In closing, I must mention briefly the general lines of treatment. Having no specific treatment, we can but treat symptoms, and our efforts are directed mainly to supporting the strength of the patient. Hydrotherapy, quinin, iron, strychnia, salicylates, guaiacol, mercurials, and various nuclein preparations are all used. Mention should also be made of the Credé ointment, the anti-streptococcus serum, the bleeding and subsequent intravenous injection of salt solution, all of which at times seem to yield results.

DISCUSSION.

DR. WILLIAM KRAUSS, Memphis, Tenn.—I have observed a number of times, particularly in the autumn, a number of cases of fever neither malarial, typhoid nor tuberculous, which, upon blood examination, showed only leucocytosis. The paper of Dr. Preble was very instructive to me, and I shall follow up those lines and try to verify the observations made.

DR. LOUIS FAUGÈRES BISHOP, New York—We often have cases in which we fail in making a diagnosis, because we insist upon putting them in long-established groups. I have been impressed with the thought that there should be a group of cases that, in popular parlance, we might call blood-poisoning. Such are the patients who run a long course of irregular temperatures after we exclude every possible disease that can be diagnosed by the microscope or by physical examination and about which we can not arrive at any definite conclusions. But such a group must not be made a dumping-place for undiagnosed cases. The suggestion of Dr. Preble that some of these are cases of typhoid is a good one. I was called in consultation to see a case in New Jersey a few months ago. The patient was a woman, of middle life, who had a great deal of pelvic trouble. She was a chronic pelvic invalid. A very clever gynecologist who examined her said there were many adhesions, but no pus in the pelvis. The woman was sick for a long time running this irregular temperature. Then it was thought it might be tuberculosis. I examined the chest and found only a slight amount of dullness at the base of the right lung; I suspected there might be pus there, but the needle introduced at that point did not show any. A careful restudy of the case led me to suspect typhoid fever which had attacked a person already an invalid. I then took some of the blood and got a very good Widal reaction. The case was entirely atypical. It was simply a chronic invalid who had commenced to run an irregular temperature. On that reaction, without symptoms of typhoid, I made the diagnosis of typhoid fever and gave a good prognosis. The woman recovered after prolonged nursing and treatment.

DR. N. S. DAVIS, JR., Chicago—The obscure cases which are difficult to diagnose and difficult to describe are made much clearer by what Dr. Preble has said regarding them. There is one point that I wish to emphasize, i. e., the liability in certain cases of infection through the gastrointestinal tract. This important point has been impressed upon my mind by several instances. I will call attention to two in the paper which I am to read here to-morrow. Another point is the frequency with which, in these cases, cardiac symptoms arise. In prolonged fevers of obscure origin we should watch for an endocarditis. And yet, when we ultimately discover the existence of a cardiac lesion we are not justified in concluding that we have had to do from the beginning with ulcerative endocarditis, but with a septic disease, in the course of which endocarditis has developed. I speak of this particularly, because in meeting other physicians and talking over cases, especially cases of malignant endocarditis, I find that it is usually an endocarditis superimposed on a general sepsis.

DR. ROBT. B. PREBLE, Chicago—In regard to the Widal reaction, I would be unwilling to make a diagnosis upon it; nor would I be willing to exclude typhoid fever in the absence of this reaction. I believe, though, that it can be obtained in the vast majority of cases.

Iron in Eggs for Invalids.—The *Gazzetta degli Osp.* of October 18 states that Dr. Viray feeds hens with salts of iron incorporated in their food. The fowls tolerate the iron perfectly and eliminate it mostly in their eggs, which are thus delicately medicated.

ACUTE ARTICULAR RHEUMATISM—ITS ETIOLOGY AND PATHOLOGY.*

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PHILADELPHIA.

