AETIOLOGY OF ACUTE RHEUMATISM

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During the last few years certain new conceptions have arisen in regard to rheumatism, which shed fresh light on the old and much-disputed views of the origin of the disease. The old idea of a specific infection may have to be modified, and attention directed away from the bacterium and focused more on the tissue reactions of the patient. For the purposes of this discussion two types of the disease will be considered—rheumatism of childhood and, more briefly, rheumatoid arthritis.

ACUTE RHEUMATISM

Influence of Infection

After watching innumerable rheumatic children through the most severe attacks of carditis and pericarditis, often with associated inflammation of the lung and pleura, it would take a great deal to convince me that infection played no part. Post-mortem findings can only confirm this view, yet the original streptococcal theory of Poynton and Payne has not been universally accepted. In an endeavour to reproduce these early researches the quest has always been for a living streptococcus circulating in the blood stream and settling itself into the tissues, there to work its pathological destiny. The results have been so conflicting that many have come to regard the time-honoured streptococcus as a secondary invader in the wake of a still unknown rheumatic virus. This view is a retrograde step, and there is surely sufficient evidence to consider the streptococcus as the fundamental cause.

Streptococcal Theories

Those who believe in a *specific* streptococcus point out that rheumatism is pre-eminently a specific disease, and therefore most likely to have its own particular organism with strict cultural characteristics. It is difficult to reconcile this view with the complete reversal of bacteriological opinion that has occurred in the last two years. Until quite recently we had come to associate a *non-haemolytic* streptococcus with rheumatism, and on the various occasions when an organism had been isolated from the blood, the heart, a lymph gland, or a joint, this was the type. Now with equal vehemence some bacteriologists insist that a *haemolytic* streptococcus is really the root of the evil. This *volte face* certainly shakes one's faith in a specific organism, and one is tempted to think that too much stress has been laid on small cultural differences on the laboratory agar plate and insufficient on the effect of bacteria in the living tissues. Bacteria are now known to be composed of carbohydrate and protein, part of the nucleoprotein being common to the various types and strains of each particular group of organisms. It is not unlikely that this part of the streptococcus will prove to be the important factor, and that therefore the possibility of a *multiple* streptococcal aetiology of rheumatism must be accepted.

The close relationship of acute tonsillitis and rheumatism was realized by some of the earliest authorities. More recently the real significance of these throat infections has been brought to light. It appears that acute rheumatism, especially a relapse in old cases, nearly always follows a streptococcal "sore throat" after a *silent period* of ten to twenty-one days. First attacks of rheumatism may also have the same sequence of events.

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In children, at any rate, the term "sore throat" is quite misleading, as often there are no subjective symptoms, and the whole nasopharyngeal infection is so mild and fleeting that it might easily pass unnoticed if a close watch on the temperature is not being kept. Whether or not the tonsils have been previously removed appears to make little difference; if they have, acute pharyngitis is just as likely to lead to a rheumatic relapse as tonsillitis. Obviously the majority of such acute infections of the upper respiratory tract are not followed by fresh outbursts of rheumatism, but it is impossible to predict in which it will occur. Any form of rheumatism may appear, from the mildest to the most severe and fatal. Some patients only pass through a short bout of renewed pyrexia unaccompanied by any other symptoms, but others are attacked by the most virulent type of pericarditis imaginable. Arthritis, chorea, nodules, and, further, myocarditis or endocarditis have all been observed. Curiously, patients who have long been convalescent from an acute attack of rheumatism are particularly likely to relapse after fresh tonsillitis. Acute rheumatism following scarlet fever has the same "silent period" before its appearance. In this case the scarlatinal streptococcal tonsillitis is responsible, and probably initiates the same train of events as other forms of streptococcal throat infections. It is significant that rheumatic subjects are much more prone to fresh rheumatism during scarlet fever than people who have never suffered before.

Certainly these observations do incriminate the streptococcus, but there still remains the problem how such nasopharyngeal infections manage to light up rheumatism in far distant areas of the body. The repeated failure of attempts to demonstrate streptococci in the inflammatory lesions of rheumatism is strong evidence against the idea of a great exodus of living bacteria from the inflamed throat into the general circulation. There must be some other way in which these violent inflammatory reactions are brought about.

Bacterial Allergy in Rheumatism

The explanation which appears most hopeful is that of bacterial allergy, on the lines of serum disease. In a non-fatal microbic infection the responsible micro-organisms are attacked by certain tissue cells and broken down. The patient thus becomes cured of the infection, but during the process the body may become sensitized to these disintegrated products, consisting largely of nucleoprotein, and will react in characteristic fashion should a fresh invasion and disintegration of the same organisms take place. Ample opportunity exists in childhood through minor throat infections for the tissues to become sensitized to streptococci. At the time these may only give rise to a deterioration in general health and pass unnoticed; but the train has been laid, and it needs merely a closely following acute tonsillitis to set off the explosion. The word "explosion" is used figuratively, as best describing those fulminating cases of pericarditis, which either rapidly reach a fatal issue or else leave a previously healthy child a permanent cardiac cripple. Other cases are undoubtedly more insidious in onset, and apparently without any preceding history of tonsillitis. Apart from the frequent symptomless character of these nasopharyngeal infections, there are other common illnesses which are associated, although less obviously, with a streptococcal sore throat. Thus rheumatism and rheumatic endocarditis can often be dated from an attack of scarlet fever, influenza, diphtheria, measles, or pneumonia. These diseases are even more frequently the starting-point of a severe relapse in mildly or definitely rheumatic subjects.

