a single tabloid was given, but, the pulse keeping about 100 and the other symptoms being still marked, the drug was stopped for four days until the pulse had fallen to 80 and the dyspnoea allayed. Two tabloids were then given for two days, the pulse rising to 90. Since that time only one tabloid has been administered each day. During the first fortnight of treatment with the tabloids the patient lost flesh although her appetite never failed, but since then she has entirely regained her usual appearance. The patient was allowed out of bed on December 21st, and has been going about her customary avocations ever since.

"So much with regard to her general condition under the thyroid tabloids; now with regard to the psoriasis. On December 10th, that is to say the day when the thyroidism manifested itself, the itching was greatly reduced. In a day or two the hyperemia became more dull, and then the scruft, which had always re-formed when it was removed, ceased to reappear. In each patch the same sequence of events took place. The itching was first lost, then the redness, and eventually the scruft. Improvement everywhere has been general and rapid. The smaller spots yielded first and the largest patch has been the last to go. And now only blotches of pigmentation mark the sites of the patches of psoriasis, and these blotches themselves are daily becoming less distinct. The tabloids are still being given.

"I may add that at first the diet in Mrs. A.'s case was restricted to plain food with no condiments, and an injunction was given to partake of animal food sparingly; but during the treatment with the thyroid tabloids ordinary diet was allowed in any proportions the patient thought fit to take. Her appetite was always good.

"I regret that, owing to a natural reluctance on the part of the patient, I am unable to furnish you with photographs or drawings of the case.

"Believe me,
"Faithfully yours,
"D. LENNOX."

THE LUMLEIAN LECTURES

ON

HEART INFLAMMATION IN CHILDREN.

Delivered before the Royal College of Physicians.

By OCTAVIUS STURGES, M.D.CANTAB., F.R.C.P.,

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III.

MR. PRESIDENT, FELLOWS, AND GENTLEMEN,—In the last lecture we were led to consider the service of percussion in estimating the area of cardiac dullness at various ages and in several conditions, together with the manner of the heart's displacement with the gradual increase of 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 feebly expressed by words, become 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 the accompaniment, of a child's first rheumatic attack of short duration and no severity. It remains to consider some further signs of endocarditis referable to the right and uninflamed side of the heart; to estimate the nature and frequency of mitral narrowing in the child as compared with the adult; and, in conclusion, to add but 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 are laid out of the case. Yet, inasmuch as endocarditis concerns the fibrous structure of the left side of the heart and extends to the papillary muscles and their tendinous cords, it is of necessity that this inflammation should produce effects beyond the valve itself. 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 reflecting on the right ventricle, as Sibson points out, leads to accumulation of blood therein, which is relieved by tricuspid reflux—what has been called "safety-valve" action. 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 inflammation as well as a token of relief.

There are some obvious reasons why this early occurrence of tricuspid murmur should often be overlooked, such as the presence of old valve murmur; the short duration of this

sign and the speedy oncoming of a louder mitral murmur to drown it. It is only, therefore, in a first attack of rheumatism, and with the happy chance of early observation, that tricuspid murmur gets noticed. Something also must be allowed for the habitual neglect of the right side of the heart in the affection we are discussing, neglect due to the knowledge that the actual seat of disease is assuredly the left ventricle not the right. Yet with all this allowance, it may still seem that, upon the hypothesis, tricuspid murmur should be met with more often 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 action and rhythm of the heart. 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 heart muscle due to the altered relation between the inflamed endocardium and the blood that visits it. Tumultuous action, variability, reduplication of the second sound and altered (lengthened, it may be, or of altered tone, such as I know but cannot put to words), these are joint signs of this irritability. Tricuspid murmur in these circumstances may so soon be followed by mitral as to escape notice. But, given a previously healthy heart, early access to the patient, and, it must be added, due respect for the right ventricle, and care to discriminate between tricuspid and mitral, and there can be no doubt that the sign upon which Sibson laid so much stress is not seldom met with.

