death; nor should it ever be waited for to complete the diagnosis in a doubtful case. It usually indicates such a profound degree of toxæmia that, unless the cause of the obstruction be an external hernia or some equally readily ascertainable intra-abdominal lesion, the patient is unable to withstand the additional shock of the operation and dies "poisoned." Were as careful attention paid to the early diagnosis of intestinal obstruction as is now expended upon that of the other, perhaps more dramatic, groups of abdominal catastrophes, the present truly appalling mortality of this condition would, I am convinced, be very speedily and very materially reduced.

The chief features characterizing this group are (1) *the persistence of the vomiting*, the early reflex vomiting passing without interruption into that of obstruction; (2) *the cessation of passage of flatus*—if in a case of suspected obstruction flatus fails to pass within a period of twenty-four hours in spite of repeated enemata the diagnosis of intestinal obstruction is established with practical certainty; (3) *progressive abdominal distension*, a most important sign, and always calling for surgical intervention. To wait until distension is general is to render the operation much more difficult, to make inevitable the escape of coils of bowel during its performance—a procedure calculated more than any other to increase the intensity of the shock—besides paralysing the bowels from overstretching and increasing the toxæmia. The absence of any involuntary muscular rigidity and of early localized tenderness are negative points of some value. I have found *auscultation* of the abdomen of great assistance, and with a little practice the sounds produced by the passage of fluid contents along dilated coils and through narrowed portions of gut may be readily recognized. Sometimes there are other signs to help us in special types of obstruction. Thus in intussusception we have the passage of blood and mucus from the rectum (even before this has occurred a finger passed per anum may be covered with blood on withdrawal), and the presence of a tumour of characteristic shape felt somewhere along the course of the colon, or per rectum; this tumour is most in evidence as the peristaltic wave passes along it, and may therefore be palpable only in a spasm of pain, when the voluntary contraction of the abdominal muscles during the child's cries tends to obscure it. However, as already mentioned, there is always, in voluntary rigidity, a momentary relaxation at the end of expiration, and this will allow of the tumour being felt.

Many cases of chronic intestinal obstruction end acutely and may be mistaken for primary acute obstruction; such can, however, usually be recognized by the presence of two features never found unless obstruction has been in existence for some considerable time: (1) The outlines of individual dilated coils may be seen beneath the abdominal wall, giving a "hill and vale" appearance, and (2) peristaltic waves may be observed passing along coils of distended bowel. In the normal state neither peristaltic waves nor the outlines of individual coils of bowel are ever visible through the abdominal walls, though rarely these phenomena may be seen beneath the thinned-out covering of large ventral herniae, in cases of wide divarication of the recti muscles, and also in extreme degrees of emaciation. Even in these instances, however, the coils observed are of normal size and not distended, and are not really exceptions to the rule that whenever we can see clearly the outlines of coils of *distended* intestine, together with a peristaltic wave passing along them, we can safely diagnose chronic obstruction, and the presence of these two features in a case whose symptoms otherwise suggest acute obstruction should lead to a recognition of its primarily chronic nature.

Time will not permit me to consider cases presenting the features of more than one of these five groups—a not uncommon combination being that of inflammation with obstruction—nor of the rarer types of acute crises which do not exactly fall into any of these groups, but perhaps I may be allowed to mention two not very uncommon emergencies of special interest—acute hæmorrhagic pancreatitis and torsion of an ovarian pedicle. *Acute pancreatitis*, though it should strictly be classified with the inflammatory group, has such a sudden and severe onset that it simulates and is usually mistaken for either a perforation of the stomach or duodenum or else an acute obstruction high up in the jejunum. The severity of the symptoms is such as to call for immediate operation, but

the actual diagnosis is not often made prior to laparotomy, when the blood-stained odourless effusion and the presence of minute areas of "fat-necrosis" in the omentum and mesentery at once direct attention to the pancreas. *Torsion of the pedicle of an ovarian tumour* is readily diagnosed if the existence of the tumour is previously known, sudden abdominal pain associated with sudden increase in size of the tumour being quite characteristic; where previously unsuspected the tumour can, as a rule, be felt either through the abdominal wall or per vaginam, at any rate in the early stages, and if of the right side is not uncommonly, in my experience, mistaken for appendicular abscess; but the size of the tumour is too large for an appendicular abscess to have attained in the given time. Later, when peritonitis of the plastic type has set in, the tumour is masked by the overlying muscular rigidity, but as this rigidity relaxes under anaesthesia it is always possible to recognize the condition prior to making the actual abdominal incision.

