

rheumatic in nature as are the cases of acute pyemic arthritis of infants.

What makes confusion still easier is the fact that tonsillar infection, with its resulting complications, is, like rheumatism, very likely to recur.

ERYTHEMA NODOSUM.

The rheumatic connection of erythema nodosum believed in by many of the older writers, and more recently by Cheadle, is not confirmed, according to some of the present-day writers, by closer clinical observations. In the absence of opportunity to make anything like wide observations in this disease I ought probably to withhold an opinion. But that discussion by those of you, who have had a larger experience with erythema nodosum, may be brought out, I want to suggest that this disease, too, is only another of the variable streptococcus infections of which the tonsils are the usual port of entry.

Two years ago Abt⁷ reported three cases of erythema nodosum to this section. In none of these cases could a history of rheumatism be established. What was especially interesting to note, however, was the fact that the one case which Abt had opportunity to observe from the very onset of the present illness, was treated by him ten days before in an attack of membranous tonsillitis. Bacteriologic examination proved the latter condition to be a streptococcus infection.

The findings in a single case of any disease may prove but little. At the same time, in a condition like erythema nodosum, the nature of which is the subject of discussion and the absolute knowledge of which is meager, close observations may suggest along what lines profitable work may be done, and may, at least, be taken as so much evidence *pro* or *con* prevailing opinion.

Finger, moreover, has already found streptococci in the inflamed tissues of erythema nodosum, and the same author, after a number of observations, concluded that the disease was a septic process. A few more studies like Finger's and Abt's will almost positively separate the disease from the rheumatic class and may prove it to be no more or less than streptococcus infection usually following tonsillitis.

As to the many other conditions considered among the manifestations of child rheumatism I have at this time very little to say, except to express the opinion that one by one they will be taken away from this connection until rheumatism will come to mean not everything, but something, or nothing.

THE CLINICAL ASPECTS OF RHEUMATIC FEVER IN CHILDHOOD,

AND THEIR SIGNIFICANCE IN THE QUESTION OF SPECIFIC
ETIOLOGY.*

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Knowledge of the disease, which is here termed rheumatic fever, for reasons to be set forth in the course of this paper, has been recently much advanced by certain experimental work and the criticisms on it. The advance, both from the incompleteness of the evidence and

the difficulty of judging as to its final value, is somewhat fraught with confusion. The lack of definite knowledge of the etiology of the disease was one of the motives for this paper. A second motive was the failure of many standard text-books fully to appreciate the marked peculiarities of this disease in early life as distinguished from adult life.

The clinical study of a number of cases was undertaken, in the hope that it might bring forward certain peculiarities of the disease in childhood, and that these peculiarities might throw some light on the question of etiology, or might at least form a basis for further study.

NOMENCLATURE.

The word rheumatism has been so much abused that some authorities advocate discarding it altogether. It has been applied to almost every condition associated with pain in the joints, muscles, fibrous tissues, and nerves of the body. In proportion to the progress of knowledge of these conditions, the terms rheumatism and rheumatic have been applied to a constantly decreasing number of conditions. Special progress has been made in the detailed study of joint pathology, with the result that many conditions, particularly of a chronic character, which were formerly included under the term rheumatism, have been relegated to their proper place in classification. Finally, this term, from the point of view of those engaged in classifying joint diseases as such, came to be limited to those conditions of arthritis which were supposedly due to infectious processes. Further objection to the use of the term rheumatism could still be made, on the ground that the condition of arthritis has been shown to occur in a variety of infections, such as scarlet fever, gonorrhea, septicemia, etc., and that for this reason the word rheumatic loses its significance. Consequently, it seemed to many authorities far preferable to discard the term rheumatic as applied to infectious joint conditions, and to substitute the term infectious arthritis as covering the entire group.

The use of this term, infectious arthritis, has its origin in the study and classification of the joint diseases as such, and it would be improper to use it to describe a disease which is not a disease of the joints as such. If there is a disease of which arthritis is only one of several co-ordinate manifestations, and if this disease has a specific etiology, we should use some other name in speaking of that disease. Again, if there is a disease of which arthritis is only one manifestation, and if this disease has a fairly constant and definite anatomic and clinical description which can be distinguished from the description of other forms of infectious arthritis, and in which the definiteness of the description suggests the probability of a specific cause, or at least of a specific reaction of the body, we should use some special term in describing that disease. The use of such a special descriptive term is only following precedent, for we classify diphtheria as a specific disease from its specific etiology, and scarlet fever as a specific disease from its definite clinical picture. Provided that rheumatic fever is such a disease as we have described, we have no more right to use the term infectious arthritis as synonymous with rheumatic fever than we have to use the term infectious angina as synonymous with scarlet fever.

The term rheumatic fever has been chosen to describe such a disease—if it exists—as being preferable to the term acute rheumatism, for the reason that it is analogous to the terms typhoid fever and scarlet fever, which have stood the test of time. The name acute articular

7. "Erythema Nodosum," THE JOURNAL A. M. A., Nov. 12, 1904.

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rheumatism is obviously a bad one, if the disease is neither primarily nor constantly articular.

It is necessary to determine, if there is a disease of specific anatomic and clinical description, to which the name rheumatic fever can properly be applied.

ETIOLOGY.

In most of the studies of the etiology, rheumatic fever was defined as a disease characterized by fever, polyarthritis, and a tendency to complications in the heart and serous membranes. Many views as to the etiology of these conditions have been successively advanced and discarded. That one of them all which has survived to the present day is the view that rheumatic fever is a manifestation of infection.

Mantle,¹ in 1887, advanced views of its infectious nature. Popoff,² in 1887, whose experiments attracted very little attention, cultivated a micrococcus from a rheumatic fever patient, and produced in rabbits by intravenous inoculation arthritis, pericarditis and endocarditis. Achalmé,³ in 1891, found a bacillus resembling anthrax in nine cases of rheumatic fever. He failed to reproduce the disease in animals. Sahli,⁴ in 1892, obtained several varieties of bacteria from the blood, urine and joint fluids of rheumatic fever patients, but could scarcely ever produce arthritis in animals. Singer,⁵ in 1898, obtained the same results. Goldscheider,⁶ in 1892, reported a case of serous pleurisy complicating a case of polyarthritis, in which a streptococcus was found, not pathogenic for animals. Dana,⁷ in 1894, obtained a diplococcus from a case of chorea following rheumatism. Widal and Bezançon,⁸ in 1894, found a streptococcus in the mouth of a rheumatic fever patient, which caused endocarditis in rabbits. They failed to produce endocarditis in five experiments with other streptococci. Thiroloix,⁹ in 1897, isolated a bacillus, apparently identical with Achalmé's, from a rheumatic fever patient, and claimed by intravenous injection in animals to have produced the entire picture of rheumatic fever. Triboulet,¹⁰ in 1898, found a diplococcus in five cases of rheumatic fever. In two of these cases it was associated with Achalmé's bacillus. Triboulet and Apert,¹¹ in 1898, isolated a diplococcus from eleven cases of rheumatic fever, which produced an endocarditis in a rabbit, but which failed to produce arthritis. Westphal and Wassermann,¹² in 1899, isolated a diplococcus from a case of severe rheumatism and chorea, which produced regularly polyarthritis in a series of eight rabbits, and was recovered from the blood and heart valves. Meyer,¹³ in 1901, investigated the bacteriology of the tonsils in rheumatic fever. There were many negative cases. In five cases he found streptococci. He investigated as a control many cases of sore throat without rheumatism, and could always find streptococci,

