THE LUMLEIAN LECTURES

ON

HEART INFLAMMATION IN CHILDREN.

Delivered before the Royal College of Physicians.

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I.

WHEN I was honoured by the invitation of the Censors' Board to deliver these 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 custom, but it is determined more imperatively in my case by the knowledge that for me there was 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 that which falls to everyone, then and then only could I hope to interest an audience such as 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—when Laennec was announcing the physical signs of heart 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 common with children) was spoken of as of rare occurrence and obscure signs. And, coming to later times and our own country, it is curious to note how the great workers upon this subject—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 adult heart, moderate in pace, of decided rhythm, and with a clear distinction of sounds and their 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. But 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 assumption that the physical signs of chronic disease might be made to apply, with some modification, to that which is more recent.

It is now frankly admitted that the early diagnosis of heart inflammation is among the most difficult problems of practical medicine, and that, at the first, it depends largely upon the presence or absence of pericardial rubbing, a sign of inflammation to us now (speaking generally) gross and palpable, but of which the significance was for long altogether missed. Laennec was not ignorant of the now familiar sound made by pericarditis. He heard and described it; but for reasons not hard to understand 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." And so, when in actual touch of the truth, he goes off upon theories of sound-producing gases, leaving others, and especially three distinguished Fellows of this College—Latham, Watson, and Stokes—to recover the path and pursue it to the end.

Thus, of the two main factors of cardiac inflammation, while pericarditis had to await investigation until a comparatively recent period, endocarditis may be said to await it still; at the same time, the conflicting opinions of to-day

upon many questions of physical diagnosis, upon the displacement of the heart in pericardial effusion; upon the meaning of doubled sounds; upon the proper signs of pericardial adhesion or the earliest indications of mitral stenosis—these, with other instances, supply evidence enough that to this hour we are seeking more light.

My purpose in these lectures is (1) to signalise some clinical forms of heart inflammation, forms both insidious and perilous, which are chiefly to be observed among children; (2) to consider the physical indications of such inflammation, their significance and modes of origin; (3) to review the main facts of heart inflammation, as it shows in children with reference to its prognosis and treatment.

It would seem at first sight that children were the most promising subjects for the study of heart inflammation. In the case of adults, there are many hindrances to accurate observation of the heart, fatness, great muscular development, the interference 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 within touch, almost within handling, its position, its shape and its acts can be measured and felt; 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.

Cardiac inflammation in children, as is well known, occurs chiefly in connection with rheumatism; if young children be excluded almost wholly in that connection (Table A). Yet

TABLE A.—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	
<i>Ages of the Rheumatic.</i>		
Between two and four years	2	54
" four and six years	4	
Six years	6	
Between six and twelve years	42	
<i>Ages of the Non-Rheumatic.</i>		
Four years and under	32	46
Between four and six years	5	
Above six years... ..	8	
Age omitted	1	

the rheumatism is often ill expressed in the way of arthritis, and sometimes spares the joints altogether. Strictly speaking, carditis—inflammation, that is to say—both exocardial and endocardial, is exclusively rheumatic. Pericarditis alone, as the table (Table B) shows, is met with in tubercu-

TABLE B.—Distribution of 83 Cases of Heart Inflammation Examined Post-mortem: I. Periendocarditis (Carditis). II. Pericarditis. III. Endocarditis.

I. Periendocarditis (carditis) ... 18	One was a case of phthisis, and in 1 the rheumatism was doubtful: with these two exceptions all were rheumatic. In 11 of the cases the endocarditis was old.	
	Tubercle	8
II. Pericarditis (without endocarditis) ... 28	Empyema	8
	Pneumonia	4
III. Endocarditis (without pericarditis) ... 7	Septic	3
	Diphtheria	2
	Meningitis	1
	Bronchitis	1
	Chronic arthritis	1
	Old mitral disease	3
	Slight beading on tricuspid in baby with broncho-pneumonia ...	1
	Slight beading on mitral	1
	Pyæmia (newborn child)	1
	Chorea	1

losis, in empyema, in pneumonia, and in septic conditions. Endocarditis alone is met with in children far less than in adults, and, were we to take *post-mortem* evidence alone, with remarkable rarity, cases of chorea alone excepted. The rheumatic heart inflammation of children when pericardial is always endocardial as well, and when endocardial is extremely likely, with the recurrence of rheumatism, to involve the pericardium also. Thus, in the heart as elsewhere—as in catarrhal inflammations and tuberculous development—the differentiation of morbid processes is less marked in the

child than in the adult, and a common sympathy more apparent.