In conclusion, surgical technique has now reached such a high level of efficiency that further improvement therein cannot be relied upon to better the statistical results of the surgical treatment of acute abdominal crises—such can be obtained only by earlier diagnosis on the part of the general practitioner permitting the application of such treatment at an earlier stage. The time that has elapsed between the onset of the attack and the opening of the abdomen is the real deciding factor in the ultimate issue of the case—far more so than the skill of the individual surgeon. Do not expend too much time in making an exact and elaborate diagnosis: content yourself with recognizing that a grave lesion has occurred which is far more likely to recover if surgically treated. I believe I speak for all surgeons when I say that we infinitely prefer to meet in consultation the practitioner whose diagnosis, *made in the early hours* of the attack, is limited to "something gone seriously wrong in the belly," than he who, *forty-eight hours later*, can give us a cut-and-dried and possibly perfectly correct description of what an operation will reveal.

REFERENCES.

- ¹ *Lancet*, May 3rd, 1919. ² *BRITISH MEDICAL JOURNAL*, January 3rd, 1914.

A Clinical Lecture

ON

THE STUDY OF HEART DISEASE.*

BY

F. JOHN POYNTON, M.D., F.R.C.P.,

PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL AND THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET.

I TRUST that your time to-day will not be wasted in listening to the remarks that I am going to make upon such a general subject as the one I have chosen. It can hardly be called a lecture, for it is only an expression of some of the ideas that have passed through my mind during the years I have studied this subject, but I hope, nevertheless, that I may stimulate your interest.

Heart disease is to me the least special of diseases because the cardio-vascular system is essentially a mechanical apparatus for distributing blood throughout the body, and when it is damaged the circulation in important organs suffers, this injuring in turn their functions and producing symptoms which are not strictly cardiac, but yet take a part in the patient's illness, and often an important one. These symptoms may be hepatic or pulmonary, or renal, or alimentary, or nervous, or a combination of them. Thus a wide knowledge of general medicine becomes essential in order to understand, and treat with confidence, such patients. If we focus all our attention on the heart we shall overlook valuable indications of heart disease, and though alive to the changes in the heart itself, be unable to grasp the exact condition of the patient.

It is not to be supposed that the heart must not be carefully studied, but even in doing this we are liable to fall into the error of restricting this part of the investigation to one part—the valves, for example—instead of considering the organs as a whole. We can see this tendency

* Delivered at the West London Post-Graduate School, December 9th, 1920.

if we glance at the evolution of the skilled study of heart affections. With the introduction of the stethoscope there necessarily followed a period in which the greatest attention was given to elucidating the various sounds, thrills, murmurs, and friction signs. Looking back we can recognize that much time and skill were devoted to nice distinctions, which, though excellent training in accuracy, were academic rather than practical, and one feels that sometimes larger issues were lost sight of in minor details. It is certainly possible to overdo the study of valvular affections, and by dwelling too much on the mechanical effects of valvular lesions, to lose sight of disease of the myocardium, and even more to arrive at a wrong interpretation of the very valvular conditions that we studied so rigidly. This I will illustrate from the combined lesion of aortic and mitral regurgitation. It is often said that in such cases the mitral regurgitation is due to a *relative* incompetence of the mitral valve produced by the effects of the aortic disease on the left ventricle. When, however, we follow many of these cases from their commencement, we find that in rheumatic cases the mitral valve is frequently damaged *first* by endocarditis, and the aortic valve *later* by the same process. Thus there are two separate lesions, and the mitral one is not dependent upon the aortic.