With the exception of a small and gradually lessening number, modern writers consider acute articular rheumatism an infectious disease. The nervous origin of rheumatic arthritis, proclaimed by the elder J. K. Mitchell¹ in 1831, and defended by Charcot, and more recently by Friedländer², who places the primary lesion in the medulla oblongata, has been abandoned. More tenacious is the lactic acid theory of Prout, Todd, Fuller, Richardson, and others, according to which rheumatism is dependent on an excess of lactic acid in the blood. I need not bring forth any arguments against this theory. As MacLagan³ says, the advocates of the lactic acid theory have taken one of the phenomena of the disease and raised it from its normal and subordinate position as a symptom to the rank and dignity of an exciting cause. Edwards,⁴ Eisemann, and lately Haig,⁵ have modified the acid theory, and replaced the lactic with uric acid, the retention of which in the system Haig considers to be the causative factor in rheumatism. He presupposes the existence of an excess of uric acid in the blood, and that this is driven into the joints, owing to a diminution of the alkalinity of the blood. But not only has he failed satisfactorily to prove that uric acid accumulates in the joints in acute articular rheumatism, he has also drawn a conclusion that is scarcely warranted even in the premise—a pre-existing "uricacidemia"—is accepted; for it is just as likely that if an excess of uric acid does occur in rheumatism, it is a consequence—a symptom, merely—and not the cause of the disease. The very fact that Haig finds uric acid retention in so many affections suggests the thought that, like fever, leucocytosis, or albuminuria, the hypothetical "uricacidemia" is only a symptom.

There are two ways of proving that a disease is infectious: by direct evidence, that is, by the unequivocal demonstration of the causative agent; and by indirect or circumstantial evidence, which rests largely on analogy. I may say that in the case of acute articular rheumatism we are not yet in possession of direct or prima facie evidence; in other words, I do not believe that the cause has been definitely discovered. It is true that a large number of investigators claim to have found the specific micro-organism, but in no instance has indubitable proof of the causative relation of the particular organism been brought. If it were that the micro-organisms hitherto found could not be cultivated, we might, as in the cases of leprosy and malaria, rest content with the mere demonstration of their invariable presence, and not demand, for final proof, the production of the disease in the lower animals; but the organisms which have been found are, for the most part, readily cultivated, and yet it has not been possible to reproduce in the lower animals the typical picture of rheumatism as we see it in man. This fact, however, does not render invalid the proposition that acute articular rheumatism is an infectious disease. The same is true of syphilis, scarlet fever, smallpox, chickenpox, and possibly of whooping-cough and measles; and yet no one doubts their specific nature. The circumstantial evidence in favor of the infectious origin of rheumatism

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is as strong as in the diseases which I have mentioned, although two of their important characteristics are not possessed by rheumatism, viz.: contagiousness and immunity after one attack. Regarding contagiousness, however, I may add that Wagner,⁶ of Leipsic, believed it possible, and made it his practice to isolate cases of acute rheumatism in the hospital. As for immunity, it is such a vague concept, and its duration in different diseases varies so much, that it is not out of the realm of possibility that there is a brief period of immunity after one attack of rheumatism, perhaps as long as that obtaining in diphtheria, which, as is well-known, is of comparatively brief duration.

I wish now to take up the facts which, although they do not demonstrate it absolutely, point in an unmistakable manner toward the infectious origin of acute articular rheumatism.

Fever.—While fever may be due to causes other than infection, it is most natural in the case of a continuous fever, like that of rheumatism, to look for a micro-organismal agent. The occurrence of hyperpyrexia is also strongly suggestive of the activity of toxin-producing organisms.

Chill.—At times, as I have seen in several instances, the disease is ushered in by a distinct chill or rigor. I do not mean by this the chilling of the surface which often precedes an attack of acute articular rheumatism, but the initial chill which inaugurates so many infectious diseases, and which indicates that the period of incubation is passed and invasion has begun.

The Occasional Epidemic Outbreak of the Disease.—McClymont⁷ and Newsholme⁸ have both called attention to this epidemic incidence, and Risse⁹ has reported the concurrence of an epidemic of acute articular rheumatism with one of influenza. Edlefsen,¹⁰ McClymont, and Stoll¹¹ have observed house epidemics of rheumatism, such as we occasionally find in croupous pneumonia, an unquestioned infectious disease.

