourth left costal cartilage with the sternum, audible four days before death. *Post-mortem*, the pericardium, which was very thin and held an excess of clear fluid, was smooth and without trace of deposit. Again, a boy of 8, also very anæmic, suddenly developed a loud double friction rub which did not long continue and which on his death, some time later, was entirely unexplained by the state of the pericardium.

With such reasons for caution, it is well to remember that friction is not the only physical sign of pericarditis: there is pericardial distension also. These signs, as has been said, arise together, but neither of them is appreciable at the very beginning of the inflammation. It takes time both for the exudation to grate and for the fluid to distend its sac.

The very first physical indication of pericarditis, it has been said, is quickened, tumultuous, uneven cardiac action, a phenomenon which, because it occurs early and varies in duration and distinctness in individual cases, because, moreover, its precise significance is undetermined as between threatening inflammation and actually signifying it (to use Sibson's phrase), is, for these reasons, too little regarded. But the fact is, that these signs of heart disturbance are signs of endocarditis rather than of pericarditis. It is true that in the case of a rheumatic child exocardial inflammation so often concurs with endocardial that when the latter is present the former is threatened and may even be present though there be neither friction rub nor displaced apex. At its very beginning, as has been already said, pericarditis is without physical signs of its own.

Leaving for a while the further consideration of pericardial effusion I would conclude the present lecture by some remarks upon the pathology of rheumatic carditis in children.

Clinically, as we have seen, the main feature is sudden cardiac failure, failure which the physical signs very imperfectly explain, being precisely the same, whether the affection be active or passive, although in the latter form there are no threatening symptoms whatever. Further, the general symptoms are in the main cerebral, or at least, nervous—delirium, constant vomiting, extreme prostration.

Anatomically the pericardium is nearly always adherent, often extensively and firmly adherent. The endocardium, generally speaking, is but little affected, and the muscular substance of the heart undergoes but slight degenerative change and sometimes seems unaltered.

I have here a series of very faithful drawings by my friend Mr. d'Esterre, showing (1) the normal cardiac muscle in a child of 13; on the next page (2) is the cardiac muscle of a child, aged 8, who died very quickly with cardiac failure

within two days of pericardial rubbing being superadded to endocarditis; the muscle is but little altered and its striæ are distinct. A further drawing from the same subject less magnified shows well the inflamed pericardium (3). By way of

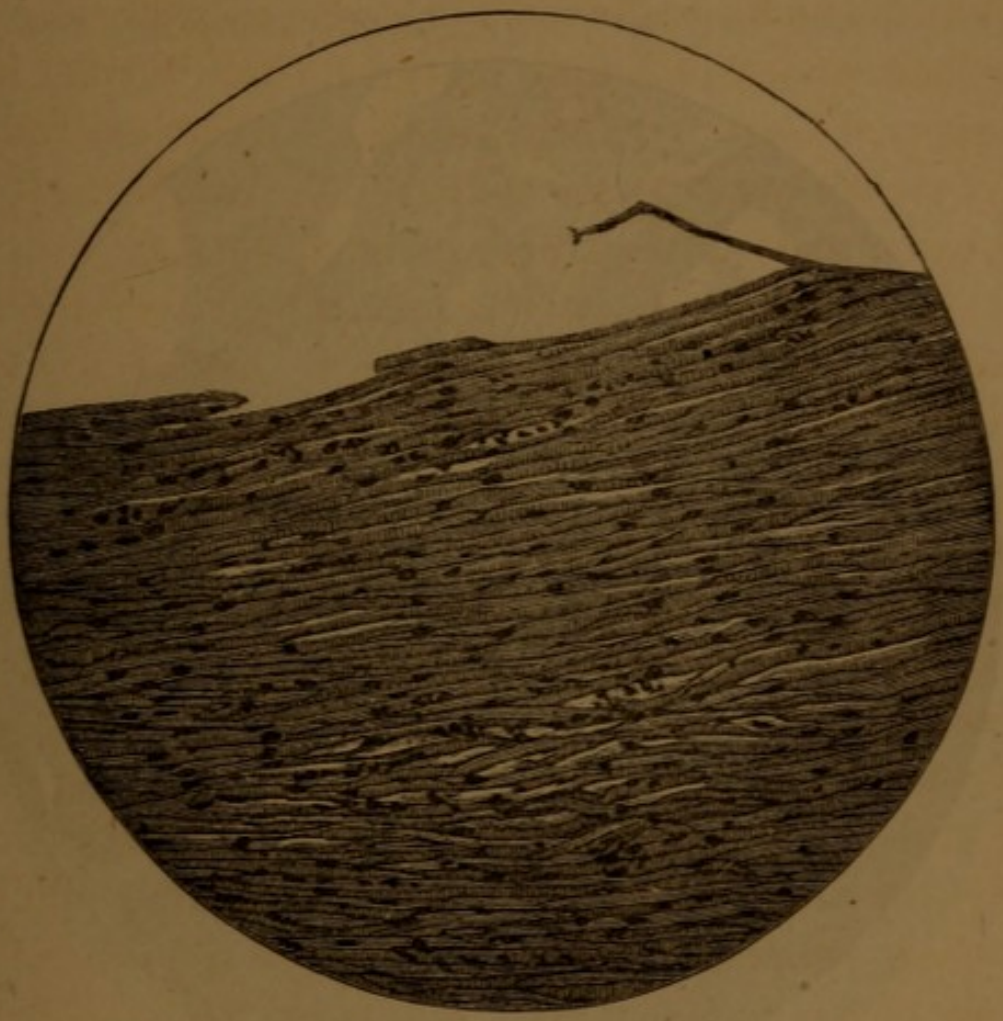


FIG. 1.

Normal cardiac muscle. E. M., aged 13, longitudinal section. The muscle fibres run parallel without much separation. Striation distinct; nuclei normal in size and number; some nuclei seen within the muscle fibres as well as on them. Zeiss D. ocular No. 2.

comparison with this acute case is a specimen (5) of the degenerate muscle of old adherent pericardium where striation is absent and the fibrillæ widely separated.[1] Midway between

[1] In the opinion of some authors (Cadet de Gassicourt, for example, p. 71, vol. ii., "Maladies de l'Enfance") adherent pericardium is necessary for the production of myocarditis in children.

these is a specimen showing distinct myocarditis (4) where the muscular fibres are swollen and the nuclei enlarged and numerous inflammatory elements present in abundance. The subject was a boy of 8, who lingered some time in a condition of extreme prostration.

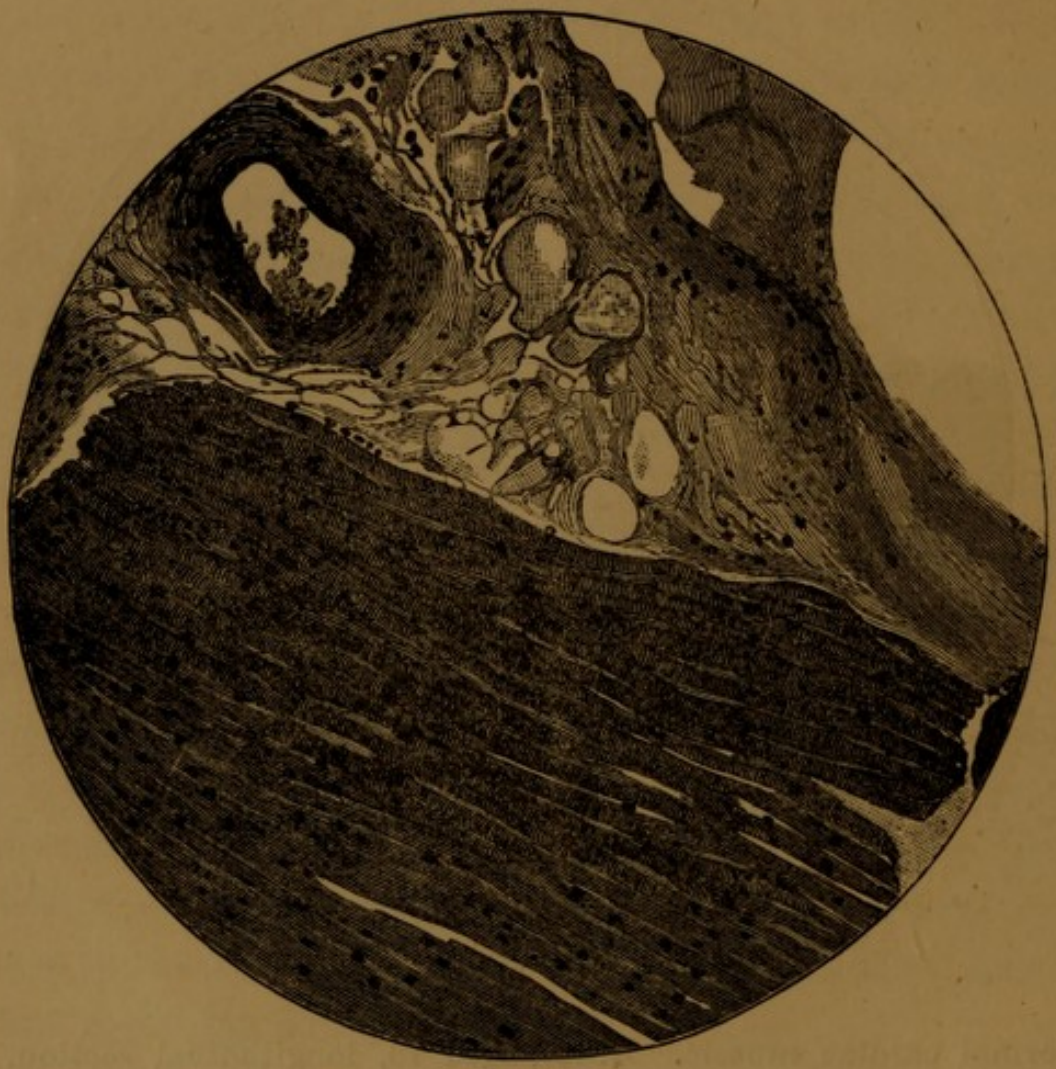


FIG. 2.

Pericardium and cardiac muscle in acute pericarditis. D. B., aged 8. Muscle fibres in longitudinal section normal, with no signs of inflammation; distinct striation; nuclei normal in size and number. Zeiss D. ocular No. 2.

Why do these patients die, and what makes the difference between dying and living? Physical signs, no doubt, are the more threatening when they indicate exocardial inflammation or the heart is already diseased owing to earlier attacks.

Again, changeful cardiac murmur with tumultuous action may at any time exhibit all the general symptoms of acute carditis. But it is not always so, and there is no physical sign whatever and no combination of signs necessarily associated with such symptoms. The first note of danger comes, in fact, not so much from the heart's physical state as from certain ominous

FIG. 3.

The same, showing thickened pericardium with ragged surface of lymph muscular substance seen beneath. Power 2 ocular No. 2.

general symptoms— restlessness, dyspnœa, delirium, prostration, vomiting—which, for reasons unknown to us, attack some of these rheumatic patients and spare others. The appearance of such nervous symptoms is a far better guide to the future than anything that auscultation discovers.

