Clinical lectures on rheumatic fever : I. Endocarditis in childhood considered as a symptom of infective diseases. II. Clinical evidences of myocardial damages in rheumatic fever. III. The parallelism between the clinical symptoms and the pathological lesions of rheumatic fever / by F.J. Poynton.

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ENDOCARDITIS IN CHILDHOOD CONSIDERED AS A SYMPTOM OF INFECTIVE DISEASES

BEING LECTURE I OF A SERIES OF CLINICAL LECTURES ON RHEUMATIC FEVER DELIVERED AT THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET, LONDON

BY F. J. POYNTON, M.D., F.R.C.P. (LOND.)

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Ladies and Gentlemen: The subject of this demonstration is perhaps commonplace and the study hardly scientific, when we bear in mind that endocarditis is not in itself a special disease, but a manifestation of several different affections. But I do not intend to approach it in quite the routine way; I wish rather to illustrate this great truth: that endocarditis in childhood is one of the manifestations of infective diseases. By means of some clinical cases I hope to show that this truth is a living one, practical in its application to diseases of the heart, and far-reaching in its power to elucidate their problems. With its realization, the juggling with cardiac murmurs and the hair-splitting details which so often surround them with mystery, assume their true proportions, and in the broader study of the natural history of the diseases which may cause endocarditis, lose their undue prominence.

How various are the causes of endocarditis is clear when we bear in mind that the infections of rheumatic fever and of scarlet fever, the gonococcus, the pneumococcus, the streptococcus pyogenes, and the staphylococci, are all well recognized as causative agents; indeed, there are few infective diseases which at one time or another have not been recorded as causes of endocarditis; though whether as a result of their direct agency or of some secondary processes has not, as yet, been thoroughly demonstrated.

But, you may ask, Cannot endocarditis arise in childhood without an infective agent? May not some poison, produced perhaps within the body itself by a perverted metabolism, give rise to this

condition without the aid of microbic infection? It would be wrong to answer this dogmatically in the negative, for the possibilities of disease appear infinite, and to-day we are the slaves of the master microbe, but this answer can I think be given. No one has shown a proof that perverted metabolism may produce endocarditis, while many have demonstrated the infective nature of the process.

I must not overlook the fact that about fifty years ago the late Sir B. W. Richardson claimed that he had produced endocarditis by injecting a 10 per cent. solution of lactic acid into the peritoneal cavity of the dog, and that he was supported in this by Rauch. Yet, later doubt was thrown upon the truth of the observation by Rayher and Johannes Müller, and, to my knowledge, the observation has never since been confirmed, and if it were, can hardly be compared to any condition that is found in disease.

It would be inopportune to enter further upon these abstruse questions, but it is well to recognize how important it is for us to ascertain whether any poisons, not due to an infective agent, can produce endocarditis. Upon the poisons of gout I would not venture to express an opinion, for in dealing with children, gout in its classical types, can be practically put aside, and it is not, I believe, common in any form. If we allow that endocarditis in childhood is the result of an infective agent, in most if not all cases, then we can proceed to investigate the history of the illness.

First and foremost in the study of this history, rheumatic fever claims our attention; it is the great cause of endocarditis, and the frequency of the occurrence is one of the strongest claims for the specific nature of the disease.

If you turn to the post-mortem records and study fatal rheumatic fever, fatal septicemia, from an infected wound for example, and fatal pneumococcus infections, you will be convinced of this: that endocarditis is almost invariable in rheumatic fever and usually absent in the wound septicemia, and that pneumonia or a suppurative pleurisy are the usual results of the pneumococcus infections. I do not deny for a moment that any one of these diseases may cause endocarditis, but in rheumatic fever only, is it the usual result.

What then is the cause of rheumatic fever? It is not my intention to discuss and argue about this question, but I will remind you that the evidence accumulated from all parts of the medical world points to an infective agent. One infective agent, I believe, to

be the cause of the disease, and that a diplococcus, belonging to the great group of streptococcal organisms.

But when that question is raised—what is the cause of rheumatic fever? The answer must be a far broader one than the mere putting forward of a microbe. Why, for example, do we see in London case after case among the children of the poor? Look through the post-mortem records of a large general hospital and count the deaths from rheumatic heart disease. Recall the number of chronic "heart cases" that occupy the beds in the wards, or haunt the out-patient departments. Then some faint idea is given of the damage that rheumatic fever is doing. It maims and often eventually kills. The loss to the nation must be great indeed from the crippling effects of chronic heart disease alone.

There is the hereditary factor as in tuberculosis, but is it overcrowding or damp, or drain-poisoning or foul dust, or over-exertion, or a combination of all these, which assists the pernicious effect of the micro-organisms?

Among much that is dubious, it appears to be established now that a "sore throat" is often an early symptom of rheumatic fever, and so not infrequently a premonition of endocarditis.

This, the first clinical case, will serve to illustrate that point.

Case I.—*Faucial Angina and Rheumatic Endocarditis.*—This case is that of a girl, aged 10 years, whose mother had suffered from rheumatism. She had been brought in the first place to the hospital in May, 1901, suffering from a sore throat, with enlarged cervical glands. In June there had been epistaxis, and later arthritis and chorea developed. The heart was enlarged and there was a mitral systolic murmur. Under treatment in the hospital she improved, and was sent to Margate. During September, October, and November, the girl remained well; there was no murmur and she became fat but remained very nervous.

In January, 1902, she failed to pay me the usual visit, and in February, when her mother brought her again, there was a distinct change for the worse. I heard this history: In January she developed a severe ulcerated sore throat—the doctor had described it as almost diphtheritic—and she has not been well since. There was now once more a definite and loud systolic mitral murmur, there was arthritis, and the child had wasted. I prescribe a formalin gargle and a course of salicylate of sodium, in addition to rest; and

gradually the patient improved. The systolic murmur now remains constant and there is clear proof of hypertrophy. Doubtless the dilatation of the earlier illness has been followed by an organic lesion, the result of the second infection.

This is a striking example, gentlemen, of the relation of faucial angina to rheumatic endocarditis.

The physician has long recognized this association of a sore throat with rheumatic fever, and I think that all English clinical authorities, experienced in this disease, admit the fact. In 1900, Dr. Paine and I confirmed the truth of the belief by isolating from such a throat a diplococcus, which on intravenous inoculation into rabbits produced endocarditis and arthritis. This diplococcus was identical with one which we had previously isolated from and demonstrated in rheumatic lesions.[1]

Here are drawings that were made from the actual microscopic sections. They are faithful representations. They show the diplococcus in the human tonsil (Fig. 1), in the mitral valve, synovial tissue, rheumatic nodule, and pleural (Fig. 2) and pericardial exudations (Fig. 3). Here again are macroscopic preparations of experimental endocarditis, both simple (Fig. 4) and malignant (Fig. 5);[2] and of human rheumatic endocarditis, simple (Fig. 6) and malignant (Fig. 7). Under the microscope you will see the diplococcus in an inflamed human mitral valve.

There is but little doubt that the micro-organisms gain access to the valves by making their way from the tonsils into the blood stream and then they survive and multiply in those tissues which favor the rheumatic infection.

Three morbid changes that they produce in the cardiac valves are worth remembering: (1) swelling of the subendothelial tissues; (2) cellular proliferation; (3) necrosis of portions of the valve.

Here are drawings which illustrate these changes in the human valve (Figs. 8 and 9), from their early commencement to the formation of the vegetation; and to complete the pathological picture,

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[1] The researches of Meyer, Menzer, and Allara, in 1901–1902, have done much to elucidate this point.

[2] The drawings and photographs of the lesions not illustrated in this paper, have been already published in the Lancet, September, 1900, and May, 1901, and Path. Soc. Trans., 1901 and 1902.

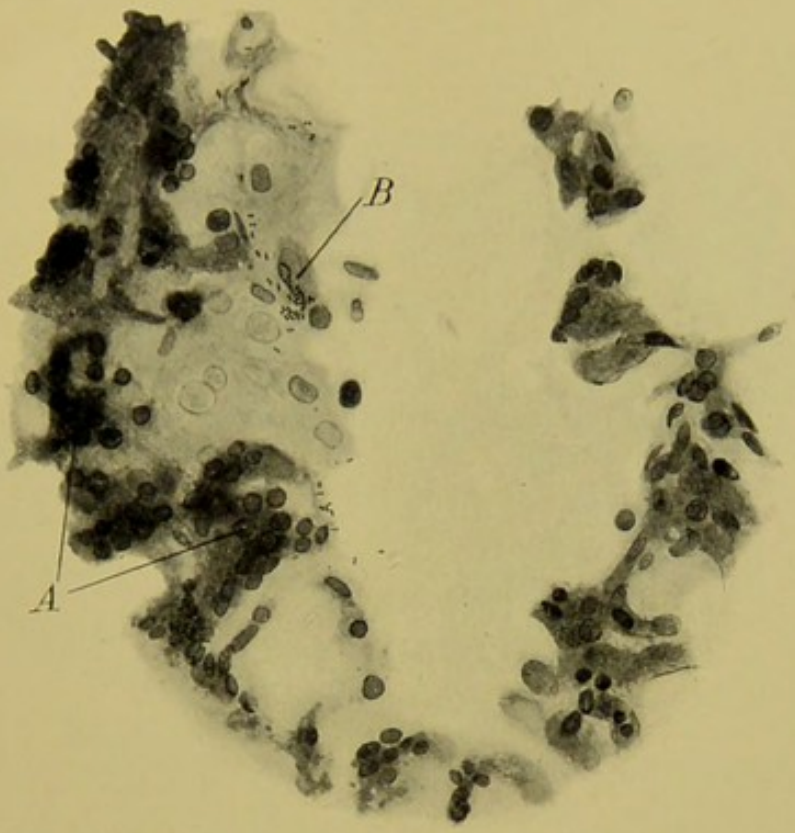


FIG. 1.—A section through a crypt of human tonsil from a case of rheumatic angina, showing diplococci. *A*, Inflammatory cells; *B*, Diplococci.

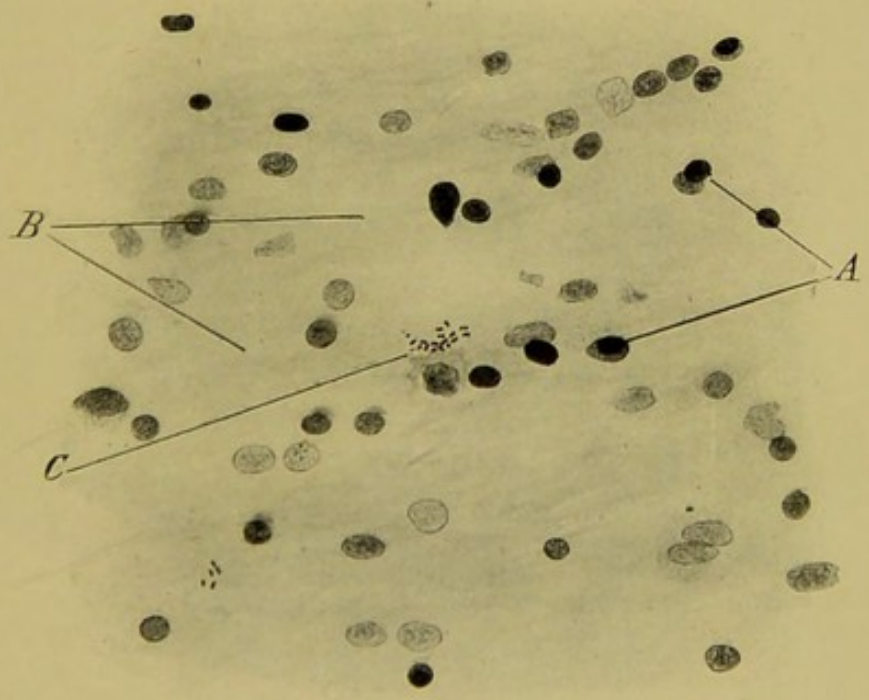


FIG. 2.—A film preparation of the exudation from a case of rheumatic pleurisy. *A*, Leukocytes; *B*, Fibrinous exudation; *C*, Diplococci.

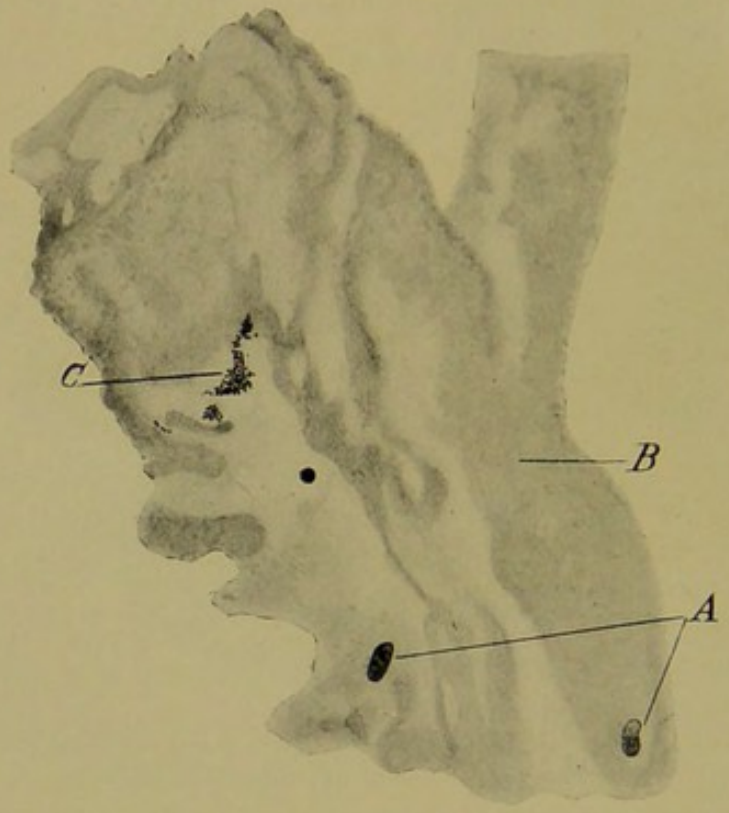


FIG. 3.—A film preparation of the pericardial exudation from a case of rheumatic pericarditis (human). *A*, Leukocytes; *B*, Fibrinous exudation; *C*, Diplococci.

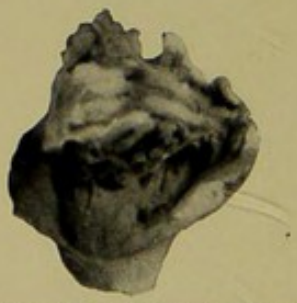


FIG. 4.—Simple endocarditis of the mitral valve experimentally produced in a rabbit.

FIG. 5.—Malignant rheumatic endocarditis of the mitral valve experimentally produced in a rabbit.

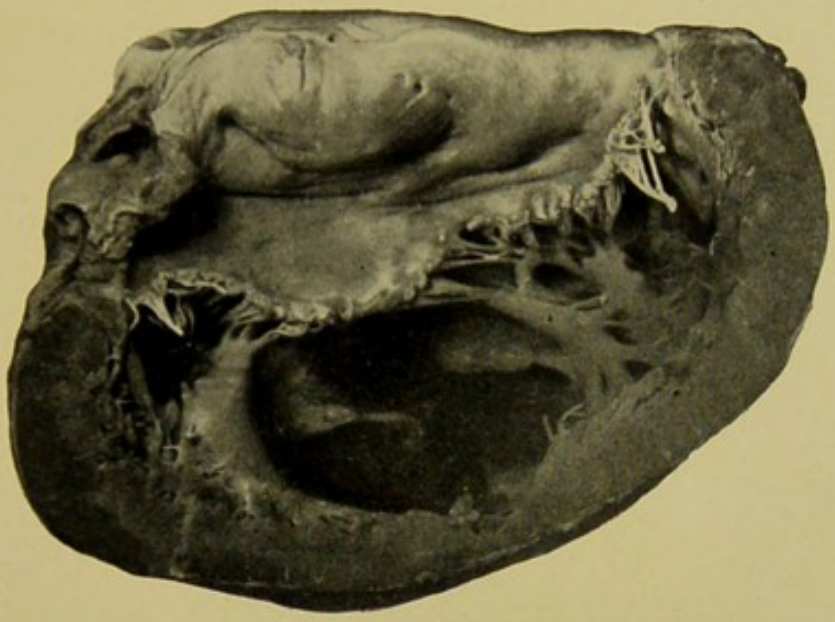


FIG. 6.—Simple rheumatic endocarditis of the mitral valve (human).