H. W., a boy of 11, was admitted to the Westminster Hospital October 24th of last year, having been ill three days with his first attack of rheumatism. Pain was in knees and ankles, and temperature 102°, but there was neither swelling nor redness of any joint, and on the fourth day of the illness, that is a few hours only after admission, the fever left him. After three days' residence the boy seemed well. The area of cardiac dullness was normal, and it was not until the fifth day from the first joint pain (26th) that anything abnormal was noticed of the heart. On that day the rhythm became uneven, and a slight systolic murmur (not blowing) was heard at the ensiform cartilage, less audible at the apex. Pulse was 84 and ill sustained, varying in force, that is to say, remarkably when continuously felt over three or four minutes. A week later (November 6th) the tricuspid murmur was still audible with that rough beginning which some christen pre-systolic. At the apex the first sound was lengthened and murmurish. It happened with this boy as with the girl Edmunds, previously quoted, that upon repeated experiment the murmur was always lost in the erect posture.

Shortly after this patient, whom we were not able to retain long, a girl of 18 was admitted on the ninth day of her first attack of acute rheumatism. There was pericardial rubbing, and at the ensiform cartilage a distinct systolic (tricuspid) murmur. Both joint pains and pericardial friction quickly disappeared, and on the girl's discharge, after three weeks' residence, a soft apex murmur had taken the place of the rougher tricuspid *bruit*.

A third case might be quoted to the same effect, a boy of 10, in his first attack of rheumatism with some heart pain. At first no cardiac murmur was audible, and the earliest physical sign of valve lesion was again tricuspid in seat and character.

And just now, in the nick of time, comes another boy, aged 12, in the third day of a slight rheumatic attack. A systolic murmur was audible at first at the ensiform cartilage; but after three days' continuance it shifted its place, and became distinctly mitral.

It is true that in practice we cannot afford to depend upon so precarious a sign as tricuspid murmur; true also that the signs we do trust we often fail to describe. Many have laboured at their description from Sibson to Potain, and all have consciously failed; they are to be learnt not from the book, but at the bedside. Thus there are many varieties of first sound, all of them more or less indicative of commencing endocarditis. The prolongation of such sound right up to the second is a precise definition enough. Not so "the accent" which begins it, while the several changes of quality—changes which every student soon learns to recognise for himself—he cannot impart to another. It is knowledge that is not transmissible, but remains each man's secret in

spite of his best efforts to reveal it. Such words as "booming," "rolling," "rattling," and many more, even if they satisfy their authors, hardly suggest to a novice what manner of sound he is to listen for, but presently the sound itself will strike upon his own ear, and become his possession for ever.

While this is true of modified sounds there is less difficulty in describing the precise place and time in the heart's cycle of added sounds; but even this is not to be decided in a moment. In the child especially the exact sequence of sounds and pauses is ill expressed. In timing the heart's acts by the ear it is not always realised that these acts are represented partly by sound and partly by silence, and that defective action is detected as well by the silence being broken as by the sound changed. In the long silence especially—silence which is not rest—in the period, that is, of ventricular diastole, whatever noise intrudes is morbid.

But let the patient be a child, with action rapid and impulse obscure, and it becomes a nice point to decide whether intruding sound is just without or just within the 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 concur respectively with distinct acts of the heart, with dilatation and contraction, yet they both join on to the first sound, and in some cases neither is much conducted.

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 structure changes that need time to accomplish. Early systolic murmur, on the other hand, a harsh commencement of the first sound, converting "who" into "through," is commoner with them than with their elders. It will sometimes arise early in the course of rheumatism, and in a first attack of it, presently to disappear, or toning down into a soft blowing systolic murmur.

I have no intention of entering upon any matter of doubt or controversy. All who had the opportunity of listening to Dr. Chapman's Goulstonian Lectures must speak with new deference of the mechanism of heart sounds. But the light-hearted way in which this word "presystolic" is flung about nowadays, coupled with some facts I am about to relate referring to mitral stenosis in children, may, perhaps, excuse a few words upon this 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 towards the end of its work. Arising precisely as such conditions would lead us to expect, within the period of ventricular dilatation, where properly there is silence, this murmur grows and gathers right up to the first sound, which abruptly ends it, extra pulmonary resistance being next indicated by over-distinctness of the second sound at the second left cartilage.