The Seasonal Influence.—Malaria, typhoid-fever, pneumonia, and influenza vary in their prevalence with the time of year and with meteorologic conditions. The same has been proved to be true of acute articular rheumatism by Edlefsen, Newsholme, and many others. The first-named has shown that, as a general rule, the incidence of acute articular rheumatism increases with the diminution of the precipitation and decreases with increased rainfall. Newsholme confirms this, and finds that rheumatism is more prevalent in dry seasons, when the ground-water is low, than at other times. He found that at Croydon and at Brighton epidemics occurred at a time when the sub-soil water was very low, especially if there had been more than one year of deficient rainfall. In London a low sub-soil water and a high earth temperature are the conditions in which the rheumatic poison finds its most favorable opportunities for activity.

The Complications of Rheumatism.—They are the complications that are pre-eminently characteristic of infectious diseases—endocarditis, pericarditis, pleurisy, and hyperpyrexia. So common is the first that it may readily be considered a manifestation of the disease, rather than a complication. Acute endocarditis is certainly an infectious process under practically all the conditions in which it occurs. It may not always be due to the bacteria themselves, for it can probably be produced by bacterial toxins. In rheumatism it sometimes happens, especially in the case of children, as pointed out by Cheadle,¹² that the endocarditis precedes the rheumatic manifestations or begins simultaneously with them.

Thus, in a case of my own, a boy of 8 years, there had been pain and swelling of the knees for less than twenty-four hours when I found a well-marked systolic mitral murmur. Such occurrences strongly suggest that the endocarditis and the joint manifestations are, under these circumstances, due to one and the same cause. Phlebitis, seen in many typical infectious diseases, may also occur in rheumatism, as pointed out by Remlinger,¹³ Censier,¹⁴ and others.

Portal of Entry.—The majority of infectious diseases have a demonstrable portal of entry through which the exciting cause invades the body, and if we can prove that in rheumatism there is also a portal of entry we have strongly fortified the infectious theory of the disease. Haygarth,¹⁵ it seems, was the first to state the connection between sore-throat and rheumatism. Trousseau¹⁶ also upheld it, and since then a number of writers have referred to the subject, among others Roos,¹⁷ Bloch,¹⁸ Buss,¹⁹ Gallois,²⁰ Sacaze,²¹ Pettersohn,²² and lately Packard,²³ who has summed up the relevant literature in an admirable manner. As to the frequency with which tonsillitis or, to use the more comprehensive term, angina, precedes rheumatism, it is variously stated: Stoll¹¹ gives 5 per cent.; Lebert,²⁴ 5 per cent.; Gerhard,²⁴ 20 per cent.; Fowler,²⁵ 30 per cent.; Whipham,²⁶ 24.12 per cent., and Stewart,²⁷ from 70 to 80 per cent. Some of these figures are unquestionably too large. They include, in many instances, attacks of angina long antecedent—in Whipham's statistics by as many as fourteen years—to the rheumatic attack.

I recently saw a case with Dr. T. R. Currie, of Philadelphia, which seemed to illustrate the pharyngeal origin of rheumatism. The patient was a man aged 31 years, who had had an attack of acute articular rheumatism of seven weeks' duration at the age of 19, and another at 21, which lasted only a few days. Two weeks before I saw him he had been taken ill with sore-throat, and this was followed by an attack of rheumatism of most severe type. In addition to the involvement of the joints, he had an acute pleurisy on the right side and was bathed in the most profuse sweats that I have ever seen in rheumatism. There was no evidence of endocardial or pericardial involvement, but the poor heart-action suggested myocardial changes.

Packard is strongly of the opinion that rheumatic polyarthritis, as well as other manifestations of what Cheadle has called the rheumatic group, may be brought about through the medium of the tonsils; but infection may also enter through other channels. Gradenigo²⁴ and Bloch¹⁸ have each recorded cases of acute articular rheumatism following otitis media, and recently a case was reported by Kronenberg²⁸ in which an attack of acute articular rheumatism followed an operation on the nose. Bloch cites instances of rheumatism following felon, furuncle, fistula in ano, and vaccination, and in a case of Sacaze's²¹ the attack occurred in a patient who had an infected wound of the foot.

