some unknown way cause a modified tuberculosis and an eruption of lupus erythematosus, is quite tenable after what these injections of tuberculin have taught us.

DR. L. DUNCAN BULKLEY, New York—In regard to the age of patients with lupus erythematosus, I certainly do not believe that the young average observed is owing to the early death of many of these patients by tuberculosis. I believe that it is rather due to the fact that the disease is prone to develop in early life, and then after some attempts at treatment, if not speedily cured, the patients give up, and do not seek further medical aid. I have certainly seen patients 60 years old with lupus erythematosus, and I have watched patients with the eruption for many years. I have had the subject of tuberculosis under thought since a possible connection was first suggested, and have repeatedly examined patients for evidences of the same, in vain. I recall only one patient who had lupus erythematosus, who died of tuberculosis; and this among many hundreds of cases in public and private practice.

DR. M. L. HEIDINGSFELD, Cincinnati—I am by no means favorably impressed with any effort to bring tuberculosis in causal relation with lupus erythematosus. I believe both clinically and histologically the types are separate and distinct forms of dermatoses, easily recognizable and distinguishable from each other. A few cases may have had both affections coincidentally associated, but I believe that the transitional forms, from one to the other, mentioned by Roth and others, have been largely errors in diagnosis, the affection being one or the other, *ab initio*. Personal experience in one or two instances strengthens me in this opinion. I concur with Pick, who states that we are not justified in bringing tuberculosis and lupus erythematosus in causal relation.

DR. J. FRANK WALLIS, Philadelphia—I would like to ask the Doctor in regard to those scrofula cases, whether in their history and in others he has found inoculation to prove that they are tubercular. I have had under my care the past year five or six cases, four in one family. There is no history of tuberculosis. Two other cases that came up later had the same affection, but no history of tuberculosis, and I wondered if any inoculation had been made in any of the cases reported. The cases are still under observation.

DR. H. W. STELWAGON, Philadelphia—I am surprised that the experience of those present is so different from my own, as in the large majority of my cases it has occurred in tuberculous families. I have never seen a case demonstrated as tuberculosis, but I have been thoroughly impressed with the history given me in my cases that tuberculosis has existed in one or more of the remote members of the family, and I think there is a good deal of clinical evidence in medical literature in the same direction.

DR. ANTHONY—In answer to Dr. Hartzell's question, if these cases are included, then tuberculosis is present in 70 per cent. of the cases; if excluded, then tuberculosis is present in about 33 per cent. of the cases where every possible evidence of tuberculosis is considered, such as the death of a relative from tuberculosis, scars in the neck from scrofula in childhood, bone disease of childhood, reaction to Koch's lymph, etc. Where these doubtful evidences of tuberculosis are disregarded, the percentage is much less than 33 per cent.

The cases which I would exclude are those like the following one of Boeck's cases: The axillary glands were removed and found to be tubercular; shortly afterward there appeared on each cheek a sharply defined, symmetrical erythema, which was diagnosed as erythema lupinosum, a transitory erythema which is not the same as lupus erythematosus. Six months later the patient had a swelling of the forearm which the attending physician supposed to be erysipelas, but which proved to be a tubercular periostitis. Then she had the eruption of folliculitis on the arms and forehead, next erythematous plaques on the face, and lastly, more than a year after the beginning of the disease, two small plaques of lupus erythematosus behind the ear.

These two small plaques are permitted to dominate the entire clinical picture; they give a name to the disease and furnish one more case to swell the statistics in proof of the assertion that lupus erythematosus is tubercular.

To my mind these are cases in which tuberculosis produces, among a great variety of lesions, one or two lesions which at the present state of our knowledge can not be distinguished from lupus erythematosus either clinically or microscopically. There are very few cases on record in which there was present in the same patient lupus vulgaris and lupus erythematosus. Brocq has reported one such case.

