Remarks
ON
THE INFECTIVE NATURE OF RHEUMATIC FEVER,

ILLUSTRATED BY THE STUDY OF A FATAL CASE.

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MR. PRESIDENT AND GENTLEMEN,—I have the honour of bringing before you to-night the results of an investigation into a fatal case of rheumatic fever in a child; I also wish to occupy some of your time in a consideration of the statement that rheumatic fever is an infective disease.

I.—CASE AND COMMENTARY.

The case is the twenty-fifth fatal one that I have investigated, 24 of which were studied jointly with Dr. A. Paine; it is a rare example of an illness, which it was possible to follow over a period of many months, to investigate carefully after death, and finally to elucidate by experimental research. I have to thank my colleague, Dr. W. S. Colman, as at one time or another I have had to thank all my medical colleagues at the Hospital for Sick Children, Great Ormond Street, for permission to make use of some of the notes of the illness. Let me first say that it is not my intention to weary you with irritating clinical or *post-mortem* details, and the descriptions that I shall give will only bear upon practical points, and upon facts which need due emphasis.

History of Early Attack.

In July, 1902, a delicate little girl, aged 9, was brought to my out-patient department suffering from chorea; the attack was moderate in severity, general in its distribution and ordinary in its type. Unfortunately there was active heart disease; the mitral valve was damaged, and the action of the heart was rapid and excited. It was clear that immediate rest was essential, and she was at once admitted into the hospital.

This was her first attack of rheumatic fever. It had commenced six weeks previously with vomiting and pains in the limbs; a week before admission there had been painful swellings of the wrist-joints, and the last symptom to make its appearance had been the chorea. There was no family history of rheumatism.

In the hospital a more detailed examination of the heart showed that there was considerable dilatation, and that there was also a to-and-fro murmur at the apex; the systolic element of this murmur was loud and blowing, and could be traced into the axilla. Her stay in the hospital was uneventful, and, with a further rest in a convalescent home, occupied fourteen weeks. After this she returned to me, and I found that the chorea was cured, but that the condition of the heart was unsatisfactory, its action was still rapid, and the systolic murmur was distinct. Rest and medical treatment were continued at home, with slow improvement, and the child reported herself to me at least once a month.

By May, 1903, the *bruit* had disappeared, and at the apex only a short first sound and normal second sound were audible. I mention this fact because the disappearance of this mitral murmur could neither have meant that the valve had completely recovered its natural state, nor could it have meant that the *bruit* was the result of dilatation of the left ventricle, and not the result of an endocarditis. The attack, I think, had been too severe and too obstinate for either of these explanations to be probable, and I had little doubt that the disappearance of the *bruit* was to be interpreted as the commencement of mitral stenosis. For this reason I watched her case with particular interest.

History of Fatal Illness.

Unfortunately, in June her mother fell ill, and did not bring the child again until August 17th. She was then so much worse that she was at once admitted into the hospital under Dr. Colman.

The history of this latter illness was as follows. Fourteen days previously she had complained of pain over the heart and in all the limbs; she had looked cyanosed, and had felt cold and shivery, and, as in the former illness, had suffered from attacks of vomiting.

On admission her temperature was 103.8°, her pulse 140, and her respirations 40. There was great cardiac distress, but, although the heart was greatly enlarged, there were no signs of pericarditis. The prominent feature on examination was a singularly loud systolic murmur in the mitral area. Examination of the lungs showed nothing definite. The spleen was enlarged and tender, the liver was enlarged, and the urine was albuminous. The temperature, in the two-hourly chart, showed irregular fluctuations; in the four-hourly, a continuous and moderate pyrexia. While in the hospital, the prominent symptoms were rapid anaemia, sweating, and abdominal pain. Ten days after admission she died suddenly.

This illness appeared to me to be an example of that form of endocarditis of rheumatic origin which is comparatively rare in childhood, and which may be called the malignant rheumatic form. For looking upon rheumatic fever as an infective disease, I recognize three types of endocarditis.

The first, simple endocarditis. In this the micro-organisms in the valve either cease to be active, or are destroyed by the living cells of the tissues.