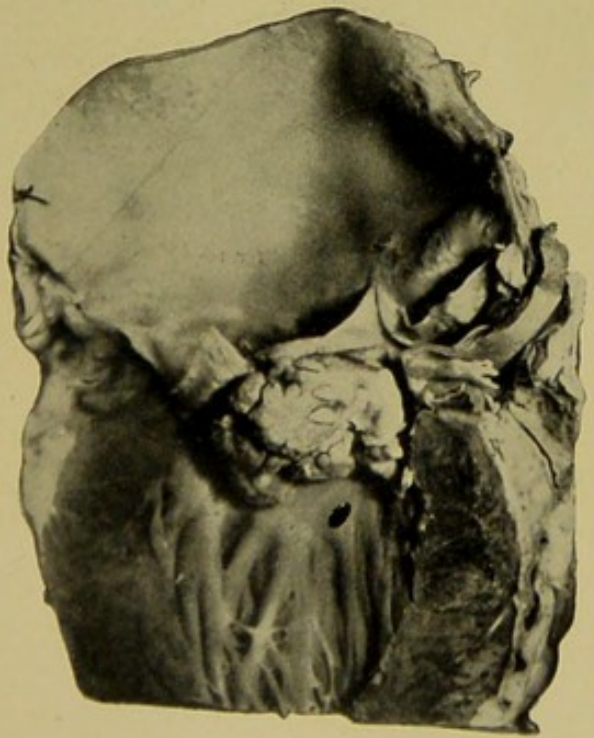


FIG. 7.—Malignant rheumatic endocarditis of the aortic valve (human).

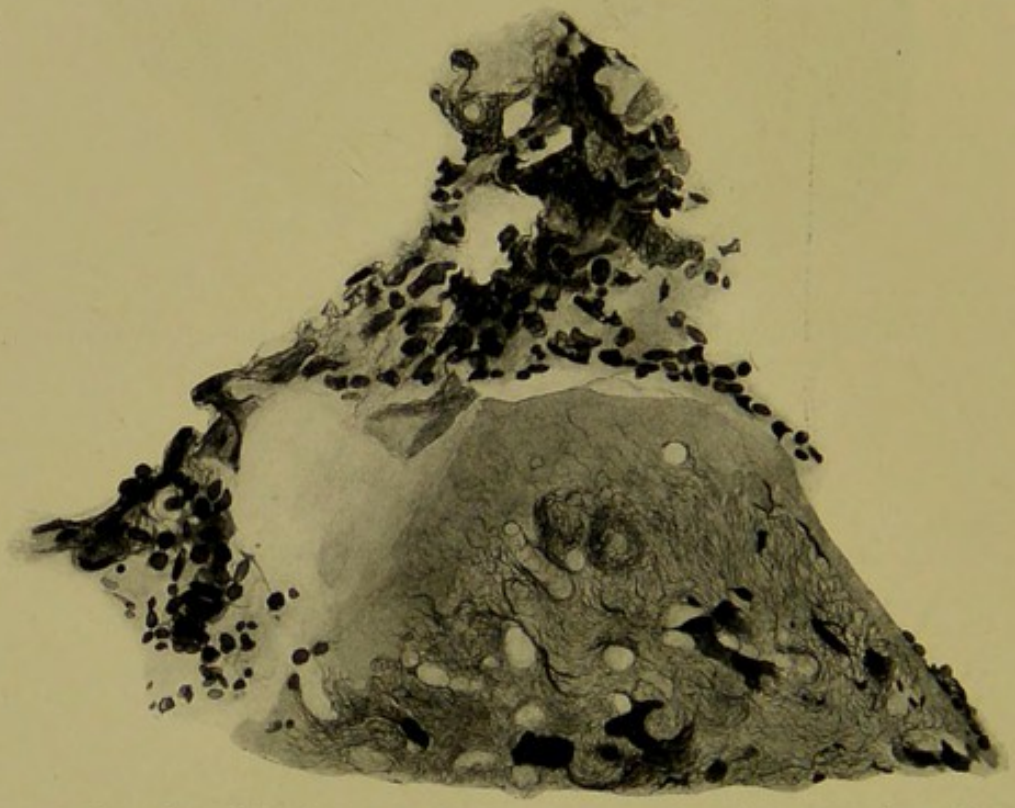


FIG. 8.—Early endocarditis of the mitral valve ; a section through a vegetation. There is in the upper part of the figure free cellular exudation, and among the cells under higher magnification diplococci well shown (human)

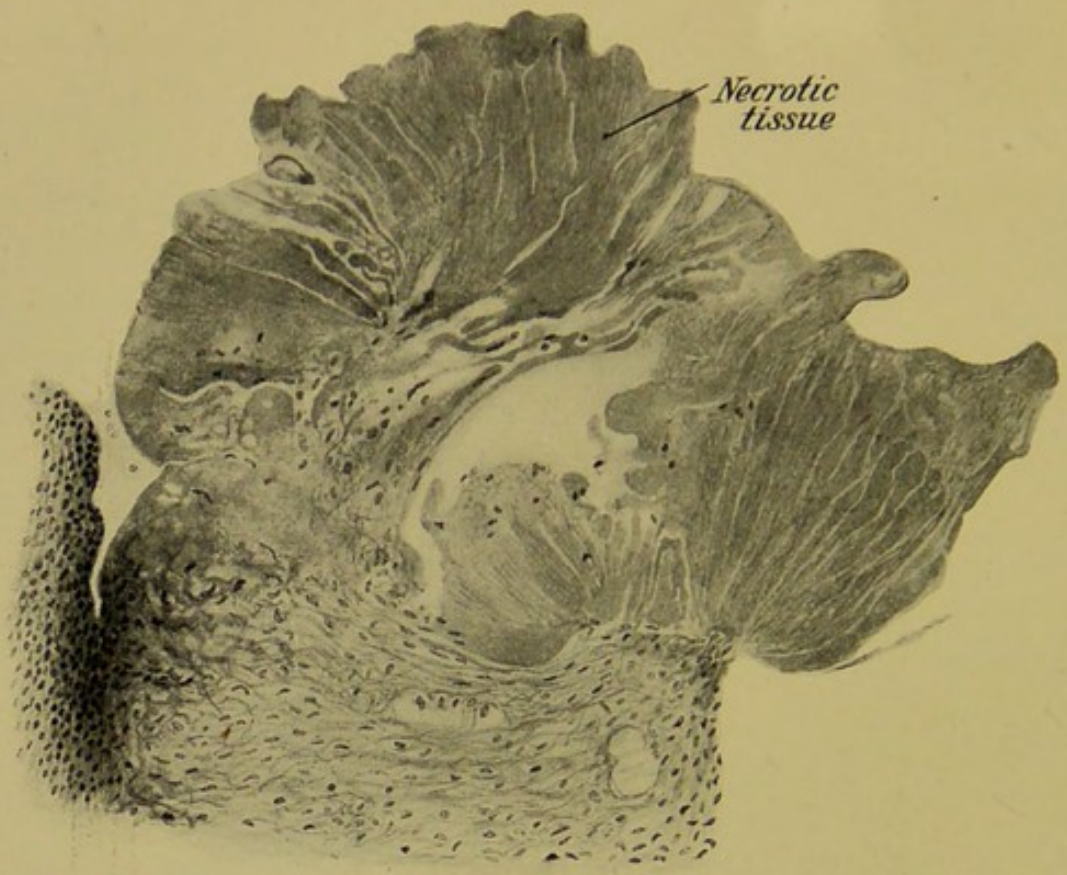


FIG. 9.—A section through a vegetation in simple rheumatic endocarditis. The stage is mor advanced than that in Fig. 8. There is much necrotic tissue, but no diplococci, and at th base reparative changes are commencing (human).

FIG. 10.—A section through a vegetation in malignant rheumatic endocarditis. The necroti tissue, in striking contrast to Fig. 9, contains myriads of diplococci (human).

here again is the macroscopic specimen of rheumatic endocarditis (Fig. 6).

There are two points to which I would direct your especial attention. The first is, that a cap of fibrin is not an essential part of a vegetation; and the second, one of great importance, that the diplococci are, as a rule, rapidly destroyed at the site of the lesion. When in man death occurs, not we know as a rule from acute endocarditis, but from concomitant pericarditis, few, if any, diplococci, will be found in the necrotic part of the vegetation upon the valve.

Contrast that drawing of the simple vegetation (Fig. 9) with this one from a case of malignant endocarditis (Fig. 10), and note the myriads of micrococci in the latter. Contrast also the macroscopic specimens, illustrating the two types of endocarditis, both in man and the animal. (Figs. 4, 5, 6, and 7.)

In childhood more than one valve is often affected. The damage, as Sir Thomas Barlow has observed, may not be great, but more than one valve shares in the process. The order of frequency is first the mitral then the aortic, next the tricuspid and very rarely the pulmonary valve. When two valves are damaged the lesions most frequently combined are those of mitral and aortic regurgitation.

Why is the mitral valve the most frequently damaged? I believe it to be because it is the most complex in structure and contains minute blood vessels, in which the diplococci travel to the tissues of the valve. The aortic comes next I believe, in part, because of its proximity to the mitral; this enables the micro-organisms to spread from the one to the other. I do not believe the difference of the blood in the right and left chambers of the heart has anything to do with the preponderating frequency of the endocarditis upon the left side, because I think that the diplococci gain access to the valves, not from the blood in the chambers of the heart, but from that in the capillaries of the coronary arteries which always contain arterial blood. For the same reason I traverse the sweeping assertion which is sometimes made, that only the left side of the heart is affected by rheumatic endocarditis. Here, indeed, is proof to the contrary. In 1898, I made an analysis of 150 fatal cases of rheumatic fever, and in 149 of these the mitral valve was damaged, in 51 of these the aortic valve was damaged, in 36 of these the tricuspid was damaged. These figures were obtained

from necropsies made by many different pathologists and they are quite free from any bias. It is clear too, that if the infection takes place through the coronary circulation, the right side will be sure to suffer in a certain percentage of cases.

Finally, before we turn to the clinical side of the subject again, it should always be remembered that there are two types of morbid changes in rheumatic fever: those due to the local lesions, which have already been detailed, and those which result from the general toxemia, the exact nature of this toxemia being at present not known.

When the clinical side of the subject is approached, the first question will be: What are the earliest clinical evidences of rheumatic heart disease? The answer is: A little fever, perhaps some palpitation, and pain, possibly even scanty breath on exertion, and a quickened low tension pulse; and these physical signs: (1) an outward displacement and feebleness of the cardiac impulse; (2) an increase in the area of the deep cardiac dulness; (3) a diminution in the length of the first sound, and an accentuation of the pulmonary second sound at the base; and (4) in some instances the development of a soft systolic murmur internal to the left nipple.

The interpretation is this: rheumatic fever causes a dilatation of the heart.

This fact has been repeatedly emphasized, and its importance insisted upon by Dr. D. B. Lees during the last ten years, and I will show you some tracings upon glass, which I made at the time I had the honor of working with him upon this subject.

To-day, it is not possible in the time at our disposal, to put before you the myocardial lesions that occur in rheumatic fever; another demonstration will be necessary. Nor would it be advisable to divert our attention from the subject of to-day—endocarditis. But, this point we must recognize: *that the early clinical evidence of rheumatic heart disease is often myocardial rather than endocardial.*

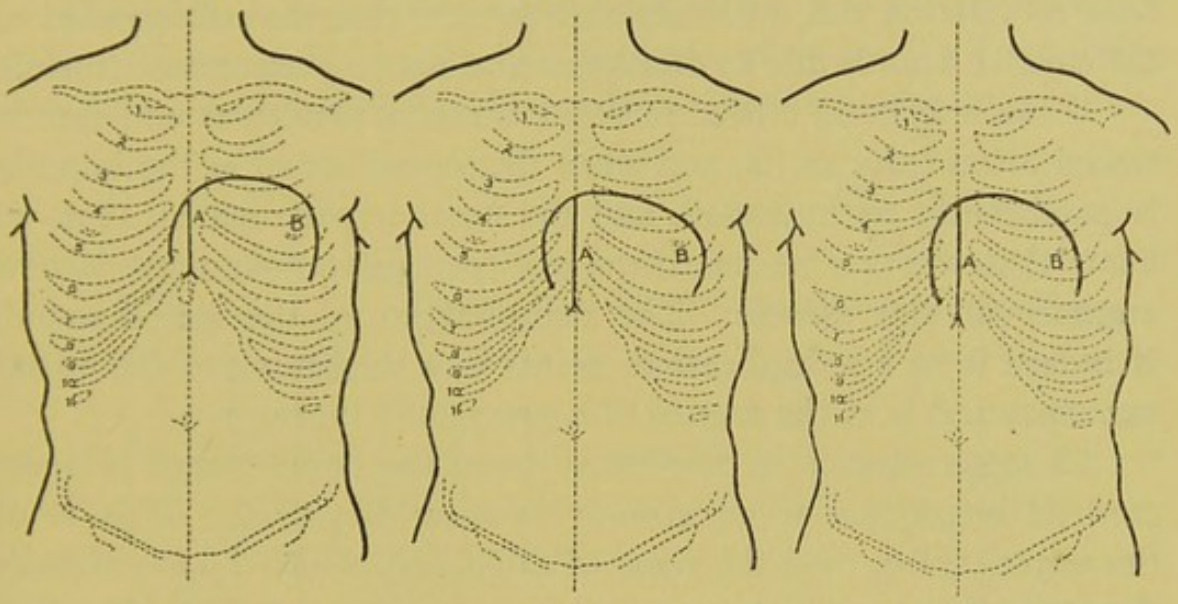
A word now about these tracings: The limits of the deep cardiac dulness were carefully percussed and outlined upon the chest wall, then transferred to tracing paper, reduced to scale and painted upon glass, by my friend Dr. Ransome. Each observation during the attack of rheumatic fever, was made independently of the other,

and each outline is painted in a different color. The phenomenon of dilatation can in this way be illustrated upon a screen.

I cannot show you clinical cases with this condition of dilatation (these glass slides, Figs. 11, 12, and 13, must suffice), for such cases require rest in bed, and cannot be well dwelt with in an out-patient department.

This early dilatation may entirely disappear—not a trace need

FIG. 11. FIG. 12. FIG. 13.



Illustrating variations in the degree of dilatation of the heart. FIG. 11.—The condition on admission—slight dilatation. FIG. 12.—After a relapse of rheumatism—increased dilatation. FIG. 13.—Convalescence—diminution of dilatation. A, The midsternal line; B, the nipple. The patient eventually recovered without a trace of permanent damage.

remain; and so the next clinical question is one of much interest. It asks whether it is possible to distinguish dilatation from early endocarditis?

If by this is meant—can we draw a hard and fast line between the two conditions, I believe not; for, in the first place, a bruit may occur without endocarditis; in the second, endocarditis may occur without a bruit; and lastly, both dilatation and endocarditis are often associated. Although, we cannot be absolutely certain of distinguishing between early dilatation and early endocarditis, we can, by experience, judge pretty accurately of their occurrence. Exceptional cases, as in everything medical, may leave us in doubt; but as a rule, if we are not in too great a hurry, a correct conclusion can be drawn.

When the cardiac area is considerably increased, and yet there is no systolic murmur or only a faint one, which is heard most

clearly internal to the nipple; and when the enlargement of the area of cardiac dulness diminishes and disappears without the appearance or persistence of a murmur, it is certain that there has been cardiac dilatation, and that there has not been, in all probability, endocarditis.

Pericarditis, you will remember, in rheumatism is usually detected by the friction rub. If the area of cardiac dulness is considerably increased, but the cardiac sounds are clear, and there has been no friction rub, you may rest assured that the enlargement of the cardiac area is due to dilatation and not to pericardial effusion.

When a systolic mitral murmur remains persistent and can be traced toward the axilla, and when the second sound at the impulse is persistently reduplicated, and the heart hypertrophied, then we know that whether or not there has been dilatation, there is now organic valvular disease. The reduplication of the second sound, if indeed it be a reduplication, points to a thickening of the mitral segments and is of the nature of a presystolic murmur.

In some cases it is possible to trace the development of endocarditis subsequent to an already existing dilatation. If it be of the mitral valve, then the signs of dilatation, which I have already detailed, being present, you notice one day an abrupt alteration in the character of the first sound, which becomes indefinite and hard to detect. And then later, a sharp systolic whiff traceable toward the axilla is heard. If this whiffing murmur seems far from your stethoscope at a deeper level than the cardiac sounds themselves, the mitral valve is, I think, inflamed.