The rheumatic carditis of childhood differs from that of later life in other respects besides those already mentioned. It is, or it seems to be, less intimately connected in time with arthritis; its clinical course at the outset is therefore more insidious, and its immediate mortality is far higher.

Three forms may be roughly indicated: the active, the passive, and the chronic—terms perhaps not without objection, yet sufficient for the purpose of labelling.

Active carditis rare in adults, has some special characters in childhood, and, owing to its sudden origin, rapid course, and high mortality, claims our chief attention.

Passive carditis—similar to the first in its physical signs—is without its symptoms and without its danger. It is free from cardiac pain or distress, and, but for auscultation and facial aspect, might even escape notice in the course of rheumatism. It is, in fact, the form of carditis which is commonest in the acute rheumatism of the adult, but it differs in two points: One, that the attending joint pain is less acute; the other, that there is much higher probability of acute carditis supervening.

Passive carditis shows less in the child than in the adult because the rheumatism on which it depends shows less. It is less acute and less enduring. In the adult, however little the special cardiac signs may intrude, they are secure of observation, because the severity of the illness keeps the patient to his bed and the state of the heart is carefully noted from day to day for a length of time. So, it is true, may it be sometimes with children; only the rarity with them 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 pains serve to remove these young patients the earlier from observation, and so in the absence of rheumatic pain apparent convalescence may be reached and the child accordingly dismissed when the cardiac inflammation is in full swing.

Of chronic carditis (or pericarditis) I shall have little to say. The amount of effusion may be measured from day to day and week to week by percussion, alternately increasing and decreasing, with no active distress, no pyrexia, or any other suffering than which is due to some dyspnoea. It is 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.

Active or acute carditis has for us now, for the reasons just stated, the chief interest. It is of this kind. In the course of a slight rheumatic attack, or it may be following it with an interval of apparent health, active carditis is suddenly announced by restlessness, increased pallor, and a curious aspect of anxiety, together with dyspnoea, 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 a common and important symptom. There may be little rise of temperature and not much quickening of breath or pulse, but the act of respiration is distressing and accompanied by the short expiratory grunt so characteristic of the acute chest affections of young children. Along with these symptoms there is a preference for the raised position, and some children with great distress for breath will lean forwards in search of relief.

The posture, the pallor, the restlessness, and look of alarm, coupled with dyspnoea due mainly to enfeebled inspiratory effort, all these are symptoms so characteristic that active carditis in a child may be detected almost at sight. Certainly when the chest is bared, and both eye and hand take part in the observation, the character of the heart's action will remove any doubt that may remain. And when these symptoms rapidly progress to a fatal end, as they often do, it is sometimes prostration, sometimes delirium, sometimes persistent vomiting that shows the most conspicuously. But the main source of the symptoms, however these may arrange themselves, is manifestly cardiac failure—failure which is often so sudden that it is not possible to attribute it to texture degeneration.

Of these several forms of carditis, anatomically similar yet widely differing in clinical characters, it is the acute alone that threatens death as an immediate result. We are wont

to regard them all—whether active, passive, or chronic—as having a common origin in rheumatism. Without impugning the correctness of this belief, it must be insisted that premonitory symptoms are by no means confined to the joints. They are, in fact, so various that it is difficult to name any of them that is invariable or common to the whole series. Sometimes, for example, it is not articular but limb pain that accompanies or precedes the heart inflammation; sometimes it is dyspnoea or cardiac pain; sometimes in the recurrence of an attack, which at the first was articular and obviously rheumatic, the old pyrexia will reappear, but in the fresh attack the heart will be actively concerned, while the joints escape altogether.

In no small proportion of cases wasting and sweating are the main premonitory symptoms, and the history of antecedent rheumatism will be extremely vague. Even in those instances, the majority no doubt, where the rheumatic nature of the affection is beyond question, it is noticeable in children how soon joint pains recede, and the condition of the heart occupies the whole attention, whether from the changeable 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 these patients is considerable. I cannot state it precisely, for 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 illustrate the good results of treatment.