The second, the fibroid type, as in mitral stenosis. In this the infection is persistent, but the resistance of the tissues is great. This type was greatly elucidated by a distinguished former President of this Society, Dr. Sansom.

The third, the malignant type. In this the micro-organisms grow in countless numbers in the valve; the vegetations may be either large or small, and the resistance of the tissues is feeble.

In this case the diagnosis of malignant endocarditis was based upon the presence of infarction with severe endocarditis, of irregular fever and rapid anaemia, and upon the absence of any true evidence of pericarditis.

Post-mortem Investigation.

I was fortunate enough to obtain permission to make an examination immediately after death, and I had the necessary apparatus at hand, and the assistance of Dr. Neave, the clinical pathologist, and Mr. Langmead, the house-physician. A bacteriologist will understand the real value of such an opportunity, which is only too rare.

The method of investigation will need only a very brief description:

1. The pericardium was exposed, dried, and seared, and two Pasteur pipettes were passed—one into the pericardial cavity, the other into the left ventricle. The pericardial exudation was clear. This and the blood from the heart were added to culture media of alkaline broth and milk.
2. The pericardium was opened. Then a thread was passed through the muscle at the apex, and the heart was pulled out and its posterior aspect exposed. This surface was dried and seared, and the left auricle and ventricle opened by one sweep of a heated knife. Assistants then held the walls open with sterilized forceps, and the mitral valve was exposed, covered with blot clot. The valve and clot were cut out and placed in the media, and another piece of the valve was put at once in fixative.
3. Cultures were taken from (*a*) the lungs, (*b*) the kidneys, (*c*) the spleen, (*d*) the gall bladder.
4. Films were made from these organs and the exudations.

Permission for examination of the brain was refused. The necropsy made it clear that there was no pericarditis, and showed that the mitral valve was the only one certainly diseased; this valve was much thickened, and upon the small piece which was placed in the fixative there were tiny vegetations. The heart itself was generally enlarged. The lungs showed patches of pneumonia and congestion. The spleen was much enlarged and contained white infarcts. The kidneys were very pale, and the transition from the cortex to the medulla was difficult to trace. The liver was large and fatty. There was no suppuration.

So far, then, as the gross lesions were concerned the diagnosis of malignant rheumatic endocarditis was upheld, and it was with much interest that I cut the fragment of the mitral valve, for small though the vegetations upon it were, the true interpretation of the case rested upon the demonstration in these sections of numerous diplococci. A section is under the microscope, and later will be thrown upon the screen. The enormous number of diplococci in the vegetation is very remarkable, and this specimen is a perfect example of a valve in the condition of malignant rheumatic endocarditis.

Bacteriological Investigation.

The bacteriological results from the cultures were as follows:

1. Pure cultures of the diplococcus were found in the tubes inoculated from the valve, the spleen, and the kidney.

2. The culture from the lung was not pure, but the diplococcus was present among other micro-organisms.

3. The tubes containing the pericardial fluid and the blood gave negative results.

The negative results obtained from the pericardial fluid emphasize a point to which, with Dr. Paine, I have already directed attention. It is that these micro-organisms, when they grow in the body, grow best in the local lesions, and although they are often present in the blood they do not thrive in it. Moreover, they are not easily found in recent exudations, for they lie in the synovial tissues, and not in the synovial fluid of the joint; in the pericardium itself, and not in the pericardial exudations. To reach these fluids they must destroy the phagocytic endothelia that line the synovial membrane and pericardium, and the reason why the micro-organisms are deposited in the tissues and not at first in the exudation is clearly because they are carried in the blood stream, and where they escape will find themselves in the connective tissues around the blood capillaries.

It is not, then, to be expected that in a slight attack of rheumatic fever, either examination of the blood or of the synovial exudation will often result in success. This is not strange, indeed it would be much stranger if one had only to take a minute quantity of blood from a finger or vein, in order to show the infective agent, or if the micro-organisms dropped into a synovial exudation like pepper from a pepper-pot. If that were the case rheumatic fever would indeed be a terrible malady.

Experimental Investigations.