but these streptococci always produced in guinea-pigs subcutaneous abscesses, whereas the streptococci from the rheumatic patients never caused suppuration, but caused polyarthritis resembling the rheumatic, and occasionally pericarditis, non-purulent peritonitis and pleurisy. In one-fifth of the animals there was a valvulitis, and he succeeded in recovering the organism from the heart valves. Menzer,¹⁴ in 1902, confirmed Meyer's work.

The most important contributions to the study of the etiology of rheumatic fever have been made by English investigators. Poynton and Paine,¹⁵ in 1900, isolated a diplococcus from eighteen cases of rheumatic fever, in five cases in pure culture. This organism was obtained from the blood, joints and heart valves. Inoculated in large doses intravenously into rabbits, it produced all the clinical and anatomic characteristics of rheumatic fever, polyarthritis, valvulitis, pericarditis, and one rabbit developed a condition suggesting chorea. The organism could be recovered from the exudates, blood, urine and cerebrospinal fluid. Beaton and Ainley Walker,¹⁶ in 1903, confirmed these results and made a detailed study of the organism. Poynton and Paine¹⁷ repeated their experiments in 1903, with the same results, isolating a diplococcus from twenty-two cases of rheumatic fever, sometimes from blood cultures during life. In rabbits, arthritis was almost constant, while pericarditis, endocarditis and pleurisy occurred. Pus was never produced. Ainley Walker,¹⁸ in 1903, obtained similar results. Beattie,¹⁹ in 1904, obtained an organism from the inflamed patches on the synovial membrane of a rheumatic fever patient, and with it, in rabbits, produced typical polyarthritis, endocarditis, and also chorea, with twitching of the head and eyes. In the same year he published a detailed study of the morphologic and cultural characteristics of the micrococcus, which failed to show a means of distinguishing it from other streptococci. Lewis and Longcope,²⁰ in this country, in 1904, isolated a streptococcus before death from a case of chorea, endocarditis and rheumatism. This organism produced constantly in rabbits a multiple arthritis, which was neither purulent nor fatal. They once obtained endocarditis. Culturally, the organism resembled the *Streptococcus pyogenes*.

As a result of all these researches, among those believing that rheumatic fever is an infectious process, two views are held at present as to its etiology.

1. The bacteriologic view, that the disease is a specific infection, due to a specific micro-organism.

2. The clinical view, that the disease is not due to a specific infection, but that it is a particular reaction of the body to a wide variety of organisms.

Under the bacteriologic view, two varieties of micro-organism present claims to be considered the specific cause, a micrococcus, called by the English investigators *Micrococcus rheumaticus*, and a large anaërobic bacillus, usually called Achalmé's bacillus. The claim of the latter is lacking in proper confirmation, and it has finally

1. Mantle: Brit. Med. Jour., 1887, vol. i, p. 237.

2. Popoff: Medit Prebavlena K. Morskowa Sboneskie, 1887, p. 401.

3. Achalmé: Compt. Rend. Soc. de Biologie, Paris, 1891.

4. Sahli: Deut. Arch. f. klin. Med., 1892, vol. ii, p. 451.

5. Singer: Aetiologie un Klinik des Akuten Gelenkrheumatismus. Leipzig, 1898; Verhandl. d. xix. Cong. f. Innere Med., 1901, p. 441; Wien. klin. Wochschr., 1901, No. xx.

6. Goldscheider: Zeitschr. f. klin. Med., 1892, vol. xxi.

7. Dana: Am. Jour. of Med. Sci., 1894, p. 195.

8. Widal and Bezançon: La Semaine Méd., 1898, p. 195.

9. Thiroloix: La Semaine Méd., 1893, p. 376, *ibid.* p. 420; Compt. rend. Soc. de biol., 1896, p. 268; *Ibid.* No. xxx, p. 882, *ibid.* No. xxxiv, p. 945.

10. Triboulet: Compt. Rend. Soc. de biol., vol. v, p. 214.

11. Triboulet and Apert: Compt. Rend. Soc. de biol., vol. v, p. 128.

12. Westphal and Wassermann: Berl. klin. Wochft., 1899, p. 29.

13. Meyer: Deuts. med. Wochft., vol. xxvii, No. vi, p. 81.

14. Menzer: Aetiologie des akuten Gelenkrheumatismus, Berlin, 1902.

15. Poynton and Paine: Lancet, Sept. 22 and 29, 1900, pp. 860 and 932.

16. Beaton and Walker: Brit. Med. Jour., vol. i, p. 237, vol. ii, p. 659.

17. Poynton and Paine: Brit. Med. Jour., vol. ii, p. 779; Med. Press and Circular, vol. cxxvi, p. 478.

18. Ainley Walker: Practitioner, 1903, vol. lxx, p. 185.

19. Beattie: Jour. of Path. and Bact., vol. ix, p. 272; Brit. Med. Jour., Dec. 3, 1904.

20. Lewis and Longcope: Am. Jour. Med. Sci., 1904, vol. cxxviii, p. 601.

been shown to be a saprophyte. The weight of evidence points toward a micrococcus as being the organism most frequently associated with the lesions of rheumatic fever.

As to the clinical view, some of its supporters regard rheumatic fever as an attenuated pyemia, the exciting cause of which may be any of the pyogenic cocci. Others maintain that simple uncomplicated arthritis is due to toxins whose nature is undetermined, while the cardiac lesions are due to secondary infection with the pyogenic cocci.

There is much experimental evidence in favor of the bacteriologic view. In England, where rheumatic fever is of a particularly severe type, the great number of cases in which the micrococcus has been isolated and the great constancy and uniformity in which the complete picture of the disease has been reproduced in animals, is a strong argument in favor of the *Micrococcus rheumaticus* being the cause of the disease. Against it is the negative evidence, and the cases in which a variety of organisms has been found associated with the rheumatic lesions.