THE ETIOLOGY OF ACUTE ARTICULAR RHEUMATISM.*

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At the outset, while it may seem trite, it may be well to define the sense in which the term etiology is used as well as to indicate what is meant by the term rheumatism. We here employ the term cause in the same sense as we do in typhoid fever, pneumonia, pulmonary tuberculosis, etc., in which we recognize that aside from the infective agent there must be what some have called the soil, susceptibility to infection varying from extreme susceptibility on the one hand to complete immunity on the other; and that the latter may for certain animals and diseases be variable in degree and also either hereditary or acquired.

In order to limit discussion the term rheumatism will be confined to those cases of non-suppurative arthritis occurring especially in children, but which also occur in the adult. This is the view of Poynton and Paine.¹ They say: "All suppurative arthritis must, we think, be excluded. There may be very possibly a suppurative form of rheumatic arthritis; there may, indeed, be rheumatic suppuration as there are pneumococcus suppurations, but as yet all suppurative conditions must be set aside as a basis for investigation."

We may start with the postulate that it is always an infection. If proof of this be demanded it may be found in the work of Fraenkel² in 1892, Sahl³ in the same year, Lanz⁴ in 1893, Singer⁵ in 1894, Dana⁶ in 1894, Lubarsch in 1896, Achalmé in 1891⁷ and 1897,⁸ Triboulet⁹ and Coyon in 1898, Wasserman, Poynton and Paine¹⁰ in 1900, Meyer¹¹ in 1901, Menzer¹² in 1901, Predtechensky¹³ in the same year. A study of this literature shows that there are four principal views in regard to the bacteriology of acute articular rheumatism.

THE THEORY OF A MODIFIED PYEMIA.

The view of Gustav Singer, first published in 1898, is that it is not a distinct disease, but due to streptococcal or staphylococcal infection; in other words, a modified pyemia. This view he again defends vigorously in a recent communication,¹⁴ in which he holds that in the face of his evidence the search for a specific organism is futile. He maintains that the diplococcus previously described by Wasserman, Meyer, Poynton and Paine and others is nothing but the *Streptococcus pyogenes*. He further maintains¹⁵ that polyarthritis is only an occasional occurrence in rheumatism and that rheumatism can not be produced in lower animals. He claims that because of the peculiarity of the circulation of the joints they may be affected by a degree of virulence less than that required to produce a pyemia and that this attenuation is brought about by passage through the adenoid tissues of the throat. The objections to Singer's views are briefly as follows:

* Read at the Fifty-third Annual Meeting of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee: Drs. Frank A. Jones, George Dock and J. M. Anders.

1. His results have not been confirmed by other observers, especially by Poynton and Paine.

2. He has not demonstrated the organisms in the joints, but only in the heart and lungs.

3. As so well stated by Poynton and Paine,¹⁶ in the five last cases reported by him, the strepto-diplococci were demonstrated in, and isolated from, the tissues, but no experiments were recorded. They say: "This organism Singer calls the *Streptococcus pyogenes* on account of the morphological and cultural characters. Yet the lesions were non-suppurative and our present knowledge of the morphology and cultural characters of several organisms in the coccus group make any assertions on these grounds as to their exact nature a very doubtful proceeding."

4. He does not admit that rheumatism can be experimentally produced in the lower animals.

5. It does not seem reasonable to suppose that micro-organisms which so frequently cause suppuration should be so uniformly attenuated as to always produce such constant and uniform phenomena and always without suppuration.

6. It does not seem quite reasonable to argue that these joints are more vulnerable because of their lessened resistance, and that yet they are as likely as any other structures to be affected by a virulent culture.

7. His hypothesis must carry with it the idea of a constitutional predisposition to suppurative infections.

8. If due to streptococcus infection, the use of the antistreptococcus serum ought to be curative or at least helpful.

ACHALME'S SPECIFIC-GERM THEORY.