One of the cultures was immediately sent to Dr. V. Shaw, who carried out experimental investigations upon rabbits and monkeys. The lesions of rheumatic fever resulted in both animals, and thus Dr. Shaw has shown that the monkey is susceptible to the infection. His paper, a valuable addition to the subject of rheumatism, was published in the *Journal of Bacteriology and Pathology*, December, 1903.

The infected rabbits died of arthritis, endocarditis, and pericarditis. One monkey died from arthritis, pericarditis, and endocarditis; another, after an attack of severe arthritis and the development of a mitral systolic murmur, recovered and was alive four months later. Dr. Shaw has kindly lent me the specimen of the heart of the monkey that died from rheumatic fever. It shows, you will see, well-marked aortic endocarditis, and also early mitral endocarditis.

These are the hearts of two rabbits which were also inoculated. And as a contrast let me also show the heart of a monkey which died from a mixed infection of the rheumatic diplococcus and the staphylococcus aureus. The pyaemic abscesses, due to the staphylococcus, are distinctly seen in the cardiac wall, and form a striking contrast to the specimen of the pure rheumatic infection. The diplococcus was again isolated in pure culture from the tissues of the animals.

This experimental investigation by Dr. V. Shaw was an entirely independent one, which in my opinion adds very greatly to its weight.

This case then is a striking confirmation of the infective nature of rheumatic fever. When I hear the great "if" with which this view is often prefaced, I sometimes feel inclined to ask: "What more proof do you want"? The infective agent is present in all cases in which there is a reasonable chance of finding it; it is present in the local lesions; it produces similar lesions in animals, and is present in those lesions also. Scientific clinical physicians have for years thought that the disease is of the pyaemic or metastatic type; the bacterium belongs to that group of micro-organisms. Suppuration is rare in rheumatic fever. It is also rare in experimental rheumatic fever. Even our old friend "acidity in the blood" can now pass into an honourable retirement, for the micro-organism, as this culture is witness, has powerful acid-producing properties, although I do not suppose it causes "acidity in the blood."

II.—Practical Applications of the Infective View of Rheumatic Fever.

In the first place, this view brings into prominence the extreme importance that must be attached to the early symptoms of the disease. In these days, when we deal with tuberculosis, our first thought is to detect the disease in the earliest stage, and when the lungs are attacked the infective agent is fortunately deposited in a position from which it is often coughed up and discovered in the sputum. This advantage we do not possess when dealing with rheumatic fever, but the principle of an early diagnosis equally holds good, and when the medical profession seriously accepts the infective view it will be bound to search for every shred of clinical evidence that can lead to the detection of rheumatic fever before it has produced gross lesions. Clinicians there are who have already done so. The gradual failure of health, the wasting, the vague epigastric and other pains, the nervousness, the night terrors, the anaemia, all these equivocal warnings, and others also, will require the closest investigation in the children of rheumatic stock. Among the hitherto unexplained pyrexial attacks of childhood rheumatic fever must be reckoned as a cause. In course of time the public will learn from the medical profession the value of this early diagnosis, and fewer cases will be brought to us already irretrievably damaged, and thus our opportunities for treatment will be improved.

Secondly, there are certain facts—none of them, I admit, new facts—which were formerly uncertain, but which now rest upon a sure basis, namely:

1. The existence of a true rheumatic broncho-pneumonia and a rheumatic pleurisy.

2. The existence of a true renal rheumatism.

3. The existence of a true rheumatic peritonitis.

Clinical, pathological, bacteriological, and experimental evidence can now be obtained in support of these statements, and although some of these manifestations may be rare, the proof of their occurrence must be of value.

The relation of tonsillitis to rheumatic fever has been recognized for more than a century, and at the present time the learned President of our Society has been among the foremost in insisting upon the importance of that relation. Now its true meaning is clear, for it is certain that one channel of infection is through the damaged tonsils in which the micro-organism is deposited. Almost as complete is the proof of the existence of a true rheumatic iritis and chorea.