You may perhaps consider this to be an excellent example of an academic distinction, but it is not so, for you will find in a long series of these combined lesions that they are results of a severe type of cardiac rheumatism and illustrate a tendency to a spread of the infection. And you will not be surprised to hear that a considerable number of these cases die at an early age of a progressive or malignant endocarditis. Many look upon this final result as due to a secondary infection, but I regard it as a local intensification of the original and lurking rheumatic infection.

Now if we can be led astray over valvular affections by concentrating our attention too closely on the mechanical aspect of heart disease, it is easily understood how far wrong we may go when we are dealing with the human complex, a patient. For example, obsessed by a murmur, we may exclude a child from all activities, when with ordinary care such a child can lead a full life; thus we create invalidism and cause needless alarm.

This error, among others, has led to a swing of the pendulum, and many of you are well acquainted with the writings and teaching of Sir James Mackenzie, who brought us new facts and thoughts on the myocardium, and was the forerunner of extensive studies with the polygraph and electrocardiograph. I think he underrated the knowledge we in London possessed of the myocardium, but the man with a blazing torch naturally looks in the direction of its light, and of his fine work we have no doubt. This new period has taught us a vast deal about disturbances of the heart rhythm, and with it we must always associate the name and work of Dr. Thomas Lewis in this country. The result—and a very valuable one—is that we are now far more balanced in our views upon the part taken by the various cardiac structures in heart disease.

I believe, nevertheless, that we shall soon enter upon another period in the history of heart affections, with the realization of the immense importance of *infection* in cardiac disease.

This will bring with it a greater breadth of vision; for no one can study the results of infection in one organ without at the same time considering its effect upon other organs. And two aspects of heart disease will be always before our minds—the first the state of the heart and patient during the active phase of infection, the second the same considerations when the infection has ceased its activity and left its scars. The first stage may be illustrated by a child with recurrent chorea and the early signs of mitral endocarditis, the second by this same child when a woman of 35 years in an adult hospital, mortally ill with advanced mitral stenosis.

Is Rheumatic Heart Disease Infective?

The realization of this conception of heart disease, however, cannot be complete until there is general agreement upon a burning problem in medicine, the importance of which I am anxious to impress upon you to-day. The problem is this: Is rheumatic heart disease infective? Here I feel that I must proceed with caution, and assure

D

you that I do not intend to enter upon a lengthy digression upon the pathology of rheumatism. I do, however, want to point out that this question is not a matter of my views or someone else's views, but one of vital interest to medicine and the public. Twenty years ago Dr. Paine and I, discussing this problem, were convinced that sooner or later it would have to be faced, and we believed that it would be brought to the front, not over the cause of acute rheumatism, but over the causes of malignant endocarditis. We felt that the study of acute rheumatism required so much investigation of the most exacting kind that few would have the time and opportunity to undertake it, but that the study of malignant endocarditis was comparatively a simple matter. No one with any experience can fail to see and isolate the infective agent in the valves of a fungating endocarditis, and the fatality of the disease gives many opportunities for verification. It is interesting to find our forecast of twenty years ago has come true.

The problem has now come to the front, and the failure to agree upon its solution stands across the path of progress. It has been brought into prominence not over the question of acute rheumatism, but over a condition named, unfortunately in my opinion, "subacute bacterial endocarditis."

These three statements will put the position clearly before you:

1. Malignant endocarditis is undoubtedly infective in nature.
2. The most frequent antecedent of malignant endocarditis is rheumatism.
3. Rheumatic patients suffer very frequently from endocarditis.

Is, then, rheumatic endocarditis bacterial and cardiac rheumatism infective?

If it is infective, then I think no fact concerning the cardio-vascular system since Harvey discovered the circulation is of more importance to sufferers from heart disease. It introduces a new system of study of heart affections with its basis the study of heart disease in the young, for it is then that infective processes are most frequent and in their earliest phases. Upon this basis the evolution of heart disease in the adult will be gradually built.