The second view is that of Achalme,¹⁷ that acute articular rheumatism is the result of infection by a specific anaërobic bacillus which is of large size and possessed of remarkably variable morphologic characteristics. In the papers published in 1891 and 1895 he claimed that the organism is found with constancy, and that rheumatic fever was produced by it in rabbits. Poynton and Paine¹⁸ failed to confirm the results of Achalme, but found a bacillus similar to the one described by him, which they found to be the lactic acid bacillus. Since then they carefully sterilized their media and incubated it over night.

Fullerton²¹ expresses the belief that Achalme's work was not well done and that it was most improbable that the organisms which he describes has anything to do with the disease. Rist¹⁹ says "that the organism described by Achalme has been found very often in a number of cases, pathologic or not, where rheumatism was out of the question."

Achalme now reviews²⁰ his former work and states his belief that his bacillus is to be found in the healthy individual and that it is an ordinary saprophyte whose development in the blood is favorably influenced by the action of heat or cold.

THE THEORY OF VARIED INFECTIONS.

The third view is that acute rheumatic fever is not due to any particular organism, but is a particular reaction to varied infections. In other words, that we may have a uniformity of lesions with a multiplicity of causes. This is too general and includes too much. It makes rheumatism a synonym of arthritis. For example, it is well known that the gonococcus, the pneumococcus, the bacillus of tuberculosis, or the staphylococcus may cause arthritis, and yet we no longer call these cases rheumatic. Gradually there is being formed a rather larger class of cases designated as arthritis, to which

the appropriate descriptive adjective is prefixed, as gonorrheal arthritis, pneumococcus arthritis, tuberculous arthritis, etc. If this is followed out to its legitimate conclusion it will be a question of simply calling all inflammatory affections of the joints arthritis and then determining the particular organism causing the inflammation. If it shall be found that all those cases of non-suppurative inflammation of a constant type are always due to a specific germ, then for these the term rheumatism may be reserved, but there is no particular advantage in doing so.

THE DIPLOCOCCUS OF POYNTON AND PAINE.

The fourth view is that of Poynton and Paine. In January, 1899, Poynton and Paine²² undertook the study of the bacteriology of rheumatism with the intention, if possible, of confirming the results of Achalme. In this they failed and abandoned the attempt. In eight successive cases, however, they obtained a diplococcus which grew in streptococcal chains, which did not thrive on ordinary agar or on serum agar, grew indifferently on blood agar and appeared to grow best in a liquid medium of milk and bouillon, rendered slightly acid with lactic acid. These organisms were isolated in pure culture on three occasions, from the blood of patients during life who were suffering from acute rheumatic pericarditis; also from the pericardial fluid after death, from the valves of the heart and the throat of the patient; fragments of granulation from the valves of the heart of human beings, and also from the joint exudation, heart-blood, urine from the bladder and cerebrospinal fluid of inoculated rabbits.

These organisms when inoculated into rabbits produce polyarthritis, bursitis and tenosynovitis, multiple non-suppurative valvulitis and pericarditis, plastic pleurisy and pneumonia, a condition of the myocardium analogous to that found in the human heart as a result of rheumatic carditis. There are multiple joint swellings, wasting and pyrexia, thus closely simulating rheumatism in the human being.

When these organisms are passed from animal to animal similar symptoms are produced, but there is a tendency to increase in the severity of the cardiac with lessened severity of the arthritic lesions. The organisms were found, not on the surface, but in the interior of the valves. In one case of chorea the diplococci were found in the perivascular lymph spaces and capillaries of the pia mater and in some parts of the motor areas of the brain.

In September, 1901, they reviewed²³ the bacteriology of the disease and brought the number of cases up to eighteen. They take the ground that rheumatism is not only an infectious disease, but that it is due to a diplococcus infection, and point out emphatically that "the most ardent bacteriologist does not pretend that the presence of a micro-organism explains all the phenomena of rheumatic fever."

The organism described by Poynton and Paine is undoubtedly the same as that previously described by Triboulet²⁴ in 1898, and Apert and Triboulet in the same year and by Wasserman and Malkoff²⁵ in 1899. Litten, as the result of the study of two cases and the inoculation of many guinea-pigs, says²⁶ that he sees no reason why the diplo-streptococcus should not be regarded as the cause of acute articular rheumatism.