Thirdly, it is now possible to produce with great constancy experimental heart disease. This I believe to be a step forward in the study of heart disease, and to be an event in the history of heart affections. Even if the accumulated evidence from all parts of the world upon the infective nature of rheumatic fever were swept away to-morrow, there would still survive after its destruction this fact which cannot be ignored. Already our practical knowledge has profited, for it has shown:

1. That simple and malignant endocarditis can be stages in the same process, and has demonstrated that they are not always essentially different, and that the malignant form does not always premise a secondary infection. It follows from this that if rheumatic endocarditis is infective it can be both simple and malignant in type.

2. It has shown that *ante-mortem* thrombosis may occur in the heart in rheumatic fever without any severe valvular damage. Although this was recognized, it could only be certainly proved by experiment.

3. Accurate knowledge can now be obtained upon the rapidity of the formation of vegetations and upon their actual structure, points which in the future must make our knowledge of the active heart diseases a more accurate one.

4. It throws light upon the formation of infarcts in rheumatic fever, which experiment shows may form without the presence of any visible valvular disease or clotting of blood in the chambers of the heart. The bearing of this fact on the pathology of chorea is manifest.

5. The myocardial changes in rheumatism which were suspected by the clinician and elucidated by the pathologist are absolutely proved by experiment, for they can be produced by this micro-organism.

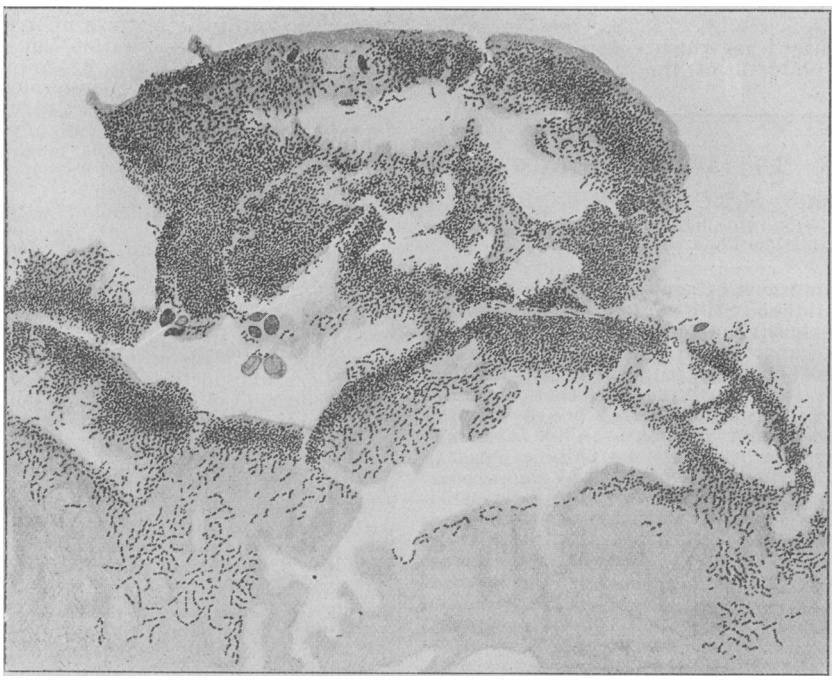
The ability to isolate an infective agent from rheumatic fever brings us one step nearer the proper understanding of the chemical problems of the disease. Triboulet, Walker and Ryffel, Shaw and Berger have already attacked this side of the question, and have shown that the micro-organism can in different media produce such various acids as formic, lactic, and acetic. Walker and Ryffel have demonstiated formic acid in the urine of the rheumatic patient, and have obtained the same acid from the bodies of the bacteria themselves—a very interesting point. The fact that different acids are found in different culture media should, I think, be a warning against any over-hasty assumption that rheumatic fever is due to one special poison, to which salicylate sodium is an antidote.

But not only does this view establish facts, it also throws out suggestions which have also a practical value. Is it not probable, for example, that the bacterium may sometimes

remain latent in the tissues, stunned as it were, but not really killed? Some years ago, with Dr. Paine, I showed that it might be found in the urine of the rabbit, ten weeks after inoculation, and the bearing that this has upon chronic rheumatic arthritis and any other form of chronic and relapsing rheumatic lesion needs no labouring. How suggestive, again, was the occurrence of a relapsing iritis in a rabbit as the result of intravenous inoculations with this bacterium, which was recorded by Dr. V. Shaw in the paper to which I have already alluded. May not this throw light upon that intractable relapsing iritis, which is met with now and again in those who come of a stock in which there is a history of rheumatic fever, rheumatoid arthritis, or gout?