On two occasions, within a space of 48 hours, I have traced the early development of aortic endocarditis. The stages are these: first of all the aortic second sound becomes faint—you have as it were to search for it and to ask for silence in the room; then you hear a soft diastolic blow and you know that the lesion of aortic regurgitation has resulted. It is astonishing in childhood how rapidly the character of the collapsing pulse will appear after the aortic valve shows signs of incompetency.

Case II.—*To Illustrate Mitral Endocarditis.*—This child, H. G., aged 11 years, has been wasting for three months—a very common history with rheumatic children. Two years ago there was an indefinite history of pains in the legs and sweating.

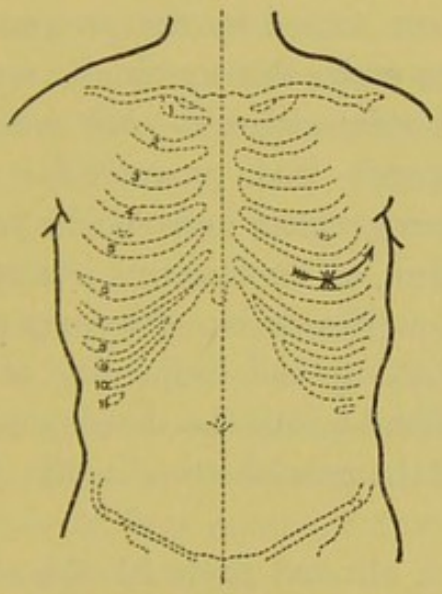
You will notice that the systolic mitral murmur is extraor-

dinarily loud and following the murmur is the curious reduplicated sound which, apart from the bruit, convinces me that the lesion is organic. The hypertrophy is very definite. When listening to such a murmur it is well to recall that the loudness is no index of the severity of the lesion.

The figure shows the position of the early presystolic murmur (Fig. 14) and the direction of conduction of the mitral systolic murmur.

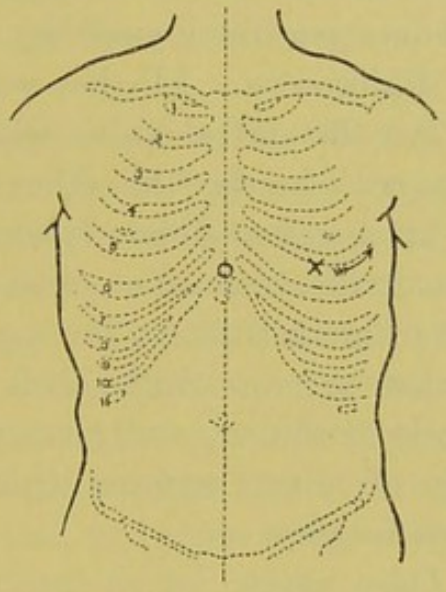
Case III.—*To Illustrate combined Mitral and Aortic Endocarditis.*—This boy, W. A., aged 10 years, was first seen in July, 1901, and had already suffered from three previous attacks of rheu-

Fig. 14.



* Mitral presystolic murmur. ⟶ Mitral systolic murmur.

Fig. 15.



o Aortic diastolic murmur. × ⟶ Mitral systolic murmur.

matic arthritis. He came to the hospital the picture of a severe case of aortic regurgitation, pale, breathless, and nervous, with the vessels throbbing visibly at the temples, with capillary pulsation, and a pulse which, on raising the arm, collapsed to such a degree as to be almost imperceptible. The temperature was 100° F.

He remained in the hospital for seven weeks. Since then I have never lost sight of him, and I managed to guide him through last winter and all this year—aided by a most sensible mother—without another attack. At present he is better than he has been for some years, but the damage is irreparable.

Fig. 15 shows the position at which the diastolic aortic and systolic mitral murmurs are most clearly heard.

We have now gradually drifted from the acute endocardial lesions to the chronic ones, and those who have studied the drawings of early endocarditis, will easily understand how those deformities are caused which form the basis of the study of chronic valvular disease of the heart.

Let me once more show you the rheumatic vegetation in the necrotic phase (Fig. 9). You will see that the base is formed by the fibrous tissue of the valve, infiltrated with living and active cells. Step by step this necrotic area is invaded by fibroblasts, and gradually this young imperfect connective tissue contracts and distorts the segments of the valve.

This photograph [1] shows you the early change in a chorda tendinea of the mitral valve of a rabbit. The swelling upon it is a connective tissue swelling, and it was caused by the presence of the diplococcus. All that was done to make this specimen, was to remove the mitral valve with two chordæ attached—one was the damaged chorda, the other was natural—and to spread it out flat upon a glass slide. It was then photographed under a low power of magnification. You must all, I think, allow that this is a specimen of much interest, for it gives you a glimpse into the early stage of that process which leads to shortening and deformity of the chordæ tendineæ, and gives you an insight into the developmental stage of mitral stenosis which cannot be gained even in the post-mortem room.

Once again, let us interpret some clinical facts by the study of these processes of healing.

It is not very unusual to find that after some months a systolic mitral murmur may disappear, and it is quite conceivable that the repair of a simple endocarditis may be all but perfect. You do not marvel over the complete disappearance of a rheumatic nodule, and the two lesions are closely analogous.

Not only may a mitral murmur disappear, but an aortic murmur also.

But there is another reason for the disappearance of a murmur in acute rheumatism, a more usual one, which is this: there has been dilatation of the heart and the murmur has been produced by relative incompetence. When the dilatation subsides, the murmur disappears.

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[1] Reproduced in the Lancet, September, 1900.

This phenomenon of the disappearance of a murmur has a very special importance in the estimation of the value of methods which are employed for the treatment of acute endocarditis. It is a clinical fact which authorities upon the subject of heart disease have long pointed out, and it is not a rare occurrence. Yet, from time to time, it comes as a revelation to some one who is studying the treatment of heart disease. He naturally ascribes the result to the treatment and the treatment is perhaps magnified by others into "a cure." But it is "kindly nature" probably who should receive that thanks, for all treatments which include rest will meet with the same result in a certain percentage of cases, and no drug is certainly known to influence acute endocarditis. I have under my care at present two examples of this disappearance of the murmur. The one was treated with an alkaline solution of quinin, and the other with anti-streptococcus serum.

Case IV.—*To Illustrate the Disappearance of Cardiac Murmurs.*—This boy, aged 6½ years, came here first in February, 1902. There is a family history of rheumatic fever, and before this illness the patient had suffered from vague pains in the limbs. In December he developed a sort throat; then followed inflammation of the mitral valve, hemichorea, and arthritis. He was admitted to the hospital and whilst there the aortic valve was attacked.

In May he came to see me again with another attack of rheumatic fever, was readmitted to the hospital, and there suffered from a severe pericarditis.

Now, to-day, though he was so very ill in June, you will be surprised when you examine his heart. There is a good impulse. I cannot hear the aortic regurgitant murmur—which had been audible for some months—and only with difficulty can I detect any mitral bruit.

The converse to the disappearance of a murmur is also met with and may be a cause of disappointment and sometimes, without due reason, of discredit to the medical attendant. I mean that a cardiac murmur which was absent during the acute attack, may make its appearance some months later. Now the causation of cardiac bruits is not at all easy to understand, in spite of the multitude of writings about them. Consider for a moment these two specimens, from a recent investigation by Dr. Paine and myself. In this specimen, you see a large vegetation upon the mitral valve (Fig. 5), yet

during life no murmur was audible over the rabbit's chest. But with this other one, during life, there were loud systolic murmurs, which led us to predict and rightly, that both the aortic and mitral valves were affected. You see exuberant vegetations upon both of them. If such gross lesions produce bruits in one case and not in another, it does not seem to me difficult to realize that a deformity in a cardiac valve, produced by the process of healing, may be the cause of the appearance of a bruit which was absent during the acute phase.

It is impossible now to make more than a few discursive remarks upon chronic heart disease. You have already seen some types in the early stages, and in conclusion, let me show you a case of mitral stenosis and a case of aortic stenosis (Cases V and VI). Mitral stenosis of an advanced type is not common in childhood, but the condition is one of the very greatest interest in the scientific study of rheumatic fever. This question will make that clear: Is stenosis the result of the healing of a single severe attack of acute endocarditis, or is it the result of a persistent smouldering infection? In the answer to this I feel convinced there lies hidden much that we need to know about rheumatism. It was Dr. Stephen Mackenzie, I believe, who pointed out that sometimes before the characteristic bruit of mitral stenosis appears, there may be a complete absence of murmurs, that is to say, a systolic murmur previously present, disappears, and then after some time has elapsed a presystolic murmur is heard.

Case V.—*To Illustrate Mitral Stenosis.*—This little girl, aged 10 years, exemplifies very distinctly the condition of pure mitral stenosis. There has never been a definite history of rheumatic fever, nothing more than occasional sore throat and some aching pains in the knees and elbows. For three years at least the heart has been damaged.

The presystolic thrill, the rumbling presystolic murmur, the disappearance of the second sound at the impulse, the loud banging pulmonary second sound, all point to an advanced mitral stenosis with little, if any, regurgitation.

It is remarkable how latent the rheumatic manifestations are apt to be in these cases of mitral stenosis, and the well-known frequency with which mitral stenosis is met with in females as compared with males, is also very curious. The child, you will per-

ceive, is stunted, for the output of blood from the left ventricle is small.

Case VI.—*To Illustrate the Bruit of Aortic Stenosis.*—This little girl, C. E., aged 8 years, is an interesting case. There is a doubtful family history of rheumatism, but no personal history. The only illnesses the child has suffered from have been mild attacks of scarlet fever and whooping-cough. All the evidence that I have been able to obtain points against a congenital lesion of the heart. I show her to-day as illustrating the bruit of aortic stenosis.

In the second intercostal space on the right side close to the sternum (Fig. 16), you hear a loud systolic murmur, harsh in character, traced very definitely into the large vessels at the base of the

Fig. 16.

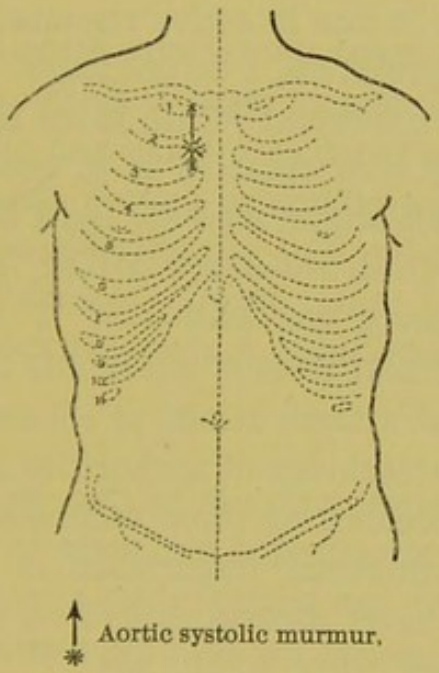
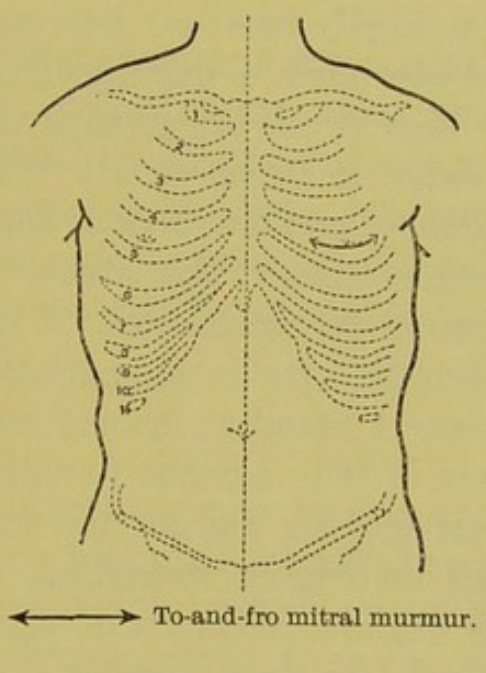


Fig. 17.



neck, and becoming less audible when traced toward the impulse. There is a systolic thrill over the upper part of the chest. The child is stunted and fragile and suffers from attacks of dyspnea and cardiac pain.

An error that is sometimes made by those who are not well acquainted with endocarditis in childhood, is to mistake a to-and-fro murmur heard over the impulse for an indication of disease of the aortic valve. Such a murmur in the adult is as a rule aortic in significance, but in the child, it points to mitral damage.

Case VII.—*To Illustrate the To-and-Fro Mitral Murmur.*—This boy, A. B., aged 11 years, has suffered from rheumatic fever off and on since he was five. When I first saw him there

was dilatation and hypertrophy of the heart, a subcutaneous nodule on the right olecranon, and a stiff neck. The to-and-fro mitral murmur is very plainly heard immediately around the impulse. It indicates a mitral lesion, the aortic valve being intact (Fig. 17).

Edema is not so common in the child as in the adult, but edema of the face is, I fancy, more common. Partly, I believe, because rheumatism in its severe visceral types damages the kidney, and partly because there is a liability to venous thrombosis of the large cervical veins.

It is remarkable how little distress even grave heart disease may cause a child, but the great test of healthy childhood, the power to rush about, in season and out of season, usually tells a pathetic tale. Children afflicted with morbus cordis are easily terrified, and often highly nervous; how much this is due to active rheumatism, and how much to impaired cerebral circulation, I do not know.

Last of all, but perhaps the most important of all, there is the question to be answered: Why, since compensation is so good in childhood and the valvular damage often slight, does the heart break down so often? The answer is that fresh rheumatic attack is the cause of the breakdown. I do not mean that overstrain is never responsible, but in an analysis of 100 fatal cases of rheumatic heart disease I found undoubted evidence of a fresh attack of rheumatic fever in 86! I doubt, in spite of the teachings of authorities upon heart disease, whether the true significance of those numbers has been thoroughly recognized. Their immense importance in considering the prognosis of rheumatic heart disease cannot be overlooked. If those numbers could speak, they would say: "Until you know more about the determining causes and the life history of rheumatism, and more about the infective agent in rheumatism, you cannot forecast rheumatic heart disease in childhood." Who can say that a case of slight mitral endocarditis of to-day may not appear next spring as a hopeless case of general pericarditis?

This concludes the demonstration, and if I have succeeded in leading any one present to-day to a clearer conception of the infective nature of endocarditis in childhood, I shall feel that the time has been well spent. My sincere thanks are due to Dr. F. G. Penrose and Dr. A. E. Garrod, for allowing me to make mention of those cases which while in the hospital were under their care.

CLINICAL EVIDENCES OF MYOCARDIAL DAMAGE IN RHEUMATIC FEVER

BEING LECTURE II OF A SERIES OF CLINICAL LECTURES ON RHEUMATIC FEVER DELIVERED AT THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET, LONDON

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Ladies and Gentlemen: This demonstration is the second of three which I hope to give upon the subject of rheumatic fever. In the first one I considered endocarditis as a symptom of infective diseases, dealing especially with rheumatic endocarditis. To-day I am considering the part taken by the myocardium in rheumatic heart disease. In the future I hope to deal with the clinical analogy between the various rheumatic lesions.

I

The aim of the first demonstration was to show by means of clinical cases and experimental evidence how clear a conception of rheumatic endocarditis can be obtained by the acceptance of the infective nature of rheumatic fever. We can trace the entire process in our thoughts, picture it in our minds, and talk of it in plain and simple language. There are, I admit, some difficult questions connected with malignant endocarditis, but even these become less obscure when rheumatic fever is thus studied. To-day I have a more difficult task, but the subject is so important that if I only succeed in making clear the reasoning upon which its study is based, I shall have given you some assistance.

We have to consider the damage to the cardiac wall that may result from one or repeated attacks of rheumatic fever. As in my former lecture upon endocarditis, I shall endeavor to deal with this question clinically and to apply it practically, and although I must approach it from theoretical and pathological stand-points, my hope

is to leave it in such a position that its clinical importance will be manifest.

If we consider the human heart, quite apart from the lessons of disease, we cannot but think that its healthy function depends more upon the soundness of the cardiac muscle, than upon the integrity of the valves and pericardium. A study of heart disease strengthens us in this opinion, for although the valves may be much damaged by disease, we repeatedly see the cardiac wall come to the rescue, and although the pericardium may have become functionless from general adhesion, yet may life be preserved for years, and death eventually result from a totally different cause.