Perhaps the views of Pribram²⁷ should be mentioned. He maintains that in most cases cultures from the joints and blood are sterile and that where micro-organisms are present they are due to secondary infection, predisposed to by the damaged joint.

The fact that micro-organisms are not found in the exudate in the joint is not against the infection theory. It is exceedingly difficult and sometimes impossible to demonstrate the tubercle bacilli in tuberculous effusions. So generally is this recognized that their absence is considered diagnostic of the tuberculous origin of the disease. Indeed, in these cases of rheumatism the germs are found in the articular structures, in the structure of the valves and vegetations, and it is only in those cases in which they come to the surface in large numbers that the name ulcerative or malignant endocarditis occurs and that many minute emboli are formed.

According to Hamburger²⁸ the bactericidal power of venous is much greater than that of arterial blood. Consequently, we find endocarditis in the left side of the heart because of its arterial blood. It is well known that the venous engorgement of valvular disease, especially mitral disease, affords almost complete immunity against pulmonary tuberculosis. A large percentage of diabetic patients die of tuberculosis because the formation of sugar restricts the formation of carbonic acid.

Brandenburg²⁹ has shown that alkali occurs in the blood in two forms, that combined with carbon dioxide and that combined with albumin. The latter are very tenacious of their alkali and the bactericidal properties of the blood are augmented by increasing the amount of diffusible alkali in it. Hamberger found that after determining the bactericidal power of the blood, the addition of carbon dioxide releases the alkali from the albumin, increasing the proportion of the alkali, and this augmented alkali tension markedly increases the bactericidal properties of the blood. He suggests that acid intoxications induce variations in the alkali tension. Hegeler, Tsuboi and Stemmetz long ago showed³⁰ that the bactericidal action of serum which has been heated to 55 C. is restored by adding highly diluted alkali.

CONCLUSIONS.

1. Many organisms produce arthritis.
2. Probably all cases of acute articular rheumatism are due to infection.
3. Probably all organisms known to cause either acute articular rheumatism or other forms of arthritis also give rise to other pathologic conditions as well.
4. It would thus seem to be not merely a local infection or inflammation, but a general infection, the commonest seat of the principal lesion being the joints, but also involving the heart, both endocardium, pericardium and muscle, occasionally the meninges and other structures and caused by a diplococcus circulating freely in the blood. The diplococcus seems to be merely a germ capable of causing widespread inflammation, and the joints are included in its sphere of action. Just as in pneumonia, we may have a general infection with local manifestations or inflammatory reaction in the meninges, the pleura, the endocardium, pericardium, post-nasal space, peritoneum, lymphatics and lungs, the latter being probably the usual site of the localizing inflammation.
5. It is quite possible that there is some hereditary diathetic or constitutional peculiarity, the nature of which is completely unknown to us, which may favor infections in general, and possibly that of rheumatic infection in particular.
6. That there may be varying degrees of susceptibility depending on exhaustion, depressing influences, as cold and the like, seems reasonable. These are the occasions and not causes. They favor but do not cause.

7. That susceptibility varies greatly there can be no doubt. Whether this depends on alkali tension has not yet been demonstrated, but it seems plausible on the ground of variation in the bactericidal action of the blood. This may explain the value of alkalies in the treatment of rheumatic affections.

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DISCUSSION.