Thus a child admitted recently to my ward with carditis, yet quiet and reposeful, had hallucinations as to people visiting him. Some days later, the physical signs (which include a variable friction rub) undergoing no change, this boy became

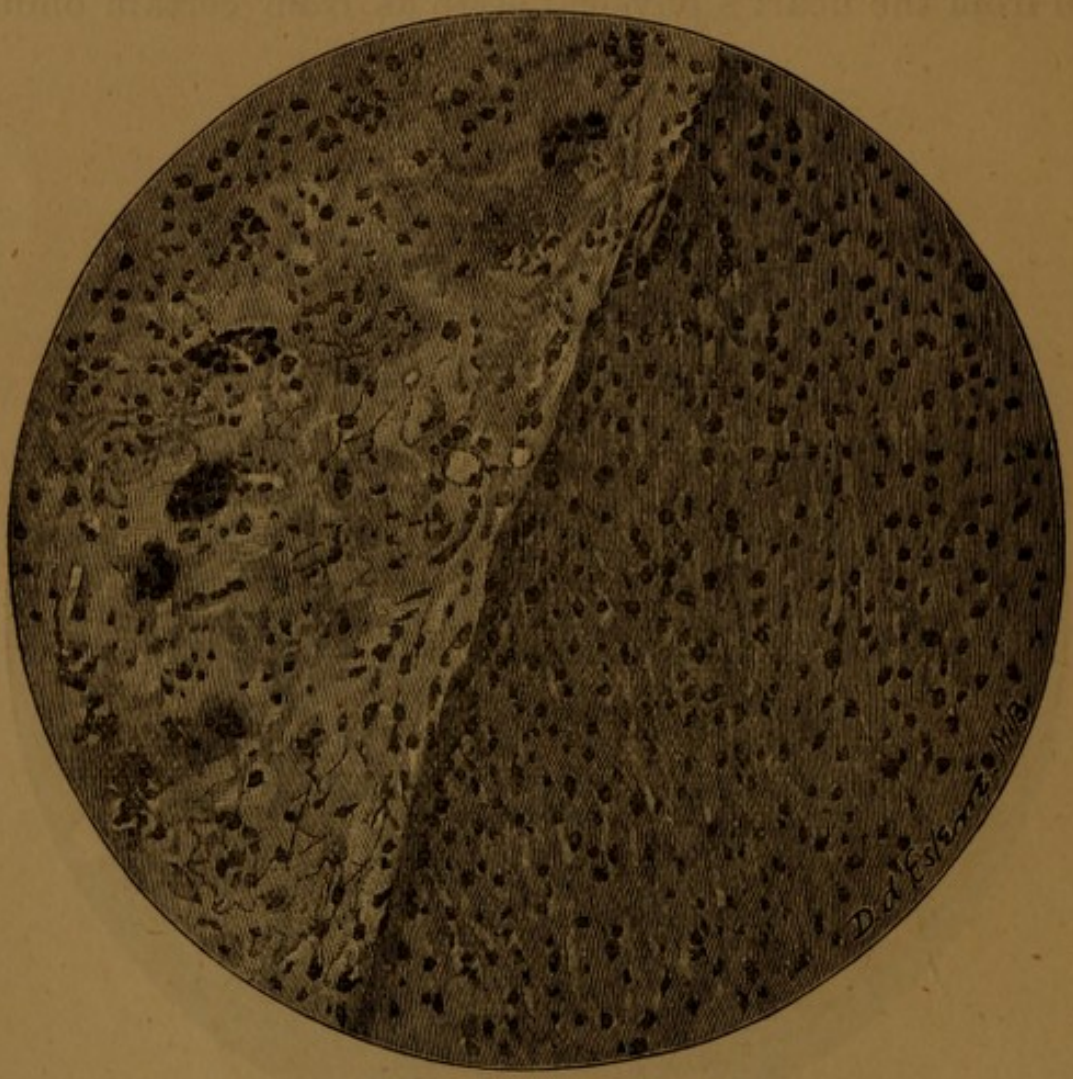


FIG. 4.

Myocarditis-pericarditis. Muscle fibres are swollen and their outlines indistinct; they stain less readily than normal. Nuclei are numerous and swollen. Zeiss D. ocular No. 2.

prostrate and soon unconscious, and he narrowly escaped death.

And just as physical signs are of small service in prognosis, so *post-mortem* examination often fails to explain the fatal event. The heart failure is too sudden to be the result of muscular

degeneration, and as a matter of fact this is not present to any great extent. The myocardium is hardly implicated except as the sequel of inflammation of long standing. Old adherent pericardium shows it best, and it is sometimes met with where there have been no clinical signs on the part of the heart to

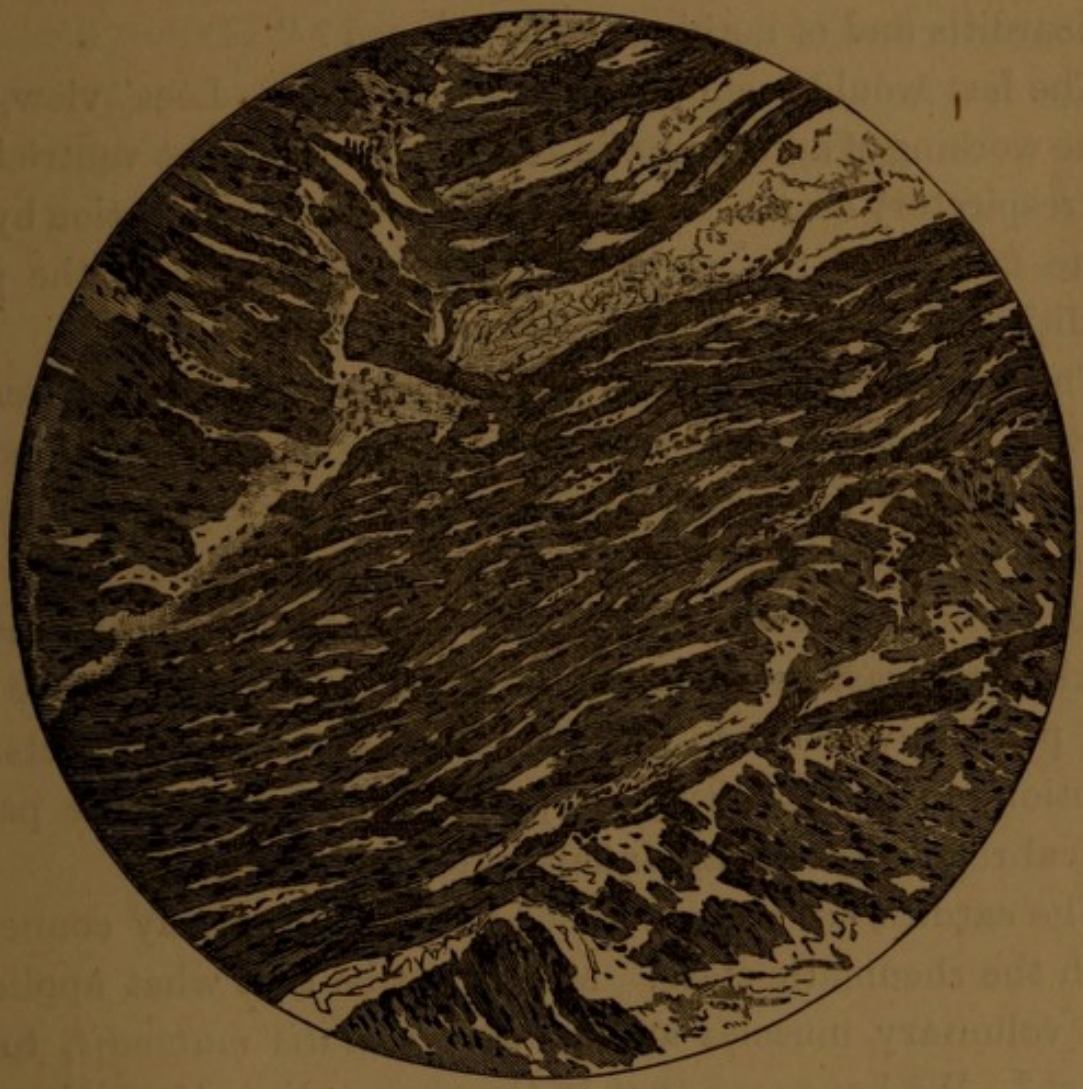


FIG. 5.

W. B. Old adherent pericardium. Muscles wavy, and more than usually separated by fibrous tissue in which the nuclei are numerous and distinct. No striation seen. Zeiss D. ocular No. 2.

lead us to suspect it. In recent inflammation, fatty changes are not generally observed in the muscular fibres. These are swollen merely, their striæ are indistinct, and blood corpuscles are seen in the spaces separating individual fibres as in the drawing.

A consideration of the marked and agonising degree of dyspnœa shown by some of these cases of acute carditis has suggested to Dr. Lees the question, "Is there a dextro-cardiac respiratory centre?" In the absence of any disease of the lungs and with but little defect in the aeration of the blood, how, he asks, is "this very obvious dyspnœa, the result of pericarditis and of nothing else, produced?"

The last would seem best explained in Dr. Lees' view,[1] by "the working of an automatic reflex from the right ventricle to the respiratory centre." Such reflex is called into action by the acute failure of the right ventricle, resulting from the pericarditis or by irritation of the pericardial nerves.

The paper is addressed to physiologists, and it is for them to answer. But whatever that answer may be, the question still remains, why does acute failure of the right ventricle and consequent distress sometimes occur and sometimes not? In other words, what most strikes the mere clinical observer is the marked contrast between two forms of carditis, the active and the passive, which are indentical in anatomical respects. A solution of the difficulty may, perhaps, be found in the pathological relations of heart inflammation.

The carditis we are concerned with is intimately connected with the rheumatic state. May it not be that what applies to the voluntary muscles applies also, *mutatis mutandis*, to the heart? We have seen that in the rheumatism of children the muscles may suffer along with or to the exclusion of the joints. The altered seat of pain makes little difference so far as the limbs are concerned, and it has no anatomical expression. But to the heart it makes all the difference in the world. Supposing that the muscular structure of the heart sometimes suffers as do the voluntary muscles, the symptoms thence

[1] "Is there a dextro-cardiac respiratory centre?" *Lancet*, October 28, 1893.

arising would be analogous to those met with in so-called muscular rheumatism, and, like this latter, without known physical basis. But though analogous, they would be far from similar. For in the one case, the case of the voluntary muscles, the pain is largely within control and can be mitigated by rest, while in the case of the heart, not only is constant movement a necessity of life, but the occurrence of pain there, in whatever connection, excites extreme alarm and just that sort of agitation we are in the habit of seeing in acute carditis.

LECTURE II.

PHYSICAL INDICATIONS.

The normal area of cardiac dulness in children—Physics of pericardial effusion—Its diagnosis—Physical signs of dilatation—Of adherent pericardium—The earliest recognisable signs of endocarditis.

Mr. President, Fellows, and Gentlemen,—In my first lecture I attempted some classification of rheumatic heart inflammation in childhood, dwelling especially on that acute form of carditis which, while it is almost peculiar to that time of life, is at once so insidious and so dangerous as to demand special attention. Reference was made to the great diversity in its earlier symptoms, the trivial character of its arthritis, and the absence sometimes of any distinct evidence of a rheumatic origin. Further, the active and the passive varieties of carditis were contrasted—the prominence of nervous phenomena and the rapid heart failure of the one, the absence of all serious symptoms in the other, with the remarkable fact that the physical condition of the heart, so far as appears, was the same in both.

We have now to consider some points of physical diagnosis, already incidentally mentioned, in reference to pericardial effusion and its effect upon the heart's position. Thence we may proceed to discuss the symptoms of inflammation within the heart and their earliest physical indications.