These studies in childhood will be undertaken at a time when we have hopes of prevention rather than palliation. There will be search for a serological or other test for the presence of active rheumatism in the system, a discovery for which there is great need. There is promise of the possibility of finding some antidote to the toxins that injure the heart; and finally we shall be driven by it to recognize that an essential step in the treatment of heart disease is its prevention, which, if not complete, at any rate may do much to minimize recurrences of active disease. Our ideal will be to regard chronic valvular disease as a surgeon does a strangulated hernia, and with such an ideal even imperfectly realized there will be advance.

You may have noticed that in speaking of "subacute bacterial endocarditis" I said that I thought the term unfortunate. I think, at the present time, it is unfortunate, because it implies a non-bacterial endocarditis, for otherwise some such term as subacute progressive endocarditis would surely have been chosen. Now, is there a non-bacterial endocarditis? And, if so, what form of endocarditis is it, and what is its cause? What non-bacterial agent produces the inflammation, necrosis, and scarring that we meet with in all forms of endocarditis that I personally am acquainted with?

Upon the answer to these questions there is little clear information, but I must not overlook a statement made by a distinguished physician at Cambridge at the discussion on subacute bacterial endocarditis. It was to this effect:

Whatever the essential nature of rheumatic fever—and as to this we are still much in the dark—it is generally agreed that the vegetations of the associated endocarditis are, as judged by our present methods of investigation, bacteria-free, as also is the patient's blood stream.

Now I have been told, when discussing rheumatic endocarditis, that I have been hammering at an open door, and that no one doubts that it is infective, or that rheumatism is the *cause* of the heart disease.

Yet, is that door open if it is "generally agreed" that the heart disease is "associated with" rheumatism, and that the endocarditis—and therefore presumably the myocarditis and pericarditis—are bacteria-free? If any meaning is to

be attached to words, I should say that the door is still tightly closed. At that meeting I put forward this counter-statement:

The heart disease in rheumatism is *caused by* and not *associated with* the disease, and the endocarditis in its early stages is not bacteria-free.

Thus you see that in a discussion on a malignant form of endocarditis this essential question in the study of heart disease has come into prominence: Is the most common form of heart disease infective?

This question cannot now be ignored unless medical research in this country, in spite of all the brave words we have heard about it, is dying. I am convinced that the younger investigators will not let it drop, but will decide the point in the near future and earn the gratitude of all who believe in our position as a scientific nation, and of all who are lovers of children. Openings for fruitful research are not so many that such an obvious and vitally important question as this will be allowed to remain unsolved.

Leaving now the point in dispute, I am going to ask you for the rest of the lecture to accept rheumatic endocarditis as infective and as caused by acute rheumatism. The latter you may be more inclined to accept when I point out that in a recent analysis of 172 cases of acute rheumatism under 12 years of age, by Drs. Paterson, Spence, and myself, in 114 there was evidence of heart disease. This surely favours cause rather than association, for it was the most frequent of all the manifestations.

I do not ask you to accept the strepto-diplococcus as the cause of rheumatism, because that is not necessary for my purpose. I only ask you to accept the carditis as infective, and seeing how constant a disease acute rheumatism is, as due to one and not divers infections.

With rheumatic heart disease infective, you will realize how great a part infections take in these illnesses. We have, among others, scarlet fever, septic infections, diphtheria, pneumococcal infections, tuberculous, syphilitic, gonococcal, influenzal, meningococcal, and typhoidal infections, all implicated. When these infections attack the heart they produce there, as elsewhere, lesions which vary with the nature of the infection and the resistance of the tissues. These fall into three chief types: Local lesions may be produced in the valves and elsewhere with connective tissue changes, leucocytic infiltration and necrosis, and when recovery occurs the necrotic tissue is replaced by scar tissue. If the endocarditis heals, it is a simple endocarditis, and if it does not, a malignant endocarditis. Or the process, as in mitral stenosis, may be less acute but more obstinate, with the result that there is a slowly advancing scar formation. Or thirdly, it may be fulminating, the toxins destroying the vitality of the heart before local lesions of any moment have time to develop.