A single example will serve to bring before you the special characters of this active and dangerous form of carditis in childhood, a form so rarely observed in after life.

A girl, aged 7 years and 10 months, anæmic and ill-nourished, was admitted to the Children's Hospital on April 28th, 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 incessantly 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, however, 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 tumultuous in action, its impulse forcible and diffuse, apex apparently in fifth space; the murmur was now 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 dyspnoea) the most characteristic signs of active carditis appeared; that is to say the child, though free from actual pain, and without marked dyspnoea, 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 *bruit* reaching the third space. It continued to enlarge somewhat for a while, as shown in the figure, but presently receded, and at death was hardly larger than on admission.

Without staying to record daily changes—the distinctive 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 31st, 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 5th. At the latest stage considerable engorgement of the lungs perhaps accelerating 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 symptoms (constant vomiting being the most prominent, pericardial effusion disappearing the while) lasted less than a week.

of the *post-mortem* examination, it will suffice to say that the heart weighed 9 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 granulations. The other valves were normal. The pericardium was universally adherent, both anteriorly and posteriorly. No fluid remained, and the surfaces were separated with difficulty.

Time will not admit of separate mention, even in this brief outline, of other examples of a similar kind. They have a strong family likeness, but the end does not always come in the same way in the fatal cases. In a certain number of which I have had two well-marked cases within the last year, one fatal and one recovering, typhoid prostration is the main feature, the patient lying apathetic and but partially conscious. In the recovering case, a boy of 9, the 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 this pattern, all patients of my own. Six were boys and ten girls; four only recovered. As many as eight—half the number—were under 7; the four oldest were between 10 and 11. The pericardium was adherent in all the fatal cases, and in only one was there extensive valve disease. In most the endocardial change was limited to small granulations on the mitral valve.

It is not easy to summarise the several symptoms. It may suffice to say that in one case alone were joint pains prominent and long continued. In one pain affected the limbs but not the joints. In two slight rheumatic pains were suddenly replaced by prostration, the while the physical signs indicated pericarditis. Four were admitted for cardiac pain and dyspnoea, 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 dyspnoea occurring six weeks before, no obviously rheumatic symptom having been noticed for the past three months. There was a history of former rheumatism in twelve; subcutaneous nodules were noted in four.

In the fatal cases, as I have elsewhere shown,¹ it is the rule to find adhesion, and the exception to find the heart free. At the same time, it is the rule to hear exocardial rubbing, often continued until death, and the exception to hear none. Diagnosis, therefore, upon this point is as likely to be wrong as to be right. Yet it may reasonably be supposed, as regards recent adhesion, that the mere act of dying favours it; so that taking all the cases together, fatal and non-fatal, adherent pericardium may be a less frequent event of rheumatic carditis in childhood than our *post-mortem* records would lead us to suppose.

TABLE C.—*Sixteen Cases of Acute Carditis.*

Boys, 6; Girls, 10.
Average age, 8.
The 3 youngest, 4.2 (D); 4.8 (D); 5 (R).
The 3 oldest 10.5 (D); 10.8 (R); 11 (D).
Recovered, 1—Boys, 1; Girls, 3.
Died 12—Boys, 3; Girls, 9.
Previous attacks of rheumatism in 12.
acute carditis in 2.

Duration of acute symptoms: longest, 8 days; shortest 1 day, except 1 recovering after 9 days' acute symptoms, and 1 who had prostration 3 weeks before death.

TABLE D.—*Pericardial Adhesion in Relation to Friction Sounds.*

- A. Characteristic friction sounds heard shortly before death in 21:
- 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.

In some instances the *post-mortem* examination discloses unusual conditions whether of peri- or endocardium, such as may probably have influenced mechanically the mode and the distress of dying.

¹ *Rheumatic Carditis of Childhood.*

Thus, in a girl under 5, who became very blue and suffered a sort of anginal attack just before death, the appendices of both auricles were pretty firmly adherent to the adjacent portion of the heart.

In another instance, a girl of 1 years old, there was sudden collapse and death; *post-mortem* besides pericarditis, a continuous decolourised clot extended through the tricuspid valve from auricle to ventricle of the right heart.