In most instances of pericarditis, if not in all, there is slight displacement outwards of the heart's apex. The sign, however, is of some delicacy, even when the heart is of normal

size and the patient is seen early; moreover, it needs for its appreciation accurate notions of the natural position of the apex at various ages.

Much labour has been expended on the determination of this point. In his observations upon the locality of the apex beat in 300 healthy children between a month and fifteen years of age, Stärck[1] found that in many cases the point was difficult to determine. Up to the fourth year it is, in his estimation, just outside the nipple line, from five to six it is in the nipple line, and after six inside that line. As to the cardiac area of superficial dulness, he found it diminished with the years both as to its upper and left boundary, falling by the width of a rib—*i.e.*, the fourth left rib—between the sixth and the twelfth year. Stärck recognises three types of superficial cardiac dulness—corresponding with a year old, six years old, and twelve years old. It is sufficient for our present purpose to notice the second and third types. The deep dulness at both six and twelve years is practically the same. The superficial dulness is lower at twelve years by the width of a rib, and having its left border on the parasternal line.

My friend Dr. Garrod has been good enough to investigate this subject of the superficial cardiac area among the out-patients at the Children's Hospital, and the life-size tracings here produced are the result. They accord generally with Stärck's conclusions. "The observed peculiarities are explained," in Garrod's belief, "by a more horizontal or recumbent position of the heart in children than in adults, the heart becoming more erect as the child grows."

A point included in the tracings before you concerns the amount of heart displacement in different positions. It was observed in a patient of my own, aged 8, that the displacement

[1] *Archiv für Kinderheilkunde*, 1888.

to the left following the child's being placed upon that side was maintained for some minutes after the dorsal position had been resumed. Drs. Ringer and Phear made a similar observation in a recently published paper.[1] They noted further the same delay in the heart's displacement following the first disturbance from the back to the left side. Dr. Walsham has made observations not only as to the movements of the heart right and left, but also in the inverted position. In the case of four boys aged 5, 7, 8, and 9, he found that no alteration whatever occurred in the cardiac area when they were placed heads downward. One of these, the boy aged 8, as I myself witnessed at Dr. Walsham's invitation, remained in that position for five minutes, and at the end of that time the cardiac area (that which had been carefully marked out beforehand) was in no way altered. These observations upon posture changes have a value in the determination of the very difficult question of old pericardial adhesion.

In many cases of acute pericarditis the variations in the area of cardiac dulness are but slight. There will be a little widening and a little heightening of this area, neither of these changes being of long duration. The period occupied by the three stages of effusion—its advance, acme, and decline—is included within a fortnight. The time of decline is apt to be longest, and the acme (or period of greatest effusion) the shortest. Sibson,[2] who made a minute study of this point, found that in as many as thirty-nine of forty-four cases the period of acme (or greatest area) lasted but a day. In chronic pericarditis these periods may be long exceeded; but even in the most prolonged the change in area of cardiac dulness will be found to vary from day to day—now increasing, now decreasing, now increasing again.

[1] *The Lancet*, Feb. 10, 1894.

[2] Sibson's Works, vol. iv., p. 10 *et seq.*

Where effusion is considerable there is, in addition to what percussion makes out, what the eye measures—namely, widening of the left intercostal spaces from the second rib downwards, with accompanying elevation of these spaces. An appearance of raised flatness is thus produced, contrasting strongly with the natural contour of ribs and spaces of the right chest. It may seem at first thought a lengthy and difficult task to map out from day to day by percussion the several fluctuations of pericardial effusion, but it is not so for two reasons: one, that the patient is a child, small, thin, easily handled, and with the heart region apparent; the other, that with certain precautions to be presently mentioned, the upper boundary of cardiac dulness taken alone indicates sufficiently by its rise or fall the daily course of the effusion.

It need not be said that the several limits of the heart's superficial dulness—upper, right, and left—are not equally defined. The right border is the most abrupt in slight enlargement, owing to the interference of the sternum. The left and the upper borders of superficial dulness are bounded by narrow regions of lessened dulness due to the interposition of thin edges of lung overlying the left ventricle in the one case and the upper portion of the heart and great vessels in the other. In extreme heart enlargement the left border is carried so far out as to be lost, so to speak, in the left side, which is itself dull owing to the contiguous left ventricle. The upper margin of cardiac dulness is best estimated by percussing gradually from the clavicle downwards; and the right and left borders by first seeking the apex place or the "centre" of dulness and thence percussing outwards in both directions.

With the percussion signs and the signs that the eye notes there occur others, not less important, which the palms and fingers perceive; but these latter vary with the amount of effusion and the consequent displacement of the heart.

How, then, is the heart affected in position as the result of fluid effusion? The question has been variously answered. Sibson, in his later work, lays much stress on the fact that the heart is displaced upwards into the higher and narrower portion of the chest. He thus explains the elevation of apex (which he was the first to point out) and the close apposition of the heart to the anterior chest wall, as well as the vibrations and grating friction. "When the effusion lessens," he adds, "the thrill disappears, the creak vanishes, friction sound softens." In his first article on pericarditis, however, in 1849[1] Sibson clearly described the effect of distension of the sac as causing the fluid to rise so as to cover the heart in front, separating successively the ventricles, right auricle, and vessels from the pericardium. This earlier view of Sibson is that which is now generally accepted. Thus Bristowe[2] asserts that the heart in effusion is "necessarily carried backwards and consequently away from the anterior thoracic wall"; while Balfour[3] and others believe that the rise of the apex is only apparent. "What we feel," says Balfour, "is not the apex, but the part of the ventricle lying above it, and appearing to rise as more and more of the heart is separated from the anterior wall with which the base of the heart always remains in contact." Latham and Walshe are in substantial agreement with Sibson, believing that, with extensive effusion, the heart is pushed upwards to the fourth, third, and second interspaces, the rub, thrill, and vibration being all correspondingly raised.

There is yet another view of the conduct of the heart in effusion, adopted by those who question the fact of the heart being pushed either upwards or backwards. It is that which

[1] Vol. iii., p. 20.
[2] Bristowe's "Medicine," p. 496.
[3] Balfour's "Diseases of the Heart," p. 323.

maintains that the heart in such cases remains where it was. Rotch[1] seeks to establish this conclusion partly by experiment and more cogently by the argument in which Balfour preceded him—namely, that the visible impulse "need not be that of the apex, but of the tumultuous right ventricle above it." Henoch, in his well-known "Lectures on Children," does not allude to this particular subject, or he would certainly have settled it to his own satisfaction.

In my own belief these apparently conflicting views just quoted are, in fact, reconcilable. They all express the truth in various circumstances. The heart may be moved either forwards, upwards, or backwards in effusion, or it may remain where it was; and of the factors that determine its conduct, pericardial adhesion, here or there, temporary or permanent, is the chief. I have repeatedly, in fatal cases of effusion, inserted needles just before the *post-mortem* examination into the proper apex place and above the fifth right costal cartilage close to the sternum without being able to detect upon opening the chest any dislocation of the heart.

The validity of such experiments may be questioned; but there are clinical facts to show that the early pushing forward of the heart, with consequent pericardial friction, increasing in area and intensity, although it may be the rule, is not without exception. The fluid may cover the heart from the first, and no friction be audible until it has been absorbed. This was so in the case of a patient of mine, a girl 9 years old, who, during the time of the acute symptoms, restlessness, pain, and some delirium, showed the physical signs of pericardial effusion merely. The first sound of friction concurred with an abatement of the worst symptoms and persisted almost to convalescence.

The diagrams before you are illustrations of the several

[1] Keatley's Cyclopædia, p. 865.

ways in which the normal area of cardiac dulness is deformed and the apex displaced in effusion. In most cases the utmost degree of effusion is soon reached. It continues in that stage but for a short time, often less than a day, receding in many cases only to return, so that, without corresponding change in the general symptoms, the alternate ebb and flow of pericardial effusion may be watched over a considerable period, the upper margin of cardiac dulness, higher or lower, sufficing, as I have said, as the daily gauge. In the more lengthened attacks the pericardial sac is observed to widen disproportionately to its heightening, and it is in such cases, when newly observed, that much difficulty may arise in distinguishing between a dilated heart and a fluid-laden pericardium. It is, in fact, not by the particular shape or extent of cardiac dulness at a given time, nor yet by any constant distinction in the character of the heart sounds in the two cases, that this differential diagnosis is safely made; it is by the daily measurement of the changing area of dulness. It has been well shown by Rotch and others that cardiac dulness, transgressing the right margin of the sternum and occupying the fifth right interspace, makes fluid effusion probable, and paracentesis has been made with success in that region, as, for example, by Dr. Dickinson in a case of purulent effusion.[1] But the sign is not infallible, even when this extension of dulness to the right is a recent event.

Thus in a case of carditis to which I shall further allude presently (Daisy B——), the area of cardiac dulness remaining unaltered, it was suggested that the pericardium might be adherent. Presently pericardium rubbing became audible, and with this the right area of cardiac dulness moved to the fifth right space. The child died with the usual symptoms of prostration; yet there was no fluid whatever to account for the

[1] *Transactions of the Clinical Society*, 1889, p. 50.

sudden widening of cardiac area. That change was due to rapid dilatation of the right side of the heart at the time of its sudden failure. The pericardial surfaces were adherent everywhere with no interposing fluid whatever. In this instance the fact that the area of cardiac dulness, though widened to the right, was not heightened, might suggest dilatation rather than effusion; but there are cases like that quoted at length in the last lecture, where rapid pericardial adhesion seems to arrest the ascent of the upper margin of dulness.

In another case of markedly dilated heart, of such width as to be clearly perceptible in the fifth right space, extensive friction rub arose some days after admission; yet there was no accompanying increase of cardiac area. *Post-mortem* a notable amount of fluid was found surrounding the heart; it lay round it, so to speak, in such manner as to make discovery during life impossible.[1] Such is often the manner of cardiac empyema, where pus surrounding the heart makes no perceptible change in its apparent size or shape, and the condition is rarely recognised until *post-mortem* examination discloses it. Of the difficulty of founding prognosis upon the observation of the fluctuations in the area of cardiac dulness a good illustration is afforded by two children, one of whom (Thomas B.) died,

[1] A further means of distinguishing between dilatation and effusion, not infallibly, is furnished by careful observation of the place and character of impulse. Pulsation in the second and third left interspaces, more or less distinctly undulatory, and becoming more obvious when the patient is recumbent, are the earliest physical signs of dilated right ventricle, a recoverable condition best seen in anæmia, and sometimes in the acute stage of pneumonia and in convalescence from rheumatic fever. See Dr. Foxwell on "Heart in Debility," *Lancet*, October 24, 1891; also two papers by Dr. Stacey Wilson, "Dilatation of Right Ventricle," published in the same journal, September 8 and 15, 1894, since these lectures were given. It is true that in effusion the heart may be so raised as to give rise to a similar pulsation in the same place. But in that case the widening and flattening of the intercostal spaces, together with daily changing of the area of cardiac dulness, sufficiently distinguishes the condition.

while the other (Henry D.) recovered. In the fatal case the worst symptoms concurred with an increasing area of dulness; in the non-fatal, but very severe and prolonged case the boy was at his worst, passing motions unconsciously, with a rapid decrease of the same area. Other diagrams on the table illustrate the same point, showing examples where the area of dulness decreased for some days before death. It is very rarely that the pericardial effusion of rheumatic children becomes so extreme as to need paracentesis. In the whole of the cases I am now reviewing there was no such occurrence.