With few exceptions, of which acquired syphilis is the most notable, these infections are more frequent in childhood, and thus it comes about that in the adult we are more often dealing with the scars of former infections and with heart disease in an incurable stage.

Heart Disease in the Young the First Step in the Study of Heart Affections.

If, then, instead of basing our study of heart affections upon the conditions in the adult, we commenced with children, and if our books on the subject devoted their early chapters to a broad review of heart affections in the young, we should approach heart disease from a very different aspect to the present one, and I am convinced there would soon be a decided advance in our knowledge.

You may object that much heart disease of the degenerative type is not met with in the young, but in the adult and elderly. This is true, and is one of the important points in favour of this method of study, for, from a deeper knowledge of heart affections in early life, we should learn much as to the antecedents of these degenerative lesions, and as the years pass there would accumulate valuable evidence upon the possible part that disorders of the heart in childhood take in determining lesions later in life.

Consider for one moment the so-called soldier's heart, effort syndrome, or D.A.H., whichever term you prefer. You find this condition arising in boyhood, and you can get a shrewd idea of the kind of individual who is likely to develop such a condition in later life.

A high-strung schoolboy, who excels both in work and games, rather by virtue of his intense nerve energy than from a robust constitution, breaks down after some severe exertion at a time when possibly he is anxious to keep ahead in his form. Possibly he has had an influenzal attack or sore throat, or in these days has not been too generously fed. What happens? His heart becomes tumultuous, and he himself nervous and agitated, and his digestion faulty. He may have a fainting attack or be unable to exert himself without dyspnoea, and complains of pain over his heart. His heart may or may not be dilated, but it is certainly unstable and irregular, and his nervous condition equally unreliable. Such a condition, the counterpart of what we see in the adult, is slow in getting well, liable to relapses and a cause of much anxiety.

We can learn and teach most of the fundamental principles from the study of heart disease in children. Take, for example, rheumatism. We can study valvular lesions, dilatation, and pericarditis. It is in children that we can realize how faulty a conception of heart disease we must get if we look upon endocarditis, myocarditis, and pericarditis, as separate entities. Mitral regurgitation, for example, as you know, is generally admitted as the least harmful valvular lesion, but we find there are cases in childhood which run a most disastrous course, because the myocardium has been simultaneously damaged. Again, pericarditis is looked upon, rightly enough, as a dangerous lesion. Yet in children again you will find cases in robust patients which run a quite favourable course, when a severe myocarditis with no pericarditis in a frail child will lead to a rapidly fatal end. Our study of these cases forces us to realize that we must not judge of cardiac lesions by their anatomical names, but by their results to the patients. If you wish to learn the true meaning of a virulent cardiac infection, you will see this in the young. What more striking examples of acute asystole of infective origin can be found than a deadly rheumatic carditis, or equally virulent diphtheria or influenzal poisoning? The clinical picture of acute asystole, with its livid pallor, gasping respiration, vomiting, restlessness, running pulse, slight oedema, and rapid hepatic enlargement, is one of the cardinal studies in heart disease. Again, we can observe the origin of multiple valvular lesions and trace the gradual ascendancy of the predominant one, be it aortic or mitral. We see also cases of primary cardiac rheumatism—a very important group—in which the whole weight of the infection falls on the heart, all other rheumatic symptoms being absent, or so fleeting as to elude observation. From these we learn how latent are the symptoms of early heart disease, and when you have once followed these cases, you can readily understand how adults with "subacute bacterial endocarditis" may continue at work for weeks and months until laid low by some sudden complication such as an embolus or acute renal complications. Then again there can be no doubt that we learn much from studying the very commencement of heart affections. For example, we find that though rheumatism is more frequent in girls, it is more fatal in boys, and we learn that the fatal group is one usually associated with tonsillitis, arthritis, and carditis. In girls, however, the type is more chronic and relapsing, and we reach in them to the origin of that most serious valvular lesion, mitral stenosis, and we find it is often associated with repeated attacks of chorea.