It might be averred that the arthritis in these cases was not rheumatic, but pyemic. In some instances, no doubt, this was true, but hardly in all of them. The course of rheumatism and the influence of the salicylates generally serve to distinguish the disease from the ordinary pyemia. Moreover, the latter has a tendency to lead to suppuration, while rheumatism scarcely ever has; indeed Hué²⁹ states that no true case of suppuration in rheumatism is on record. He found a total of forty-four alleged cases, but in all of them he discovered internal evidence that made it improbable that they were examples of rheumatism. Körte,³⁰ in 1880, de-

scribed six cases of suppuration in rheumatism, all occurring in women. It seems strange that one observer should see so many examples of what must be considered a complication of maximum rarity.

There is another possible source of infection in rheumatism—the intestinal tract. I have no evidence to offer in proof of this point, but I believe that careful attention to it may yield some interesting results. That the intestines may at times be the infection atrium has also been suggested by Chvostek.³¹

Direct Transmission from Mother to Offspring.—This, if proved, would be a powerful argument in favor of the infectious nature of rheumatism. Two rather convincing instances are on record—those of Pocock and Schaeffer, which are quoted by Singer.³²

Contagion.—I have already referred to the subject of contagion. One indubitable example would be decisive, but there are so many possibilities of error in interpreting cases of contagion in rheumatism that we must look upon reports of such an occurrence with scepticism.

BACTERIOLOGY.

I want now to dwell briefly on the bacteriology of acute articular rheumatism. Diligent search has been made for a micro-organismal cause. Some, as MacLagan³ and Kahler,³³ have thought that this would be found in a miasm allied perhaps to that of malaria, but it seems to me that the resemblance between malaria and rheumatism is very slight.

Bacteriologists have examined the blood, the joints, the urine, the cerebrospinal fluid, and the lesions of the heart, pleura, pericardium, etc. Organisms were found by earlier observers, such as Guttman,³⁴ Wilson,³⁵ Mantle³⁶ and Bouchard and Charrin.²¹

Guttman found the staphylococcus aureus; Wilson found a bacillus in the pericardial fluid; Mantle, a bacillus and a micrococcus in the joints in seven cases, and in the blood in sixteen, both in acute and chronic and in gonorrheal rheumatism. Bouchard and Charrin discovered the staphylococcus albus, but only in subacute and chronic cases. Anyone who has made cultures from the living body knows how difficult it is to eliminate the possibility of contamination, and I am inclined to think that the observers I have named, whose studies were made before the present more exact methods were in vogue, dealt with accidental germs. This is the view also taken by St. Germain.³⁷

The observations of Sahli,³⁸ made in 1892, attracted much attention. He found the staphylococcus citreus in the synovial membrane, pericardial exudate, endocardial vegetations, blood, and bronchial glands of a case of articular rheumatism, but not in the fluid of the joints. He concluded that rheumatism was an abortive form of pyemia.*

Very painstaking studies were made by Singer,³⁹ of Vienna, who, distrusting the examination of the joints and believing that if the organism were in the blood it would be present in such small numbers that it might escape observation when culture was made, examined the urine, hoping that the bacteria at work in the body would be in part eliminated through the kidneys. The elimination of micro-organisms by the kidneys has been abundantly proved—experimentally by Biedl and Kraus⁴⁰; clinically, by Singer, Gwyn⁴¹ and others. Singer's examinations yielded positive results in 59 out of 84 cases of acute articular rheumatism. He found the different forms of staphylococcus and the streptococcus pyogenes, and believed with Sahli that

rheumatism is an attenuated form of pyemia. Chvostek³¹ took exception to Singer's observations on the ground that organisms eliminated through the urine might have entered accidentally through the mucous membranes, and might have no bearing whatever on the primary disease. His own bacteriologic studies were entirely negative.

Wassermann⁴² quite recently, in a case of rheumatic chorea, found a streptococcus which he thinks differs from the streptococcus pyogenes. It grows best in a strongly alkaline medium, which the ordinary streptococcus scarcely tolerates, and is most easily cultivated in hog bouillon containing 2 per cent. of peptone. The observation was carefully made in a single case, but no animal experiments were undertaken to test the virulence of the germs, and I think that we must keep our opinion under advisement.