Before leaving the subject of pericarditis I would say a word as to chronic adhesion of the pericardium, cardiac symphysis as the French call it. It is not denied that this condition is out of the reach of physical diagnosis, but that admission is somewhat obscured, at least in the text-books, by the rehearsal of certain physical effects due to adhesion between the pericardial sac and the anterior chest wall. Adherent pericardium is one thing, costo-pericardial adhesion is another thing,[1] and it is only the latter has special pathognomonic signs. When the pericardial sac sticks to the heart and to the chest as well—a quite exceptional condition—certain consequences must ensue with the cardiac acts, the pulling in of the chest wall with systole and its elastic rebound with diastole being the most striking. Such signs are more or less obvious and informing according to the vigour of the heart, the site, and extent of costo-pericardial adhesion and the age of the patient. But they do not occur at all, there is nothing to make them, unless the pericardial sac is adherent both within and without and the

[1] Dr. Ord in a paper on "The Diagnosis of Pericardial Adhesion" makes the needful distinction between a "simple growing together of the two layers of the pericardium or simple adhesion" and adhesion of its outer surface to the parts in contact with it, or "compound adhesion." Of "simple adhesion" he says: "We may at once broadly state that it gives no trustworthy signs."—*St. Thomas's Hospital Reports*, 1887, p. 188.

yielding ribs and cartilages are thus brought, so to speak, within the heart's influence.

Apart from the creaking that is heard in some cases over a long period, often an obscure sound easily mistaken for something else—old adherent pericardium as we usually encounter it has no special physical sign, nor should we expect any. The mere fact of adhesion alters neither the shape of the heart nor its sounds. And accordingly, as everyone knows, adherent pericardium is met with from time to time *post-mortem* when not at all expected in the bodies of adults dead of various diseases and of whose early history little is known.

But in children, and especially in rheumatic children, the case is different. There the patient's antecedents, coupled with certain physical indications, may furnish such strong presumption in favour of pericardial adhesion as, in our fallible art, we have to be content with in place of demonstration in many parallel cases. Thus a succession of rheumatic attacks in most, if not in all, of which the heart has been concerned, makes adhesion probable. The probability is increased when the history tells of the occurrence in one or other of the attacks of friction rubbing. It is further increased when, with no more than a mitral murmur, a large heart is felt beating over a wide area with a flapping rather than a heaving impulse, while, at the same time, cardiac compensation seems failing.

On the other hand, the probability of adhesion is lessened when there is an account of repeated and notable effusion lasting over a long period after the manner I spoke of as occurring in some cases of chronic pericarditis. Extensive effusion, thus enduring, tides over the time during which the pericarditis is recent and ready to stick, keeping the two surfaces apart until they can safely come together.

It remains to consider the assertion of some clinical observers that a fixed position of the heart within the chest,

whatever the position of the body, indicates an adherent pericardium. "Whenever one finds on percussion," says Potain,[1] "that the position of the heart and of the apex beat remains unchanged, whether the patient lie on his back or side, this is to be regarded as certain and conclusive evidence of the existence of pericarditis with adhesions, a fact first demonstrated by Galvagni in 1876."

In using this emphatic language the Professor makes no limitation of age or sex, though it is obvious that with many adult subjects, especially women, the test is not applicable, owing to their physical conformation. And besides, wherever there is great dyspnœa or prostration, the turning of the patient from side to side for deliberate exploration of this kind is impossible. Even with children suspected of adhesion the experiment is by no means the simple one it appears when performed upon healthy subjects. We know with some accuracy the amount of movement proper to the normal heart of childhood in various positions, but we know much less as to its movement when enlarged, assuming the pericardium free, the lungs healthy, and the pleuræ not adherent. Nothing short of a marked difference of heart mobility between one set of patients and another, coupled with the fact that in the less movable the antecedent history makes adhesion probable, will suffice to prove the case. I have made frequent experiments without finding any such difference. Cardiac immobility is met with from time to time when there is little cause to suspect any heart disease, while in some cases, where there has been repeated rheumatism and pericarditis, the heart moves freely.

Let us turn now to the consideration of endocarditis. While the signs of rheumatic heart inflammation are in full progress as regards the pericardium, the endocardium, as we

[1] *Medical Week*, May 26, 1893.

know, is suffering also; but just as pericarditis does not show at the first either by increased cardiac dulness or audible rubbing, so endocarditis at its commencement does not impair the efficiency of the valves so as to prevent reflux—in other words, the obvious and familiar physical signs we are in the habit of associating with endocarditis refer less to it than to its consequences.

The early observers were not ignorant of the fact that mitral reflux might occur late. Latham especially, though regarding the advent of such murmur as of itself and without qualification indicating endocarditis in cases of rheumatism, was fully persuaded that the first onset of the inflammation might precede this particular sign. His were the days of enthusiasm for physical signs, when a single one was held sufficient to indicate a particular diseased state. Mitral murmur in the course of rheumatism was for Latham the signal for bringing into play the whole array of antiphlogistic remedies, just as the subcrepitant râle was a sure indication of pneumonia and warrant enough for a like treatment. But no one was more alive than he to the significance of certain cardiac signs antecedent to murmur, and he often saw reason for commencing his remedies before this sound appeared. "There may as yet," said he in 1845, nearly fifty years ago, "be no endocardial murmur, but the systolic sound may be unnaturally prolonged and unnaturally rough. I hardly ever knew a case in which unnatural length and roughness of sound, such as a practised ear could well discriminate, has not become an unequivocal murmur in twenty-four hours. How often have I anticipated that the murmur would be formed to-morrow. It is almost as certain a sign as murmur itself. I no sooner perceive it than without waiting for the murmur I begin the treatment of endocarditis at once."[1]

[1] Latham's Works, p. ii., 69, New Sydenham Society.

A period prior to any audible murmur may, in Latham's opinion, cover all the essential morbid processes of the disease, and even outlast the time when it can be successfully treated. And accordingly, with a precision and accuracy not exceeded in the present day, Latham[1] proceeds to describe three distinct relations of mitral murmur in respect of endocarditis: (1) in some murmur is coincident in time with the very commencement of the inflammation—the natural sound is changed into the genuine murmur without notice or preface; (2) in some (and most frequently) murmur does not arise until inflammation has somewhat advanced; and (3) in some (and least frequently) murmur does not arise until inflammation is on the decline or has ceased.

From the days of Latham and Watson up to to-day the earliest physical indications of endocarditis have been much discussed, and the general conclusion has been reached that no single sign, perhaps no combination of signs, indicates with certainty its very beginning. And if this be said of a first attack, how much greater is the difficulty with subsequent attacks. One or two quotations will suffice to show that as much as this is admitted. Thus Walshe[2] would base the diagnosis of endocarditis on "febrile action, cardiac uneasiness and endocardial murmur occurring in a person presumably free from prior cardiac disease." At the same time, and together with this reservation, he alludes to the possible occurrence of dynamic murmurs, and admits that "endocarditis may exist without murmur." Dr. Bristowe frankly admits that the symptoms of endocarditis are "not striking," and that "it is by the development" of the valve lesion that we mainly assume its presence and trace its progress, while Rosenstein believes that the diagnosis of endocarditis is purely arbitrary.

[1] Latham's Works, pp. 72, 73.

[2] "Diseases of Lungs and Heart," p. 615, second edition.

Professor Potain[1] is more confident. Admitting the great diversity of opinion as to early endocardial signs, he considers all those hitherto put forth as absolutely unreliable. What *is* reliable, according to this authority, is the change which takes place in the quality (timbre) or tone of the normal sounds. This change, expressed in a word, is progressive obscurity of sound. Sometimes the first sound is obscured, sometimes the second, sometimes both. The gradations may be thus expressed: "slightly muffled," "muffled," "obscure," "very obscure," "inaudible." In a certain proportion of cases he admits that the changes were very slight, and considerable experience and a practised ear were needed for their detection, nor "was it possible to form an opinion except by comparison of the day before." He goes on to describe the several morbid changes productive of these altered sounds, but inasmuch as death very rarely occurs at this early stage we may safely assume that the description is no more than conjecture.

My own experience, as I shall presently relate, does not coincide with that of M. Potain, except as regards the changeful character of the heart sounds and rhythm and the value of successive observations from day to day. Inaudible first sound is familiar to all of us, and in its connection with typhus fever was first pointed out by Stokes. It is met with, no doubt, in some cases of rheumatic endocarditis; but, according to my observation, occurs late rather than early. In a recent instance, however, I noticed partial and very temporary effacement of the first sound preceding by some days the signs of pericarditis. The patient was a girl aged 13, admitted with a third attack of acute rheumatism, with old mitral murmur. Fourteen days after admission (when the temperature had been normal for ten days, the pulse steady at 68, and the joints free from pain), the first sound

[1] *La Semaine Médicale*, December 8, 1893, p. 581.

became very indistinct. A week later the temperature rose without any return of the joint pain, and four days later, when the first sound had regained all its distinctness, there was increased fever, and some soft friction could be heard for two days, the patient making a rapid recovery. Here Potain's sign was not distantly connected with carditis, but with an interval. Similarly Dr. Barr[1] considers a "dull first sound with delayed radial pulse" amongst the earliest signs of endocarditis.

Midway between such frank expression of imperfect knowledge as I have quoted and the more confident statements of the French Professor we may place the elaborate researches of Sibson in the same field. His are "tentative conclusions," as his careful and discriminating editor, Dr. Ord, remarks. "Conclusions such as a man would arrange before him to aid his final judgment, a multitude of facts in which the reader finds himself lost, and is conscious that on many points he is not put in possession of clearly stated inferences."[2] For such reasons it is difficult to estimate the precise value of Sibson's work, and not always easy even to follow him; yet, without doubt, we owe much to this indefatigable worker. His vivid description of the aspect and demeanour of commencing endocarditis; the minute and accurate account of the several declensions from the normal sounds, from such signs as are to be regarded as "threatening" endocarditis, to those which, in their further development, make the "actual presence of that inflammation probable," thus precisely defining the degrees and the limits of knowledge in this respect. And we may notice, by the way, and with gratitude, Sibson's very careful and guarded—it may be, too guarded—use of the word "presys-

[1] *Liverpool Medico-Chir. Journal*, January, 1894.
[2] Sibson's Works, edited by Dr. W. M. Ord, vol. i., p 12.

tolic," a term which, I believe, except in one place, he hardly allows himself to use without qualification.

On the other hand, it is certainly true that he gives no very clear account of the physical signs of mitral narrowing: it may seem to some that he exaggerates the frequency of tricuspid murmur, mistaking it sometimes for the true presystolic "grave and even vibrating," as he puts it, "beginning with an accent or shock"; while he is somewhat inconsistent in placing tricuspid murmur among the signs indicative of the very commencement of endocarditis, and also as coinciding "in almost all the cases with great general illness." These may be shallow criticisms, and in any case we are to remember, as Dr. Ord remarks, that "the hand of the artist was stayed while he was still engaged in fashioning his work—that we have but the clay model for a work in marble."

On the threshold of this subject of the early signs of endocarditis, so exhaustively discussed by Sibson, it is well to remember what he so well urged, namely, the fact that both from the structure and action of the mitral valve, as well as from the situation of the inflammatory product, as seen after death, it is apparent that blood reflux is not an early but a late result of endocarditis. "The border of contact in systolic closure is not a mere edge, but a surface or line of adaptation made up of small bead-like cells that dovetail into each other along the margins of the flaps. Endocarditis affects not the very edge or rim of the flaps, but the line of contact just within the edge. When the mitral valve is inflamed it is this border of contact that shows a frill of small bead-like granulations, quite insufficient at first to prevent accurate and complete closure. Presently, however, each of these little prominences gets capped with fibrin, and it is by this addition, sooner or later, that adequate adaptation of the valve flaps becomes impossible, and reflux begins."[1]

[1] Sibson's Works, vol. iv., p. 234.