Again, the demonstration of the infective nature of rheumatic fever is a powerful stimulus to a study of the action of bacterial poisons in the pathogeny of gout. It will help to rescue us—as have the writings of Woods Hutchinson and Chalmers Watson—from the slough of despond into which many have fallen, because searching in a swamp of facts for the explanation of gout they have rested all their weight upon some crystals of biurate of sodium.

Between rheumatism and gout there are great differences, but the acute outbreak, the attacks, are in some respects wonderfully similar. There are French writers—for example, Guyot—who hold that gout is a result of the rheumatic infection acting in a special constitution, the gouty. This, as all other views upon gout, lacks proof, but my sympathies are with these French authors thus far, that they put forward a view capable of proof or disproof, and write upon the subject of gout with a freshness of thought which is exhilarating. I hold that the demonstration of the infective origin of rheumatic fever throws open a wide field for thought and suggestion upon the subject of gout. And I would again direct attention to the production of arthritis by certain sera—as, for example, by Menzer's antirheumatic serum and by the the antitetanus serum, as recently reported by Mr. Annan in a case under the care of Dr. Rose Bradford. If any substances free from bacteria are likely to produce the arthritis of gout I hold that, on the evidence at present forthcoming, such substances are more probably of the complex nature of sera than of the simple nature of sodium biurate.



Section through a vegetation on the mitral valve, showing numerous diplococci and chains in the necrotic tissue. (Zeiss objec. $\frac{1}{12}$, Oc. 3.)

Another application of the infective view is the one I hope to show more clearly by the aid of a short lantern demonstration. It is based upon the demonstration of the identity in structure of the cardinal lesions of rheumatic fever. This identity has been long surmised, and now it rests upon good evidence. Endocarditis, pericarditis, arthritis, pleurisy, and nodule formation are lesions of the same type, when due allowance is made for their anatomical surroundings. It is then I think reasonable to suppose that the cardinal deviations from this type will also be comparable. Thus, the chronic smouldering mitral stenosis becomes comparable to chronic indurative pericarditis and chronic periarticular arthritis. Malignant endocarditis is comparable to malignant subacute pericarditis, and to some forms of rheumatoid arthritis. That is to say, our ideas of the life-history of rheumatic fever will be cautiously enlarged, and the shattered fabric of rheumatism can be slowly rebuilt upon foundations which can be tested by accurate methods. The bearing of this upon rheumatoid arthritis is especially close, and may eventually lead to an agreement upon what is really to be understood by that condition.

The question of treatment appeals most forcibly to our profession and to the public. The truth of this I cannot put more tersely than did a doctor who looked at a section of a valve containing the bacterium, and remarked, "What is the good of it? it doesn't help us to treat muscular rheumatism!"

I think that this view *does* help us in treatment, and that our treatment *is* improved. I say nothing to-night of serum-therapy, because that method is in a very critical period of its development. With its discovery we hailed the advent of rational methods, but hasty conclusions and scanty knowledge of its difficulties and imperfections are fast making its use as empirical as that of any crude drug. But our treatment is advanced in two directions; first in its application to the individual; and secondly in its application to the community.

We can now see more clearly the two phases in rheumatic fever—the active, when the infection has the upper hand, and the reactionary, when the infection is being vanquished and the healing processes have commenced. Thus we see more clearly when to bring into action the assistance of hydropathic treatment, radiant heat, electricity, massage, climatic influence, and all those many aids to recovery. Such methods are no fetishes, and it is well to be able to see their functions more plainly. We see more clearly, too, when to use tonics and liberal diet, and what we can promise a patient in the way of cure. These are not little things; it is good for us and our patients that some of the mysteries surrounding the treatment of rheumatism should be cleared away.