Riva,⁴³ on a serum made from horses' joints and synovial fluid, obtained the growth of a polymorphic, spore-bearing organism in eight cases, cultures being made from the joints, the pleural fluid, and the blood. It did not grow on ordinary media. As far as I can determine, no animal or other control experiments were made, yet Riva concludes that he has found the cause of rheumatism. I think that we must adopt the same attitude in this case as in that of Wassermann's discovery.

More important than any of the preceding is the observation of Achalme,⁴⁴ who, as far back as 1891, discovered an anaerobic bacillus at autopsy in the body of a man who had died of cerebral rheumatism on the fourth day of a second attack of acute rheumatic polyarthritis. In a second case of rheumatism examined six years later he found the same organism, and subsequently discovered it in the blood of the living patient in six cases—four times pure and twice associated with a micrococcus. The best culture-medium was found to be milk or a mixture of equal parts of milk and bouillon. Animal inoculation produced, among other changes, a marked edema, the effects being the more intense the younger the animal. Guinea-pigs died in from twenty to thirty-six hours, but when inoculated with some of the edematous fluid they perished in ten hours. Achalme does not state whether or not any joint symptoms were present in the animals.

An anaerobic bacillus had been found in 1892 by Lucatello,⁴⁵ who seemed to be unaware of the prior work of Achalme. The latter's observations were confirmed in 1897 by Thiroloix,⁴⁶ who found the organism in the blood *intra vitam* in five cases of rheumatism; also in one case in the pleural fluid, as well as in the blood. He noted no changes in the joints after intravenous injection in rabbits, but intra-articular inoculation produced marked edema of the joints and of the entire extremity. With the edematous fluid from guinea-pigs he obtained in rabbits, arthritis, pericarditis, and pleurisy. The blood from the human subject had no agglutinating action on the organism; that from the infected guinea-pig had at times such an action. The growth of the organism was markedly retarded by the addition to the culture-medium of salicylates in doses smaller than those necessary to check the growth of other bacteria.

Reports of the discovery of the same bacillus in rheumatism have since been made by Petit and Achalme,⁴⁷ Papillon,⁴⁷ and Carrière,⁴⁸ and recently also by Sawtchenko.⁴⁷ The latest observations are those of Pic and Lesieur,⁴⁷ who isolated the organism during life from a case of cerebral rheumatism. They found that it grew best in sterile milk, but only under strictly anaerobic conditions. It did not grow on gelatin, but developed

*Der Rheumatismus ist eine Staphylococcenkrankheit." Stewart had expressed the same view as early as 1881.

in secondary culture on bouillon. It is a long, slender bacillus, slightly motile, and stains by Gram's method. It is pathogenic for guinea-pigs, rabbits, and mice. In guinea-pigs it produces dyspnea, tachycardia, slight fever, and at the moment of death generally a sub-normal temperature. Suppuration does not occur. At the point of inoculation a gelatinous edema, poor in leucocytes, but rich in bacilli, develops. There is also pericarditis, but no arthritis. Injection of the edematous fluid into rabbits is fatal in ten hours, the principal lesions being focal necrosis of the liver, edema of the lungs, pleurisy, and pericarditis. The liquid products of the micro-organism seem devoid of toxic properties, but after their injection the animals appear to be immune to otherwise lethal doses of the bacteria. The bacillus is not agglutinated by the serum of patients suffering from acute articular rheumatism or chorea.

Triboulet and Coton⁴⁹ in eleven cases isolated a diplococcus which was associated with Achalmé's bacillus in two cases. While not asserting that the diplococcus is the cause of rheumatism, they deny the specificity of the bacillus of Achalmé, believing that the latter is present only in complicated cases.

Of the various micro-organisms that have been discovered in rheumatism, the bacillus of Achalmé has apparently the strongest claims to be considered the cause, but I do not think the time is ripe for a definitive opinion. In the first place, the various experimenters have not succeeded in producing a disease in animals comparable to articular rheumatism in man; secondly, the organism has not been found with sufficient constancy. Thus, Pic and Lesieur failed to find it in seven instances out of the eight examined by them.