This early condition of endocarditis is seen best, not in rheumatism, but in chorea. For in rheumatism death does not occur without further morbid changes, which chorea as such does not share. In fatal cases of chorea, apart from rheumatism, we see endocarditis in its purity, so to speak, and it is well known that the bead-like granulations often found in chorea, chiefly in connection with the mitral valves, have sometimes sufficed to produce murmur and sometimes not.

Some special circumstances of childhood help to explain why at that age the earlier physical signs of endocarditis often escape notice. Chief of these is the fact that the early joint pains seem so trivial that the child is seldom brought to hospital on account of them. It is rare to meet with a child at an early period of its first rheumatic attack; so it happens that the physical signs we get to associate with endocarditis belong properly, not to its early and active stage, but to its later consequences.

Having dealt thus far with the views of well-known authorities as to the earliest recognisable signs of endocarditis, and shown, by their want of accord, the difficulty of the subject, I will endeavour to point out and to illustrate from patients of my own, (1) the manner in which endocarditis comes about in the case of rheumatic children, and (2) the mode or the modes of its development.

The earliest physical indications of endocarditis in children acquire significance by their association with rheumatism. Tumultuous, quickened and uneven heart action; sounds that are changeful from day to day, especially the first; sounds reduplicated, especially the second at and above the apex (*not* at the base); a temporary tricuspid murmur; marked accent commencing the first sound, whether mitral or tricuspid; these are amongst the commonest of such sounds.

But it is not in all cases that endocarditis signs are thus gradually developed. Sometimes mitral reflux occurs abruptly, without previous notice or warning. Thus a child aged 3 years and 8 months (Rose E.) was admitted on October 4, 1893, for some general œdema, but without albuminuria or cardiac murmur. She had had neither scarlet fever, sore throat, nor rheumatism. In two days the œdema disappeared, and the child seemed to be well. Ten days later, however—*i.e.*, on the 16th—some desquamation was found over the legs, and the child was thereupon removed from the general ward, the heart sound at the time being perfectly natural. On November 1 the child was again admitted with swollen and painful joints of five days' duration, the temperature being 102°. There was now well-marked blowing murmur at the mitral which was conducted into the axilla, and accompanied at first by an occasional "squeak." In this instance it seems almost certain that a period of no more than five days sufficed to develop a murmur having all the characteristics of regurgitation.

In the usual course the progress is more gradual and of the kind I have just mentioned. Here again, we may invoke the example of chorea. It is generally admitted nowadays that the heart signs of chorea—or at least the later and more pronounced of them—are due to endocarditis. These signs are in the order of occurrence, unevenness of action, slight and inconstant murmur, murmur that is constant, and murmur both constant and conducted; never thrill (rheumatism being absent), never accentuated beginning of the first sound, never any sound that its most passionate advocate could mistake for presystolic. There can be little question that these signs concur with an endocarditis briefer and having a much more limited history than the rheumatic. They represent the utmost of choreic endocarditis, but only an early stage of the

endocarditis that is rheumatic. But at this moment of time their physical indications are identical.

Endocarditis in children, there is reason to think, occurs most often along with the joint pains, and not (as it seems to occur) at some indefinite period afterwards. But the first heart signs are obscure and ambiguous, while the joint pains are slight and brief. And so it habitually happens, as I believe, that the gradual development of endocarditis signs in a first rheumatic attack—the passage from "the threatened" to "the probable," and from "the probable" to "the certain" signs of that inflammation—concurs with the time when the child, apparently recovered, has returned home and is out of medical reach.

This is well shown in the case of a girl (Rose E.), aged 9 years, admitted to the Westminster Hospital on October 5 of last year with her first attack of rheumatism, the joint pain being of two weeks' duration. The pain was slight, and disappeared altogether in two days, as did the pyrexia. The patient, unlike most children with rheumatism, was of ruddy complexion and well nourished, but it was reported that she sweated much at night. With the pressure of more urgent cases I do not know that for this symptom alone the child would have long remained an in-patient, but for what I am about to relate, and in view of these lectures, she remained with us several months. Now, on October 14, when the child was to all appearance well, the finger placed along the fourth space detected with ease and nicety the variable character of impulse and the unequal intervals between one revolution and the next. The ear confirmed the finger and added these further signs: a lengthening of the first sound, or what some would call a just perceptible systolic murmur, and together with this reduplication of the second sound, not at the base (where it is wished to be for the sake of the explanations, but rarely is), but at or a little above the apex.

Two days later the signs had altered thus: the uneven rhythm remained, the reduplication of the second sound had disappeared, and now there was an undoubted blowing murmur following the first sound. On the two next days the heart's action became almost regular, but it faltered occasionally; the pulse was 96, perceptibly uneven, and the mitral murmur was less obvious. During the succeeding three weeks (the patient continuing in bed, though bright and cheerful, and without fever) the murmur varied *and the pulse was ill sustained.* It was even as to rhythm, but unable to maintain an even force through the minutes, a feature which only becomes apparent when the observer will have the patience to keep his finger on the wrist for two or three minutes at a time. This child, as has been said, was kept under notice from early in last October until late in January. Not to weary you with repetitions, let me call attention to two points: (1) That at the end of our watching a loud, blowing murmur was established, together with signs of some heart enlargement; and (2) that for a considerable time the murmur, quite apparent in the recumbent position, ceased when the girl was erect. Thus mitral murmur was not fully established in this case until the end of two months, months wholly free from disturbed health; while for some time after its establishment it was dependent on position, insomuch that had the patient been attending as an out-patient the discovery of valve lesion might have been long delayed.

This gradual development of mitral murmur applies to a considerable number of rheumatic children, and is the source of much misconception. The patient is dismissed, as this child might have been, heart whole. He returns presently with a second attack of rheumatism and a mitral murmur; this latter has, in fact, developed very slowly and insidiously as the result of the first attack, and in the interval between the two

—it is the sequel of endocarditis, not endocarditis itself. But the physician, consulting the notes of the child's first admission, takes it for new; he quotes it as proof that a first attack may fail to touch the heart, and that permanent reflux is not always the work of time. Both statements are perhaps true, but they have fewer illustrations than at first seems.

The influence of posture upon murmur and reduplication of the second sound may often be observed in these early cases. At first, with the upright posture a more vigorous systole serves altogether to abolish these morbid sounds. After a while this effect is only accomplished for a short time, the murmur coming out as soon as the effort of sitting up causes fatigue, and at last the murmur becomes permanent, and posture affects it but little. In noticing such posture changes Walshe asks: "Is this changeableness of a murmur proof of its inorganic nature?" "My experience," he answers in his laconic way, "would support the negative."

If once more we make the comparison between the endocarditis of chorea and of rheumatism (allowing for their distinctive characters), it might seem that what is the common event in the one case is the less common event in the other. In chorea the murmur grows, it becomes distinctly blowing, and is conducted into the axilla. It is then recognised by most as a sign of endocarditis. But presently it begins to lose these characters, and eventually the normal sounds return. Such is the common experience of chorea, rheumatism apart. We are not without evidence that similar recovery sometimes obtains in rheumatic endocarditis also, but for reasons to be presently mentioned we are without accurate information upon this point.

LECTURE III.

DIAGNOSIS AND TREATMENT.

Signs of endocarditis referable to the right side of the heart—Altered and modified sounds—The place of murmurs in the cardiac cycle—Prognosis—Treatment.

Mr. President, Fellows, and Gentlemen,—In the last lecture we were led to consider the service of percussion in estimating the area of cardiac dulness at various ages and in several morbid conditions, together with the effect upon the heart of gradually increasing pericardial fluid. The earlier physical signs of endocarditis were then discussed, and the opinion was expressed that these signs, varying widely in various cases and scarcely within reach of words, became significant in their combination and by the comparison of to-day with yesterday. Finally, examples were given both of the sudden and of the gradual development of morbid sounds, mitral murmur occurring not seldom as the sequel, and not as the accompaniment, of a child's first rheumatic attack.

It remains to consider some further signs of endocarditis, especially those referable to the right and uninflamed side of the heart, to estimate the precise significance in diagnosis of certain endocardial murmurs and altered sounds, and in particular of the so-called presystolic murmur in connection with narrowing of that orifice, the case of the child being compared with that of the adult. In conclusion, I would add a few words on prognosis and treatment.

Endocarditis does not at first, we say, produce regurgitation,

or, at least, does not produce it by any defect on the part of the mitral valve itself, muscular incompetence, and the share in closure belonging to the muscular fibres surrounding the mitral orifice being left out of the case. Yet inasmuch as endocarditis concerns the fibrous structure of the left side of the heart, and not only so, but extends to the heart itself, to the papillary muscles and their tendinous cords, it is of necessity that this inflammation should produce physical changes beyond the valve. And the effect is this: the muscular weakening of the inflamed left ventricle impedes the onflow of blood and tends to engorgement of the pulmonary vessels. This in its turn, as Sibson has pointed out, reflecting on the right ventricle, leads to accumulation of blood therein, which is relieved by tricuspid regurgitation, what has been called "safety valve" action.[1]

If this be so it follows that in endocarditis it is not the left and inflamed side of the heart that will give the first note of suffering; it is the right and uninflamed side, and tricuspid murmur is the early signal of endocarditis as well as a token of relief.

There are many obvious reasons why this early occurrence of tricuspid murmur should be overlooked—the existence of old valve disease, the short duration of this sign and the speedy oncoming of mitral murmur to obscure it. It is only in a first attack and with the happy chance of early observation that tricuspid murmur gets noticed.

[1] The expression I used is by Sibson, but it originated, believe, with Mr. T. W. King in a striking and elaborate essay printed in 1837 in the *Guy's Hospital Reports*, vol. ii., "On the Safety Valve Action of the Right Ventricle of the Human Heart and of the Gradations of this Function in the Circulation of Warm-blooded Animals." As bearing on our present subject King's conclusion may be quoted: "That the tricuspid valve, naturally weak and imperfect, closes less and less accurately according to the increasing degrees of the ventricular distension" (p. 139).

Something also may be allowed for the habitual neglect of the right side of the heart, due to a knowledge that the actual seat of disease is assuredly the left ventricle and not the right. Yet, with all this allowance, it would still seem that upon the hypothesis tricuspid murmur should be met with oftener than it is as an early sign of endocarditis. The explanation is, I think, that there is a concurrent or even a yet earlier effect of endocarditis—namely, disturbed cardiac rhythm.

However early the transmitted effect of left-sided endocarditis upon the right ventricle, the immediate effect, as regards the left ventricle itself, must be earlier still. This effect is no other than an irritability of the cardiac muscle, due to the altered relation between the inflamed endocardium and the blood that visits it. Tumultuous cardiac action, its variability, reduplication of the second sound (apt to alternate with true diastolic murmur at the apex) and altered length and tone of the first are joint signs of this irritability. They are not wholly reliable signs of endocarditis, but they are, in Sibson's phrase, its "threatening," and especially in connection with rheumatism they make it "probable."

Tricuspid murmur, I say, in these circumstances may so soon be followed by mitral as to be overlooked; but given a previously healthy heart, early observation of the patient, and, it must be added, the care to discriminate tricuspid murmur from mitral, and there can be no doubt that the sign upon which Sibson laid so much stress is not seldom met with.