DR. HEINRICH STERN, New York—The diversity of the methods employed and the contradictory findings of the various investigators clearly demonstrate that in spite of an apparent clinical justification the supposition of a microbic causation of rheumatic fever is in reality based more on speculation than on facts. Achalmé's results are directly contradicted by those of Westphal, Wassermann, Malkoff, Meyer, Predtetchensky and others. Sawthenkow, on the other hand, obtained the same results as Achalmé, namely, a specific bacillus. Dr. Webster, if I am not mistaken, made liberal use of the articles of Poynton and Paine. In my estimation it is the easiest thing in the world to be a follower of the theory of bacterial origin of disease. It is convenient and for the time being, at least, it is made to serve a certain purpose. The bacterial theory of the causation of rheumatic fever has relegated to oblivion the old lactic acid theory. Who yet believes in the lactic acid foundation of acute articular rheumatism? If we want to determine the cause of acute articular rheumatism I think we must start from a different point altogether. In those affected with the disease we find two pronounced abnormalities in the blood and the urine. This is, firstly, the increased fibrin formation, and secondly, the deficient output of chlorids. This is the basis on which we should work. Where does the fibrin come from; how did it originate? We know there is an intravascular formation and accumulation of fibrin in acute articular rheumatism. This can only be brought about by the presence of fibrinogen and some salts of calcium. The third factor essential to the formation of fibrin does not interest us at the present moment, and I abstain from mentioning the different contentions as to its nature. We can not have thrombosis or intravascular fibrin formation unless the calcium factor works together with prothrombin and the third factor. Looking now for the anatomic substratum of the affection, we will find that the only tissue involved is white fibrous tissue, principally that of joints. The changes found in the inelastic fibrous texture are the same whether they are located in the articulations themselves, in the adjacent structures, or in the heart. White fibrous tissue is found more particularly in those parts habitually used in locomotion. White fibrous tissue is made up of bundles of fibers held together by an agglutinating substance.

Lime or baryta water, or other weak alkalis affect this cementing material. The alkalis may dissolve the cement completely out of the texture. Now it is possible that the calcium salts which bring about intravascular fibrin formation which at this period exists either in larger quantities in the organism or exerts an increased reactivity, also causes the extraction of the agglutinating material from the white fibrous texture. As a natural result of the lesions in the membranes thus produced we will have a disturbance of the osmotic equilibrium, that is, material will pass through the seat of injury which could not penetrate or diffuse through the normal tissue. The deficiency of urinary chlorids may be also explained in this way. The accumulation of ectogenous material in the synovial sacs, the inflammation of the membranes and structures making up the articulations, and all other phenomena of acute articular rheumatism can be explained by the existence of such a lesion in the white fibrous structure.

DR. FRANK BILLINGS, Chicago—Why is it self-limited?

DR. STERN.—That is easily explained. A given case of acute articular rheumatism is dependent either on the degree of the anatomic lesion and the activity of the fibrin-calcium factors or on the calcium material alone. However this may be, the attack will continue as long as the reactivity of the calcium factors continues. The abrupt onset of the affection, its shifting character, etc.—these and all other pertaining characteristics of acute articular rheumatism are better understood on the basis of this physico-chemical substratum than by taking refuge to pathogenic micro-organisms whose influence as regards production of rheumatic fever, with all due deference to the various observers, has not as yet been demonstrated to me.

DR. J. M. ANDERS, Philadelphia—There is perhaps a unity of opinion that we know little about the so-called arthropathies and in cases of acute articular rheumatism we have instances that belong clearly to the intoxications or infections. As you know, we class both under one large head, acute infectious disease or acute infections. The specific micro-organism may be seen; still, I do not see why they should not be multiple as in ulcerative endocarditis or pneumonia. I do not believe that, at the present day, we can definitely say that any special micro-organism is a constant cause of the disease.