This association of repeated attacks of chorea and mitral stenosis affords us an excellent example of what may be gained by studying the heart disease as a part of the general infection of the body. One of the most important points, as you know, in the history of mitral stenosis is the advance of the scarring process and consequent progressive narrowing of the mitral opening. If the process stops with the opening still of a fair size, life will be prolonged, but if the contraction steadily advances, the time must come when the heart begins to fail from the strangulation of the blood stream at the mitral valve. It is most difficult to estimate whether the process in the valve is advancing, because it is so slow in its progress, but we learn a great deal as to the probabilities by shifting our attention to other rheumatic lesions, and in particular to chorea, because that is such an obvious symptom and so closely associated. If we study chorea, we shall very soon see how obstinate it is in some children, how insidious, how apt to recur, and from it we get a clear idea of the dogged obstinacy of the rheumatic infection. If rheumatism can haunt the brain as it does, we can readily understand how quietly and unobtrusively it can progress in the mitral valve. Whenever, then, a patient with mitral stenosis

shows signs of active rheumatism elsewhere, be it in the brain or joints or muscles, we must suspect an advance of the lesion in the valve, or a sapping of the strength of the myocardium. It is because I have convinced myself of this persistent character of the rheumatic infection in the young that I believe in the adult malignant endocarditis as being frequently of rheumatic origin, the local virulence in the valves intensifying with failure in the patient's general health.

There are in children a considerable number of cases in which rheumatism does not appear to injure the valves, but causes a dilatation from damage to the myocardium, and I believe that much may be learnt about "weak" hearts in adults by tracing the history of the child's heart, which has been thus damaged through the early years of adult life. We know that in children such cases are very obstinate, and liable to break down under stress, and that these children are often highly nervous—a factor of deep meaning in the history of cardiac affections.

We have also opportunities in children of studying the adult type of heart failure, for we see mitral cases with oedema, ascites, orthopnoea, pulmonary oedema, albuminuria, and hepatic enlargement. We also see anginal attacks and embolism.

There is, I think, no doubt that disturbances of rhythm take a much smaller part at this age than later in life, and this fact itself affords us hope that we may learn much from a comparative study of these points at various ages, particularly as in recent years considerable additions to our knowledge of these arrhythmias in the young have been made by various investigators.

If for a moment we transfer our attention to the adult, and if we have thoroughly mastered the pathology of infective heart disease in the young, we can see more clearly the explanation of many of the symptoms in the adult, and we are in a much better position to estimate the importance of occupation and other factors in the final defeat of a damaged heart. I am convinced also that if more attention is given to early heart disease we shall soon see organized efforts in this country for the protection of adolescents with damaged hearts, and the gradual disappearance of those sad cases in which, from ignorance or lack of assistance and opportunity, young people thus handicapped are thrown into employments which mean for them certain death within a short space of time. To quote one case:

I had a boy under my care, badly damaged with aortic and mitral endocarditis, who, at the age of 14, had a terrible attack of pericarditis, from which recovery seemed almost impossible. He, however, did recover, and after many months got about again. He was a sensible boy, with good parents, and they got him work as a clerk. He is now 19, and has done steady work as a useful citizen, coming to report at the hospital every three months. I have not a shadow of doubt that if he had been put into a position requiring physical strain he would have been dead in six months or less. All the skilled nursing and care and money expended on him in hospitals would have been wasted, and the country would have received no return for his existence.

Is there any event in the history of heart disease more remarkable than the development of rheumatic nodules in children? Is there any better example of the truth that the student of heart disease must not keep his eyes glued to the heart? In themselves they are painless and of no practical importance, and yet they are signs of great danger. I give you some figures from the recent analysis I have already mentioned:

In 172 cases of active rheumatism in children under 12 years, 33 developed these nodules; 12 of them died; 16 became permanent cardiac invalids; and all but 2 had severe organic disease.