I may quote a few recent examples:—

H. W., a boy aged 11, was admitted to the Westminster Hospital on October 24 of last year, having been ill three days with his first attack of rheumatism. He was suffering from pain in the knees and ankles; the temperature was 102°. There was neither swelling nor redness of any joint, and on the fourth day—that is, a few hours after admission—the

fever left him. On the third day the boy seemed well. The area of cardiac dulness was normal, and it was not until the fifth day from the first rheumatic pain (26th) that anything unnatural was observed of the heart. On that day the rhythm became uneven, and a slight systolic murmur was heard at the ensiform cartilage, less audible at the apex. The pulse was 84 and ill-sustained, varying in force, that is to say, markedly, when continuously felt over a period of three or four minutes. A week later, November 6, the tricuspid murmur was still audible, not blowing, but with that rough beginning that some christen "presystolic." At the apex the first sound was lengthened and murmurish. It happened with this boy (as with the girl E. mentioned in the last lecture) that on standing up the murmur was immediately lost. The experiment was repeated several times, always with the same result. We were not able to retain this patient for prolonged observation like the last.[1]

Shortly after this a girl aged 18 (A. H.) was admitted on the ninth day of her first attack of acute rheumatism. On admission there was pericardial rubbing, and at the ensiform cartilage a systolic tricuspid murmur. Both joint pains and pericardial rubbing quickly disappeared, and on the girl's discharge, after three weeks' residence, a soft apex mitral murmur had taken the place of the rougher tricuspid murmur.

A third case might be quoted occurring at about the same time—that of a boy aged 10 in his first attack of rheumatism

[1] A remarkable fact was that with the disappearance of tricuspid murmur in the upright position there occurred on three separate occasions a perfectly distinct true diastolic murmur, replacing exactly, as appeared, the proper second sound at the apex, and immediately disappearing when the boy was recumbent. The phenomenon, which was observed by many, was lost before his discharge. Doubled second replacing and being replaced by diastolic murmur is not uncommon, but here doubled second sound was never heard.

with heart pain. At first no cardiac murmur was audible, and the earliest physical sign of valve lesion was again tricuspid in seat and character.

In these and in other cases that might be given the rheumatic attack is the first, and is neither severe nor prolonged. The tricuspid murmur sometimes is transferred to the mitral, and sometimes upon its disappearance the heart sounds again become normal.

There are many varieties of first sound that depone more or less certainly to commencing endocarditis—for instance, the first sound becoming loud or prolonged, or doubled; but these are all characters of which the value depends on the comparison of one day with the next. A prolonged first sound in a patient suffering from rheumatism presently becomes murmurish, though no definite murmur can be alleged. And the *quality* of the sound is altered, as well as its duration; it becomes rough, sometimes, in children especially, begins with an accent, and is continued right on to the second sound. Unfortunately these morbid sounds are difficult to describe. Such words as "rolling," "rattling," "churning," and many more, hardly suggest to a novice what sort of sound he is to expect. They are to be learnt, not from the book, but at the bedside, and after patient and, as it may at first seem, profitless practice they will suddenly strike upon the ear and become a possession for ever; but it is knowledge which is not transmissible, and remains each man's secret in spite of his best efforts to communicate it.

There is less difficulty in describing the precise time and place of added sounds in the heart's revolution, but even that point is not determined in a moment. In the child the heart rhythm—that is, the exact sequence of sounds and pauses—is ill expressed. Moreover it is not always realised that the

acts of the heart are represented partly by sound and partly by silence, and that defective action is detected rather by the silence being broken than the sounds altered. In the long silence especially—silence which is not rest (in the period, that is, of ventricular diastole), whatever noise intrudes is morbid. But with children, and especially when the impulse is obscure, it is a nice point to decide whether intruding sound is just without or just within the proper long silence—whether, in other words, such sound coincides with quite the end of diastole or just the beginning of systole—whether it be presystolic or early systolic. These two murmurs represent, of course, perfectly distinct acts on the part of the heart, ventricular dilatation and contraction; yet they both join on to the first sound, and in some cases neither is much conducted from its place of origin. And there is another point of which Dr. Paul Chapman reminded us in his valuable Goulstonian lectures. Before any final statement can be made as to the significance of cardiac sounds we must know whether such sound is valvular or muscular, connected with the closure of the valves or with contraction of the muscular fibres.

The term "presystolic" is made use of just now with little precision, and when no more is intended than that the first sound begins with an accent. Presystolic murmur is far from common among children—it depends upon structural changes that need time to accomplish. Early systolic murmur, on the other hand—a harsh beginning of the first sound converting "who" into "through"—is commoner with them than with their elders. It may arise early in the course of a first attack of rheumatism and quickly disappear or tone down into a soft, blowing systolic murmur. Its disappearance, no less than its quite recent origin, puts mitral stenosis out of the question.

I have no desire to enter upon any matter of doubt or controversy, but the light-hearted way in which the word "pre-

systolic" is sometimes used, coupled with the peculiar circumstances of the child in reference to mitral narrowing, may perhaps excuse a few words upon the subject.

The real presystolic murmur is express, as are the causes on which it depends. It indicates not only difficulty in the auriculo-ventricular passage, but the particular way in which that difficulty is overcome—namely, by the suddenly aroused energy of the auricle at the end of its work. Arising precisely as this mechanism would lead us to expect, within the long silence of ventricular dilatation, it grows and gathers with a thrill right up to the first sound, which abruptly ends it; a further sign consisting in over-distinctness of the second sound at the second left cartilage, due to extra-pulmonary resistance.

For the production, therefore, of presystolic murmur there needs not merely a contracted mitral orifice, but the service of a hypertrophied auricle. When either of these elements is wanting, whether the narrowed passage or the extra power necessary to overcome it, there is no sound heard within the proper silence—no audible pushing, so to speak, with a view to supplying the ventricle efficiently with the blood, upon which it will presently close, no presystolic murmur.

And so it is common to meet with extreme constriction of the mitral after death where no presystolic murmur has been audible in life. That is the case, said one of wide experience, Dr. Hilton Fagge, "in the large majority of cases." It is common also in life to find the auricle failing temporarily or permanently to perform its extra work; and in that case, inasmuch as every stenosed mitral is also incompetent, presystolic murmur becomes replaced by regurgitant murmur.

No murmur is to be called presystolic unless it can be demonstrated that it precedes the impulse. I know that some observers have demurred to this test, asserting that in mitral

stenosis it is the right ventricle that initiates ventricular contraction, the left being a thought behind. All are agreed, however, as to the difficulty of timing the sound. "It must not be thought," says Dr. Sansom, "that the question can be determined without considerable care and difficulty"—"difficulty so great," said Dr. Stokes long before, "that we cannot resist altering our opinion from day to day as to which *is* the first sound, and which the second." It is exceedingly dangerous to accept diastolic mitral murmur occurring anyhow and anywhere—at *some* time within the long silence—as being evidence of a *degree* of stenosis.

While, therefore, the well-marked characteristic presystolic murmur, with its gathering force, its sudden end, succeeding shock, and accentuated pulmonary second, are reliable physical signs of notable mitral narrowing, we cannot trace by physical means the degrees of this change or watch the progress of mitral stenosis from little to much.

Now, as a matter of fact, it can be shown that the conditions necessary for the production of presystolic murmur are not often met with in children. I have carefully noted the *post-mortem* experience of eleven years at the Hospital for Sick Children. During that time, 1,503 cases were examined, 102 of these having died of heart disease. These supply but a single case of "buttonhole" contraction of the mitral valve, and this one, a boy aged 9 with cellulitis, was not connected with rheumatism. There are 2 cases of undoubted mitral narrowing in connection with rheumatism and 2 with but slight stenosis in the same relation. These 4 presented the physical signs of narrowing, including "presystolic" murmur during life. But this same term "presystolic" is by no means confined to them; it appears in the histories of at least nine, probably eleven, of the children who showed no mitral stenosis *post-mortem*.

My own clinical notes of the last three or four years referring to 50 children with rheumatism, point to a like conclusion. In 11 of these the mitral murmur was described as "presystolic," but after death no stenosis was found. There were 5 where this same murmur had been observed who showed *post-mortem* degrees of stenosis classed as follows:—"Slight stenosis" in 2; "some stenosis" in 1; "mitral admits thumb" in 1; "stenosis" (not further described) in 1. Furthermore, taking a series of 12 cases of my own, of whom 4 died and 8 recovered, "presystolic" murmur having been attributed to the whole series, I find the following result. Of the 4 that died no mitral narrowing was found in any, but in 2 the mitral valves were thickened. Of the 8 that recovered (or rather, were discharged relieved), 5 had "presystolic" murmur on admission, but it did not persist, and they left hospital free of it. In 3 the so-called "presystolic" murmur referred to three children in their first attack of rheumatism, and on admission within three weeks, in the oldest case, of the commencement of such rheumatism. The time is far too short for the production of real stenosis.

It is stiffening, often with subsequent thickening of the mitral valves, that produces the murmur that is thus misunderstood, and may produce it very early and but for a time. Stenosis is a later matter.

If we take by contrast the case of adults it will be found that the "buttonhole" form of stenosis is by far the commonest. Thus in 47 cases by Dr. Fagge, 46 were of that kind against 1 funnel-shaped, while in 40 under Dr. Sansom's care 19 were of that form, against 15 funnel-shaped and 6 not particularly described. It may be added that Dr. Sansom reckoned but half his cases to be rheumatic, while Sir Dyce Duckworth found 60 per cent. of that origin. It thus appears that *post-mortem* evidence upon the vexed question of presystolic, as opposed to early systolic, murmur, wears a some-

what special aspect in the case of children, owing to the rarity of that marked chink-like narrowing of the mitral orifice, which alone is to be depended on (provided there be auricular hypertrophy) to furnish the combined physical indications of stenosis.

The chief form of rheumatic endocarditis met with in children at an early stage consists, as is well known, of a ring of minute granulations edging, or just within the edge of the mitral valve, but not constricting it. These minute bodies effect no more, indeed, than to cause the blood to pass over a rough surface instead of a smooth one. The dynamic effect of the early active inflammation is another matter. A later stage of this inflammatory process as regards the mitral flaps and chordæ tendineæ is not in the way of narrowing but of stiffening. Such a condition seems fitted to produce delay in closure, and perhaps audible creak as well, rather than any material hindrance to the onward blood flow. "It is the case of a valve delayed in its closure by rigidity, but which yet shuts at last." Valves so circumstanced permit regurgitation at the first part of systole, and a murmur which is not presystolic but early systolic. "This presystolic murmur falsely so-called," says Dr. Dickinson, "is short and well marked, begins low, rapidly increases, and ends abruptly with a slap or knock. This is supposed to precede the systole, but actually accompanies it, as determined by its relation to the impulse. The early part of the first sound is obscured or replaced by the murmur, the terminal knock or slap is the only part of the first sound that remains. It is caused by the delayed closure of the mitral valve, which puts an end to the murmur, which is truly mitral regurgitant. The knock which puts an end to the murmur (or to which the murmur runs up) is exactly synchronous with the carotid pulse which occurs towards

the end of the cardiac systole. The murmur is therefore *in* the cardiac systole." [1]

What I would chiefly insist upon is that the rough or accented beginning of the first sound, commonly called presystolic, has nothing to do with stenosis. I do not say that it is unconnected with some hindrance, often temporary, to the onflow of blood, or that persons who have exhibited this physical sign in childhood may not get mitral stenosis later on; but the earlier stages of this chronic process do not suffice for the production of true presystolic murmur, but merely of its rough counterfeit. The notion that mitral stenosis can be watched through all its changes from early childhood onwards is, I am persuaded, erroneous.