The pulse in these cases is small and of low tension. The sphygmographic exhibition of pulse tracings is less satisfactory than in adults, but in some of the examples of acute carditis of which I am now speaking there is marked dirotism with some unevenness. It is Dr. Sanson's opinion

that excessive degrees of dirotism are not met with unless a severe form of endocarditis be present. It may be so; but I

Henry Drinkwater, aged 9.
December 29th.—A.C.D. increasing. Loud friction. Patient pale and prostrate.

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

January 11th.—Slowly recovering. A.C.D. fixed, and as on January 1st.
Dicrotism still marked.

doubt whether the degree of dicrotism is an accurate measure of the patient's condition and progress. for in some instances, as in the one here represented, there was but little difference in this respect between the tracing 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 carditis is seldom much raised, and its variations are difficult to interpret. So long as the articular pains remain—which, as has been said, is not long—we have the irregular pyrexial trace of acute rheumatism. Later on, and when the heart has begun to claim chief attention, there is a fall of temperature to the normal or a little above or a little below normal, the only constant feature being irregularity of temperature from day to day.

The two charts here shown refer to two children. The first is the one whose case I related, and who had a long period of prostration. The second, also fatal, was admitted with joint pains, which very suddenly disappeared, leaving the child much exhausted and with severe heart pain. In the first of these cases, as in two or three others, hæmaturia was observed during the activity of the carditis.

Of the physical signs of rheumatic carditis in children, whether active or passive, the pericardial are the more obvious and the more immediately important. The concurrent endocarditis—whose presence may be assumed—difficult enough to appreciate in any case at an early stage, is more or less masked by the presence of exocardial signs.

The course of pericarditis, like that of other serous inflammations, is conventionally divided into distinct stages, which are described as successive but are in fact 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 is no orderly passage of the inflammation as a whole from stage to stage. There may be more of plastic or more of fluid exudation, or either the one or the other. In the case of pericarditis especially, it is important to notice that its earliest physical signs are conjoint, due partly to the distension of its fluid product, and partly to the audible attrition of the product that is more consistent.

Pericardial inflammation indeed at the first neither distends the sac nor causes audible rubbing. It is recognised, or rather it is suspected, by fallible signs such as altered rhythm, with quickened and tumultuous cardiac action. Extra area of cardiac dulness does not appear until the fluid is sufficient to push aside the edges of the lungs; friction rubbing does not appear until the earliest exudation has acquired some consistency. But the two signs, friction and extended area of 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 is measured not by the rub, but by the daily changes in the area of cardiac dulness. In the acute cases the increase of fluid is usually rapid, and reaches its term within three or four days when the tide turns, the ebb being slower than the flow. In the chronic cases the changes of area are also frequent, but the frequent 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 pericard-

itis, as we shall presently see, the effect of pressure upon the heart is not always the same. Commonly the organ is displaced upwards into the narrower portion of the thorax, where it presses against the sternal and costal cartilages, and friction is not abolished, but made more loud and grating.

But though the area of friction and its intensity are apt to be greatest when effusion is at its height, there is no rule with regard to rubbing sound that is without exception. The increase of fluid may separate the ventricles, saving the upper part of the right ventricle and the great vessels (that is, from the first to the third spaces) from the chest wall by a layer of fluid, a layer so thin that firm pressure with the stethoscope will serve to bring the pericardial surfaces in contact and restore the friction sound.

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, rubbing sound is not inconsistent with extensive adhesion.

Thus the hazard of friction rub as a physical guide 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 audible when actual attrition is rendered impossible by firm adhesion. And it may be said further of this friction sound as of other things in Nature, that the noise it makes is not proportionate to its importance. The slightest scratching early in the disease may be the prelude to severe, perhaps fatal, carditis; on the other hand, loud and extensive rubbing sound may become audible when effusion is subsiding, and the improved aspect and manner of the child clearly indicate that the danger has passed.

Fallible as it is, let us make the most of it. Observe especially the cardiac tenderness that accompanies it in acute cases, a most suggestive sign; the change that it undergoes (or rather the changes) with varying pressure of the stethoscope, and sometimes also with altered position and with time.