Then, again, when surgical measures are considered, the indications are more certain. I hope I am among the last to approve of hasty appeals to surgery in rheumatic arthritis, or to think that the opening of a large joint is a light measure. But when occasion arises the procedure is surely a rational and prope one when we know that there is an infective agent to be dealt with. It is equally clear that, in early rheumatic arthritis, in order to rid the joint of this infective agent by surgical methods it would be necessary to remove the synovial membrane, and that, I feel, is a sufficient answer to any who would counsel such a measure. Surgery in these matters has a very definite limitation, and, as Mr. Howard Marsh stated in his recent Bradshaw Lecture, in many cases only partial success can be expected. Its position, nevertheless, is much strengthened when it is remembered that there are cases of arthritis—and non-suppurative ones—in which literally thousands of micrococci have escaped the barrier of the endothelium and have made their way into the synovial fluid.

It is, however, in its application to the treatment of the community that I think the greatest hope lies.

Rheumatic fever in this country, and especially in London, must be looked upon as one of the most common and most damaging of all diseases. So far as the poorer classes are concerned it is as much to be dreaded as cancer, for it attacks the young, and though tuberculosis is more rapidly fatal to life, I doubt whether even it is a lesser evil. If rheumatic heart disease was not so often looked upon as heart disease, but rather as a mutilation from rheumatism, as phthisis is

looked upon as a mutilation from tuberculosis, the true magnitude of the evil would, I think, strike home more forcibly. We should hear far more of efforts at prevention than of academic discussions upon cardiac *bruits*, and of hopeless attempts at treating scar tissue. It is with this aim in view that I have again brought forward a study of the infective nature of the disease, so clearly foreshadowed in this country by Dr. A. Mantle nearly twenty years ago, yet then so little regarded. The logical step from a practical interpretation of these facts is the prevention of rheumatic fever. It matters not whether, as some hold, there is a specific micro-organism, or as others, that there are several different infective agents, the essential fact remains unaltered. Rheumatic fever is infective—much heart disease results from infection—the first step in treatment must be preventive.

The time, I believe, has come to study once again by the light of this view the predisposing causes of rheumatic fever. To trace once more the influence of climate, of cold and damp, and of sudden changes in temperature. To inquire into the incidence of the disease on those who live in new and jerry-built houses, into the effects of overcrowding and malsanitation. To study epidemic outbreaks and cases of apparent contagion. To investigate the part taken by school life among the poor, with its outbreaks of sore throats and its strain upon the system of the ill-fed and ill-clothed. Surely there is hope from such investigations that the prevention of some of the rheumatic fever which causes such pitiable suffering among the children of the poor will be thus obtained.

ABDOMINAL PAIN IN ACUTE RHEUMATISM.

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AMONGST the minor symptoms of acute rheumatism of frequent occurrence in childhood Still pointed out some years ago[1] that pains in the epigastrium and about the bases of the axillae were of the first importance. Poynton,[2] too, refers to the frequency of "obscure abdominal pains" in the rheumatism of childhood, and gives it as his impression that they "are generally paroxysmal, and usually referred above the umbilicus to the epigastric region." Donkin, in his *Diseases of Childhood* (1893), p. 212, states that "the painful onset of rheumatic attacks is sometimes accompanied by rigors, occasionally by vomiting, and not very rarely, especially when there is cardiac involvement, by marked precordial or epigastric pain." Much might be said about each of these features, but it is to the pain in the upper abdomen that I wish to draw attention now; for having seen a good deal of acute rheumatism recently, especially in children, I have been much struck not only by the frequency of this symptom, but also by some characteristic features not previously noted, so far as I know, which are present in a certain number of cases.

The manifestations of acute rheumatism in childhood are so manifold, and occasionally their advent is so insidious and ambiguous, that it is rather surprising how little some of the "minor symptoms of frequent occurrence" have been studied and emphasized. The epigastric pain is not peculiar amongst these minor symptoms in repaying a more thorough investigation than has hitherto been granted them, but in this paper I shall confine my attention to this symptom alone. Perhaps the frequency of aches and pains about the abdomen in children has interfered with the detailed examination of the "epigastric pain" of rheumatism, and the common occurrence of "pains all over" in many a febrile infection accompanied by pains in the limbs has led to a callousness on the part of clinical observers. In my experience few pay attention to the abdominal symptoms occurring in the acute rheumatism of either children or adults, yet often it is these symptoms that are the important ones in diagnosis and treatment. In children pain is the most frequent of the abdominal symptoms, and it is often of help diagnostically. In its most typical form it conforms to the following description.