What a valuable warning these lesions are to us to be not pessimistic, but very cautious in our prognosis in such cases!

I come now to my last and most important point. When we are studying heart disease in childhood we are dealing with it at a stage which holds out much greater hope than later in life. If we can do good then, it may well be permanent good. Failing this, if we can prevent advance of the infective processes, we know that with increasing years the infective phase becomes less frequent. In the case of rheumatism the incidence is most frequent between the seventh and ninth years, and then it begins to decline, though still far too rife through late childhood and adolescence.

I have no hesitation in saying, from the evidence of incontrovertible pathological facts, that in the adult much of our treatment must, in the nature of things, be entirely palliative. All the skill and science devoted to the perfection of cardiac tonics will never make them more than heart splints; and no drug, short of one that will replace scar by healthy tissue, will ever do more than support the heart.

It is to prevention that we are bound to turn—a task of much difficulty in the case of rheumatism, but with great possibilities which have not been thoroughly explored. There are isolated writings of great value, such as those of Sir Arthur Newsholme on the nature and relation of the disease, of Dr. Shrubbsall pointing out the relation of the incidence to river courses in the London area, of Dr. F. Langmead on the throat and rheumatism in children of the school age, of Dr. C. Coombs on fatal heart disease in Bristol. These give us suggestive indications, but as yet no serious organized attempt has been made in this country to study the distribution of rheumatism in the young throughout the land, viewing the disease as an infection.

On one point there seems agreement: that the disease as we see it in our hospitals is infrequent in the well-to-do, and this points to careful systematic inquiry into housing and clothing and school influences. Of school influences we are learning more each year from the medical officers to the London County Council schools. I was once told by a doctor that wearing clogs in the wet season diminished the incidence of rheumatism in the schools where they were worn. Do not suppose that upon this evidence I am about to advocate that the Government provide free clogs for all scholars. I only mention it because it illustrates lines of inquiry which are practical, and which must end in our learning more facts about the natural history of the disease. The need for special arrangements for cases of early heart disease to have a long period of rest have been repeatedly spoken of, and in course of time I believe these arrangements will be forthcoming.

Why is chorea so frequent during school life in London? In one year we admitted 104 cases out of 172 of acute rheumatism! Would not a prolonged meteorological study of the disease help in warning us when to expect an outbreak of acute rheumatism if we had the organization needed? Do we know enough about the influence of dust and crowding as a factor in the causation of acute tonsillitis and rheumatism?

Is the general public sufficiently alive to the danger of this disease? Had the same energy been expended on acute rheumatism as has been on tuberculosis and venereal affections, I believe the study of heart disease would have already made substantial progress. We should not hear so often as we do wonder expressed at children suffering from rheumatism, or the belief that heart disease was an affection of adults, and that chorea was caused by fright. You may not be aware that there was less chorea at Great Ormond Street during the worst year of the air raids than in 1914, 1918, 1919, or 1920!

In conclusion, I have endeavoured to-day to put before you my belief that we shall soon enter upon a new stage in the history of heart disease, one of prevention based on its study in children.

I am not so blind as not to recognize that at the present time, borne down as we are by the aftermath of a great war, our first duty is to make life possible for the strong and healthy, for, with thousands of unemployed, no attempts of ours to prevent a disease can balance the evils produced by unemployment. One hopes that the rally will come, the country steady itself, and the vast burdens on the active and vigorous be gradually lightened, and then will come this country's time to make a determined effort to grapple with the problem of organic heart disease. Meanwhile we should have time to think out the organization in a spirit of moderation and plain sense, but not of fanaticism. If there is not light in this direction I am at a loss to see where to look in the present state of our knowledge of the treatment of heart disease.

THE Food Controller has appointed an Inter-Departmental Committee to consider the provisions of the Bread Acts and the Bread Order relating to the sale of bread by weight. The members of the Committee include Dr. A. C. Farquharson, M.P., and Dr. J. M. Hammill.