Summing up the bacteriologic evidence then, it may be said that the cause of rheumatism is not definitely determined.

We may consider rheumatism etiologically from two points of view; either as a group of diseases having a multitude, or at least a variety, of causes, which is the view taken by Singer, Chvostek, and others; or we may hold that there is a distinct form of acute polyarthritis, to which the term rheumatic may be properly applied, which has a single cause. The latter seems to me the more correct attitude. I believe that we may have in the case of joint affections a condition somewhat analogous to the pseudomembranous inflammations of the throat. Formerly these were nearly all classed together under the name diphtheria, but now we know that there is only one disease diphtheria—that due to the Klebs-Loeffler bacillus; nothing else deserves the name. With improved methods of investigation we shall no doubt find the organism which is the specific cause of rheumatic polyarthritis.

PATHOLOGY.

I have taken so much time in the consideration of the phases of the subject hitherto discussed that I must make my remarks concerning the pathology of rheumatism very brief.

Joint Inflammation.—In a majority of instances the fluid has been found sterile and the synovial membrane and the periarticular tissues free from germs.* This

*Since reading this paper I aspirated the ankle-joint in a young colored woman who was suffering from typical acute articular rheumatism. Only a few drops of fluid were obtained and these were at once placed into a mixture of milk and bouillon (two tubes) and glucose agar (one tube). The glucose agar had been previously melted. After inoculation it was quickly solidified and overlaid with an equal bulk of fluid glucose agar, which was then also congealed. This placed any bacteria present under moderately anaerobic conditions. No growth was obtained on the glucose agar, and of the milk-bouillon tubes one remained sterile; in the other a few diplococci, the result of contamination, developed.

leads us to the conclusion that the joint changes are probably toxic in origin, a view emphasized by Chvostek, who points out the resemblance of the rheumatic changes to the occasional joint swellings seen after the injection of diphtheria antitoxin. However, anaerobic cultures have not often been made and might lead to different results from those hitherto obtained. The synovial membrane in acute cases is swollen and injected and at times somewhat ecchymotic, and may be covered with a fibrinopurulent exudate. The fluid is more watery than the synovia and is reddish or yellowish in color. Ollivier and Ranvier⁵⁰ found proliferation of the cartilage cells and capsules and circumscribed ulceration.

Rheumatic Nodules.—These were first clearly described by Jaccoud and later by Troiser,⁵¹ although they may have been recognized long ago by Froriep. They usually occur in children, but also in adults, as multiple, pin-head or pea-sized bodies—sometimes a little larger—are situated under the skin and are tender on pressure. Microscopically they show a necrotic focus with round-cell infiltration, dilatation of the lymphatics, and thrombosis of the small arteries. Riess⁵² believes that they have an embolic origin.

Endocarditis.—You are familiar with the small vegetations that dot the valves, usually just behind the free edge at the lines of contact, and little need be said regarding their general structure. Recently, however, Achalmé⁵³ has made a contribution to the subject of their minute anatomy that merits attention. He divides rheumatic endocarditis into three phases: 1. The edematous microbial phase. 2. The proliferative phase. 3. The cicatricial phase.

The naked-eye appearance of the first stage in one case that came to autopsy a few hours after death consisted in a thickening of the valves to five times their normal thickness; the auricular surface of the valve had lost its polish, and there were slight elevations a few mm. above the free border. The myocardium was softened and of a dead-leaf color. Microscopic examination revealed an edema of the interstices of the tissue of the valve, together with an enormous bacterial invasion and an extensive transformation of the connective-tissue cells into Ehrlich's mast-cells. The edema occupied the entire valve, but predominated at certain points. Least affected was the fibrous expansion of the ring, but the cellular tissue extending from it into the auricle was markedly infiltrated, and the elements almost completely dissociated. The elastic tissue of the auricular layer of the valve was much thickened by the edema and had lost its homogeneous aspect. This was also true of the elastic layer at the free border of the valve. The bacterial invasion with Achalmé's bacillus was greater than that seen in any other disease. There might have been some growth after death, although one autopsy was made four, and the other twenty-five, hours post-mortem. The transformation of the connective tissue-cells into the cells of Ehrlich is the most characteristic feature of rheumatic endocarditis. It is much more marked than is seen in any other affection. I have myself observed these cells in rheumatic pericarditis and found them penetrating deeply into the myocardium. The granules which they contain, and which are splendidly brought out by thionin, are often so large as to resemble micrococci.