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 usual also 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 gets 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 contraction, the left being a thought behind.¹ But all are agreed 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 must be exceedingly dangerous, therefore, to accept diastolic mitral murmur occurring anywhere within the long silence as being evidence of "a degree" of stenosis.

While the well-marked presystolic murmur, with its thrill, gathering force, sudden end, and accentuated pulmonary second, are reliable signs of notable mitral narrowing, we cannot trace by physical means the successive degrees of this change or watch the progress of 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. The *post-mortem* experience of eleven years at the Hospital for Sick Children supplies, so far as I can find, but a single example of "button-hole" contraction of the mitral valve; and this one, a boy of 9 with cellulitis, was not known to have had rheumatism. In that connection there are but two cases of marked (not "button-hole") mitral narrowing; one of these had and one had not presystolic murmur in life. "Slight" stenosis, with what was named presystolic murmur, was possibly present in five, not all accurately described. But the same term, presystolic, is by no means confined to these. It appears in the histories of at least nine (probably eleven) of the children who *post mortem* exhibited no stenosis whatever. In my own clinical notes of the past three or four years I find the following illustrations of this point. There was marked stenosis with its proper physical signs in one; slight stenosis with "presystolic" in one; in five, thrill and "presystolic" murmur (so-called) showed no corresponding stenosis *post mortem*; in five, "presystolic" murmur, noted at the first, disappeared later on.

"Presystolic" Murmur in Connection with Mitral Narrowing.

A.—POST-MORTEM SUMMARY.

I. Marked stenosis ("button-hole") with presystolic murmur (not rheumatic) ...	1
II. Stenosis without presystolic murmur ...	1
III. A degree of stenosis with presystolic murmur in ...	5
Very "slight" in ...	2
"Some" in ...	1
"Mitral admits thumb" in ...	1
"Stenosis," not further described, in ...	1
IV. No stenosis with "presystolic" murmur in ...	11
N.B.—In 8 out of 19 mitral thickening noted, not stenosis.	

B.—CLINICAL SUMMARY.

The murmur disappeared in 5 (1, 2, 6, 10, 11).

The murmur appeared within two or three weeks of first rheumatic attack in 3 (3, 5, 11).

No narrowing was found *post mortem* in 4* (4, 7, 8, 9).

* In 2 of these mitral was thickened.

If we take by contrast the case of adults, it will be found that the "button-hole" form of stenosis is by far the commonest. Thus, in cases collected by Dr. Fagge, 46 were of that kind against 1 funnel-shaped; while in 40 of Dr. Sansom's, 19 were of that form against 15 funnel-shaped and 16 not particularly described. It may be added that the same authority found rheumatic antecedents in but half the cases, and Sir Dyce Duckworth in 60 per cent.

It thus appears that *post-mortem* evidence upon the question of presystolic murmur wears a somewhat special aspect in the case of children, owing to the rarity of that chink-like narrowing of the mitral orifice, which, together with auricular hypertrophy, can alone be depended on to produce the combined physical indications of stenosis.

Now, the chief form of rheumatic mitral endocarditis met with in children at an early stage consists, as is well known, of a ring of minute granulations edging the valve but not appreciably constricting it. These effect no more, after the active stage of inflammation has passed, than to cause the blood to pass over a rough surface instead of a smooth one. A later stage is not in the way of narrowing but of stiffening as regards the mitral flaps and chordæ tendineæ. Such a condition seems fitted rather to produce tardy closure than any material hindrance to the onward blood flow. "It is the case of a valve delayed in its closure by rigidity," as Dr. Dickinson expresses it, "but which yet shuts at last." Valves so circumstanced permit regurgitation at the first part of systole; they are slowly raised during that act, and in the rising the stiffened valve apparatus itself may emit

¹ Dr. Barr, *Liverpool Medico-Chir. Journal*, January, 1894.

sound. The early thrill is caused, as Dr. Seymour Taylor has pointed out,² by these stiffened flaps "held edgewise to the regurgitant stream."