There are some morbid sounds of the heart apt to be imputed when not actually present, notably presystolic murmur, as I shall presently try to show; there are others more often recognisable than recognised; of these latter, friction rub is the chief; and the main reasons for its being overlooked are:

1. That at its beginning and at its end it is often murmur like.
2. That it is often fitful and only encountered by chance; and
3. That the observer does not always employ the right methods for eliciting it, namely, varying pressure, frequent examination, comparison of its character to-day and to-morrow in various positions of the patient.

In some instances a rub-like exocardial sound will be heard when *post-mortem* examination discovers nothing to make it. The occurrence in the case of extensive and firm adhesion of the pericardium is not uncommon, as has been shown. But rarely what is taken for friction by expert observers is found after death to consist with a smooth and apparently healthy pericardial surface.

Thus a girl, aged 12 (N. H., August 26th, 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 contained an excess of clear fluid, was smooth, and without trace of deposit. Again, a boy (J. R., aged 8) with extreme anæmia, suddenly developed a loud double friction rub which did not long continue, and which, on his death some time later, was entirely unexplained on *post-mortem* examination.

Happily, then, pericardial friction rub is not the only physical sign of pericarditis; there is pericardial distension as well. 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 is quickened tumultuous uneven action of the heart, which, because it is difficult to express in precise language, because it varies more or less in individual cases; because, moreover, its precise significance, whether as “threatening” endocarditis or actually signifying, it may be open to question. But a little later and of more definite

physical indication there are these two: the rubbing of the inflamed surface of the heart and the ever so slight displacement of its apex.

There are certain characters, both clinical and anatomical, which distinguish these cases and deserve a moment's consideration.

Clinically.—(1) The cardinal feature is rapid, sometimes quite sudden cardiac failure, failure which the physical signs very imperfectly explain, being precisely the same as may occur with passive carditis where there is no similar failure and no threatening symptoms whatever; (2) the general symptoms are mainly cerebral, or at least nervous delirium, constant vomiting, sudden prostration. (3) The fatal cases very closely resemble the non-fatal in clinical respects; but when death is escaped, recovery is complete and sometimes rapid.

Anatomically.—(1) The pericardium is always adherent, often extensively and firmly adherent, the adhesions being more or less recent. (2) The endocardium, generally speaking, is but little affected, the tiny granulations upon the mitral valve (which is the characteristic appearance) being quite insufficient to render the valve incompetent. (3) The muscular substance of the heart undergoes but slight degenerative change.

Why do these patients die, and what makes the difference between living and dying? Physical signs, no doubt, are the more threatening when they show exocardial inflammation, or when the heart is already diseased owing to earlier attacks. Changeful endocardial murmur with tumultuous action, yet with no general disturbance, may at any time suddenly exhibit pericarditis together with all the symptoms of active carditis. But the first notice of danger comes not so much from the heart's physical state as from certain ominous general symptoms, restlessness, dyspnoea, delirium, prostration, vomiting—for the most part sudden and quickly fatal, which for no reasons known to us attack some rheumatic patients and spare others. The early appearance of such nervous symptoms is the best of all guides as to the future. Thus a child admitted with signs of carditis, but quiet and reposeful, had hallucinations as to people visiting him. Some days later he became prostrate and then unconscious, and narrowly escaped death; and just as physical signs are of small service in prognosis, so *post-mortem* examination fails to explain the fatal event. The heart failure is too sudden to be explained by muscular degeneration, and, as a matter of fact, this is not present to any great extent. The myocardium is chiefly implicated when the inflammation has been of long standing. Old adherent pericardium perhaps shows it best, but in recent inflammation, according to Dr. Byrom Bramwell and other high authorities, fatty changes in the muscular fibres are not generally observed. "The muscular fibres are swollen and their striæ indistinct, and leucocytes and blood corpuscles are seen in the spaces which separate the individual fibres."

I have here a series of very faithful drawings by my friend, Mr. d'Esterre, showing (1) the normal cardiac muscle; (2) the cardiac muscle from a child who died very quickly of cardiac failure within two days of pericarditis being super-added to endocarditis; the muscle is but little altered, its striæ being distinct; a second drawing from the same subject, less magnified, shows well the inflamed pericardium; (3) by way of comparison with the last is a drawing showing the degenerate muscle of old adherent pericardium, where striation is absent and the fibrillæ are widely separated. (4) Midway between these is a specimen showing distinct myocarditis, inflammatory elements being present in abundance; the specimen is taken from a boy who lingered some time in a condition of extreme prostration.