Chvostek,²¹ in 1895, and Kraus,²² in 1896, failed to obtain any organism in cultures from rheumatic fever patients. McCrae,²³ in 1903, reported blood cultures during life from a large number of cases of rheumatic fever, with uniformly negative results. He advanced the suggestion that, inasmuch as the organisms described by other observers were usually found in fatal cases, they might represent merely a terminal infection. He fails to explain satisfactorily the constancy of the experimental results in animals. Philipp,²⁴ in 1903, in twenty-one cultures from the blood and six from the joints, in a great variety of media, obtained negative results. Von Leyden,²⁵ in 1894, obtained from six cases of malignant endocarditis, supposed to be rheumatic, four cultures of streptococci differing from the *Micrococcus rheumaticus* in being very pathogenic for animals.

Sahli and Singer, who obtained a variety of pyogenic cocci from cases of rheumatic fever, are strong supporters of the clinical view. But they could scarcely ever reproduce arthritis and endocarditis in animals. von Leyden's experiments are inconclusive, while the evidence of Chvostek, Kraus, McCrae and Philipp is merely negative. In fact, all this evidence is of comparatively little value when advanced against that based on the very positive results of the English observers.

The principal argument against the specificity of the *Micrococcus rheumaticus* is the fact that it can be distinguished neither morphologically nor culturally from other varieties of streptococci. Although some of the English observers have pointed out minor peculiarities in the behavior of the *Micrococcus rheumaticus* toward various methods of cultivation, yet these are too slight to establish the separate identity of the organism. They have also advanced the Marmorek test in favor of the specificity of the organism, since the *Micrococcus rheumaticus* grows freely in media exhausted by other strains of streptococci. Recent work, notably that of Meyer²⁶ and of Aronson,²⁷ has thrown great doubt on the Marmorek reaction as a test of specificity. It must be admitted that the proof that the *Micrococcus rheumaticus*

is the specific cause of rheumatic fever must rest entirely on the fact that it produces specific results in animal inoculations.

But the supporters of the clinical view maintain that this is not the case and that other varieties of micrococci can produce in animals the lesions of rheumatic fever. Menzer,¹⁴ in 1902, advanced the opinion that rheumatic fever is one of the manifestations of a variety of organism, which under certain circumstances assume specific properties. Glaser,²⁸ in 1904, observed polyarthritis with other strains of streptococci. There are many reported cases of arthritis with various streptococci in which, however, the kind of arthritis is not specified. Harris,²⁹ in 1905, experimented with streptococci from various sources and succeeded in producing arthritis. He concludes that rheumatic fever is a streptococcus manifestation. The arthritis obtained by him in those rabbits which were autopsied was invariably purulent, and he failed to produce cardiac lesions. Cole,³⁰ in 1904, experimenting with streptococci from seven different sources where there was no suspicion of rheumatic fever, produced arthritis in rabbits with every strain, and twice produced endocarditis. He assumes that the claim of the *Micrococcus rheumaticus* to specificity rests on the demonstration of its ability to produce in animals arthritis and endocarditis, and that there is no essential difference in the nature of the lesions from those produced by him with other streptococci.

Beattie,³¹ in his latest article, 1906, criticises Cole's conclusions. In Cole's first series of experiments, working with an organism which he admits might be the *Micrococcus rheumaticus*, he obtained results in marked contrast to his later ones. In no case was the exudate purulent, but resembled that obtained by Beattie and the other English observers with the *Micrococcus rheumaticus*. On the other hand, in Cole's experiments with other streptococci he obtained usually either a purulent exudate or turbid fluid in the joints. Cole's inoculations frequently resulted in the death of the animal from septicemia, another marked difference from the results of the other English investigators. Beattie concludes that Cole's experiments fail to prove that other varieties of streptococci produce the same results in animals as the *Micrococcus rheumaticus*, but merely show that purulent arthritis in rabbits is a frequent manifestation of streptococci injected intravenously, the result resembling a pyemia. Endocarditis is less frequent.

There is considerable further evidence in favor of the specificity of the *Micrococcus rheumaticus*. Poynton and Paine,³² in answer to the view of Singer that rheumatic fever is an attenuated septicemia, point out that the organism is very virulent, and is found, associated with very severe forms of ulcerative endocarditis. It never produces pus nor a picture resembling that of streptococcus septicemia. Weichselbaum,³³ as early as 1889, working with various organisms, found it necessary to injure the heart valve before experimental endocarditis could be produced. Pasquale,³⁴ in 1893, working with streptococci, produced subcutaneous abscesses, general septicemia and a great variety of lesions, but

21. Chvostek: Wlen. klin. Wochft., 1895, No. 26, p. 469.

22. Kraus: Zeits. f. Heilkunde., 1896, vol. xvii.

23. McCrae: Amer. Med., Aug. 8, 1903.

24. Philipp: Deuts. Arch. f. klin. Med., 1903, lxxvi, p. 150.

25. Von Leyden: Deuts. med. Wochft., 1894, No. 49.

26. Meyer: Berl. klin. Wochft., 1902, No. 39, vol. xl, p. 936.

27. Aronson: Berl. klin. Wochft., 1902, No. 42, p. 979.

28. Glaser: Verhandl. des XIX Cong. f. Innere Med., 1901, p. 471.

29. Harris: Trans. Chicago Path. Soc., June 12, 1905.

30. Cole: Jour. of Infect. Dis., 1904, No. 4.

31. Beattie: Jour. of Med. Research, vol. xiv, No. 2, p. 399.

32. Poynton and Paine: Medico Chir. Trans., London, 1903, vol. lxxxv, p. 211.

33. Weichselbaum: Ziegler's Beit., 1889, vol. iv.

34. Pasquale: Ziegler's Beit., 1893, vol. xli, p. 433.

never mentions arthritis nor endocarditis. In the *Journal of Hygiene*, April, 1903, is an account of autopsies on rabbits used in experiments on the value of antiseptics. All died from acute infection, but none showed an abnormal endocardium.

The attempts to show that other organisms can produce the specific results claimed for the *Micrococcus rheumaticus* have not resulted in any very convincing evidence. If an organism can base its claim to specificity on specific results in animal inoculations without means of identification other than these results, the weight of evidence inclines toward the *Micrococcus rheumaticus* as the specific cause of the disease. Those who regard rheumatic fever as a specific reaction of the tissues to a variety of infections may in time prove right, but at present they need further evidence to show that a variety of organisms or the streptococcus group can produce such a specific reaction. It must be admitted that these organisms can at times produce arthritis and endocarditis, but it remains to be proved that they produce the anatomic lesions and clinical picture of rheumatic fever with a constancy and accuracy approaching that seen in experiments with organisms derived from rheumatic patients.