The rough or accented beginning of the first sound 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 children who exhibit this physical sign may not get mitral stenosis later on. But the early stages of this chronic process do not suffice for the production of a true presystolic murmur but only for its rough counterfeit. Early rheumatism no doubt favours eventual mitral narrowing, but the notion that mitral stenosis can be watched through all its stages from early childhood onwards is, I am persuaded, erroneous. Without denying, therefore, the special significance of those signs of stenosis first pointed out by Fauvel and Gairdner, there is evidence enough, both clinical and anatomical, that these signs are met with sparingly among children, hardly ever among very young children.

Of all the morbid sounds of the heart whose meaning we guess at with more or less of probability, there is none that to me seems more obscure than true diastolic murmur at the apex. In the easy language of the wards, we often hear of "double mitral," what is meant being sometimes a presystolic with a systolic murmur, and sometimes, and most often in the case of children, a rough accent commencing the first sound and a blowing murmur following it. But neither of these is properly "double mitral." That phrase must imply murmur with the first sound, murmur with the second sound, and both at the apex. It is so far comparable with the double aortic murmur whose mechanism everyone understands. How can a diastolic murmur at the mitral exactly replace the second sound? Walshe meets the difficulty by observing that the diastolic murmur is rarely loud enough to cover the second sound completely. "It is commonly spoken of," he says, "as diastolic in rhythm, but in point of fact is rather post-diastolic and presystolic that precisely coincident with the diastole." Another suggested explanation, and in some instances no doubt the right one, is hardly applicable to children. It is that the diastolic murmur, apparently mitral, is in fact aortic. "In a small proportion of cases," says Dr. Bristowe, "systolic murmur is associated with a true diastolic mitral." This true diastolic, however, he defines as "running up from the second sound, and tending to die out in the diastolic silence." "It may be confounded," he adds, "with the diastolic aortic." And in Dr. Bristowe's³ view, Laennec's diastolic mitral (and probably Hope's also) was in reality aortic. But what of the little children with whom aortic disease is rare and double mitral common?

Dr. Dickinson, as is well known, does not so interpret Hope's murmur, but regards it as in fact mitral diastolic, and clearly distinguishable from the so-called presystolic, which in his hearing is really a reflux murmur as determined by its relation to the impulse. "The early part of the sound," he says, "is obscured or replaced by the murmur; the terminal slap or knock is the only part of the second 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."

I have tried to show that in the case of children at all events *post-mortem* evidence is all in favour of such an explanation of that rough and early murmur which habitually goes by the name of presystolic. Yet still true mitral diastolic exactly covering the second sound is unprovided for; and still, as I cannot but believe, mitral stenosis together with hypertrophied auricle—a disease of women rather than of children—has physical signs of its own, and that these have been accurately defined by Gairdner.

Mention may be made in this place of a morbid sound noticed by Laennec, and, so far as I know, not greatly enlarged upon since his day—a creaking like that of a rusty hinge or an ill-greased wheel. It is not accurately in time with the cardiac sounds, and, owing to its peculiar quality, easily audible over and above these. This metallic "squeak," which is not constant, is sometimes so loud as to be heard

away from the chest, and I know an instance in a child where from time to time it disturbs the family quiet, though causing no distress to its subject. Laennec avers that this squeak is sometimes loud enough to be heard in the next room. Usually it seems to be mitral in place, but I have a striking example where it was diastolic aortic, occurring in a boy, to whom it gave very little inconvenience. It is a curiosity, and has no significance apart from that of the valve disease (usually mitral) that it accompanies.

A few words will suffice for all that can be usefully said of prognosis. Rheumatism we say is the main cause of heart inflammation in children. But what is rheumatism? What definition can be contrived to compass the whole of it and yet retain distinct form? Of late years its area has been more and more extended, and in the process its substance has so thinned out that its proper shape is hardly discernable. Pleuritis, pneumonia, tonsillitis may all be rheumatic, we are told, though unattended by joint or limb pains.⁴ And further, if further there can be, Sir Dyce Duckworth, in a lecture⁵ published this very week, announces "rheumatism of the brain" (probably of the cortex), of which the chorea of children is the outward and visible sign, a trifling ailment for so alarming a lesion. It is not as yet anatomically known, but it is thus foreshadowed; the effect is familiar, the assumed cause awaits subsequent discovery. Rheumatic phenomena being thus indefinitely multiplied, 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. 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 *Noûmenon* of the old philosophy.