In consideration of the marked and agonising degree of dyspnoea shown by some of these cases of acute carditis, Dr. Lees suggests the question, "Is there a dextrocardiac respiratory centre?"² In the absence of any disease of the lungs, and with but little defect in the aëration of the blood, how, he asks, "is this very obvious dyspnoea, the result of pericarditis and of nothing else, produced?" Some might be con-

tent to answer that there is not pericarditis alone, but carditis also, and that the implication of the whole substance of the heart must so weaken it as to account for the symptom in question. But the extremity of the dyspnoea would seem better explained in Dr. Lees's view 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

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 between the muscle fibres as well as in them. Stained with hæmatoxylin, Ziess D, ocular No. 2. From a drawing by D. d'Esterre, M.B.

Fig. 2.—Pericardium and cardiac muscle in acute pericarditis. D. B., aged 8, muscle fibres in longitudinal section normal in appearance, no signs of inflammation, distinct striation, nuclei normal in size and number. Stained with eosin and hæmatoxylin, Ziess D, ocular No. 2. From a drawing by D. d'Esterre, M.B.

of the right ventricle resulting from the pericarditis or by irritation of the pericardial nerves." The paper is formally addressed to physiologists, and it is for them to answer.

In conclusion, something remains to be said of passive

² "Is there a Dextro-cardial Respiratory Reflex?" *Lancet*, October 28th, 1893.

carditis—of the signs of inflammation, that is to say, whether endocardial or exocardial, appearing without active general symptoms in the course of rheumatism, as is habitually the case with the adult. There is no more striking or

Fig. 3.—D. B. Showing thickened pericardium with ragged surface of lymph; muscular substance seen beneath. Power 2 in., ocular No. 2.

Fig. 4.—T. B. Myocarditis pericarditis. The muscle fibres are swollen, and their outlines indistinct; they stain less readily than normal. The nuclei are numerous and swollen. Zeiss D, ocular No. 2.

more important feature of heart disease as it happens to children than the close relation between endo- and pericarditis. A recent rheumatic endocarditis, fully developed in the case of the adult, leaves little fear of pericarditis presently supervening; with the child that fear is never absent, while in some instances, as we shall presently see, owing to the peculiar character of exocardial inflammation, it remains

Fig. 5.—W. B. Old adherent pericardium. Muscle fibres 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.

doubtful to the end whether or not the pericardium has been implicated. This near sympathy makes the distinction between endo- and pericarditis less discernible in children, and, so long as the inflammation is recent, justifies the term carditis as applied both to the active and the passive form.

A CASE OF ENCEPHALOCELE: REMOVAL: HYDROCEPHALUS: DEATH.

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On July 14th, 1893, I was called to Mrs. O., a thin, anæmic woman, with an external strabismus of one eye and a chronic ulcer of the leg. Her confinement was expected about the middle of August, but labour had already commenced. Examination revealed a normal presentation, but in addition a fluctuating tumour, the nature of which could not be determined with certainty. A few sharp pains, however, soon cleared up the diagnosis, for the child (female) was speedily born, and presented a large fluctuating tumour attached to the occiput. This, as events afterwards proved, was an encephalocele. It was lemon-shaped, and in size almost, if not quite as large as the child's head. Both from the mother's reckoning, and its general appearance, the child was evidently premature—I should say from three to four weeks. It was unable to take the breast, and had to be fed with a spoon. On the third day, the covering of the tumour showed unmistakable signs of congestion and ulceration, and a slough was in the act of separating when removal was determined on in the barest hope of prolonging the child's life.

With the aid of Dr. Taylor of this town I passed a strong catgut through the centre of the pedicle, tied a Staffordshire knot, and then quickly detached the tumour. It was found, however, that the ligature included brain substance. Stitches were accordingly passed on each side, the stump of brain matter pushed inwards, and the scalp firmly stitched over the opening in the bone, the Staffordshire knot meanwhile serving to restrain the hæmorrhage, which altogether was but trifling.

Within the next twenty-four hours the child had several fits, in which both eyes were turned towards the left shoulder.