Nevertheless, until we have some means of recognizing the *Micrococcus rheumaticus* other than by its results in animal inoculation the possibility remains that other organisms may produce specific results, though lacking in experimental support. At present it seems advisable to accept the *Micrococcus rheumaticus* not as the absolutely proven specific cause, but as the probable specific cause of rheumatic fever.

CLINICAL STATISTICS OF RHEUMATIC FEVER IN CHILDHOOD.

Inasmuch as we can not regard it as absolutely proven that there is a disease of specific etiology, to which the term rheumatic fever may be properly applied, it remains to consider whether the clinical and anatomic description of certain cardiac and articular conditions is sufficiently definite and constant to warrant the use of the term. For this purpose a detailed study was undertaken of a number of consecutive cases treated in the wards of the Children's Hospital. Every case of arthritis, endocarditis and pericarditis admitted to the hospital for a considerable period, somewhat over five years, was included in the study.

In this entire course of time only five cases occurred which were obviously due to other causes than the infection provisionally defined as rheumatic; 300 cases occurred in which no other cause was found. These 300 cases were divided into the following classes: 1, With arthritis on admission, 102 cases; 2, with endocarditis only on admission, 140 cases; 3, with pericarditis on admission, 58 cases.

From another standpoint the cases were divided as follows: 1, Acute infections, 223 cases; 2, chronic endocarditis, 77 cases.

The interest in these figures lies in the fact that they show the great frequency of rheumatic fever infection, or at least of infection of a certain type, of unknown etiology, in children as compared with cardiac or articular manifestation due to other known causes. Of still greater interest is the fact that they show the much greater frequency of cardiac manifestations in children as compared with the articular. Of the 223 cases of acute infection, there were 121 cases showing at the time of admission no evidences of the localization or manifestation of infection anywhere but in the heart.

In 30 cases there was a rheumatic family history, but only in those cases with present or recent joint symptoms were the parents particularly questioned in regard to this point.

There was no case in the entire series of 300 cases under 3 years of age. The frequency of rheumatic fever apparently increased from the third to the seventh and eighth years, and then decreased from the ninth to the twelfth.

THE ARTICULAR CASES.

These were cases having joint symptoms at the time of admission to the hospital. The first striking point was the great comparative mildness of the joint symptoms. Whereas all these children had joint pain or pain on motion, it was in general slight as compared with adults. A certain number of cases were observed with the redness, heat, swelling and extreme pain on touch or motion, so familiar in adults. But cases of this type formed a small minority of the whole number. There was distinct relation between the age of the children and the severity of the joint symptoms, the severe cases resembling the adult type being much commoner in the later period of childhood, most of them occurring in children of from 10 to 12 years. Another striking point was the brief duration of the joint symptoms after treatment was begun, although again a small minority in the older children, resembled the adult type.

Duration of Joint Pain.—One day or under, 64 cases; two days, 12 cases; three days, 14 cases; from four to six days, 10 cases; one week or longer, 2 cases.

Objective Manifestations.—Redness, swelling, heat and tenderness to pressure were very noticeably infrequent as compared to arthritis in adults, more than half showing none: No objective signs, 52 cases; redness, 30 cases; tenderness, 52 cases; swelling, 45 cases.

The next point of interest lay in the number of joints affected, which showed the comparative infrequency of marked polyarthritis. More than one joint was usually affected, the arthritis being confined to one joint in 10 cases and to two joints in 28 cases. There were but 2 cases in which all the joints were affected. The ankles were most frequently affected, then the knees: Ankles, 73 cases; knees, 60 cases; wrists, 30 cases; hips, 15 cases; shoulders, 10 cases; elbows, 8 cases; fingers, 7 cases.

A very noticeable feature was the frequency of signs of endocarditis. Of the whole number, 85 cases showed signs of valvular endocarditis, and but 17 children left the hospital with an apparently normal heart. Seventy-two cases had signs of endocarditis on admission, and 13 developed signs during the stay in the hospital. Eighty-three per cent. is a very much larger proportion of cases of arthritis to develop signs of endocarditis than is met with in adults.

A comparatively large number of the cases showing heart signs also had symptoms referable to the heart. Here, again, is a marked contrast to the rheumatic fever of adults, where cardiac lesions are so apt to develop insidiously. Only 27 patients had no heart symptoms, whereas 45 patients had precordial pain or dyspnea. Thirty patients had dyspnea and distinct signs of failing compensation, which developed during the febrile attack. Fifteen patients had precordial pain and palpitation without marked dyspnea.

In a number of cases the cardiac symptoms preceded the articular by a variable and often considerable period of time.

The modes of onset of the attack were as follows: Fever and joint pain, 67 cases; fever only (at first), 15 cases; fever and dyspnea, 10 cases; fever and precordial pain, 5 cases; fever and sore throat, 5 cases.

It may be seen from these figures that while fever and joint pain is the commonest mode of onset, nevertheless, even in the articular cases, in 35 out of 102 cases several days elapsed before the development of articular symptoms.

The following tables show the degree and duration of the febrile reaction:

TABLE 1.

	Fever on admission.	Maximum reached.
99-99.8	0 cases.	0 cases.
100-100.8	32 cases.	17 cases.
101-101.8	20 cases.	20 cases.
102-102.8	25 cases.	20 cases.
103-103.8	15 cases.	30 cases.
104 or over.	10 cases.	15 cases.

TABLE 2.

Time.	Duration of fever. Cases with cardiac symptoms.	Cases without cardiac symptoms.
1 day.	2 cases.	10 cases.
2 days.	0 cases.	19 cases.
3 days.	5 cases.	9 cases.
4-6 days.	13 cases.	11 cases.
1-2 weeks.	8 cases.	16 cases.
Over 2 weeks.	17 cases.	3 cases.

An exceedingly interesting point in connection with the duration of this fever is the fact that in general it was much longer in cases with heart symptoms. Moreover, in every one of the 17 cases in which there was no sign of any heart lesion the temperature fell to the normal within three days. On the other hand, in cases with cardiac symptoms, the duration was often notably prolonged. In comparison with the time of disappearance of the joint symptoms, this prolongation of fever becomes especially interesting. In the majority of the cases without cardiac symptoms the temperature fell to the normal with the disappearance of the joint symptoms, and in only 15 cases did it persist longer than two days after articular symptoms subsided, and in only 4 cases longer than one week. In all the 45 cases with cardiac symptoms, the fever persisted more than two days after the disappearance of articular symptoms, and in 25 cases over one week. This suggests that after the subsiding of an active infectious process localized in the joints, there may continue to exist an active infectious process localized in the heart. It also suggests that in children in whom this infectious process affects the joints and the heart, its activity persists longer in the heart, in so far as fever is a measure of the activity of the process.