One theory regarding this affection has not been mentioned by Dr. Webster, and that is that the symptoms may sometimes be classed with gout, both being due to a prolonged intake of proteids and carbohydrates in excess. Again, other determining factors may be absorbed from the alimentary canal, resulting from the action of bacteria on proteids. Still, I think this view simply explains the predisposing, rather than the specific cause of the disease. To my mind, acute articular rheumatism is a disease entity; there is no difficulty in recognizing this in its more typical phases. In the typical forms, and in chronic rheumatism, all probably of some specific nature, we are constantly confused with other forms of arthritis and I know of no way to discriminate these clinically. We should attempt, by every therapeutic resource known, to separate acute articular rheumatism from gonorrheal rheumatism; this is usually easy. We should also separate it from acute polyarthritis and gout. In many instances the tophi in the ears will be of great value in discriminating between them. To discriminate between the two diseases clinically is oftentimes not very difficult. Symptoms of purpura occurring in connection with acute articular rheumatism I believe are not common; when it does occur what is the interpretation? It is probably a toxic purpura, yet it may be due to emboli. Quite recently I saw an instance of acute rheumatism, of typical form, in which purpura developed. Here we are confronted with the question, are they *Peliosis rheumatica*, or Schönlein's disease? Polyarthritis and purpura and wheels guide us in making a diagnosis of acute articular rheumatism. *Peliosis rheumatica* is of rheumatic origin, it is stated. I can not ascribe to this view because the cases that I have seen did not show any cardiac complications. Cardiac complications occur so rarely in *Peliosis rheumatica* that it tends to show that it has a different etiology. I see no more reason for speaking of rheumatic diathesis

than for speaking of pneumonic diathesis, or of influenza diathesis. I think such a term is obsolete.

DR. WEBSTER, in closing—Dr. Stern says I claim this to be a type of infection and that I have no reason for such a claim. I said in my paper that an organism had been found which we believed to be at least one of the causes of the disease; that this organism had been found in the blood, in the heart, in exudations in joints, and in various other structures and fluids in the body in both the human being and in the lower animals. These organisms taken from the human being have been inoculated in animals and produced identical lesions in them. If carried through a series of animals the arthritic phenomena were again produced in them. If anyone is not convinced by such evidence, I do not know what evidence is called for; I think it is sufficient to convince anybody. With regard to the calcium salts, etc., I have not gone into this part of the subject. The bactericidal powers of the blood are well known, but the Doctor does not demonstrate anything to us; he has been giving us theories; what I have given has been demonstrated.

ETIOLOGY AND PROPHYLAXIS OF THE CARDIAC MANIFESTATIONS OF ARTICULAR RHEUMATISM.*

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The scope of this paper is limited to the consideration of two phases of rheumatic cardiopathies which, notwithstanding the relative frequent occurrence of these manifestations and their great importance because of their far-reaching effects, have, until a recent period, been almost as imperfectly determined in the one case and ineffectually managed in the other as when Pitcairn, in 1788, noticed the connection between rheumatism and pericarditis, a relation more fully explained by Dundas, Wells and Latham, or when Baillie and Kreisig observed the association of acute rheumatism and endocarditis, the definite connection of which was shown by Bouillaud, who also introduced the term endocarditis.

RHEUMATISM AS A FACTOR IN HEART DISEASE.

Among the extrinsic causes of diseases of the heart rheumatism has long held the most important position. It has gradually absorbed into its etiologic relations the so-called idiopathic inflammations of the pericardium and endocardium, and even certain myocardial conditions of an acute nature. It is even admitted that many of the so-called hereditary diseases of the heart are probably of rheumatic origin, while it is generally conceded that those cardiopathies of early life which obtain with the entire absence of clinical history, or, at most, an indefinite mention of "growing pains," are of rheumatic origin. In several hundred ambulatory cases of acute and chronic disease of the heart observed during the last ten years, fully 30 per cent. failed absolutely to give any history of rheumatic manifestations, though usually some such history could be traced in the parents or other members of the family. In those presenting primary conditions or exacerbations of chronic states the exhibition of rheumatic remedies invariably gave relief. Sansom believes that practically all cases of heart disease in individuals under 35 years of age are rheumatic, and that practically all fetal endocarditis is rheumatic.

THE ETIOLOGY OBSCURE.

The exact etiologic nature of the relation of cardiac disease to rheumatism has long been in discussion.

* Read at the Fifty-third Annual Meeting of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee: Drs. Frank A. Jones, George Dock and J. M. Anders.