It may then seem strange that only of late has it been thoroughly recognized how important must be the condition of the cardiac wall in the very disease which above all damages the heart—rheumatic fever. We have heard a vast deal about endocarditis and pericarditis, but in proportion very little about the myocardium in rheumatic fever.

But the reason is not far to seek, and when it is recognized how much dependence is placed upon the stethoscope in the clinical detection of heart disease it becomes apparent. As a general rule the diagnosis of endocarditis without the evidence of a cardiac bruit is insecure, although there are, no doubt, some cases of advanced valvular disease, as, for example, advanced aortic regurgitation, which may be detected without a stethoscope and with absolute certainty. So, again, with pericarditis. In the absence of any pericardial friction sound it needs all the skill of the clinician to diagnose pericarditis. The stethoscope, then, is of immense value, but its very usefulness has brought with it this drawback,—a tendency to follow a process of false reasoning, which argues, unconsciously, I admit, that when there is no unusual sound heard with the stethoscope there is nothing much the matter with the heart.

Disease of the myocardium gives rise to no characteristic bruit, and often enough we are obliged to judge of its existence from the symptoms rather than by the physical signs. This is true when there is damage to the myocardium alone, but when, as in rheumatic fever, we have often to deal with a concurrent pericarditis or endocarditis, you can quite well understand how difficult it is to get any clear notion of the part taken by the cardiac wall. It is then this absence of definite physical signs which has made

the detection of myocardial disease so difficult, and the bruits and pericardial friction sounds of rheumatic heart disease have added to this difficulty by diverting attention from it.

But do not let me given you the impression that damage to the myocardium in rheumatic fever has been altogether overlooked at the bedside. That would be a false impression. About 1870 one of the greatest of our English physicians, Sir William Gull, was in the habit of pointing out, in the wards at Guy's Hospital, that acute rheumatic pericarditis was liable to give rise to a rapid dilatation of the heart which was mistaken for pericardial effusion. His opinion was quoted by Dr. James Goodhart, in 1879, when he showed at the Pathological Society of London the heart of a youth, aged 17, who had died from rheumatic pericarditis, with endocarditis. The heart was dilated and there was no pericardial effusion, but only a soft fibrino-cellular coating of exudation. In 1882 Dr. Samuel West showed the same condition of dilatation and demonstrated fatty degeneration of the muscle. Again, in Dr. A. E. Sansom's writings, we find allusions to the swollen heart of rheumatic fever. There are other scattered observations to be found, but I believe I am correct in saying that the real importance of these observations was not realized, and, indeed, they were, generally speaking, unknown.

It is to Dr. D. B. Lees that we chiefly owe the establishment of the clinical value of this condition of dilatation in rheumatic fever, and for ten years he has been upholding its importance with unshaken faith. In 1896 Dr. John Broadbent, in a monograph on adherent pericardium, from clinical observations at the hospital, brought forward important evidence in support of this view. In 1897 I had the honor of working with Dr. Lees upon the subject, and in two papers read before the Royal Medico-Chirurgical Society, in 1898, we were able to put the matter beyond the stage of controversy; while Dr. Theodore Fisher, of Clifton, arrived at the same goal independently.

To state our conclusions with the greatest brevity, we held that dilatation might occur independently of endocarditis or pericarditis, that it might be the first sign of rheumatic heart disease, and that when it occurred with pericarditis and endocarditis it was not entirely a result of those inflammations, but a result of the rheumatic poisoning. In those days we did not recognize the microbic excitant of rheumatic fever.

So much for the clinical history of the condition. But are there any pathological changes in the myocardium to explain these clinical observations of dilatation of the heart?

It has long been known that in cases of rheumatic pericarditis fatty changes are found in the muscle under the inflamed membrane. There have been also scattered observations upon more diffuse myocardial changes, but they are scanty, and in most cases these changes have been interpreted as secondary to endocarditis or pericarditis, and no especial attention has been directed to them.

At this point let me ask you to divert your attention for a short time from rheumatic fever to diphtheria, for the latter will assist us considerably in the study of the pathology of the myocardium. Diphtheria, we know, only too well, often damages the heart; not, as a rule, by causing endocarditis or pericarditis, but by damaging the nerve endings and muscle. In this disease we can then get an insight into myocardial affections unaccompanied by endocarditis or pericarditis.

After an attack of diphtheria death may sometimes occur with dreadful suddenness, or there may be a more gradual cardiac failure —part of a paralysis more or less general. In passing I may say that in this hospital it is remarkable how rarely we find patients who die suddenly without some warning or other, and there is almost invariably a certain amount of cardiac dilatation.

Now, diphtheria is an infective disease, just as is rheumatism, though differing in its contagious character, and in the distribution of the lesions. It is, then, of especial interest to us to know what the pathological changes are in the cardiac muscle, and the light thrown upon diseases of the heart wall by those who have investigated the myocardium in diphtheria has, I think, been very great, and their investigations most suggestive.

This drawing (Fig. 1), which was made from a section of the heart wall of a case of fatal diphtheria that I investigated in 1899, illustrates some of the changes which result and which have been for many years well recognized. The fatty changes, the nuclear changes, the alteration in the shape and size of the muscle fibers can all be seen in this drawing.

From it, I think, you will get some notion of the action of a bacterial toxin on the cardiac wall, and be prepared to hear of the changes that take place in rheumatic fever, which is also an in-

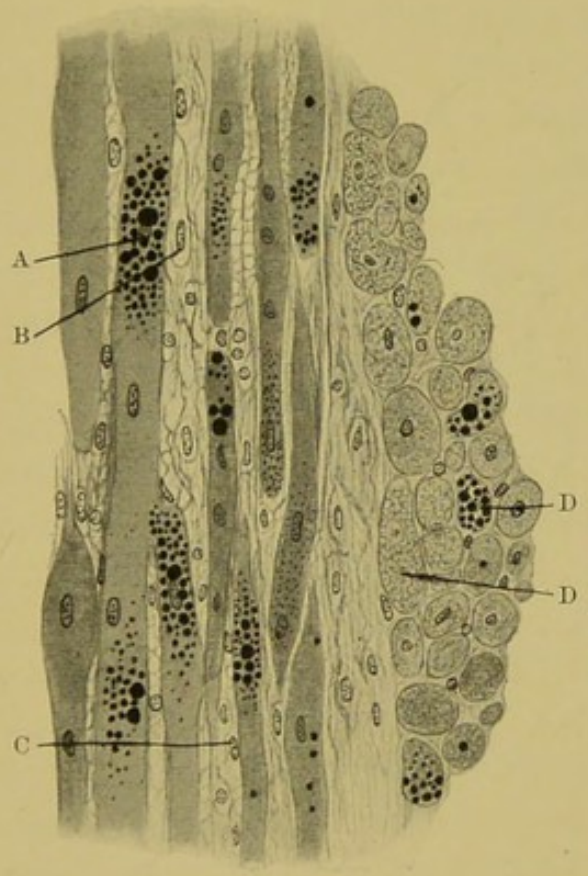

FIG. 1.—A section through the left ventricle of the heart, from a case of diphtheritic paralysis. A, fatty change; B, a muscle fiber almost destroyed; C, a muscle fiber destroyed all but the nucleus; D, damaged muscle in transverse section. (Osmic acid preparation.)

fective disease that frequently damages the heart and often causes dilatation.

In 1895 Neill and Barjon [1] gave a detailed account of the myocardium in a case of rheumatic myocarditis. In 1898 I brought before the Royal Medico-Chirurgical Society a paper upon the subject of rheumatic damage to the cardiac wall which was based upon a study of 4 cases, and in 1900 I had investigated 18 cases of acute rheumatic carditis, and Dr. Fisher, of Clifton, had investigated cases of chronic rheumatic carditis.

Later in 1900 Dr. Paine and myself were enabled to add a further link by the production of myocardial changes of a fatty nature in the hearts of rabbits injected with a diplococcus which we believed to be a cause of the disease. These pathological observations can now be put side by side with the clinical ones.

I will give only a very brief description of them and under two headings: (1) those affecting the cardiac muscle, and (2) those affecting the blood-vessels and interstitial tissues of the heart wall.

Under the changes affecting the heart muscle come (*a*) fatty changes in the muscular fibers, not only close under the pericardium, but also scattered throughout the heart wall, more especially in the neighborhood of the minute blood-vessels (Fig. 2). These changes may be slight or severe, but, in my experience, there is seldom the destruction of the muscle fibers which is found in diphtheria. (*b*) Loss of striation, exaggeration of striation, and segmentation. And (*c*) nuclear change—division of nuclei, hyperchromatosis, and possibly a granular change spreading from the poles of the nuclei.

Among the second series of changes those affecting the blood-vessels and interstitial tissue are found: (*a*) Cellular exudation around the blood capillaries (Fig. 3). (*b*) Exudation into and swelling of the interstitial tissues—which when extreme give rise to the appearance of an intramural nodule. (*c*) In the chronic cases perivascular fibrosis (Fig. 4) and the occurrence of new-formed strands of connective tissue running in the intermuscular septa, and replacing in part the muscular tissue (Fig. 5).

The papillary muscles of the mitral valve, upon which depends in great part the effective action of the valve, are often seriously damaged.

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[1] Arch. de Méd. Expér., 1895.

I have arranged these pathological changes in the cardiac wall in this way for a special reason. I am anxious for you to rid yourself of the word myocarditis. Inflammation is only one process of disease, and that probably a reparative one dependent on the presence of blood-channels. In rheumatic fever, as in other infective diseases, there are at least two processes: (1) those the result of the toxins damaging, for example, the muscle, not by inflammation, but by a poisonous action, and (2) the inflammatory changes taking place in the regions of the minute blood-vessels.

In some cases the deleterious effects of the toxins are more marked than in others, and there are on the other hand, I am sure, some cases in which, after repeated attacks of rheumatic fever, the muscle remains sound. To speak, then, of rheumatic affections of the myocardium as a rheumatic myocarditis is to take too narrow a view of the question. Nor, for my part, have I any belief in the inflammatory changes spreading into the heart wall from the valve rings or pericardium. I think there is in rheumatic fever a blood infection of the entire heart through the coronary circulation, and if the heart wall suffers it is a direct result of this infection, as are endocarditis and pericarditis.

The drawings illustrate:

Fig. 2. The fatty changes in the muscle.

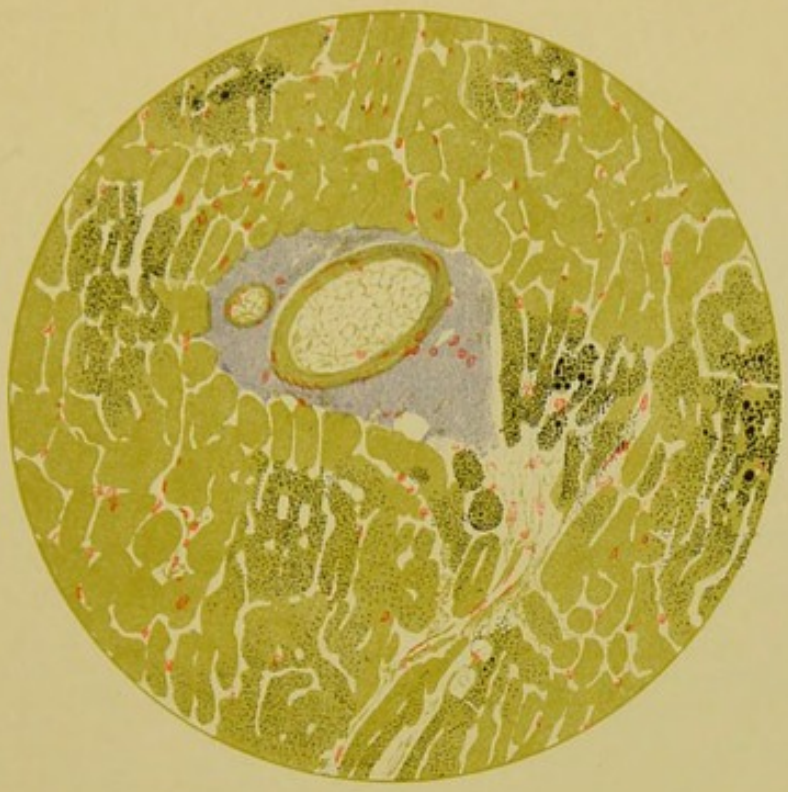
Fig. 3. The cellular exudation from a blood capillary.

Fig. 4. Interstitial cellular exudation and fibrosis.

Fig. 5. Perivascular fibrosis.

This macroscopic specimen, preserved by the Kaiserling method, shows what you may have seen yourselves in the post-mortem room —the pallor of the cardiac muscle in a case of severe rheumatic carditis. And this macroscopic specimen is a most striking example of rheumatic dilatation. The patient died in a first attack from progressive heart failure. There was no pericarditis, and though there was endocarditis of the mitral valve, there was also great myocardial damage.

It is most unfortunate that the right ventricle has been removed in order to get the specimen into the jar, for when I came to examine the wall of this ventricle, I found upon the outer surface two projections which I can only compare to the blister-like excrescences upon a Higginson's syringe which has been overstrained. At those two spots there was practically no muscle at all, only the visceral



IG. 2.—A section through the left ventricle of a case of rheumatic carditis to show fatty change in the muscular fibres in the neighborhood of a capillary blood-vessel.

FIG. 3.—A section through the left ventricle of a case of rheumatic carditis to show cellular exudation in the interstitial tissues. A, cellular exudation.

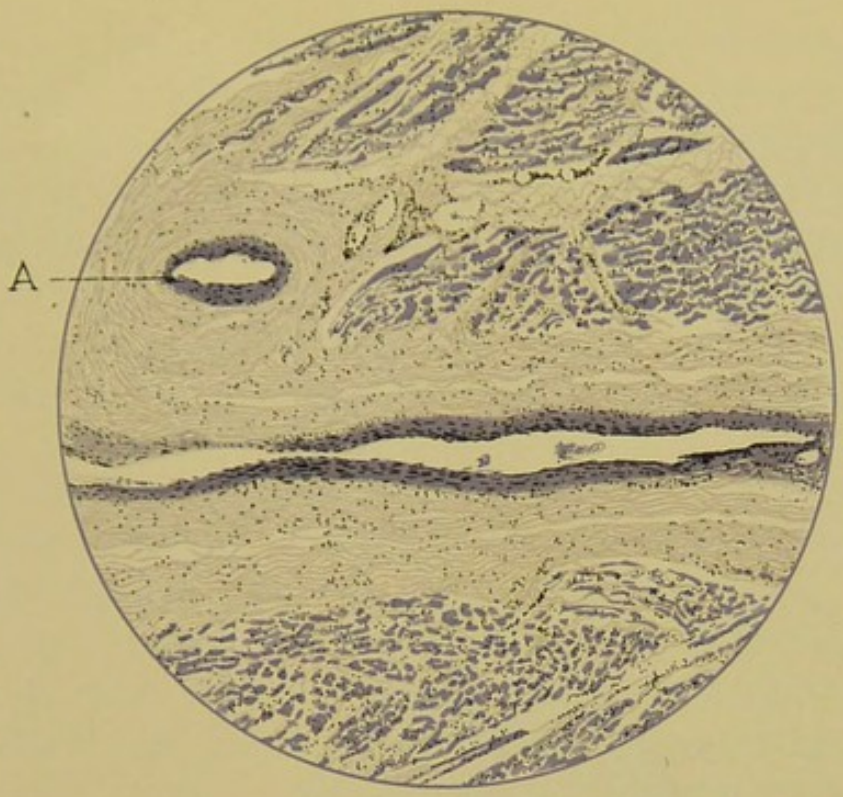


FIG. 4.—A section through the left ventricle of a case of chronic rheumatic carditis. A, a blood-vessel with much perivascular fibrosis.