The proliferative phase is distinguished by a proliferation of the flat cells of the superficial elastic layer. All the space previously distended with fluid becomes filled with mononuclear cells which displace the elastic

bundles. The latter become granular and indistinct and their affinity for orcein is lessened. New blood-vessels form, the walls of which are friable and are apt to rupture at the base of the vegetations, leading to minute hemorrhages. The characteristic feature of the second period of the second phase is the appearance of vegetations. According to Ziegler, the primary process in their formation is the deposit of fibrin on the valves. According to Achalme, the first change is an alteration in the tissue of the valve, consisting in a dissociation of the superficial elastic tissue and the formation of a prominence on which fibrin is subsequently deposited. The next step consists in a proliferation of the endothelium of the blood-vessels leading to a narrowing of the lumen. Proliferation of the cells then ceases and cicatrization begins, and brings about the well-known results: stenosis and insufficiency of the valvular orifice. As the earliest changes in the valve do not produce a murmur, we may infer that endocardial lesions are more common than is clinically demonstrable. It is probable that the muffling of the heart-sounds noticed so often is due to edema of the heart-valves, and indicates the earliest organic changes.

As to the cause of the endocarditis and of the inflammation of other serous surfaces, as the pleura, the pericardium, and possibly the peritoneum, it is probably the same as that giving rise to the joint inflammation, but it is conceivable that under the damaging influence of the toxins of the rheumatic germ the heart-valves become prepared for the reception and activity of the common micro-organisms of mixed infection—the streptococci, staphylococci, and pneumococci. This would be in keeping with what we know of other diseases, such as typhoid fever and diphtheria, in which endocarditis may be due to specific organism of the disease, but is more often caused by secondary infection, the toxins bathing the valves having facilitated the invasion of pyogenic bacteria.

Rheumatic Pneumonia.—This has been described, but I have never seen an instance. Lebert⁵⁴ found only 2 in 140 cases, although Valliard⁵² makes the proportion very much greater; Stoll¹¹ gives the percentage as 1.1.

Nephritis occurs occasionally, but presents nothing characteristic. I have already referred to the occurrence of *phlebitis*, to which attention has recently been drawn by Censier.¹⁴

Cerebral Rheumatism.—The pathology of so-called cerebral rheumatism is unknown. In a case reported by Soques,⁵⁴ in which there was also hyperpyrexia, the blood and cerebrospinal fluid at autopsy were sterile, and injection of the latter fluid into animals was without effect. Souques believes that the cerebral localization of the disease is favored by a neuropathic taint.

Changes have been described in the spinal-cord—rheumatic myelitis—but the cases hitherto reported are in no way convincing.

CONCLUSIONS.

The following conclusions, based on what has been said before, seem to me to be warranted:

1. Acute articular rheumatism is a specific infectious disease.
2. It is not an attenuated form of pyemia in the sense that it is due to pyogenic organisms of reduced virulence.
3. Its true cause has not been definitely discovered, although the bacillus of Achalme may prove to be the long-sought micro-organism.
4. A number of joint affections resemble acute articular rheumatism, just as certain pseudomembranous in-

flamations of the throat resemble diphtheria, and various diseases of the lung resemble typical lobar pneumonia.

5. These joint diseases should, wherever possible, be separated as *rheumatoid* or pyemic affections.

6. They are best designated according to their cause, as streptococci, staphylococci, gonococci, or pneumococcal arthritis.

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COLOSTOMY FOR PERMANENT FECAL FISTULA.*

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The operation I desire to demonstrate deals entirely with those inoperable lesions which cause occlusion of the rectum or lower portion of the sigmoid flexure of the colon. Its chief feature is the formation of a very considerable loop of that portion of the colon which is immediately above the artificial anus, making what might be called an artificial sigmoid flexure or store-

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