But if rheumatism, whatever its criterion, be the cause of carditis, what is its own cause? Not a tithe of the cases, even of unequivocal acute polyarthritis, have their origin in exposure. In 608 consecutive examples collected by my friend, Dr. Syers, only 33 could be thus attributed.⁶ Let us doubt that number, and even so hardly a tenth can be so counted. In the great majority the cause of rheumatism is conjectural, like so much else connected with it, so that in particular cases of carditis little help towards prognosis is likely to come from consideration of the nature of the rheumatism that attends it.

Yet there are certain circumstances of undoubted service in prognosis; for example, 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 præcordial tenderness; the degree of pallor, and the rate of wasting.

As for age, rheumatism occurring at an unusually early period of life, say at 4 or 5, is very nearly allied to carditis, and sometimes shows itself chiefly by heart inflammation. It may be said without qualification, other things being equal, 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 successive attack of rheumatism, however little severe in itself, is a fresh injury to the heart, the effect of these multiple attacks is obvious. Older childhood is far less subject to this same frequency of rheumatic visitation; and on that account, as well as because the increase of years of itself lessens the danger of carditis, the child past 8 or 9, 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

⁴ Mitchell Bruce, BRITISH MEDICAL JOURNAL, April 26th, 1890.

⁵ Clinical Journal, March 14th, p. 307.

⁶ Westminster Hospital Reports, 1881-89.

² Lancet, November 12th, 1887.

³ Lancet, November 12th, 1887.

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 is more or less—it can never be absent—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 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 became extremely dilated, and the child very quickly died.

It thus appears, both in regard 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 are altering their character from day to day, so long is there active endocarditis; and in the same way, as was shown in the last lecture, 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 children and affects its mortality. The appearance, therefore, of such symptoms in whatever degree at an early stage of endocarditis—vomiting, severe headache, weariness, passing delirium, uneasy sleep with night terrors—ought properly to cause anxiety, however favourable the child's progress in other respects.

In one of the cases of acute carditis already quoted, a boy of 9, in his first attack of rheumatism with 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. Headle, so ably detailed in his Hunterian Lectures on Rheumatism in Children as to the evil import of subcutaneous nodules.

The clinical picture of carditis in children, gloomy as it is, is not without light. 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 as chronic sufferers, and at last they either perish quickly with acute suffering or else linger on with hearts incompetent and attending 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 child 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 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 quite 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; there is no harm done; if you had discovered it you might have treated it."

It is of interest now to observe the point of view whence

rheumatic carditis was once regarded and the plan of treatment that resulted therefrom. It is of yet greater interest to note the remarkable fact that the characteristics of child's rheumatism, and notably its moderate fever and brief pain, did not serve to exempt children from all the rigours of the antiphlogistic treatment.

"I know of no disease," says Latham,⁷ "in which febrile heat is greater and the pulse harder and more forcible than in acute rheumatism. What are the signals for copious bloodletting 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." That this is no picture of child's rheumatism need not be said. It seems to follow that treatment might reasonably be modified in the case of children so as to suit the symptoms. But the old authors admit of no exception. A single passage throws vivid light on the treatment 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 foetor 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 happily 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, but bleeding alone would not suffice, mercury must go along with it; and this not in stated doses, but in such amount as would as quickly as possible "touch the gums." When this particular symptom of mercurial poisoning was delayed the drug was "pushed" to the utmost degree. Yet some (these two healthy patients in the prime of life, for example) would die unsalivated.

It is easy to criticise at this safe distance of half a century. Therapeutics is a healing 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 doubt whether the early advent of salivation, with the consequent suspension of mercury, did not bring a blessing to the patient less by virtue of its healing than of its poisonous qualities.

Contemplating the old antiphlogistic treatment, we are, perhaps, too much in the habit of assuming that the rate of mortality of certain acute diseases, notably of pneumonia, is less now than it was early in the century. It is a delusion, as statistics prove. But in the case of pericarditis, anyone who will read its literature will, I think, become convinced that it was more fatal under the old methods than now. The whole portraiture of the disease gives evidence of this. One of its chief and 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, putting children aside, the immediate danger to life is very small.