In no respect is the presystolic better contrasted with the early systolic murmur, or accent, than in this, that the former, depending upon precise and definite morbid changes—mitral constriction, auricular hypertrophy—is always of one pattern, be it more or less distinct, while the other varies with the time and according as the endocarditis is acute or chronic, new or old. Without denying, therefore, the special significance of these signs of stenosis, first pointed out by Fauvel, there are reasons enough, both clinical and anatomical, why those signs should be found very sparingly among children, almost never among very young children.

Of all the morbid sounds of the heart, whose meaning we guess at with more or less probability, there is none that to

[1] Dr. Dickinson adds: "There is a true diastolic mitral, which has been recognised by Hope and others. This is from the auricle to the ventricle, and immediately precedes the so-called presystolic when both are present together, which they sometimes are. The true mitral diastolic sometimes appears to run into the false presystolic. It ends gradually, the false presystolic begins gradually, so that where the one begins and the other ends is not always readily distinguishable. They, however, belong to different phases of the heart's action, one to the systole, the other to the diastole." See *Lancet*, October 19, 1889.

me seems more obscure than true diastolic murmur at the apex. Such a murmur is sometimes heard in perfection at an early stage of endocarditis, a blowing diastolic bruit at or a little above the apex, exactly covering the second sound and hardly outlasting it, inconstant, affected by position and usually alternating with doubled second, both phenomena disappearing at a later period of the inflammation.[1]

In the easy language of the wards we often hear of "double mitral murmur." What is meant is, sometimes a presystolic with a systolic murmur, and sometimes, in the case of children especially, a rough accent beginning the first sound, with a blowing murmur following it. But neither of these is strictly double mitral, a murmur *with* the first sound, and a murmur *with* the second sound comparable with the familiar and easily understood double aortic murmur.

How, indeed, it may be asked, can a presystolic murmur exactly replace the second sound, as in the proper double mitral it certainly seems to do? Dr. Walshe meets the difficulty by observing that "the diastolic murmur is rarely loud enough to cover the second sound completely. It is

[1] Shortly after this lecture was given the following case occurred in my practice. Kate C., aged 16, complained of pain in the knees and at the heart, and was short of breath. She had had scarlet fever at the age of 11 and rheumatic fever at 14. The heart's apex beat was in the fifth space internal to the nipple line. Upper limit of cardiac dulness reached to the second interspace; there was friction rub and pericardial thrill; the second sound was doubled at the apex. There was marked præcordial tenderness. After an interval of three days, during which she had increased dyspnœa and cardiac pain and a temperature of 103·4°, pulse 116, but no return of joint pain, and the cardiac dulness had extended to the right of the sternum, the doubled second at the apex was converted into a true diastolic murmur, and for some days the doubling and the murmur alternated distinctly. These symptoms did not last more than a few days, and when the girl was dismissed after three weeks' observation the area of cardiac dulness had become normal and there was nothing remarkable to note of the heart.

commonly spoken of," he says, "as diastolic in rhythm, but in point of fact it is rather post-diastolic and presystolic than precisely coincident with the diastole."

Another suggested explanation (and in some instances no doubt the right one) is that the diastolic murmur is really aortic. In Dr. Bristowe's belief the "diastolic mitral" of Laennec was in reality aortic, as probably was Hope's also. But such an explanation hardly applies to young children, with whom aortic disease is rare, while this form of murmur, a true double mitral, is common. Dr. Bristowe asserts further that "in a small but interesting proportion of cases systolic murmur is associated with a true diastolic mitral." Yet he adds, in further description of such diastolic murmur, that "it runs up *from* the second sound and tends to die out in the diastolic silence." It does not cover or replace the second sound, therefore, and it is not, in the sense I speak of, accurately diastolic.

Time forbids my dwelling upon this point. I would rather revert to the fact that the stiffened but not stenosed mitral valves (even, it may be, of early endocarditis) permit regurgitation at the first part of systole, then are slowly raised during that act, and in the rising the valve apparatus itself may possibly take part in the sound and give, or help to give, the rough accent with which systole begins; the early thrill being caused, as Dr. Seymour Taylor[1] has pointed out, by these stiffened flaps "held edgeways to the regurgitant stream."

I have tried to show that in the case of children[2] *post-mortem*

[1] *Lancet*, November 15, 1887.

[2] Dr. Theodore Fisher, Registrar to the Bristol General Hospital, in an able and instructive paper, has recorded twelve examples of presystolic murmur without stenosis. All but one are children. He offers three suggestions in reference to the differential diagnosis of such murmurs:—

(1) "It is always low-pitched and probably never simulates the harsher presystolic murmur of mitral stenosis.

(2) "It is always accompanied by a systolic murmur which is longer than that often associated with mitral stenosis.

evidence is all in favour of such an explanation of that early and rough murmur which habitually goes by the name of presystolic. But *post-mortem* evidence shows, moreover, that the case of the adult is different, and mitral stenosis not uncommon. And I cannot doubt that marked stenosis with hypertrophied auricle—a disease of women rather than of children—has physical signs of its own, and that these signs have been accurately described by Fauvel and Gairdner.

A few words will suffice for all that can be usefully said of prognosis.

Rheumatism, we say, is the main cause of heart inflammation. But what *is* rheumatism? Of late years its area has been more and more extended, and in the process the substance of it has so thinned out that its proper shape is hardly discernible.

Pleurisy, pneumonia, and tonsillitis, we are told,[1] may all be rheumatic, though unattended by joint or limb pains. And further—if further there can be—Sir Dyce Duckworth, in a lecture published this week,[2] announces "rheumatism of the brain, probably the cortex," not anatomically known, but having the chorea of children as its outward and visible sign, a trifling ailment for so alarming a lesion; the effect is patent, the alleged cause awaits discovery.

Rheumatic phenomena being thus indefinitely multiplied,

(3) "The first sound is not short and sharp and may be replaced by the systolic murmur.

(4) "The murmur is most commonly associated with adherent pericardium occurring in children; and consequently a presystolic or diastolic apex murmur in a child where there is a history of pericarditis should be regarded with suspicion."

See "Diastolic and Presystolic Apex Murmurs without Mitral Stenosis or Incompetence of the Aortic Valves," *Medical Press and Circular*, October 10, 1894, p. 361.

[1] Dr. Mitchell Bruce, *Brit. Med. Jour.*, April 26, 1890.

[2] *Clinical Journal*, March 14, 1894, p. 307.

who shall say what rheumatism is in itself? The forms of gout are multiform, but they are held together in some sort by reference to urinary changes common to them all. But nothing holds rheumatism, it has been hunted to death. There is an "idea" of rheumatism, but it is fast becoming an abstraction like the Eternal Noumenon of the old philosophy.

But if rheumatism—whatever its criterion—be the cause of carditis, what is its own cause? It is called a chill disease, but not a tithe of the cases, even of unequivocal polyarthritis, have their origin in exposure. In 608 consecutive examples collected by Dr. Syers only 33 could be so attributed.[1] Let us double that number in virtue of the class represented, many of them careless labouring people, habitually exposed, and not nice to reckon as to more or less exposure; even so, hardly a tenth can be so counted. In the great majority the cause is conjectural. Sometimes it seems to be the result of bodily fatigue, sometimes of mental distress; sometimes, in children at least, of improper feeding. Where the causes are so various the effect will vary too. And no help to prognosis is likely to come from any consideration of the supposed origin of the rheumatism that attends it.

Yet there are certain circumstances of undoubted service to prognosis: age, previous attacks and the disposition to rheumatism, whether more or less, which these imply; and, as applying to the particular case, nervous implication; the presence of subcutaneous nodules; the changeful or the fixed character of the physical signs; the prominence or otherwise of heart pain or uneasiness and of precordial tenderness; the degree of pallor and the rate of wasting.

As for age, rheumatism occurring at an unusually early period of life, say at four or five, is very nearly allied to carditis, and sometimes shows itself chiefly by heart inflam-

[1] *Westminster Hospital Reports*, vols. i.-iv.

mation. It may be said without qualification, other things being equal, that the younger the child the greater the peril to life.

There is a form of rheumatism almost peculiar to children, which carries, I believe, a special danger. It is when a succession of attacks come so near together that they are often reckoned as one prolonged attack. Sometimes there is a sort of periodicity in such seizures, the mother saying that they happen every month or every fortnight. If we reckon, as in the case of a young child we safely may, that every one of such successive attacks (however little severe in itself) is a fresh injury to the heart, the ill effect of these multiple attacks is obvious. Older childhood is far less subject to this manner of repeated rheumatic visitation ; and on that account, as well as because the increase of years of itself lessens the danger of carditis, the child past eight or nine, the subject of acute carditis, is far more secure than he would be at an earlier age.

In the same bad category must be placed cases such as I have already quoted, where an acute rheumatic attack is presently repeated, in respect of its pyrexia and sweating; but this second time the symptoms are cardiac, and not articular.

Of physical signs in their bearing on prognosis it may be said that, so long as such signs are recent and refer only to the endocardium, the fear of pericarditis and of the development therewith of the active signs of carditis, a fear which can never be absent, is more or less, according as the heart's conduct varies or does not vary from day to day.

So long as the physical signs, any or all of them, that I have tried to describe as indicative of early endocarditis are *changeful*, so long in my experience, however quiet and restful the child, there is real danger, a danger which only lessens with the settling down, so to say, of the heart.

In a case I have already quoted these changeful physical signs continued for forty days with no general symptoms whatever to cause anxiety; but on the forty-first day pericarditis appeared, with constant vomiting, delirium, and extreme restlessness, the right ventricle becoming extremely dilated, and the child very quickly died.

It thus appears in regard both to external and internal inflammation of the heart that the physical signs of both gain significance from their changefulness. So long as endocardial murmurs and cardiac rhythm alter in character from day to day, so long is there active endocarditis. And similarly, as has been shown, daily changes in the area of pericardial effusion, whether of increase or decrease, are chiefly significant as showing that the inflammation is still acute.

It has been abundantly shown how largely the nervous element enters into the natural history of carditis in childhood and affects its mortality. The appearance, therefore, of such symptoms in whatever degree at an early stage of endocarditis—vomiting, severe headache, sudden weariness, passing delirium, uneasy sleep with night terrors; any of these ought properly to cause anxiety, however favourable the child's progress in other respects.

In one of the cases included in my review a boy aged 9 in his first attack of rheumatic endocarditis had, on admission, some passing delusion as to his father being at his bedside. At the time he had no other bad symptom, but presently acute carditis developed, and he narrowly escaped death.

Finally, my experience fully accords with that of Dr. Cheadle, so ably detailed in his Lectures on Rheumatism in Children, delivered before the Harveian Society, "Manifestations of the Rheumatic State," as to the evil import of subcutaneous nodules.

The clinical picture of carditis in children, gloomy as it may

seem, is not without light, and may even be brighter than it first seems. We call to mind a large number of rheumatic children appearing and reappearing in their successive attacks with shorter and shorter intervals of ease. We see them presently becoming chronic sufferers, and at last either perishing quickly with acute suffering, or else lingering on with hearts incompetent and dyspnœa, dropsy, and other distress. Long observation of these children in their coming and going, the hope of recovery repeatedly disappointed, the growth of friendship between doctor and patient as time passes—all these things make our memory of such patients peculiarly distinct and vivid, and their fate distressing. But we are to consider that it is only those that thus decline that are thus remembered. The rest are forgotten. There is no returning to give thanks on the part of those that recover. Thus, whatever the number of these, circumstances conspire to make them seem less than they are. And there is at least this happy feature in the child's case which age may envy. Children have no future, and the anxiety and despondency of their elders in a like state are unknown to them.