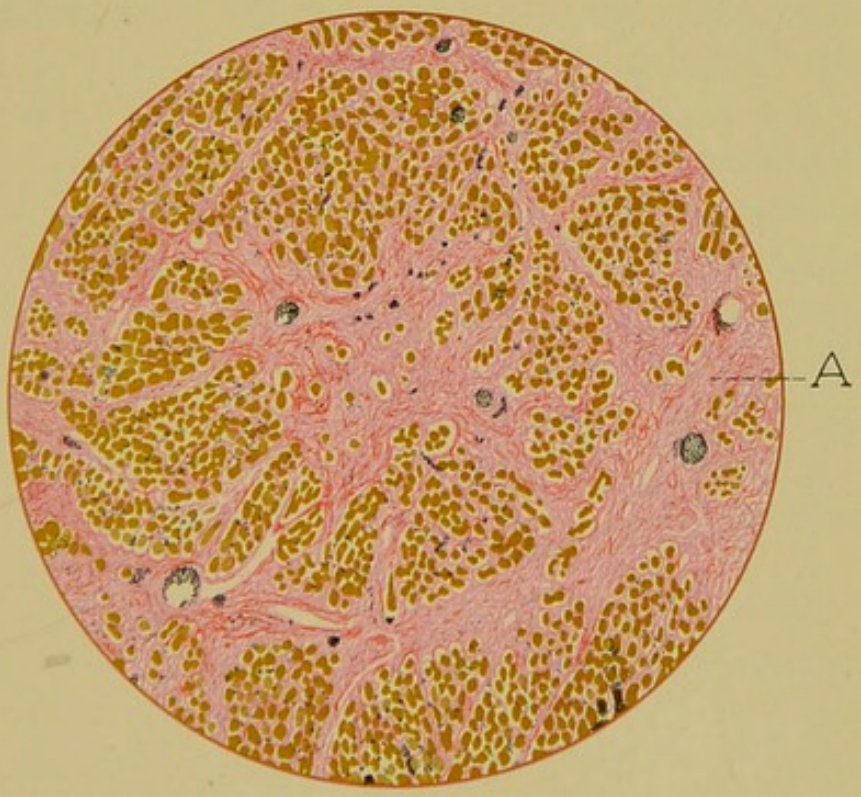


FIG. 5. A section through the left ventricle of a case of chronic rheumatic carditis showing interstitial fibrosis. A, the fibrous tissue.

pericardium intervened between the blood in the right ventricle and the pericardial cavity.

Imagine what might have happened if this dilatation had been mistaken for pericardial effusion and paracentesis attempted!

I fear these details have been somewhat wearisome, but they have enabled me now to approach the clinical side of the subject with the confidence of a basis of some assured fact, and I will now turn to some of the important clinical bearings.

II

The first point of clinical importance is this: the earliest evidence of rheumatic heart disease may be a dilatation.

I do not mean for one moment to say that other infective diseases do not cause dilatation, for we know that they do: instance only diphtheria and influenza, upon which an important paper was written by Dr. Lees.[1] But it is very important to remember that dilatation may be the first evidence of rheumatic heart disease.

The earliest physical signs of this dilatation I gave in my first lecture, but let me repeat them again here. They are: (1) An increase in frequency and a lowering of the tension of the pulse. (2) An outward movement and feebleness of the cardiac impulse. (3) An increase of the deep cardiac dulness to the right and left. (4) A shortening of the first sound over the impulse and an accentuation of the second sound at the pulmonary base. (5) In some cases a soft systolic murmur heard most clearly internal to the nipple.

Now, amidst all the uncertainty that there is about the treatment of rheumatic heart disease, it really does seem clear that rest is of great value, and may (who can say does?) cut short a severe attack. The earlier rest can be obtained the better I think is the outlook, and so the detection of early dilatation is a practical and valuable achievement.

The second clinical fact of cardinal importance is this: The great enlargement of the cardiac dulness which may occur in acute rheumatic pericarditis is more often the result of dilatation than of a large pericardial effusion.

You will at once see the importance of this as a guide in treatment. If this great enlargement of the cardiac area was usually

[1] British Medical Journal, January 5, 1901.

the result of effusion, we should be led to the conclusion that this effusion must gravely embarrass the heart, and be an imminent cause of danger. Further, we might hope to relieve the heart by a timely paracentesis. But if it is a dilated and feeble heart which is the main cause of the cardiac enlargement, not only should we be disinclined to introduce a needle, but we should actually fear to do so, lest, piercing the diseased ventricle, we precipitate death.

To make this the more clear, I will give you first the impression I gained of acute pericarditis from my education when a student. I learnt that there were three phases: (1) The early stage of cardiac excitement and early pericardial friction; (2) the second stage of pericardial effusion; and (3) the third stage of resolution and late pericardial friction. The second stage, that of effusion, was characterized by an enlargement of the cardiac area, muffling of the heart sounds, and a tumultuous action of the heart. If the effusion was very great, the fluid would need to be drawn off.

Now, there is no doubt that a great enlargement of the cardiac area in rheumatic pericarditis is a very frequent occurrence, and I have seen enormous enlargements more than once. The inference would be, then, that a great effusion was frequent in rheumatic heart disease.

But when, in 1898, I examined our post-mortem records and collected 150 fatal cases of rheumatic heart disease, I found that in only 35 had the amount of fluid in the pericardium even attracted especial attention, and in only 12 of these cases had more than 2 ounces been measured, and in only 6 cases, more than 3 ounces. The explanation is clear. The great enlargement of the cardiac dulness in rheumatic pericarditis is, as a rule, due, as Sir William Gull taught, to dilatation of the heart, rather than to effusion. If you wish for more evidence still, it may be added that investigators have introduced needles through the chest wall after death, and have found that instead of passing them into a distended pericardial sac, they have passed them into the cavity of the heart itself, and on more than one occasion during life, blood has been drawn off instead of exudation, and even a fatal event has resulted from such a procedure.

The lesson, then, that is learnt is a very real one. Picture yourselves face to face with a severe rheumatic pericarditis. The child is breathless, distressed, and, you fear, going to the bad. You are

harassed by conflicting thoughts; you know that a large effusion oppresses the laboring heart, and you know that rheumatic pericarditis is not, as a rule, fatal. You feel that if you draw off the fluid you may bring relief, but that if you fail or pierce the heart you may turn the scale against the child. What an aid it is—and I can speak from experience—to realize that an effusion sufficient to oppress the heart is rare in rheumatic pericarditis, but that dilatation is very common. For now it is clear that unless you have the most certain indications of a large exudation, you do not interfere.

I think it is my duty to attempt to give you some points in the differential diagnosis between a dilated heart and pericarditis with effusion, for it may happen to any one of us to be confronted with an exceptional case in which there may be very great difficulty in settling this point.

In the first place, we are much helped by a knowledge of the course of the disease. The doctor in every-day attendance is in a better position to judge than the consultant who sees the case once. For when a large effusion follows on pericarditis, you may trace the heart sounds becoming more faint and muffled day by day, and only the attendant who pays frequent visits can truly realize this sign. It is a very important one, for in rheumatic dilatation the sounds, though they may be feeble, are usually clear and distinct. I must admit that there are cases in which the heart is bound to the front of the chest by adhesions, and in which fluid collecting in the pericardial sac behind pushes it forward, but these exceptional cases do not alter (to any appreciable extent) the value of the point I have already mentioned.

The pulse, in a case with large effusion, may be small, rapid, and very irregular; in dilatation, though the tension is low and the pulse rapid, it is not such a small and irregular pulse, for the movements of the heart are not embarrassed as they are by a large effusion.

Disappearance of the impulse is in favor of a large exudation, a diffuse impulse in favor of a considerable dilatation. You are more likely to feel the impulse in dilatation than in pericardial effusion. As for dulness in the fifth right interspace (Rotch's sign)—I have been disappointed with this sign in rheumatic pericarditis, and I am convinced that a dilated right auricle will give the same sign in those very cases in which you are likely to be in doubt.

A toneless dulness in sharp contrast to the pulmonary resonance, and a dulness which gives a marked sense of resistance to the fingers, point to fluid.

The angle which the right lower margin of the cardiac dulness makes with the hepatic dulness is perhaps more useful. Dr. Ewart has especially insisted upon it. But it is a refinement which requires exceptional skill and accuracy. Further, it is not always reliable, as I have proved for myself in the post-mortem room. This angle, formed by the right margin of the cardiac dulness meeting the horizontal hepatic dulness, should be acute in dilatation because of the curve of the auricle, but obtuse in a large effusion, because the pericardial sac becomes distended. This is made quite clear by these simple diagrams (Figs. 6 and 7).

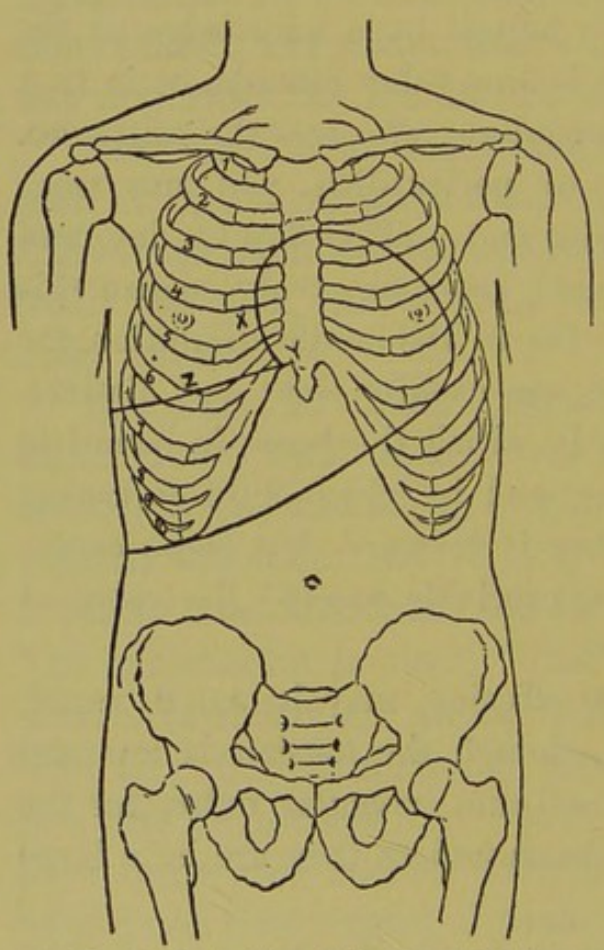


FIG. 6.—Z, the line of the hepatic dulness; X, the curved line of the right auricle; X Y Z, the *acute* angle formed by their junction.

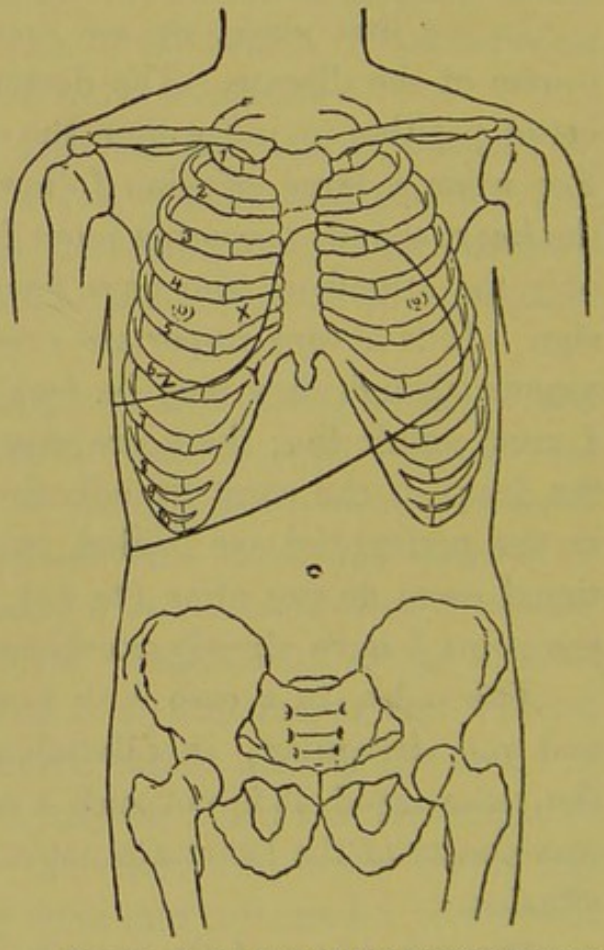


FIG. 7.—Z, the line of the hepatic dulness; X, the line of the distended pericardium; X Y Z, the *obtuse* angle formed by their junction.

The third and last clinical point I shall deal with is the one which I wish to illustrate to you to-day by clinical cases, and I will introduce it to your notice in the form of an interrogation.

Is it not possible that in rheumatic fever the cardiac wall may

be damaged out of proportion to the cardiac valves or pericardium? The answer, I feel sure, is yes.

There are two clinical types,—the acute and fatal cases, which are very rare, and the chronic cases, which I think are much more common, and of which I show you four examples.

The acute cases I must, for lack of time, dismiss in a few words. They are cases of rheumatic fever which die rapidly of cardiac failure, and in which after death no pericarditis is found, and perhaps no endocarditis or only the very slightest and earliest.

But the condition I am especially interested in now is a more or less persistent cardiac weakness following a well-defined or vague attack of rheumatic fever. There has not been any pericarditis and perhaps no endocarditis, but the heart remains weak and large, the patient breathless and nervous, and recovery is evidently imperfect.

They form an important group of cases, not only in the diseases of childhood, but, because of their bearing upon the etiology of obscure cases of dilatation of the heart, in adult life.

Case I.—This boy first came under observation in September, 1900, and was then 10 years of age. Four weeks before being seen he had got wet through twice in one day, and shortly afterward developed a sore throat and pains in the head, followed by pains in the chest and abdomen. The house in which he lived was damp, but he is very well looked after by his mother. When I saw him he looked ill, there was arthritis of the knees and hips, and a rapidly acting heart with a systolic mitral murmur. The temperature was raised to 100° F. The boy was admitted to the hospital, and when I saw him again in October a mitral murmur was still present.

It was not until May, 1902, that I saw him again, and during those 18 months he had kept well; but a fortnight before this second visit he had developed a sore throat and pains in the limbs. I found on examination no murmur; only a dilated, rapidly acting heart.

Since May, 1902, he has been again an in-patient, and I have never lost sight of him for any length of time, but have closely followed his case.

What is his condition now? Briefly this: he has an enlarged heart and the impulse is diffuse and well outside the left nipple line.

There has been no murmur, and no thrill or other evidence of a contracted or incompetent mitral orifice. There is no aortic disease, and there has never been to my knowledge an attack of pericarditis. The pulse-rate is always above 100. He cannot run, he is breathless on any sudden exertion, and with exertion there is pain over the heart. He is highly nervous and easily becomes "fagged" and anemic.

During the last few months there has been some improvement, and you do not see him now at his worst. I consider that in his case the myocardium is damaged, and that it is the heart wall and not the valves or pericardium which are in fault.

He is well worth careful examination, for you might easily underrate the gravity of the condition, because there are only the symptoms and the enlarged heart to guide you.

Case II.—This case is of exceptional interest, and well illustrates the difficulty there is in separating the nervous and the muscular factors in any study of the heart wall.

I first saw this child in March, 1902, when she was suffering from a definite but not very severe first attack of chorea. She lived in a damp house close to the Thames, and her father had suffered from rheumatism. The feature of her case was tachycardia, and although she was taken into the hospital at once, the pulse-rate varied between 140 and 200 from March until the end of May. Long after the chorea had disappeared this tachycardia continued, but there was no swelling of the thyroid or protrusion of the eyes. During the illness the heart became dilated, and the condition was for many weeks very grave.

By September there was much improvement; later when I saw her, the pulse had fallen to 78, but, nevertheless, the heart was large and the impulse heaving. There was no cardiac bruit, and except for a short time while in the hospital there had never been the suspicion of a murmur, but there was dilatation of the heart, and later there was hypertrophy.

This child is very excitable, is easily rendered breathless, and although very much better the heart is not yet really strong.

It may well be that the poisons in this case acted more upon the nerve than upon the muscle, but the point I want to make is this: the heart wall suffered rather than the valves or the pericardium.

Here, again, we have a large heart, without a murmur. A highly nervous child, easily terrified, easily tired, short of breath, and apt to become anemic. Her breath is short, and the heart very irritable. I mean irritable in this sense, that on very slight provocation it beats rapidly and often irregularly.

Case III.—This case is a more recent one. She is a little girl, aged 10, who had an attack of rheumatic fever at 3 years of age, and off and on since. The grandmother and grandaunts on the mother's side had suffered from rheumatism. In January, 1903, she was operated upon for adenoids, and about a week afterward a fresh attack of rheumatism commenced with a stiff neck.

She is a nervous, excitable child, always short of breath. The pulse is rapid, and when I saw her first was 125 to the minute. The impulse is excited, the area enlarged, but there is no murmur, and there has not been one since I saw her first in January. She is anemic, and the pulse easily becomes excited and irregular. At one time I suspected albuminuria, but did not find any in the urine, although it is quite probable there may be albumin occasionally. She is improving.