CASES OF ENDOCARDITIS.

These 140 cases were diagnosed as endocarditis, from the fact that endocarditis was the only lesion found on their admission to the hospital. There were also numerous cases of endocarditis among the cases regarded as primarily articular or pericardial, so that in all, out of the 300 cases, 281 showed at some time signs of endocarditis and 19 none. While it can not be argued from these figures alone that endocarditis is a commoner manifestation of rheumatic fever in childhood than arthritis, it can, nevertheless, be inferred that endocarditis is an exceedingly common manifestation of rheumatic fever in early life.

As to the lesions seen in the 140 cases of endocarditis, the mitral valve was very much the most commonly affected. In fact, it was affected in every case but one.

Lesions.—Mitral insufficiency alone existed in 70 cases; mitral stenosis alone existed in 1 case; aortic insufficiency alone existed in 1 case; mitral insufficiency

and stenosis existed in 56 cases; aortic and mitral insufficiency existed in 7 cases; aortic and mitral insufficiency, mitral stenosis existed in 5 cases.

The cases showing endocarditis as the chief manifestation are divisible into three classes as follows: 1, Chronic endocarditis with cardiac symptoms, 47 cases; 2, chronic endocarditis without cardiac symptoms, 30 cases; 3, acute endocarditis, 63 cases.

CHRONIC ENDOCARDITIS.

These patients were either admitted to the hospital on account of cardiac symptoms or were admitted for some other reason in which the cardiac lesions were discovered in the course of routine examination. Twelve patients of this latter type were admitted for chorea. Of the 47 cases with cardiac symptoms, the majority began with the usual symptoms of failure of cardiac compensation. In 8 cases the cardiac symptoms were immediately preceded by articular symptoms and fever, which had since subsided. In 2 cases the cardiac symptoms developed in the course of chorea.

The connection of these chronic cases with rheumatic fever is very noticeable.

Previous History of Seventy-seven Cases of Chronic Endocarditis.—Joint symptoms only, 32 cases; chorea only, 12 cases; joint symptoms and chorea, 14 cases; scarlet fever, 5 cases; no cause found, 14 cases.

Forty-six patients out of the 77 had had joint symptoms; in all, 28 had had chorea. In 19 cases there was no evidence of any rheumatic infection. In 5 cases the symptoms dated from an attack of scarlet fever, leaving 14 cases of chronic endocarditis in which no cause was found.

ACUTE ENDOCARDITIS.

It is not possible to determine in how many cases of rheumatic fever there is acute endocarditis. In most of the cases of acute infection already considered, in which arthritis was the principal manifestation, there was evidence of endocardial lesions. While in those patients who had had no previous attack of rheumatic fever, such evidence points toward an acute endocarditis, nevertheless in those admitted with a heart lesion, it is impossible from the history to exclude a previous attack as the cause of the lesion. From the frequent mildness of the joint symptoms, such an attack could easily have been overlooked. On the other hand, it is certain that in the 13 cases which developed a cardiac lesion while under observation, there was acute endocarditis. The special interest in connection with this form of acute infection lies in those patients who at some time while under observation were suffering from an acute febrile disease, but who showed no symptoms nor signs at that time suggesting localization of the infection elsewhere than in the endocardium. These are the cases particularly defined as acute endocarditis. Those cases admitted for articular manifestations, in which the fever and general appearance of acute infection persisted long after the articular symptoms had completely disappeared, would come under this definition. In 29 of these cases the fever persisted longer than one week, without signs other than in the heart; also, among the cases of pericarditis, 11 showed only signs of endocarditis on admission, the pericardial lesion developing later. So that of the entire 223 cases of acute infection in the series, 134 had at some time acute endocarditis while under observation, whereas only 102 had arthritis. Therefore 60 per cent. of the cases of acute infection showed the distinct appearance of endocarditis at a time when

there were absolutely no articular manifestations, but cardiac symptoms in a large proportion.

Onset in Sixty-three Cases of Acute Endocarditis.—Fever only, 3 cases; fever and dyspnea, 27 cases; fever and joint pain, 18 cases; fever and precordial pain, 3 cases; fever and sore throat, 1 case; fever and chorea, 1 case; fever, joint pain and dyspnea, 10 cases.

This summary shows the various modes of onset of these cases, the commonest being with fever and dyspnea.

The range of fever and appearance of the temperature chart in these cases is strikingly similar to the articular cases.

TABLE 3.

Temperature. Not taken.*	On admission.	Maximum reached.
99-99.8	2	2
100-100.8	15	0
101-101.8	14	7
102-102.8	15	17
103-103.8	14	20
104 and over.	0	10
	3	7

On the other hand, the duration of the fever shows a difference from the articular cases in general, being more apt to be long.

Duration of Fever.—One day, 2 cases; from two to three days, 15 cases; from four to six days, 3 cases; from one to two weeks, 10 cases; from two to four weeks, 5 cases; from four to twelve weeks, 8 cases; terminated fatally, 20 cases.

A very common type in children is characterized by a temperature chart of one or more weeks' continued moderate fever with no symptoms other than the cardiac.

When articular symptoms were present at the onset of the attack, they usually subsided rapidly. The cardiac symptoms, dyspnea and cough usually persist one or more weeks. Edema occurs in about 25 per cent. of cases with cardiac symptoms, chiefly in the severe and fatal cases. Precordial pain is comparatively uncommon. Articular symptoms frequently develop after a considerable period of cardiac symptoms only. Sweating, headache and digestive disturbance are not marked, although these symptoms occur at times. There may be no subjective symptoms at any time throughout the disease.

On physical examination the characteristic endocardial murmurs are heard. The cardiac rhythm is often irregular, though regular in the majority of cases. The rate is usually much accelerated, there being often a curious irritability of the heart, even in those cases where cardiac symptoms are slight or absent.

What proof or evidence is there that the cases which develop without any evidence of localization of acute infection in the joints are in any way connected with rheumatic infection? There is evidence of varying character. Of the 63 cases of acute endocarditis, 56 had some history of previous arthritis and only 7 cases had nothing to connect them with rheumatic fever, except their own clinical picture. But there is a strong presumption that these 7 cases also were actually manifestations of the same infection, for their clinical descriptions were precisely similar to those of the cases which had had a previous or shortly preceding arthritis, and were widely different from 3 cases of malignant endocarditis due to the pyogenic cocci, observed during the collection of the series, but not included. The chills, irregular fever, often extremely high, and the metastatic suppurations observed in these malignant cases bore no resemblance to the fairly regular temperature chart, moderate fever and absence of symptoms other than cardiac in the 7 cases under discussion. Moreover,

there were in the series a number of cases which for many days ran a course precisely similar in every way to these 7 cases, but which suddenly, in the course of the disease, proved themselves to be rheumatic by developing joint symptoms for a brief period. It seems probable that these 7 cases were due to the same infection, but in them the articular manifestations did not happen to develop.