I would add a few words on the treatment of active carditis.

So undoubting was the reliance on drugs fifty years ago that Dr. Latham (of whose labours and success in search of the very earliest signs of the disease I have already spoken) exclaims in an ecstasy, "The gain of a single day in the treatment of endocarditis is a gain indeed." In a different spirit an eminent physician of our own day, replying to a practitioner who regretted that he had overlooked pericarditis in a case of rheumatism they were seeing together, is reported to have said —perhaps not quite seriously: "Make no apologies—if you had discovered it you might have treated it."

It is of interest now to observe the point of view whence rheumatic carditis used to be regarded and the plan of treat-

ment that resulted therefrom. It is of yet greater interest to note the remarkable fact that the special characteristics of child's rheumatism, and notably its moderate fever and slight joint pain, did not serve to exempt children, so far as appears from all the rigours of the antiphlogistic treatment.

"I know of no disease," says Latham, "in which febrile heat is greater, and the pulse harder and more forcible than in acute rheumatism. What are the signals for copious blood-letting if these are not? There is no disease in which pain is more prominent, abiding, and characteristic; hardly any in which it is more severe and more extensive. It is the sort of pain which arouses and excites, and however long it may continue, even for several weeks perhaps, it still rouses and excites to the last."

That this is no picture of child's rheumatism need not be said. It seems to follow that the treatment might reasonably be modified and relaxed in the case of children, so as to suit the milder symptoms. The old writers admit no such exception. A single passage throws vivid light on the practice of those days and its consequences.

"In eighteen cases of pericarditis," says the physician I have just quoted, "I lost three. All were treated by mercury; some were brought under its sensible influence very largely, and some very slightly, but all in a certain degree except two —two of the fatal three. These two, healthy subjects in the prime of life, would not become salivated. Though mercury was given in large quantity, and for a long period, yet was there no sensible ptyalism, no fœtor of the breath, no complaint of soreness of the gums. Every conceivable circumstance was present which could promise success to medical treatment. Healthy subjects in the prime of life, and the disease detected as soon as it arose. Not a moment was lost in the application of remedies. They were venesection, cupping, leeches, and

blisters and opium, and from first to last mercury. But the mercury did not produce the peculiar effects of mercury in the slightest appreciable degree." They would not become salivated, and so (as is inferred) they died.

Here is the keystone to the treatment of that day. Bleeding was beginning to be used with some restraint (and Latham especially was wisely moderate in this respect), but bleeding alone would not suffice, mercury must go along with it, and this not in stated doses, but in such amount as should as quickly as possible "touch the gums." When this particular symptom of mercurial poisoning was delayed the mercury was "pushed" to the utmost degree. Yet some (these two healthy subjects in the prime of life, for example) *would* die unsalivated.

It is easy to criticise at this safe distance of time. Therapeutics is a halting art, and there is abundant evidence of the shrewd and careful observation of the old masters. Nevertheless, with our later experience it may be open to believe that the early advent of salivation was really beneficial, not for any virtue belonging to ptyalism, but because on its appearance the use of an injurious drug was discontinued.

We are, perhaps, too much in the habit of assuming that the rate of mortality of certain acute diseases—notably of pneumonia—in less now than it was early in the century. It is a delusion, as statistics prove; but, in the case of pericarditis, anyone who reads its literature will, I think, become convinced that it was more formidable under the old methods than now. The whole portraiture of the disease gives evidence of this. One of its earliest historians, Sir Thomas Watson, speaks of the great danger that belongs to every case of acute pericarditis, "of the danger of speedy death," and so forth (speaking, remember, of the adult, not of the child). Such language would be inappropriate at the present time. No one doubts the gravity of rheumatic pericarditis as regards the

future, but except in the case of children (to whom the passage does not refer) the immediate danger to life is very small.

Such considerations as these would seem to furnish the very strongest reason for departing from ancient traditions without more ado; but it is not so. The old treatment was at least precise, emphatic, uncompromising, and it lingers still robbed of all those characteristics. Fearing to break away altogether from the old practice, we have toned it down, reduced it to a dead formula, and catch at every excuse for evading it. Hence arises an uncertain voice and even some disagreement. "The treatment of acute heart affections" says Henoch, "must, of course, be antiphlogistic; we require local blood-letting, icebags, calomel, digitalis, and blisters." Dogmatic enough, but wanting in precision and the specific direction that marks a firm faith, such as Latham's. "Counter-irritation is of considerable value,"[1] says one. "Blisters should be shunned," says another. "Blood may be taken from the arm," says one text-book, "but preferably by cupping or leeching." "Venesection is never admissible," says another.[2] "A few leeches may *sometimes* be applied to *robust subjects*, but, as a rule, heat and moisture are to be preferred, though whether heat or cold is the better application is a matter of doubt." "The treatment of pericarditis," says yet another, "merges itself in the treatment of some other disease. Why should we change our treatment because the pericardium happens accidentally to become inflamed?"[3] All these are literal quotations from as many recognised text-books of the present time.[4]

[1] Bristowe's "Medicine," seventh edition, p. 530.

[2] Roberts' "Practice of Medicine," fifth edition, p. 501.

[3] Balfour, p. 326.

[4] I am not ignorant that both in America and France blistering and wet cupping are very confidently commended (see, for instance, Dr. Wm.

With all their difference, what is it that they concur in indicating? Is it not that the treatment of pericarditis just now, like the treatment of pneumonia, of pleuritis, of meningitis, is the treatment of symptoms, and nothing more? The old remedies are still invoked, but there is no ring of sincerity in the invocation. What we really believe more and more—what Parkes, Hughes Bennett, Gull, and others pointed out—is that disease processes are not arrested, but may, in a measure, be directed. The easing of pain by local appliances, the procuring of sleep therewith, the timely withdrawal of blood to relieve venous congestion and cardiac dyspnœa—all these are measures that are truly life-saving; but the course of inflammation is little, if at all, affected by such means.

Commenting upon such views as contrasted with those of the past, Dr. David Lees, in an able and interesting paper on the treatment of pericarditis,[1] pertinently writes: "How many of the old formidable measures are in use to-day? Three or four leeches, or a single small blister, represent the maximum therapeutic attack on the disease from without, whilst a little morphia alone remains as the representative of the active medication of former days; that is to say, that we have given up the idea of curing pericarditis."

Pepper, "Pract. Med.," ii., 257 and M. Cadet de Gassicourt, "Maladies de l'Enfance," ii., 197). "I am convinced," says Dr. Pepper, "that the early and repeated use of blisters affects profoundly the course of the disease." Now, in cases where the symptoms are active and life immediately threatened, it is easy to understand how certain observers become persuaded of the special value of a certain remedy, be it bleeding, blister, ice or what not; we can only lament their want of accord. But when it is asserted generally—in respect of a disease of which the patient habitually recovers, at least for the while, leaving us pretty much in the dark as to pericardial adhesion or valve damage—that blistering (or anything else) "profoundly affects the course of the disease," one is tempted to ask what is the nature of the evidence on which so positive an assertion is based.

[1] *The Lancet*, July 22, 1893.

Most truly; and in the modern sense of that word "cure" we have given up the notion, not merely of curing pericarditis, but any visceral inflammation whatever. Without at all disputing the formidable, even the dangerous nature of the old measures, especially in the case of pericarditis, I would urge that Dr. Lees does some injustice to such means of relief as we possess, by placing together, without distinction of relative value, bleeding, counter-irritation, and morphia. In the abstraction of blood (by leeching, as a rule) we possess, in my belief, the most potent means of relief for those sudden attacks of dyspnœa which in the case of children are apt to be so rapidly fatal. Blisters do neither good nor harm, and it is pretty plain that the text-books think so in urging as sufficient objection to their use their interference with the use of the stethoscope. Digitalis, still, to quote my own opinion (though it is that of high authorities as well, notably of Dr. Stokes[1]) is worse than useless in the acute cases we are now considering.

For the rest, I should be in full agreement with Dr. Lees as to nursing and the means for relieving pain, though demurring a little to the statement that "we have made the discovery that the salicylates are curative of the rheumatic process." There are instances, I think, where the salicylates effect absolutely nothing in that use. It has been well said[2] that if they possessed a real efficacy against rheumatism taken as a whole, they would be good all round, for the visceral manifestations, especially the cardiac, no less than for the joints, like mercury in syphilis. But it is not so.

"Have we then," says Dr. Lees, "no means of combating pericarditis as a local inflammation?" And he answers himself, "I maintain that we have; it is the local, persistent application of cold."

[1] "Diseases of the Heart and Aorta," p. 86.

[2] Cadet de Gassicourt, ii., 196, "Maladies de l'Enfance."

Time will not serve for stating the arguments upon which this opinion is based. All who are acquainted with Dr. Lees will be prepared to find in his paper a lucid, logical, restrained, if somewhat enthusiastic, statement of the case. And yet I cannot persuade myself, looking back upon many cases of fatal carditis, that chilling the surface of the chest by means of ice would have saved them, or some of them. My trials of that remedy have not, as a matter of fact, had that result. The application of the icebag, whether in pericarditis or in pneumonia, is, in my experience, sometimes grateful and soothing, sometimes taken with indifference, and sometimes so strongly resented by the patient that no one would think of persisting in its use. It has seemed to me to be of greater service in relieving the distressing pleural "stitch" of pneumonia than in pericarditis, where pain is less common and more fitful.

Everything depends on the selection of patients, and surely there is no disease whatever wherein it is more easy to separate critical cases from the rest. Passive carditis, be the physical signs what they may, does well enough. It is precisely at the moment when such symptoms intrude, as I have repeatedly dwelt upon, restlessness, dyspnœa, delirium, that danger begins, and very grave and immediate danger. If by the application of ice to the heart in cases like these the children habitually recover, Dr. Lees' contention is made out. But only so. Recovery without classification of cases proves nothing. The efficacy of the remedy can only be tested in emergency when the need for it is obvious and pressing.

In conclusion, it may be said with confidence that the outlook of pericarditis where adults are concerned is much more favourable than it used to be, owing mainly to a wise abstinence in the use of supposed remedies; and, if it be true also that children have not fully shared in this improvement, it must be

remembered that in their case, for the reasons I mentioned at the first, there has been less time for observation. But already the insidious advance of heart inflammation under cover of trivial joint pain and but slight declension from health, until such time as the heart gives grave warning of distress, is a well recognised event, and the timely warning means often the saving of life. Moreover, the condition is one that especially invites and has now secured therapeutical inquiry. Sober investigation, however, is of slower pace than the demands of the public or the assertions of the drug market. Hopeful work is being done, and with drugs. But it is as yet too early to announce conclusions from that point of view.

In bringing these lectures to an end I cannot omit mention of the ready help and co-operation of my colleagues, especially Dr. Garrod, Dr. Batten, Dr. Walsham, and Mr. Winckworth. Indeed, I have been helped by so many that, could I be certain that they would warrant my facts and accept my conclusions, what has been put before you might be taken as a joint contribution.

It only remains for me, Mr. President, to express to you and to this audience my thanks for the kind attention with which you have honoured me.

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[*Names of Authors are not included.*]

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