In the course of a relapse a "silent period" of ten to twenty-one days has been mentioned. It is an important

observation which as yet is improperly understood, although several ingenious explanations have been offered. The one that fits in best with the allergic theory just propounded is that the patient's defence mechanism only comes into full force ten to twenty-one days after the throat infection, and there is a violent reaction. One of the difficulties of explaining the basis of rheumatism through the agency of streptococcal infections is the fact that this organism flourishes equally in all classes of the community. Tonsillitis is endemic to the same degree in the public school as in the council school, and yet the incidence of acute rheumatism is very different in these two institutions. This raises one of the points foreshadowed at the beginning of this paper: that is, the importance of taking into consideration the patient's tissues—the soil, as it were, in contrast to the seed of infection which may become implanted.

Predisposing Causes

Certain environmental features of rheumatism have stood the test of clinical and statistical inquiry so steadfastly that they may now be regarded with some certainty as predisposing causes. One of the chief factors is the atmosphere. Damp has rightly been blamed, but rapid changes of temperature can play an equally important part. Poverty no doubt has much to answer for, but in regard to rheumatism it is probably the overcrowding, improper sanitation, and general lack of hygiene that are at fault. If, as some suggest, diet is a predisposing cause, there is no evidence that the disease appears for lack of food, although its quality may be open to question. Certain debilitated children with chronic ill-health are particularly predisposed to rheumatism. The truest picture of such a child is one whose lymphoid tissues are in a permanently catarrhal state, whose nervous system is easily upset, and whose digestion has been disordered by lack of protein and excess of carbohydrate. The emotional and highly strung nature of rheumatic children is proverbial, and it is therefore not surprising to find a disproportionately high incidence of chorea among Jewish children. The nervous strain of city life must have some effect, and no doubt accounts to some extent for the greater liability to acute rheumatism of the children of large towns as compared with their country cousins. Race and heredity also play a considerable part. Rheumatism certainly runs in families, and the children of rheumatic parents seem often to be born with an inherited predisposition of the tissues to the disease, a state probably best described by the term "diathesis."

In considering these various factors it soon becomes apparent that there is no one predisposing cause, but a combination of many. Together they produce a minor state of ill-health in the patient, so that he is unable properly to overcome the common infections. A bad cold, tonsillitis, or otitis, for example, instead of rapidly clearing up in such children, tends to pass from an acute into a chronic state. The chapter of each acute streptococcal attack remains unclosed, the patient's defence mechanism is continually overworked, and this is probably why his tissues reach that hypersensitive state already described. Along these lines the streptococcal theory and the varied predisposing causes can be brought into harmony, but further research is required before the whole problem is completely elucidated.

RHEUMATOID ARTHRITIS

Fortunately for our theories there are many cases of rheumatoid arthritis in which acute infection is obvious. Still's disease in children, with pyrexia, leucocytosis, enlargement of the spleen and lymphatic glands, and frequently pericarditis at necropsy, is sufficient

evidence of this. Adults may suffer from a very similar acute form, but usually the infective element is not so much in evidence. Nervous strain has been mentioned as a predisposing cause of chorea, and it is interesting to note the same influence in some cases of rheumatoid arthritis. It is perhaps encountered most frequently in young women, who, after some mental shock or anxiety due to domestic or commercial worries, promptly become afflicted by this incapacitating malady. How this is brought about is largely a mystery, but it is thought by some that a disturbed sympathetic or autonomic nervous system plays a part by upsetting the balance of the endocrine secretions. Still the most tenable hypothesis in rheumatoid arthritis, as in acute rheumatism, is again that of bacterial allergy, acting in this case through the more localized agency of hypersensitive joint tissues.

CONCLUSION

In the foregoing remarks an endeavour has been made to correlate some of the salient points about rheumatism and to suggest that the disease may be the outcome of an overacting response on the part of the tissues. There are indications of these excesses of nature, this perversion of the once physiological defence mechanism, in serum disease and anaphylaxis. Viewed in the same light, the exanthemata—and measles in particular—would present a new aspect, and complications such as scarlatinal nephritis and otitis following tonsillectomy might be explained. Graves's disease is another example of the occasional effect of an emergency call precipitating a disaster. In this case, in answer to some alarm or extra exertion, the endocrine and sympathetic nervous system overstep the mark and are not able to return to a normal state of balance.

Innumerable further instances might be given, but a great deal of research will be required along the lines indicated before we can go further than hint at a new conception of disease.

AN UNUSUAL CASE OF LYMPHATIC LEUKAEMIA

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The many points of interest presented by this case will obtrude themselves as the narrative proceeds.

HISTORY OF CASE

A male, 11 years old, was taken by his mother to a doctor's surgery at 7 p.m. on December 24th, 1932. He complained of feeling faint, and vomited some dark material, which was found to contain blood. The doctor ordered him to bed, and visited the boy at 10.40 p.m. the same day; he was asleep, of good colour, and breathing regularly. At 1.45 a.m. on December 25th the boy died. The doctor reported the case to the coroner, who ordered a necropsy. (What follows is an abstract of the mother's statement.)

The boy had never been attended by the family doctor, had always appeared healthy, and had gone to school regularly. On Wednesday, December 14th, he was caned across the buttocks by the schoolmaster. He had not complained, but on December 19th, when his mother asked him about some bruises on his legs, he told her about the caning. After breakfast on the same day he complained of feeling sick and vomited. The vomit contained streaks of blood. He went to school as usual. He vomited once on December 20th and once on December 21st, but attended school. He was sick once on December 23rd and twice on December 24th—at 6 p.m. after a meal which consisted of shredded wheat and milk, and at about 7.40 p.m. in the doctor's surgery. He