Case IV.—This boy, aged 9½ years, came to me in May, 1902, with a history of not having been in good health for some time. He had suffered with headache, pains in the abdomen and limbs, and sore throat. He was also very nervous and irritable. When I saw him his heart was acting at the rate of 132 per minute and there was a diffuse impulse. The temperature was 99° F.

His mother preferred to keep him at home rather than leave him in the hospital. He improved under treatment, and I lost sight of him until September. He then returned with more pain and an excited heart. This time he was taken as an in-patient, the temperature being 100° F. and the pulse-rate 160. With these there were slight chorea and a diffuse impulse.

The heart was enlarged, the action rapid and excited, but there was no murmur, and there has not been one to my knowledge. I have never lost sight of him for any length of time, and have not any reasons for believing he has suffered from pericarditis, for he comes here whenever he is threatened with rheumatic fever.

This boy is slowly improving, and lately he has made decided progress, but the impulse is still diffuse and the cardiac area larger

than normal. He, just as the other children, is very excitable, easily tired, out of breath, and soon becomes anemic.

I have had other instances of this condition, but to multiply the cases might be only to obscure the point which I wish to make clear,—namely, that these children have weak hearts, not from pericarditis or endocarditis, but from some damage to the cardiac wall.

You may think that this interpretation of these cases is fanciful, but I have given you strong reasons for its acceptance, and further let me add that to my personal knowledge, such cases, both acute and chronic, have proved fatal, and post-mortem examination has shown the reality of the condition. Only last autumn Dr. Theodore Fisher showed me an example of the acute condition. And more recently Dr. John Broadbent showed me the damaged muscle fibers from one of these chronic cases, which had proved fatal with much dilatation at the age of 17 years.

What is the prognosis in this group of cases? To be frank I cannot tell you. I am not aware that they are very generally recognized, and I have not traced their histories myself for a sufficiently long time. I look upon them as grave cases, for they are very slow to improve, and easily upset by slight illness. I suspect that they will always be liable to palpitation and breathlessness on sudden exertion, but, provided they have no more attacks of rheumatic fever, I do not see why they should lose ground, and should not, on the other hand, slowly improve, as these have done.

Should cases such as these later in life take to alcoholic excess, I think they will appear in the ward of some general hospital as breathless, edematous patients, with large dilated hearts which refuse to react to treatment.

I mention this because I think the temptation to take alcohol is very strong in this class of patients. They cannot get through their work with comfort, and are easily depressed, because of their feeble hearts. How tempting to fly to stimulants! How incautious oftentimes are we also with such people! I feel sure that when you see these highly nervous children, you must realize how, through no fault of their own, in later life they are easily influenced and may take to the use of stimulants. Yet nothing could be worse for them than alcoholic abuse.

Finally, a few words upon their treatment. Whenever after rheumatic fever the heart remains feeble and rapid, rest is all

important. Glance once more at those drawings, and you will understand that the reparative changes *must* take time. When complete rest is abandoned advance cautiously. Graduated exercises, saline baths, and slow, steady, voluntary exercise, first on the flat, then up a gentle incline, now have their place. Many cannot afford these refinements. You can help them almost as much by curtailing and arranging their exercise.

A mother will bring her child and say to you: His spirit is stronger than his body. There is no better definition of a rheumatic child that I know, than this. Let us accept the accuracy of the definition and take the hint. Make these excitable children rest for one hour in the forenoon. The discipline itself is good for them, and if they are thin it is especially useful. I like to see rheumatic children get fat.

A practical difficulty in dealing with such cases is to discern how much of the cardiac excitement is due to nervous influences, how much to organic damage. The symptoms and physical signs must all be taken into consideration, and you must not be guided only by the pulse.

Drugs are of undoubted value, and our study of the pathology gives a clear notion of their rightful place. The best cardiac tonics will be those drugs which improve the general health and thus assist the reparative processes. In other words, you treat the child, he will treat his heart. Tonic doses of quinin in an alkaline preparation, cod-liver oil and malt, or a course of arsenic for some weeks at a time, carefully guarded by alkalies, as recommended by Dr. W. B. Cheadle, have helped me in the stage of convalescence. I do not like iron for rheumatic children, except in the most gentle preparations of the drug. The digestion is easily upset by iron, and then it does more harm than good. Bromids are valuable when tachycardia is largely the result of nervous excitement, rather than evidence of severe organic disease of the cardiac muscle.

Direct cardiac tonics such as digitalis and strophanthus are, as a rule, disappointing, and though I admit this to be a matter of opinion, I do not like the use of salicylates in large doses in these cases. The digestion and the bowels of these children require constant supervision.

I am often asked about the question of eating meats. I should like to divorce myself entirely from the " uric acid" enthusiasts, for,

in my opinion, they have gone far beyond facts and sometimes beyond reason. I maintain in opposition to them that there is no proof that meat does harm to the rheumatic child when he is convalescent, or in a state of rheumatic cachexia. On the contrary, I think it does good, and I always advise it in quantities suitable for a child of the particular age, with, I consider, good results.

Lastly, the rest treatment may be overdone. Children, I believe, suffer from this over-treatment. You see or hear of them feeling their own pulses! exaggerating every pain or throb! We must avoid this if we possibly can, and by using all our judgment try to bring about a happy result.

THE PARALLELISM BETWEEN THE CLINICAL SYMPTOMS AND THE PATHOLOGICAL LESIONS OF RHEUMATIC FEVER

BEING LECTURE III OF A SERIES OF CLINICAL LECTURES ON RHEUMATIC FEVER DELIVERED AT THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET, LONDON

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I

Ladies and Gentlemen: In this demonstration, the third of a series upon rheumatic fever, I shall attempt with the aid of a lantern to show the importance of the conception of the disease as a far-reaching infection. If there is as yet any practical good to be obtained from the proof of the presence of an infective agent, it lies, I think, in that conception. It is not a new one,—far from it,—but one that is more definite and more easy to grasp at the present time than heretofore.

We do not yet understand the toxins of the disease, and we have not, much though we may wish to do so, come upon a curative serum. Nevertheless, though it is a humbler path, I firmly believe we can follow with more confidence the clinical course of acute rheumatism by the light of recent knowledge. If this is the case, that result is valuable, for we leave once and for all the blind gropings in the labyrinth of "rheumatism," and start with a clear idea of our direction.

Many clinical observers have held that rheumatic fever is a far-reaching disease, above all when it occurs in childhood. They have gone further and dwelt upon the likeness of the various pathological lesions to one another. I am going to take up this position. I am going to show the identity of the lesions, and then comment upon the resemblance in the clinical symptoms they produce.

It is essential for this that you should put aside the view upheld by some writers, and tacitly accepted by many of the profession,

that there is such a disease as acute articular rheumatism. To speak strictly, there is not such a disease as pulmonary tuberculosis; that is only one manifestation of the tubercular infection. There are many other manifestations which go to make up the disease tuberculosis. It may seem hair-splitting to thus cavil over what is and what is not to constitute a disease, but it is necessary to be strict in the use of words when we study rheumatism. If articular rheumatism represents to you a definite disease, what does this condition in children which I am calling rheumatic fever represent? Is it something different, or something added? Is it not rather the wider conception of a malady of which articular rheumatism is one manifestation? Whether it be right or wrong, I want you at any rate for this one hour to fall in with that view and study the clinical aspect of rheumatic fever as it is thus presented.

Acute rheumatism has many manifestations and many pathological lesions. Some of these we know well enough,—as, for example, pericarditis and arthritis. Others are not generally recognized; such are iritis and bronchopneumonia. But our first step must be to seek the most elementary lesion, the simplest in structure and the least complicated by its surroundings. We wish to study the unit of the rheumatic process, as we would the tubercle in tuberculosis. The subcutaneous or rheumatic nodule, is, I think, the most elementary lesion; but I am rather surprised to find that even physicians of note are not always well acquainted with them, and many doctors do not recognize them. In a recent German work on gout, the author described what were clearly rheumatic nodules as gouty manifestations! It is possible that in England, where rheumatism is so common, these nodules are comparatively frequent; but whatever the explanation may be, there is no doubt that, as Dr. Warner, Sir T. Barlow, and Dr. Cheadle pointed out long ago, they are extremely important evidences of rheumatic fever.

On that account I venture to give you a brief description of these nodules and to throw on the screen slides which illustrate their structure.

They are small swellings under the skin, gently raising it without any severe pain or tenderness. Often you can see them better than you can feel them, and they should be looked for over

bony prominences and in tendon sheaths. The occiput, the olecrana, the patellæ, the tendons of the extensors of the digits,—these are very usual situations. They are often called fibrous nodules,—but this is a misnomer, and, curiously enough, a misnomer which has a very important bearing upon this paper.

They appear and disappear sometimes with great rapidity, even within three days. They cannot then be fibrous nodules.

Dr. Still and I, in a paper before the Pathological Society of London, in 1899, showed that the evolution of the nodule is somewhat as follows: A dilatation of the minute capillary blood-vessels, with exudation between the fibers of the subcutaneous tissue, swelling of this tissue, necrosis, and fibrinous exudation. From the region of the distended blood-vessels there is also a free cellular exudation. Later, especially when the nodule is unusually large and unusually chronic, there is a formation of fibrous tissue, and it is then a fibrous nodule; but this is clearly an end-product and not of the essence of the disease.

In a section through an early nodule you will see, then, three zones: (1) A central zone of necrosis (as Hirschprünger pointed out) with exudation; (2) a zone of swollen and altered connective tissue; and (3) a zone of cellular exudation. This section shows these zones; but do not push this description of zones to the bitter end, as is so often done in medical philosophy,—it is only a convenient and reasonably accurate description.

In the early stage of their development there is another element, namely, the presence of micro-organisms in the form of minute diplococci. Dr. Paine and I have found them in three different nodules, and in one case produced rheumatic fever in a rabbit by a culture from one of them.

The essential feature of the nodule is, then, that it is a true rheumatic focus; a local infection. To what can we compare it in other disease? Clearly to the local subcutaneous abscess in pyemia.

The clinical course of these nodules is variable; they may last for months, and then in their intimate structure there may be a definite fibrous tissue, or a structureless material such as you see in the vegetation of a chronic endocarditis. They are more common, far more common, in childhood, but, as with all rheumatic manifestations, they have a curious unexplained, seasonal preva-

lence, and at such a season you may see several cases in adults. Dr. Cheadle associated them with grave forms of rheumatic fever, and that association must stand good if we accept the statement as it was intended. It is a useful, practical rule, but there are some exceptions.

One interesting point is this, that in some aberrant forms of rheumatism nodules have been noted more deeply seated than usually, attached to the bones themselves, and very chronic in their course. We are in such cases clearly coming very close to a condition of rheumatic periostitis and are reminded of the curious "growing out" of the ends of the long bones in some rheumatic children.

In the nodule, then, you recognize pathologically a micro-organism, certain tissue changes, and certain processes of recovery. You recognize clinically certain physical evidences, the influences of season and age, a course varying in duration, and the general tendency to recovery. You notice, too, aberrant forms of nodule the meaning of which is not as yet clearly understood.

In the vegetation on a valve, in the inflamed pericardium, the joint, the pleuræ, or the brain, I believe you find the same processes, though some elasticity of thought and freedom of imagination are necessary to adapt them to the differing function and anatomy of the particular part. For example, when a serous membrane bounding some cavity is attacked, there would naturally be much more exudation than in the nodule or the arid cardiac valve. Again, in the brain, with its minute and terminal blood-capillaries, thrombosis may well be more frequent than elsewhere. While if the process is very acute and the part affected exposed to pressure and friction, many minute, distended blood-capillaries may be ruptured, as in a joint such as the knee-joint.

I shall now demonstrate to you by means of the lantern the pathological identity of these various lesions. (Figs. 1, 2, 3, 4, and 5.)

The first part of this lecture is now completed, and I shall turn to the clinical bearing, after a few words of warning and some discussion upon certain objections.

It would be wrong to give the impression that it is an easy matter to demonstrate these lesions. It is not a question of weeks or of months, but literally it will take years to do so. These slides, imperfect as I know them to be, have taken six years, during four of which I have been assisted by Dr. Paine. You may demand,

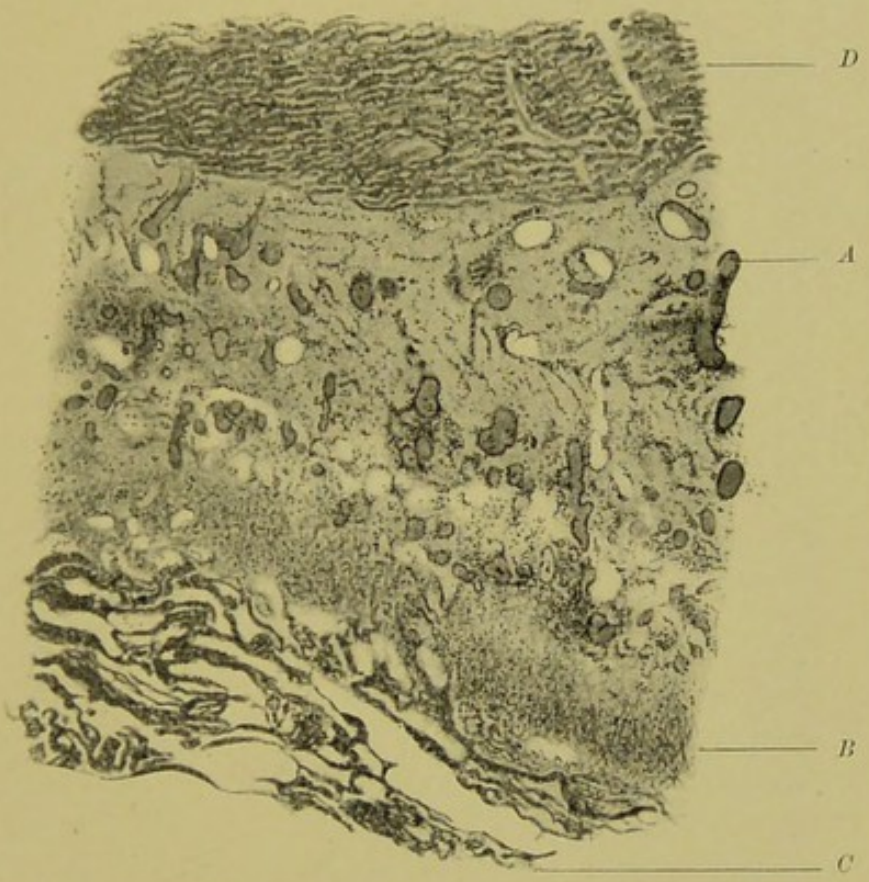

FIG. 1.—Pericarditis. Section through the visceral pericardium of man (rheumatic pericarditis). *A*, zone of swollen connective tissue; *B*, zone of cellular exudation; *C*, zone of necrosis and fibrino-cellular exudation; *D*, cardiac muscle.

[The illustrations are designed to show the similarity in structure of some important rheumatic lesions, and to represent graphically the central argument of the lecture.]