Such considerations would seem to furnish reason enough for departing from ancient traditions without more ado. But it is not so. 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. Latham's treatment was at least precise, emphatic, uncompromising, and it lingers still, robbed of all those characteristics. We have in place of it an uncertain voice and some disagreement. For example: "Blood may be taken from the arm," says one textbook, "but preferably by cupping or leeching" (Roberts, 501). "Venesection is never admissible," says another. A

⁷ New Sydenham Society, vol. i, p. 116.

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 matter of doubt" (Bristowe, 580). "Counter-irritation is of considerable value," says one; "Blisters should be shunned," says another. Finally we are told not to treat pericarditis at all as such (Balfour, 326). "Its treatment merges itself in the treatment of some other disease." All these are literal quotations from as many recognised textbooks of the present time.

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; and 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 dyspnoea, 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 opinions as contrasted with those of the past, Dr. David Lees, in an able and interesting paper on the treatment of pericarditis, 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 morphine 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."

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. But 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 lumping together without distinction as of equal value bleeding, counter-irritation, morphine. In the abstraction of blood (by leeching, as a rule) we possess, in my belief, the most potent means of relief for those sudden dyspnoea attacks 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 textbooks think so in urging as a sufficient objection to their use the 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), 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 of relieving pain, though demurring a little to the statement that "we have made the discovery that the salicylates are curative of the rheumatic process." "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."

Time will not serve for stating the arguments or the evidence upon which this opinion is based. All who are acquainted with the author 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 ice bag, whether to pericarditis or to 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 cases, and surely there is no disease whatever wherein it is easier 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, dyspnoea, 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's contention is made out, but only so. Recovery without classification of cases proves nothing. The efficacy of the remedy can only be tested when the need for it comes.

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; and if it be true also that children have not as yet shared in this improvement it must be remembered that there has been some neglect in their case and less time for accurate observation. There is strong hope for the future in the pathology of heart inflammation, for it represents, as I have shown, an organ for the time disabled, yet structurally but little altered. The condition is one that especially invites therapeutical aid, and already there are indications that such aid is forthcoming. Sober investigation, however, is of slower pace than the demands and assertions of the drug market. Hopeful work is being done, and with drugs, but it is yet too early to announce conclusions.

In bringing these lectures to an end I cannot omit mention of the ready help and co-operation of my colleagues, especially of Dr. Garrod, Dr. Batten, Dr. Walsham, and Mr. Winckworth—indeed, I have been helped by so many that, could I but be certain that they would warrant my facts and accept my inferences 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.

AN ADDRESS

ON

APPENDICULAR COLIC.

[WITH COLOURED PLATE.]

Read before the Leeds and West Riding Medico-Chirurgical Society.

By T. R. JESSOP, F.R.C.S.,

Consulting Surgeon to the Leeds General Infirmary.

THE objects I have set before myself in writing this paper are to urge a closer attention than has hitherto been given them, in this country at least, to those conditions of the vermiform appendix which stop short of, or precede, the peritonitic complications described under the heads of "appendicitis," "typhlitis," "caecal abscess," etc., and to advocate a more precise and discriminating nomenclature by which these early conditions may be indicated. There is no need to dwell upon the advantages to be derived from a recognition of those pathological changes within the cavity of the appendix which, whilst they may prove to be of fleeting or of only a temporary importance, may yet be the precursors of an acute peritonitis productive always of serious illness, and not seldom leading to a fatal termination.

The exciting causes of appendicitis are probably various. We are familiar with the foreign bodies, stercoral concretions, melon or grape seeds, cherry stones, and the like, because we meet with them when, in the later stages, they are cast off along with the inflammatory products they have given rise to, or when we incise the abdominal wall for the evacuation of pus collected around the vermiform appendix; but these, so unmistakable when recognised, are comparatively rare. It is not to these I would ask your attention, but rather to those commoner varieties in which there is no such obvious causation—cases presenting all the signs of a so-called acute typhlitis clearing up, it may be, without leaving a trace behind, or characterised by recurrences more or less frequent and of varying severity, or again ending in the formation of abscess, which wherever, or in what way soever, evacuated presents no features by which its origin may be divined.