The most striking type in the rheumatic fever of childhood is the primary endocarditis. By this is meant those cases of acute infection in which endocarditis is the first, and for a time or throughout the only rheumatic manifestation. There were in the series of 63 cases admitted for endocarditis 35 cases of this type. Fifteen had had previous arthritic attacks, but at a period too remote to have any connection with the present infection, during the entire course of which there was no other localization. Thirteen developed articular symptoms subsequently to a varying period of fever and cardiac symptoms. The remaining 7, as we have already seen, had no other localization of infection at any time. In these cases of primary endocarditis, a child has an attack of fever, seems unwell; there are usually cardiac symptoms, frequently dyspnea and cough. After a varying period the fever subsides, while the cardiac symptoms may or may not persist. During the attack there may develop joint pain, usually of very brief duration. Occasionally an attack of chorea develops in the course of, or shortly after, the febrile attack.

It must be remembered that sometimes there are no symptoms at all, or slight precordial pain only. There is simply fever and a heart murmur. Such cases are frequently diagnosed as grip or febricula, the heart murmurs not falling under suspicion of connection with the present infectious process. After a time fever subsides and the child may be sent home without a satisfactory diagnosis having been made. There were three cases in my series of precisely this character. I have learned that every one of these patients, within a year after discharge, had an attack of arthritis and one of them had chorea. The possible rheumatic origin of cases of this kind should be recognized and the frequency of acute endocarditis as the only manifestation of rheumatic fever in childhood should be remembered. Given such a case, in the absence of any other demonstrable cause the case may be safely ascribed to rheumatic fever; and if these cases are followed the tendency to recurrent manifestations of various kinds will often prove the diagnosis correct.

ACUTE PERICARDITIS.

There were 58 cases of this kind of localization of the infection. In 3 the records were partly unsatisfactory.

Mode of Onset.—A. Cases with pericarditis on admission, 45 cases; fever, precordial pain, dyspnea and cough, 27 cases; fever, joint pain, dyspnea, precordial pain, 8 cases; fever, joint pain, precordial pain, 7 cases; unknown, 3 cases.

B. Cases with acute endocarditis only on admission, 13 cases; fever and joint pain, 3 cases; fever and dyspnea, 10 cases.

Fever and precordial pain are almost invariable accompaniments of acute pericarditis at its onset. There may be dyspnea and cough also. In some cases at the onset there is also joint pain. Other cases, in which the localization of the infection in the pericardium occurs later, may begin with fever and joint pain only, or fever and dyspnea, and show for a time only evidences of endocarditis, arthritis, or both.

35. These patients were brought in moribund.

The symptoms and signs of acute pericarditis in children show no notable difference from those in adults. Dyspnea is apparently largely independent of the presence or amount of effusion, but seems rather to a certain extent to be a measure of the severity of the infection. Of course, there is usually a notable increase in the dyspnea when effusion is sudden, or suddenly becomes marked, but very severe dyspnea occurs without signs of effusion, and on the other hand, there were in my series a number of cases with appearances of a very large effusion, in which dyspnea was comparatively slight. It is known that myocarditis is a frequent accompaniment of acute endocarditis and pericarditis. Is it not possible that the presence and severity of the dyspnea may be dependent on the presence and severity of the accompanying myocarditis, as well as of effusion?

TABLE 4.		
Temperature subnormal.	Fever admission.	Maximum.
99-99.8	5 cases.	5 cases.
100-100.8	5 cases.	5 cases.
101-101.8	12 cases.	2 cases.
102-102.8	23 cases.	10 cases.
103-103.8	3 cases.	8 cases.
104-104.8	5 cases.	18 cases.
105 or over.	3 cases.	10 cases.
	0 cases.	3 cases.

TABLE 5.—DURATION OF FEVER.		
Time.	Cases.	Termination.
1 day.	0 cases.	..
2-3 days.	2 cases.	..
4-6 days.	7 cases.	2 died.
1-2 weeks.	13 cases.	3 died.
2-4 weeks.	10 cases.	3 died.
4-12 weeks.	19 cases.	5 died.

Fever is about the same in degree, but persists much longer in the pericardial infections than in the endocardial infections, and at times may last five or six weeks.

On admission, 22 cases gave signs of fibrinous pericarditis only, and 36 of effusion. Of the fibrinous cases, 13 subsequently developed effusion, while 9 remained dry.

Most of the cases of pericarditis showed signs of endocarditis also; 48 showed endocardial murmurs on admission. Of the 10 showing no endocardial murmur on admission, 8 developed signs of mitral disease subsequently.

Three cases of pericarditis showed chorea on admission, with a history of its having developed subsequently to the pericarditis. Two cases developed chorea while under observation. Two patients had had chorea shortly before the pericarditis, and 3 had had previous attacks of chorea.

The evidence in favor of the pericarditis being a rheumatic manifestation is fairly strong: A. History of joint symptoms, 33 cases; previous attacks, 13 cases; shortly preceding the pericardial symptoms, 10 cases; accompanying the pericardial symptoms, 7 cases; developing after the pericardial symptoms, 3 cases.

B. No history of joint symptoms, 25 cases; chorea, 10 cases; tonsillitis or sore throat, 5 cases; no rheumatic manifestation, 10 cases.

Those cases in which the connection with the general picture for rheumatic fever is not clear, precisely resembled in their clinical course the cases in which the pericardial manifestations were associated with arthritic manifestations. A number of cases were aspirated, including several of those without other rheumatic manifestations. The fluid was always serous, never purulent. There was a marked contrast clinically between all these cases and 2 cases of purulent pericarditis which occurred

during the same period. Both of these latter complicated pneumonia and showed a temperature chart with a high, widely varying fever, and a septic condition very different from the cases in the series.

Primary pericarditis, that is, pericarditis without other manifestations, is fairly common in children. There were 30 cases, in which pericarditis alone or pericarditis with endocarditis, were the sole manifestations of infection. They are analogous to the cases of primary endocarditis already considered. In some of them there were several days of fever before any definite symptoms developed, then precordial pain and dyspnea followed. This point should be remembered. If a child is suffering from an acute febrile disease, if a friction rub is heard, the diagnosis of rheumatic fever is probable. If no friction rub is heard, the possibility of rheumatic fever should still be remembered, as any one of its three principal manifestations may subsequently develop. There were numerous cases of this kind in the series. A child is seen with a mild febrile attack, perhaps slight headache, and vague pains in the limbs, but nothing abnormal on physical examination. One such patient, a girl, was discharged with the diagnosis grippe. On the evening after discharge she had a rise of temperature and severe precordial pain, and returned to the hospital the next day with a dry pericarditis. Later an effusion developed and an endocardial murmur. After two weeks she had severe pain in the knees and shoulders, lasting two days. Fever persisted four weeks. About the time the temperature reached normal she developed chorea.

