RHEUMATIC FEVER

FACTORS IN ITS OCCURRENCE[1, 2]

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IT IS the purpose of this paper to mention and discuss the circumstances under which rheumatic fever seems to flourish. The justification for doing this lies in the fact that we are still in the stage of exploration in so far as methods for the control of this disease are concerned. There are no public health procedures at our disposal which can be regarded as effective preventive measures as yet, and in the absence of such measures one of the next best things that can be done is to examine the conditions under which rheumatic fever is most frequent on the one hand; and to compare them with conditions under which it is most infrequent on the other. For, if it can be found that the disease flourishes under conditions that can be altered or improved, this fact in itself might indicate that there is something preventable about the disease which has not as yet been tried.

First, as to its geographical distribution. With few exceptions it is agreed that rheumatic fever is a disease of temperate climates and that it is rare in tropical or subtropical regions. It is very common in New York City. From 1 to 2 per cent of the school children in this general area have been found to have rheumatic heart disease. It is apparently less common in Florida, and it is uncommon in southern Arizona. A measurement made in the western part of this country has shown that rheumatic heart disease is ten times more frequent among children living near the Canadian border than among those living near the Mexican border. This fact probably has something to do with the living conditions in the north and

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south, and the way people are crowded within doors during northern winters. However, it is not, necessarily, a fact that can be readily utilized for therapeutic purposes, or even for purposes of prevention except perhaps in the case of the fairly well-to-do, for the practical use of climate in this disease has not been worked out yet. Certainly we are not ready for the wholesale exodus of our rheumatic children to warmer climates. Eventually climatic treatment may find its place among the list of things which are important to do for the care of patients with rheumatic heart disease, but at present one should proceed cautiously before recommending its use.

Secondly, rheumatic fever is known to be a familial disease. It runs in families—definitely. That does not mean that it is *essentially* hereditary, and in spite of a good deal of work on this subject one can hardly improve upon the statement made by Cheadle fifty years ago, that, "The tendency to rheumatism is transmitted...." In other words it does not mean that everything about it is hereditary. Other things run in some families besides inherited traits, such as, for instance, scabies and lice. But whatever the significance of its family prevalence is, it is of great interest to anyone concerned with the control of this disease to know that if one parent has or has had rheumatic fever, the chances of their children acquiring it are about 10 to 30 per cent; whereas if both parents have had it, the chances are much higher. This fact alone makes it evident that any control program in this disease should really center upon the family, and in doing this we are again following a lead already established by workers with the other two major chronic infectious diseases which beset our civilization in this part of the world, namely, tuberculosis and syphilis. They too, are family diseases, although a good deal more is known about them than is known about rheumatic fever. But in all three diseases the family approach remains as an opening wedge in the field of prevention. Private physicians, hospitals, and dispensaries will probably accomplish more in the way of prevention by taking advantage of this fact, than by any other means now

at our disposal. The mechanism for dealing with the rheumatic family has not been standardized, but if one child, or even one parent in a family, is found to have the active disease it is fair to assume that the conditions which favor the presence of this illness (whether they are *hereditary* or *environmental*) are present in that household; and the least that one can do is to begin by examining the other members of the family.

And next, and quite important, is the fact that rheumatic fever is a city disease and a crowd disease, although it does not seem to be evenly distributed through some city populations. It is, apparently, a disease of urban, middle classes. Most pediatricians practicing in the city or its suburbs will tell you that rheumatic fever patients are uncommon in their private practices in comparison to the relative number seen in the hospital wards. But from the work done in New Haven on this subject one cannot say that its prevalence follows poverty directly. It is true that our school surveys have shown rheumatic heart disease to be eight times as prevalent in children from schools in the poorest districts of the city as among children who attended private schools, but this does not mean that it is absent among the well-to-do, because that is not the case at all.

From a number of district surveys which have been made in nine areas in New Haven we have learned something about this fact.[4] These surveys of small areas have been carried out over a period of six years and the methods used have been described in an earlier number of this *Quarterly*.[5] Briefly it will suffice to say that we have gone from house to house in these districts in an effort to dig out the story of rheumatic fever, and the evidences of heart disease among every inhabitant in the district, which usually amounted to about 100 or more people. The districts were representative of the worst and of the best living conditions in the City of New Haven;

[4] These surveys have been aided by grants from the Milbank Memorial Fund.

[5] Paul, J. R.: Methods of Determining the Prevalence of Rheumatic Fever in Cities and Small Communities. The Milbank Memorial Fund *Quarterly*, January, 1935, xiii, No. 1, p. 52.

some were damp and some were dry. The study is not quite finished but there are a few things to report already. Again we can say that the prevalence of rheumatic fever did not seem to be directly related to the quality of the living conditions. We found the greatest concentration of cases in one single district among what might be called a middle-class group living in damp surroundings. We did not find eighteen cases in three houses, as was reported from Germany during the past century, but we did find that sixteen cases of rheumatic fever and rheumatic heart disease had occurred among the children of thirty-two families which lived in this district, which is at the rate of one case of rheumatic fever to every other family. It would seem that it is to such areas as these that our attention should begin to be directed, namely, to concentrated foci of the disease, whether they occur in families, or houses, or districts. At least I am sure that a tenement house full of cases of rheumatic fever offers as much promise for clinical investigation today as does a ward full of rheumatic patients.

And finally as to the actual bacterial cause of this disease. The question is too complex for discussion here but it is safe to say that *hemolytic streptococci* have something to do with the cause of rheumatic fever. This causal relationship can be shown from the manner in which an epidemic of rheumatic fever follows close upon the heels of an epidemic of streptococcus infection. It can be shown from immunological tests too. Obviously, therefore, whatever the conditions are which promote the spread of hemolytic streptococcus infections, they should be numbered also among the environmental factors which have something to do with rheumatic fever. Such conditions probably include crowding and "poor" hygienic surroundings, and, in the state of our present knowledge, that is about all one can say.

But certainly the hemolytic streptococcus does not tell the whole story. It is the nature of susceptibility to infection by this organism which remains the greatest single question in this disease. Why is it

that one child with tonsillitis of hemolytic streptococcus origin has a short disease with no sequellae whereas another develops rheumatic fever following his attack of tonsillitis? This is one of the main problems which demands solution. It is conceivable that there may even be two or more infections or types of illness which combine to give the clinical picture. Perhaps we have a situation like that of tuberculosis in which a "bad influence" upon the human host, such as that of exposure to silica dust, rouses a latent case of tuberculosis into activity.

But the final point to be made is this: we cannot cure rheumatic fever with serums or with medicines, although it is possible that something may be done in the field of prevention by the careful and prolonged use of sulfanilamide, as proposed by Coburn[6]—a procedure which is still in the experimental stage. We cannot give those rheumatics who are peculiarly susceptible (either through their inheritance or environment) a new body or a new heart. But occasionally one can improve the environment for the rheumatic child. And I would like to make one suggestion, which is, that efforts to do this be considered from the point of view of the whole family as well as to the individual patient with the disease. Rheumatic fever is, I will reiterate, a familial infection, and as such it lends itself peculiarly well to this sort of a household approach. What effect some of the housing programs may have upon rheumatic families is one of the features which may give us some interesting information on this point in the course of the next few years.

[6] Coburn, A. F. and Moore, L. V.: Prophylactic Use of Sulfanilamide in Streptococcal Respiratory Infections, with Especial Reference to Rheumatic Fever. *Journal of Clinical Investigation,* January, 1939, xviii, No. 1, p. 147.