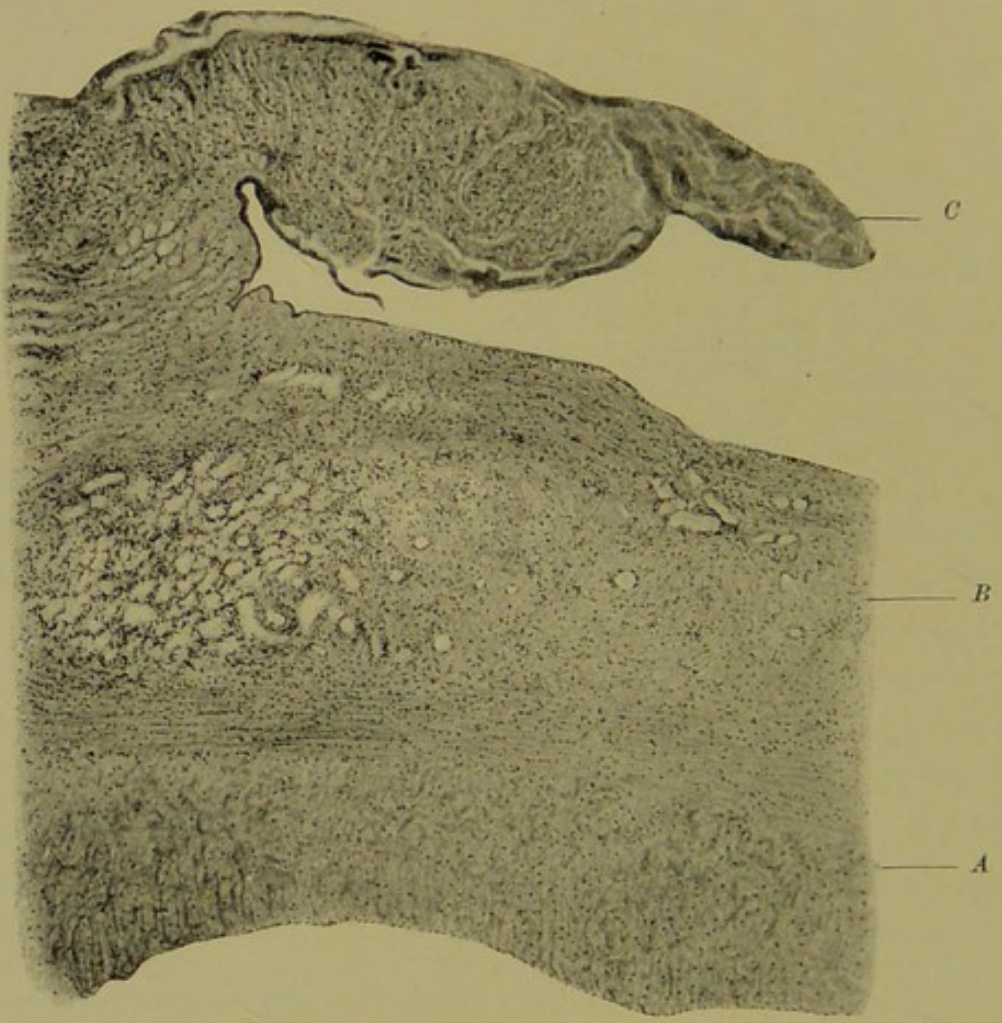

FIG. 2.—Endocarditis. Section through a mitral valve of man (rheumatic endocarditis). *A*, zone of swollen connective tissue of the valve; *B*, zone of cellular exudation; *C*, area of necrosis in the vegetation.

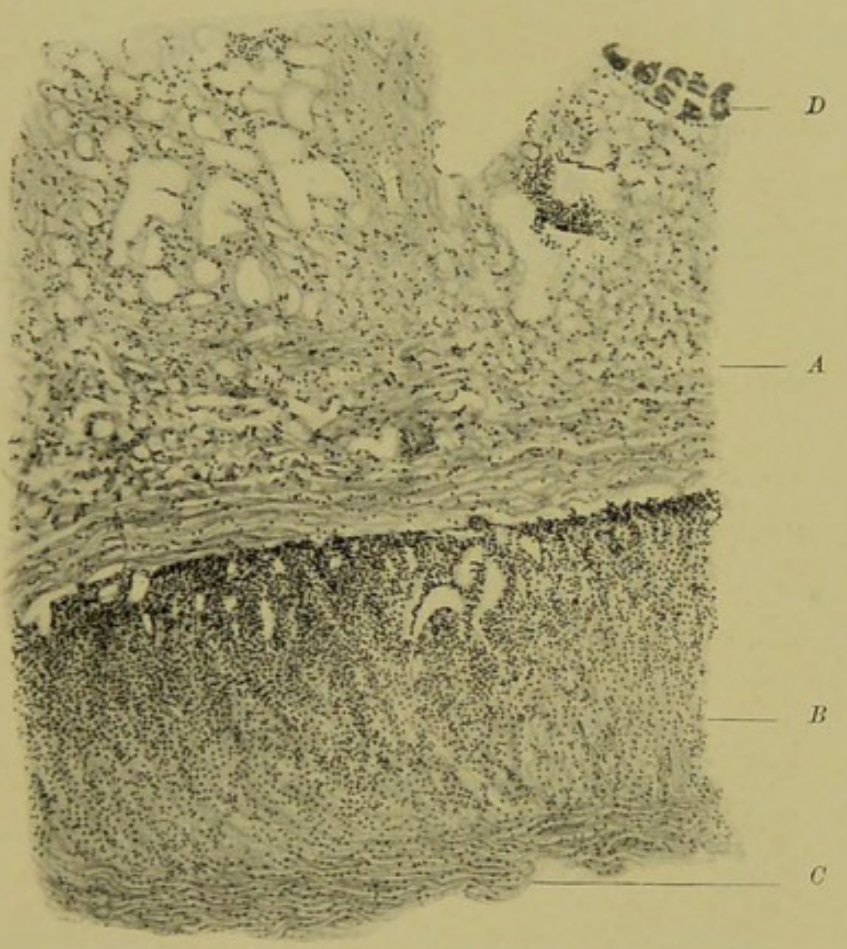

FIG. 3.—Arthritis. Section through the capsule of a knee-joint (rheumatic arthritis). *A*, zone of swollen connective tissue; *B*, zone of cellular exudation; *C*, zone of necrosis and plastic exudation; *D*, muscle fibers.

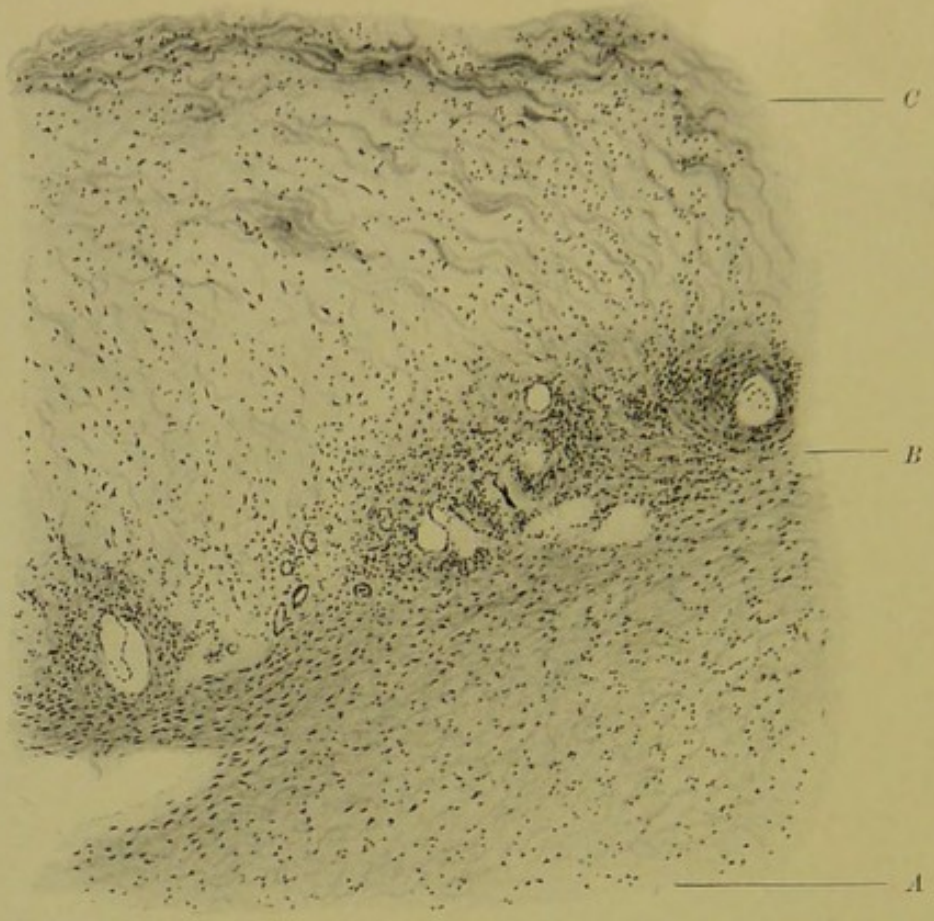

FIG. 4.—Nodule formation. Section through a rheumatic nodule of man of about six days' duration. *A*, zone of swollen connective tissue; *B*, zone of cellular exudation; *C*, zone of necrosis and exudation.

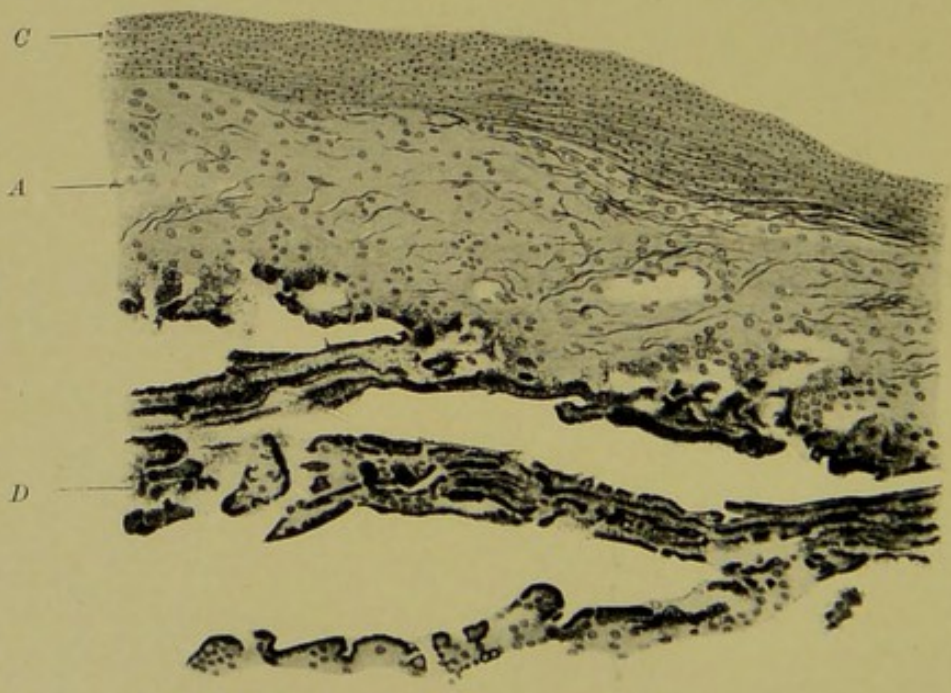

FIG. 5.—Experimental iritis. Section through the iris of a rabbit (experimental rheumatic iritis). *A*, swollen connective tissue of cornea; *C*, plastic exudation and necrosis; *D*, ciliary body.

and with right, absolute reasons for these difficulties, and I willingly give you what I believe these difficulties are.

(I) Difficulties in finding the organism. (*a*) It is very minute, and in the tissues does not fix Gram's stain tenaciously. It is therefore difficult to see in any but good preparations. (*b*) It is taken up by the living cells of the body, and even if the case is fatal, death may be lingering and the micro-organism itself destroyed. It is absolutely certain that micro-organisms, as a rule, do not kill of themselves, but they do so by their poisons, and it is a fatal error to think that the life of a micro-organism and the effect of its poisons are of equal duration. (*c*) Fatal rheumatism is not common, and the lessons of failure in method or opportunity learnt from one examination are apt to become dim in the memory before another chance occurs.

(II) The difficulty in obtaining experimental results. (*a*) The micro-organism loses virulence, and has not usually a high virulence, neither can the virulence be easily raised. (*b*) All our methods of cultivating micro-organisms outside the body are but poor representations of their conditions of life within the body. When, for example, we obtain this organism contaminated with others, and isolate it by the plate method, this, even though successful, may only obtain for us a micro-organism the virulence of which has been lost in the process. (*c*) An insufficient dose is used. Here again it is an error to suppose that all micro-organisms are either pathogenic or non-pathogenic to man or animals. They act in animals as in man. Sometimes the infection is insufficient, at other times it may produce indeterminate symptoms, and again it may produce the characteristic symptoms of the disease. With some animals the susceptibility to an infection may be at either extreme, but with others it is a question of the severity of infection, and there is then no hard and fast line between pathogenic and non-pathogenic micro-organisms.

(III) The name of the micro-organism has proved a great difficulty. It is a micrococcus, and yet a streptococcus, because it may grow in chains; a diplococcus because its elements are usually coupled; and a staphylococcus because on solid media it may grow in bunches.

Now, the mention of the word streptococcus at once calls up to many Streptococcus pyogenes. But why, I ask, *the* Strepto-

coccus pyogenes, when we admit that various organisms grow in chains, and that no one can absolutely define pus? Why, then, *the* Streptococcus pyogenes? It may perhaps be *a* Streptococcus pyogenes,—that will greatly depend upon the particular definition of pus to which you may incline.

There are many who are satisfied with an attitude of this kind. They say, "Oh, this is our old friend the Streptococcus pyogenes under a new name. We all know the streptococcus is a cause of rheumatism, and that the disease is an attenuated pyemia." They support this statement by pointing out that there are no means of distinguishing the diplococcus from the Streptococcus pyogenes. The answer to them seems to me to be this: It has not been known until recently that the Streptococcus pyogenes is a cause of rheumatism, for it is only of late that two cardinal lesions—endocarditis and arthritis—have been experimentally produced by a micro-organism isolated from the disease. Until at least these lesions had been produced, however much he might *suspect,* no one could *know* that any streptococcus was a cause of rheumatism. In the second place, it is, as already mentioned, an assumption to say *the* streptococcus. The pneumococcus will grow in chains and will produce pus; are we, then, to include it as the Streptococcus pyogenes? Are we to make pneumonia, rheumatism, erysipelas, puerperal fever, etc., all varieties of one disease? We know from clinical study they are not. In fact, all our efforts are turned to distinguishing them. If you accept that view, it appears to me that you only demonstrate the futility of bacteriological methods. To speak of rheumatism as an attenuated pyemia is surely but a mere pretence of knowledge, for we cannot define pyemia, and to grow an organism on some unsuitable medium outside the body, and to say it is attenuated, is not convincing. May it not be altered altogether rather than attenuated? Does not rheumatic fever too sometimes become virulently fatal, and yet not become a pyemia? Much more important points appear to me to be either to show some different organism as a cause of the disease, or to show that some poison irrespective of living bacteria can produce it. Nevertheless, do not suppose that I cannot see the weakness of the position I am upholding. It is clear enough, for if I am asked to show a test of the specific nature of rheumatism, I can only answer: I cannot show it, nor do I believe in specific tests. But I trust to the teach-

ings of clinical medicine rather than to the unripe doctrines of the bacteriologist. Give time; rheumatic fever is a disease which has puzzled us for centuries, and we cannot expect to unravel it in a few years.

I touch upon these abstruse questions, for it is well to recognize their difficulties, some of which have been forced upon us, by the cramping influence of words, while others turn upon our ignorance of bacterial poisons. Are we not apt, considering the extent of our ignorance, to rush to conclusions as to the specific test for a micro-organism; to mistake the micro-organism for the disease, of which it is only one factor; to say this stain shall be the test, whether the disease is or is not present; or this serum reaction is to be our certain guide? Diseases are vastly complex, and are hardly to be dealt with so. The idiopathic clinical symptom of a disease has failed us, the idiopathic bacteriological test will probably follow when we know sufficient to judge of its value.

Meantime the words of Job are in my ears.

"Canst thou draw out leviathan with an hook? . . . Or bore his jaw through with a thorn?"

It is well to remember that one organism can produce the lesions of rheumatic fever. The questions that arise around mixed infections are very difficult; but for the present we can be content with the ability of one organism to produce the disease. And now with this brief and inadequate allusion to points in dispute I turn to the second part of the demonstration.

II

A. PERICARDITIS

The first rheumatic lesion that I am going to consider is pericarditis. It is the lesion which most frequently causes death, and so we learn from it the nature of the *terminal processes* in rheumatic fever, and it is interesting to find how rarely during life we confuse fatal rheumatism with pyemia, or after death find the appearances of pyemia. But we must be very careful now in the use of our words. I mean, when I say we do not find pyemia, that we do not find scattered collections of a yellow fluid in the serous membranes, muscles, joints, or elsewhere. We do not find what we expect to occur after a suppurating wound, or from a deep-

seated abscess, or from a post-mortem infection. I speak clinically of a clinical condition which occurs with sufficient frequency to deserve the name pyemia. But if you press me for a definition, I must refuse to give it. I only say that it is an outcome of various infections, that it possibly represents a peculiar type of reaction, and that it is a useful word for the clinician. It is so, too, with the word pus. Many micro-organisms form pus, and pus, just as pyemia, is a useful clinical word—nothing more. It may well be that there is a rheumatic pus; and, in my opinion, the fluid that you find in the pericardium or joint in severe rheumatic pericarditis or arthritis is very like pus. You need not accept my word for this. You can, when an opportunity serves, make a film for yourself and look at it with an unbiassed eye.