CHOREA.

The frequent occurrence of chorea in the series is strongly in support of its rheumatic origin. There were 86 cases of the 300, or 29 per cent., which gave a history of chorea. There is, of course, no evidence that chorea is always a rheumatic manifestation, as a large number of cases were admitted into the hospital during the period without other rheumatic manifestations: Admitted for chorea, 121 cases; existing or previous rheumatic fever, 69 cases; no rheumatic history, 52 cases.

Even including all the cases admitted for chorea during the same period, 57 per cent. were in patients with some rheumatic history.

The chorea does not seem to bear any definite relation in time to the infection. At times it occurred in previous attacks, at other times it shortly preceded or followed the manifestations of infection.

It is possible that chorea may be the result of the action of toxin on the nervous system, as diphtheritic paralysis is the result of the action of the diphtheria toxin. This theory, however, does not explain the cases in which chorea precedes the cardiac or articular manifestations. With the tendency of rheumatic fever in children to take the form of recurrent mild infections, it is possible that there may have been an infection preceding the chorea, which was overlooked. It is also possible that such a mild infection might not even produce fever, or any signs of infection, other than the mere physical sign of endocarditis. This would explain the development of a cardiac murmur in cases of chorea under observation in which there is no other evidence of an active infectious process. That chorea can occur without evidence of rheumatic infection, or as the result of fright or other recognizable condition is no argument against the analogy of chorea with diphtheritic paralysis, as a manifestation of specific infection, for neuritis and paralysis can also occur from other causes.

THE THROAT IN RHEUMATIC FEVER.

A great deal of attention has recently been directed toward the throat in rheumatic fever, both as the portal of entry for and the seat of development of the infecting agent. As early as 1789, Eyerlen, in his "*Materia Rheumatica and Tonsillas Deposita*," recognized a relation between rheumatism and the tonsils. Roos,³⁶ in 1894, called attention to the frequent connection of acute rheumatism and tonsillitis. Bloch³⁷ called attention to the same point in 1898. The experimental work of Widal and Bezancon,⁸ Meyer¹³ and of Menzer¹⁴ has already been mentioned.

There is great difficulty in obtaining clinical evidence on this point. Children frequently do not complain of sore throat, even when considerable inflammation is present, and always the evidence of the parents is of little value. In spite of these obstacles, during the latter part of the period covered by this series of cases, special pains were taken to collect data on this point. In the whole series of 300 cases, 98 gave a history of sore throat: Frequent previous attacks, 65 cases; preceded rheumatic manifestations, 13 cases; present at the onset, 20 cases.

SPECIAL PECULIARITIES OF RHEUMATIC FEVER IN CHILDHOOD.

In considering as a whole the acute infection in its varied manifestations making up this conception of rheumatic fever, certain facts stand out as marked peculiarities of the disease in early life:

1. The comparative mildness of the articular manifestations.

2. The relative frequency of cardiac manifestations.

3. The large number of cases in which there are only cardiac manifestations. This number is actually greater than the number of cases having only articular manifestations.

4. The frequent occurrence of endocarditis or pericarditis as the primary manifestation.

5. The most severe manifestations are the cardiac. In acute endocarditis, and to a still greater degree in acute pericarditis, the severity of the case, as measured by subjective discomfort and duration of fever, as well as by danger to life, is greater than in acute arthritis.

6. The marked tendency to recurrent attacks, with varying manifestations. A very large number of the patients had had frequent attacks of fever, with articular or cardiac symptoms, previous to the attack for which they were then under treatment. It is not uncommon for a child to have arthritis at 4 years; a febrile attack with dyspnea and precordial pain at 5; several attacks of fever, joint pain and dyspnea during the next three years; chorea at 9, and again the following year; and finally, acute pericarditis at 11. These recurrent attacks show no particular order of occurrence. Sometimes the first is the most severe; sometimes the mildest. At times chorea occurs first; at times arthritis, at times fever and dyspnea, at times pericarditis. Sometimes joints, endocardium and pericardium are affected together; at other times endocarditis may occur alone, to be followed after months or years, by an articular attack, and later still by a pericarditis. The whole of childhood seems to be a period when, at various times, endocardium, pericardium and articular synovial membrane are particularly susceptible to this infection.

7. A seventh point suggested by a study of the rheumatic fever of early life is interesting in connection

with what is known as "broken compensation." Many of our text-books, in speaking of valvular disease of the heart, say rightly that the lesion is compensated by cardiac hypertrophy, but they go on to say that the symptoms, dyspnea, edema, etc., are caused by failure of this compensation, and mention overexertion as the chief cause of this condition of broken compensation. The throwing of more work on a heart with damaged valves by overexertion, or by an increase in the resistance in the peripheral circulation, is made the most prominent cause of these symptoms.

After studying the cases of so-called broken compensation in my series, I became convinced that as far as this condition occurs in children, the above statements in the books cover only one side of the question, and that not the most important. Obviously, the symptoms can only occur from inability of the heart properly to perform its work. But the idea that the failure is due to the valvular lesion plus more work required does not seem to conform to the facts. In my series 121 cases had signs of so-called broken compensation. Of these, 49 patients were suffering from acute endocarditis only, and 25 from acute arthritis and endocarditis at the time when the compensation failed; that is, in 74 cases the symptoms called failing compensation dated from and accompanied an acute rheumatic fever infection. In 10 further cases there was a history of a shortly preceding infection, so that in 84 cases out of the 121 the cardiac symptoms seemed to be caused by or at least to follow acute infection. In only 2 cases was there any history of overexertion. On the other hand, none of the cases of chronic endocarditis without heart symptoms gave a history of any shortly preceding infection.

A natural inference is that the principal cause of the development of cardiac symptoms in childhood is not overexertion, but a fresh infection. That this fresh infection is effective as a cause by increasing the valvular stenosis or insufficiency is not probable, because many cases of acute endocarditis show severe cardiac symptoms from the start, before there is much probability of marked damage to the valves, and sometimes even before the advent of the murmur. While it is possible that any infection may increase the work thrown on the heart in some way, this would hardly explain the specially severe cardiac symptoms so frequently seen in the acute endocarditis of early life. It seems probable that the heart in these cases of rheumatic fever is weakened by an accompanying myocarditis, in accordance with the well-known fact that myocarditis is a frequent accompaniment of acute endocarditis. In such a condition it is very possible that the rôle of the valvular lesion in contributing to the failure of the heart to do the work required is very slight; and in any event it seems that the view of disturbance of a condition of compensation developed on account of a valvular lesion by increased demands on the heart, as a cause of symptoms, is by no means established. The term cardiac insufficiency is suggested as preferable to broken compensation in that the latter points to the damaged valve as the chief agent in producing the condition. However its mode of action, the evidence is very strong that the fresh acute infection is usually the chief agent.