SITE AND CHARACTER OF THE PAIN.

The abdominal pain is nearly always situated about the upper half of the abdomen. The frequency with which children are liable to refer all intra-abdominal pain to the umbilicus makes it striking that this pain is seldom referred solely to that spot, for though occasionally it is said to run across the middle line a little above this landmark it is rare for the child to point there only. It usually seems to start from a point a little below the costal margin, close to the mid-clavicular line on either side, and to run from there either towards the umbilicus or straight across the epigastrium. At first I thought it was rather commoner on the left side than on the right, but recently I have seen a number of cases where it has been confined to the right side, and until I have collected a rather larger number of cases I am not prepared to say upon which side it is commoner for it to begin. It often occurs on both sides, and then may be either equally common on both sides, or more frequent on one than the other. Occasionally it is more purely epigastric, and about the middle line.

It is not a superficial pain, but is invariably said to be "inside," yet it does not go through to the back nor travel round the trunk. It is always described as being of a sharp nature, and in many cases that I could relate has been a persistently recurring symptom giving rise to considerable suffering. It is more acute than most of the ordinary stomachaches, but by those children who have experienced both this pain and a stitch in the side it is described as being less severe as a rule than the stitch. It is "quite different" from the pain that may be produced as the result of the administration of aperient drugs, but most children are unable to put into words the features upon which this difference depends. It is of short duration but recurs. Each paroxysm lasts from a minute to four or five minutes, though occasionally its duration is somewhat more prolonged than this. "Running," "crossing the street," and "going to bed," are the three commonest exciting causes of paroxysms. It appears to be brought on, in other words, most frequently by muscular exertion, by excitement, and by other factors the exact nature of which are somewhat obscure.

The pain is not associated with nausea or vomiting, nor with any of the ordinary symptoms of gastric derangement, except accidentally. It is not the result of any irregularity or indiscretion in the diet, nor does it appear to be in any way causally connected with either constipation or diarrhoea. There is no abdominal distension accompanying it, and as far as I can make out it is not associated with any gurglings or ordinary colics of the gut. Loss of appetite is not necessarily, nor usually, an accompaniment of it. The pain comes on independently of food, and the nature of the diet the child is taking does not generally influence its occurrence. It does not as a rule double the patient up, but should it come on during exercise it will most likely cause the patient to keep quite still, and often to sit down also. It does not appear to be associated with soreness of the abdominal walls nor with much tenderness, either superficial or deep. The attacks vary very much as to the frequency of their occurrence. I am inclined to think that a typical case would be one in which six or seven paroxysms occur daily for a few days, then three or four days come with fewer attacks, or perhaps none, followed by another period of increasing frequency culminating at about the end of a total of ten days in an illness that manifests itself as acute rheumatism by the occurrence of some of the well-recognized phenomena of that malady. But it is perhaps futile to talk of a typical case when the variability of the frequency of occurence of these attacks is so marked and when the number of cases illustrating the feature is not yet very large.

This symptom is, I think, commoner in girls than in boys. The cases conforming most accurately to the above description have been in fairly well-nourished girls of 9 to 16. When much younger than this the patients are generally incapable of describing their symptoms with precision, and amongst patients older than this the symptom is not so common. It does occur more or less typically, however, in older children, and probably occasionally in young adults. The phenomena of rheumatism that are likely to be associated with this symptom follow no rule. As far as I can make out, any of the better recognized manifestations of acute rheumatism may occur in association with these paroxysms of abdominal pain, and the latter are not more likely to be associated with cardiac manifestations than with some of the other phenomena, except, of course, in so far as is explained by the great frequency of cardiac affection at this age. The pain does not appear to have any direct connexion with the severity of the rheumatism, either as it affects the heart or as it affects other structures. The peculiar paroxysms moreover appear to be rare in relapses of rheumatic arthritis.