There may be (and why not?) a rheumatic pus, but it still holds good that fatal rheumatism does not produce what you or I in clinical parlance call pyemia, nor, if there be a rheumatic pus, does it follow that this should be identical with the pus of ordinary pyemia.

I think a study of the final processes in rheumatic fever as exemplified by pericarditis is of great value. For example, it may be that to-day we see a pericardium completely adherent, and find that the process is of recent date; we realize then how great an effort nature has made toward recovery. Another day we see the parietal pericardium enormously thickened and actively inflamed, and we are impressed with the necessary slowness of recovery from such a condition if it were to attack some less vital part such as a joint. Yet, again we find the cardiac muscle terribly damaged, but the pericarditis quite recent, and we are forced to recognize that this type of rheumatism is virulent.

Lastly, we meet with a case in which apparently the inflammation has spread through the parietal pericardium, and implicated the pleural and mediastinal tissues, thus soldering the heart into a fibrous case. In this way we slowly build up our idea of the average condition of rheumatic pericarditis and gain an insight into the chief deviations from the type. But, as I have shown in the early part of this lecture, the types of all the common rheumatic lesions are similar. Is it not, then, probable that their deviations are also comparable? In other words, from a study of pericarditis, the most fatal rheumatic lesion, we are led to understand the aber-

rant forms of rheumatic endocarditis or rheumatic arthritis, which are rarely fatal.

B. ENDOCARDITIS

The study of endocarditis teaches, I think, another lesson. From it we get a very clear idea of the *healing processes* in rheumatic fever. This is because, though not in its simple form fatal, it is liable to be associated with pericarditis. In a percentage of cases this pericarditis proves fatal, and we obtain a necropsy, and thus are able to trace the healing processes in endocarditis.

One truth it brings home to us. A simple endocarditis is often only another name for a healing endocarditis, and does not represent, as we are so liable to assume, a process of a quite different origin to that of malignant endocarditis. It may be long before the opportunity of tracing in man the various stages from the simple to the malignant form occurs to one.

We must realize how slowly the evidence about rheumatic lesions accumulates. But the study of endocarditis is so interesting and so important, because you see the healing of the valve, and you may also see the failures. The usual sequence is well enough understood, the reddening of the valve, the bead-like vegetations, the final thickening. But now and again, after many weeks from the onset of a first attack of rheumatic fever, the valve remains unhealed, and tiny cauliflower projections are visible, perched as it were upon its free margin. Let me remind you of the remarkable specimen I showed in my first and second lectures. In that you saw the actual proof of what I am saying. The history attached to it was this: A youth suffers from a first attack of rheumatic fever; the onset is clearly marked; there are arthritis and definite clinical signs of endocarditis. The arthritis subsides, but he does not improve; he becomes more and more anemic, there is persistent slight fever, and he reacts to no drugs. The heart is always excited, and is clearly the site of active disease. Finally, after three months' illness he dies. The necropsy shows no pericarditis, but great dilatation of the heart, much myocardial disease, and unhealed vegetations upon the mitral valve. Not large vegetations, but a little larger than those you find in simple rheumatism. I would compare the condition of the valve to the virulent type of pericarditis; and such a case is, in my opinion, on the border-line between the simple and malignant type of endocarditis. How often, I wonder,

does endocarditis smoulder on like this, and how often in cases of malignant endocarditis of rheumatic origin should we find that the patient has not really been well since the last attack of rheumatism? or has struggled against the disease for weeks before lying up, and thus converted the simple into the malignant type?

In rabbits experimentally inoculated you can trace every step in this process; and it is one of the great advantages of the experimental method that you can work out the intermediate stages, because you can kill the animal at any time during the illness. It is interesting also that the diplococcus which Dr. Paine and I isolated from a case of rheumatic fever could produce severe endocarditis in a rabbit after growing for six months outside the body in subcultures.

Now, if the organism can remain in the local lesions and develop a new virulence as a result, let us suppose, of privation or pregnancy or malsanitation, it seems clear that it can produce in the valve a malignant endocarditis. Such a condition as this must be in its details somewhat peculiar, for the valve is in direct contact with the swift rush of the systemic circulation, and that is, I believe, why we look upon the condition so often as a thing apart from true rheumatism. Do not mistake me as saying that all malignant endocarditis is rheumatic, or that it may not sometimes be a mixed infection; I only maintain that the micro-organism is of itself capable of producing the condition of malignant endocarditis single-handed, and that some of these cases are the direct result of the rheumatic infection. It is very probable that knowledge will be gained about this malignant type of rheumatism from a study of rheumatism in the puerperal state. At that time, as with other infections, the virulence rises, and it may possibly be that all that is called septic in those days is not so, but on occasions a virulent type of rheumatic fever.

It would seem that in malignant endocarditis the diplococcus, only partially defeated, lies quiescent in the rheumatic lesion, as does the bacillus of tubercle in a tubercular gland, and is ready to become virulent again under those mysterious influences which confer virulence. Surely this view is much more reasonable and much more in accord with the lesions of other infective diseases than that crude one of a secondary infection.

The great difficulty to deal with about malignant endocarditis

is its rarity in childhood, when rheumatism is so common. The explanation sometimes offered that septic infections are rare and easily overcome in childhood does not appeal to me in the least. Osteomyelitis is common in childhood; so are middle-ear affections and contagious impetigo. Children fall about much more than adults, they are always breaking the skin over some part of their body, and are not greatly impressed with antiseptic notions as to keeping these wounds clean. There is some mysterious reason why an infective disease so often tends to be general in the young and remains localized in the adult, and bound up in it probably lies the explanation of the rarity of malignant endocarditis.

The virulence of malignant endocarditis is essentially a local virulence; the cardiac valve is the seat of the active disease, and in the vegetation there may be millions of micro-organisms. The other organs are damaged and the general system poisoned, because the lesion is most unfortunately in contact with the whole force of the systemic circulation.

In tuberculous disease you find the same principle of local virulence in disease of the lungs. The disease may remain localized to these organs, or even to a part of one. Though localized, it may be very active, and in the end the patient succumbs. This localization of a stubborn disease to one organ is much more common in adults. It reaches its maximum, of course, in cancer. Phthisis is uncommon in childhood, but a more or less general tuberculosis extremely common. When diseases are active they tend to be general; so with rheumatism. A stubborn arthritis such as you see in the adult is rare; a stubborn, active, localized endocarditis is rare; but general rheumatism is very common. I admit this is no explanation, but I believe hidden away in it lies the truth. I cannot bring myself to believe in the crude view of secondary infection as the solution of this problem, for malignant endocarditis would then be very common in childhood.

That form of endocarditis which results in mitral stenosis is particularly interesting. I think it is comparable to that type of rheumatic pericarditis in which there is the gradual formation of adhesions, not only between the two layers of the pericardium, but between the pericardium and surrounding structures. It represents, I believe, a smouldering type of infection rather than one attack of severe endocarditis, and I should compare it to an insid-

ious subacute arthritis. There is about the process the persistence of the malignant type, but not the virulence. In this form of cardiac rheumatism the onset may be so insidious that you can find no history at all of rheumatism, or even of heart-disease, and in my first lecture I recorded the case of a little girl who only betrayed the secret by an initial attack of chorea, and two years afterward by the very slightest return of twitchings. When I first saw her, she had early rheumatic dilatation; two years afterward, though apparently she had recovered except for slight persistent chorea, she had well-marked mitral stenosis. This condition of mitral stenosis in the advanced stage is rare in childhood, but if it were the result of one severe attack of endocarditis it would be common; rheumatism of the heart may occur at four or five years of age, and this would leave plenty of time for the sclerosis to take place, if it were the result of one attack of endocarditis. At twelve years of age we should meet many cases of severe stenosis, but this is not the case. In 150 post-mortem examinations at this hospital on rheumatic children under twelve years, nine only were said to have marked mitral stenosis. It is in early adult life that you find it becoming common.

C. ARTHRITIS

Rheumatic arthritis is the clinical manifestation of rheumatic fever which has always attracted the greatest attention. The pain, the swelling, the metastatic character, the rapid resolution, these naturally enough impress themselves upon our minds. From rheumatic arthritis, on the other hand, we gain very little pathological knowledge, because the condition is not a fatal one. It is here I venture to think that error has crept in. Whenever we study clinically a condition which is rarely fatal, we lose that solid basis of fact which pathology presents to us and are apt to substitute some basis less secure, as, for example, that of treatment. This, I think, has happened with rheumatic arthritis. I will not go so far as to say that the only action of salicylates is to lower temperature and relieve pain; but will admit that it has some good, even specific, effect on rheumatic arthritis. Yet I do not concede it to be a specific to rheumatic fever, a direct antidote, and a test of the disease. It is most unlikely that such a disease has only one poison; more probably it has many. And even if the drug has this antidotal effect in rheu-

matic arthritis, it requires very close watching to trace any such in other rheumatic lesions. I cannot, then, allow that an arthritis which does not react to salicylates is not rheumatic, and that the history of rheumatic arthritis, still less rheumatic fever, is to be written on this assumption. If, on the other hand, we study the arthritis from a clinical stand-point, it appears clear that we must recognize the usual type with its acute onset, pain, swelling, heat, and sometimes redness; we must recognize, too, its metastatic character, rapid subsidence, and the completeness of the recovery. But we must also recognize, as in the other lesions, intractable types, such as the chronic form, with much synovial implication, and sometimes periarticular inflammation, and the more virulent type, in which the cartilage and bone are affected and the muscles atrophy.

Now, it appears to me that when such intractable forms are met with, the tendency is either to explain them away under the name of rheumatoid arthritis or to call them, if they are dealt with surgically, by that vaguest of vague names septic. This procedure is encouraged by the fact that the salicylates do not cure these forms of arthritis, and I feel strongly that we are getting to a deadlock over these questions, because rheumatic arthritis is so often studied from quite a different stand-point to the other rheumatic manifestations. Why does arthritis in rheumatism usually subside so quickly? Partly, I think, because the resisting power of the joint is very considerable, and partly because it is a milder type of the disease which attacks the joints. I cannot accept the view that the joints have a low recuperative power, though this may be the case with the cardiac valves. Endocarditis, though it illustrates so well the healing processes, gives us a false impression of the natural curability of rheumatism, because—unlike the joints—we cannot accurately detect by clinical methods the earliest stage in the process, and really do not know how many slight cases of endocarditis occur which entirely subside. Why is arthritis so painful? This is a question of anatomical surroundings. The inflamed structures are encompassed by unyielding bone, cartilage, and ligament, and are disturbed by the slightest movement and by the abrupt contraction of muscles whose tendons are also inflamed. They are often pressed upon by the weight of the body, and the nerve filaments either directly damaged by the disease or pressed upon by exudation.

One more word before I pass from the question of arthritis. I do not wish to imply that all cases of rheumatoid arthritis are due to rheumatic fever, but only this: that rheumatic fever may be a cause of intractable arthritis affecting the periarticular tissues and even the cartilage and bone, and that it may be accompanied with much muscular atrophy.

D. THE NODULE

I have considered the nodule in the first part of this lecture, and you will recognize how closely it follows clinically the course of the other rheumatic lesions.

E. CHOREA

The last manifestation I shall consider is chorea, but this I shall do with a full sense of the difficulties which surround it and of the imperfection of our present evidence. Nevertheless, there are points about which we can be confident. We are absolutely certain that the great cause of chorea is rheumatic fever; there may perhaps be other causes, but none approach this in frequency. This important fact we owe to the older clinical physicians, and they have put us in a position to meet the next problem, which is this one: How can such a condition as rheumatic fever cause chorea? Here it is that this pathological study becomes of assistance, for necropsies on cases of chorea are so rare that it is absolutely necessary to give direction and method to our inquiries by reasonable inferences from other rheumatic lesions.

The particular lesson which I have learned from chorea is that rheumatic fever, like other great infective diseases, takes a very firm and subtle hold on the constitution. The remarkable movements of chorea enable even those who are not medical men to recognize the symptoms early, and we thus gain a very accurate idea of its course.

Chorea is, I think, the most accurate clinical index of the character of the rheumatic infection, and shows us how protracted and insidious it may be. Its usual course is first by a premonitory stage of weariness, headache, and vague pains. Lessons worry the child and are dreamt about. Outbreaks of petulance are noticed. In the daytime the pet dog who jumps upon the child every day of

his life now does so and strikes terror; the darkness is peopled with grotesque forms. You take the temperature, and find it is raised to 99.5° F., or thereabouts. The finest movements—those of the mind—have been first damaged, now those less easily disturbed are also damaged. The child fidgets, drops things, makes grimaces, and is brought by the frightened mother to you as suffering from St. Vitus's dance. You give all sorts of medicines, which perhaps do more harm than good; however that may be, the condition for some weeks gets slowly worse, then stationary, and then slowly subsides. "Six weeks," that ancient cure for rheumatic fever, is a favorable time allowance for chorea.

Yet all cases of chorea are not like those. Some begin as abruptly as articular rheumatism. I have in mind the case of a little girl who while walking with her father complained of pain in her head; she stumbled, and on her return was seen to be suffering from severe chorea; also of a little boy who while at dinner complained of his head, and began to drop his knife and fork. One of the oldest observations about disease is that abrupt and acute illness generally cures, if it does cure, rapidly. This is very true of chorea. The acute cases often terminate rather abruptly, the subacute are often most tedious. So we must recognize that chorea may be extremely chronic; you may perhaps hardly realize how chronic. For years after a first attack the movements may never be quite natural, and from time to time there are fresh outbreaks, recrudescences rather than new attacks.

The course of chorea is, then, quite in accord with that of other rheumatic lesions, and in this respect chorea is well explained as a rheumatic affection.

In some ways the condition may remind us of tubercular meningitis. First the premonitory vague illness, then the early cerebral disturbance shown first in the damage to the emotional centers, and lastly the declared disease.

To me chorea is quite as understandable as the result of an infective process as is tubercular meningitis. A great deal has been written about fright and chorea, but I do not think it alters the position. Injuries to the head have with good reason been associated as a predisposing agency with tubercular meningitis. In the same category I should put sudden fright in rheumatic chorea. As for those inadequate examples of fright (which are the most

frequent) I should look upon them as an expression of an already rheumatic brain.

Chorea is more common in childhood; so is tubercular meningitis; and the explanation, I think, must be sought in both instances in the mysterious tendency for infections to disseminate in the young.

The infective view of chorea points, I think, to the pia mater both of the brain and cord as being sites where the infection is to be most easily detected. I should imagine that chorea and tubercular meningitis were much alike in the distribution of their lesions. In both they are wide-spread, especially in the meninges and superficial portions of the brain; in both the lesions are minute and discreet. But tuberculosis of the brain and its meninges is usually fatal, and chorea very rarely indeed.

I have departed from the general plan of this lecture in omitting to speak about the morbid anatomy of chorea. It is not possible to deal with that with any confidence, the results are so discrepant. Multiple lesions have been repeatedly found. Such are minute thromboses, perivascular exudation, necrotic changes in the nerve-cells, leptomeningitis, small hemorrhages. Lastly, micro-organisms of the streptococcal group have been detected in the pia mater and the cerebral cortex, and have been isolated from the cerebrospinal fluid, and such micro-organisms can produce in rabbits rheumatic lesions and remarkable twitching movements.

It is interesting to observe that, when the rheumatic infection obstinately attacks one system, it often spares the others. All of us recognize cases of advanced mitral stenosis with no history of rheumatism, and also obstinate chorea with the same absence of other symptoms. Articular rheumatism is so frequent as to have been described as a special disease, and I have seen cases in which numerous nodules were practically the only index of this infection.

In concluding these lectures permit me once more to insist upon their chief aim. I have attempted to put before you the conception of rheumatic fever as an infective disease. There are faults,—there must be,—there may be errors, but I claim this virtue, that I have dealt with it in earnest and in no half-hearted fashion. If this disease is infective—and what explanation of its symptoms approaches this one for accuracy and completeness?—if, I repeat, it is infective, let us approach it as we do other infective diseases,—

on the broad lines of prevention. Let us bring to it the teachings of general hygiene and cast away the narrower views of diet and uric acid. These have been tried long enough and have failed. Above all, let us guide ourselves by broad clinical observation, and not be led to trust in sophisms from the laboratory, dramatic chemical tests, or experiments upon animals, unchecked by the eternal truth that we are studying a disease in man. The prize is a great one, for the prevention of much organic heart-disease is one of its chief aims.