A number of convalescent cardiac cases are from time to time sent to the Convalescent Home of the Children's Hospital at Wellesley. At one time these children were allowed to play and run about more than is proper for cardiac cases. I know of no child who developed cardiac insufficiency from overexertion at the Convalescent

36. Roos: Berl. klin. Wochft., 1894, Nos. 25 and 26.

37. Bloch: Münch. med. Wochft., 1898, Nos. 15 and 16.

Home. Several patients did develop such symptoms there, but in every case the symptoms were ushered in by a fresh febrile attack, a recurrence of infection in short, both with and without articular manifestations. I do not wish to imply that precautions against over-exertion in cardiac cases should be relaxed, but simply to point out that recurrence of infection, being so important a cause of heart failure, at least an equal amount of attention should be paid to so regulating the child's life as to exposure, etc., as to minimize as far as possible conditions favorable to fresh infection with rheumatic fever.

DIAGNOSIS OF RHEUMATIC FEVER.

The figures in this series of cases give some indication of the frequency of occurrence in early life of arthritis, endocarditis and pericarditis of a certain type, as compared with other types. The particular type described above is overwhelmingly the most frequent. Given, therefore, any case of acute arthritis, especially if there are the physical signs of endocarditis, or given any case of acute infectious disease in which there are no other evidences of localization than in the endocardium or pericardium, the probability is strong in childhood that it is a case of rheumatic fever. A history of previous cardiac or articular attacks, or of recurrent attacks, strongly increases this probability. Chorea and sore throat, either previous attacks, or as an accompanying manifestation, also increases this probability. On the other hand, the clinical picture seen in endocarditis and pericarditis due to the pyogenic cocci, is very different from that in the type which forms the majority of cases, and can easily be recognized. In those much less common cases of endocarditis, pericarditis or arthritis which occur as complications of other recognized infections, the cause of the cardiac and articular processes is obvious. In cases with no recognized other cause, we probably have to do with rheumatic fever.

PROGNOSIS.

In this series of 300 cases, 55 died in the hospital, making the mortality of rheumatic fever in hospital cases, judged from this comparatively small number, as high as 18.33 per cent.; 10 fatal cases, however, showed no evidence of acute infection while in the hospital, although in most of them the beginning of cardiac failure which ultimately caused death dated from an infectious attack. Taking the 223 cases of acute infection in the series of which 45 were fatal, the mortality was 20 per cent. This figure is much higher than in adults, where acute infection is more exclusively confined to the joints. I think many physicians do not realize that in a case of rheumatic fever in early life the chances of death may be as high as one in five.

If all the 300 cases were followed throughout their subsequent years of childhood, I think it very possible that the mortality might be still higher: Cardiac failure was the cause of death in all cases. Of the articular cases 7 died, or 7 per cent. Of the acute endocarditis cases, 20 died, or 14.3 per cent. Of the acute pericarditis cases, 18 died, or 31 per cent.

The fatal articular cases all had acute endocardial manifestations. As a whole, they represent the mildest form assumed by rheumatic fever in early life. But in such a case with cardiac symptoms the prognosis is the same as in the cases of acute endocarditis. The severest form is pericarditis, with a high mortality.

Only 17 patients in the entire series were discharged well. The remainder were discharged with a valvular lesion.

THE RELATION OF THE CLINICAL PICTURE IN CHILDHOOD TO THE THEORIES OF THE NATURE OF THE DISEASE.

The chief characteristic of the clinical picture in childhood is its definiteness and the strong resemblance of all the cases one to another. The general course, range of temperature, symptomatology, recurrences, associated manifestations, give a picture strongly suggestive of a definite disease. If this clinical picture merely represents a specific reaction of the body to various causes, it must at least be admitted that the reaction is very specific. It rather suggests an infection, and more, a specific infection, in which the infecting agent has a marked tendency to select constantly, and over and over again certain definite parts of the body. Whatever its portal of entry, it localizes itself with a remarkable constancy in the synovial membranes of the joints, in the endocardium and in the pericardium. When it has so localized itself, it produces a definite and constant febrile reaction, and definite and constant lesions, chiefly a non-purulent inflammation. A certain number of these causes came to autopsy. The finding showed only minor deviations, being essentially the same pathologic process, which has for years been described as occurring in acute rheumatic arthritis, endocarditis and pericarditis.

That these lesions can be produced by the organisms is possible, but remains to be further proved. There is at least a marked contrast between the lesions in all these cases and those produced by the ordinary virulent pyogenic cocci. The severity of the cardiac manifestations points toward a virulent infection, and hence is against the view that the manifestations are due to a variety of pyogenic cocci in an attenuated form.

I think the definiteness of the clinical picture alone is sufficient justification for regarding rheumatic fever as a definite entity, and most probably a specific infection to be placed in the same category as scarlet fever and measles.

Whether the specific cause has been discovered is another matter. The fact that the *Micrococcus rheumaticus* has not been proved to be the specific cause must be admitted, but it must also be admitted that its claims are strong. When those who deny that it is the specific cause base their denial on the ground that specific results in animal inoculations are not to be taken as a criterion of specificity, and require a further method of differentiating this organism, they are right, but are only pointing out that the chain of proof is not absolutely complete. If we assert only the probability of its being the specific cause, a probability based on the experimental and clinical evidence in support of this position, a certain burden of proof is thrown on those who believe otherwise. In so far as they can show that other organisms give experimental and clinical results identical with those constantly produced by the micrococcus isolated from cases of rheumatic fever, they lessen this probability. Up to the present so little has been done in this direction that there is justification for the provisional acceptance of the *Micrococcus rheumaticus*.

[FOR THE DISCUSSION ON THE PAPERS OF DRS. SNYDER AND DUNN, SEE PAGE 542.]

Mind in Medicine.—Medical psychology, therefore, can no longer be limited to pathologic psychology, but must embrace the whole study of the connection of the sound mind with disease both in cause and cure. It is, indeed, true that this connection is everywhere tacitly acknowledged, everywhere exploited by quacks, and yet that it is nowhere taught in our schools or scientifically studied.—Schoefield, in *Brit. Med. Jour.*