

On  
I N F E C T I V E      A R T H R I T I S  
of  
U N D I F F E R E N T I A T E D      T Y P E .

With special reference to Tonsillar and other Foci of Infection:  
A Personal Experience and a Critical Review..

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A THESIS for the DEGREE of M.D.,  
presented by

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## INTRODUCTION.

The misuse of the word "Rheumatism" has tended to obscure the true nature of many types of arthritis.

"Rheumatism" or its root "rheuma" is one of the most ancient of medical words, and simply meant "catarrh" and this ancient meaning can still be traced in the very prevalent idea that rheumatism is caused by cold. To the ancients, a disease affecting a joint was an "arthritis" ("articulorum passio"), and indeed, it was not until the 17th Century that the word "rheumatism" was used in its present sense by Ballonius or Baillou (1) (1635) who was the first to distinguish between acute articular rheumatism and gout. Since then, rheumatism has been a convenient term to apply to every ache or pain to which the body is liable, and has been the diagnosis pronounced over many cases of arthritis which, although undoubtedly arising from various causes, yet have these in common that they (a) are painful, (b) affect the joint structures and mechanism of locomotion, (c) are inflammatory but never suppurative, (d) are extremely unamenable to specific treatment, and (e) are very chronic, with a more or less definite progressive tendency.

The present day vogue appears to be to record differences between diseases where our predecessors saw only the points/

points of similarity, and so arises the difficulty in tracing historical references to any particular disease identity, and especially among the rheumatic group. Where it is found that rheumatic gout, chronic articular rheumatism, chronic rheumatic arthritis, rheumatoid arthritis are terms used indiscriminately to describe one disease, and where there are certain signs that examples of what we know to be different diseases are described under the same headings, it makes it very difficult to dogmatise as to what any particular author really did think of this bewildering subject. Still from the 17th Century onwards can be traced a steady break-away of various identities from the huge class called "rheumatism", and, when we consider how long it was before acute rheumatism was differentiated, we cannot wonder that we have not yet elucidated all the mysteries of the far greater class of chronic rheumatism.

The endeavour to classify types according to end-results and morbid anatomy has certainly not hastened the grouping of the cases. Apart from pain, the great feature of rheumatism is its chronicity, and so, although one certainly has the opportunity of watching cases commence and develop, yet, unless careful notes are made, the first stages of the disease have slipped past unrecorded, and where joints, tendons, muscles and ligaments are concerned, I believe that prolonged/

prolonged disuse and mal-position may so influence the later picture and the post-mortem appearance, that the final states may be almost indistinguishable in cases of arthritis from various causes. Crippling, deformity, and wasting can all result from disuse and malposition without the added factor of a trophic disturbance. I feel, therefore, that it is to the etiology and the pathology that we must turn to aid us in finding the differences between the various forms of arthritis.

As has been already noted, the French physician Baillou (1) in the 17th Century drew the distinction between gout and acute rheumatism. In the 18th Century, Cullen (2) gave a clear differential diagnosis between acute, chronic, and muscular rheumatism. Later, with the beginning of the 19th Century great advances were made, and chiefly through the work of Landré-Beauvais (3) (1800), Heberden (4) (1804), and Haygarth (5) (1805), gout was finally distinguished from rheumatism, and both were differentiated from the diseases now classified as "arthritis deformans". Then Charcot described the joint conditions associated with certain diseases of the central nervous system, and finally the other arthropathies associated with chronic pulmonary diseases, and with haemophilia, have been classified.

In 1881, M. Bouchard (6) demonstrated the difference between/

between acute rheumatism and the types of arthritis which are due to specific infections. Since then, our knowledge of these infective arthritides has advanced rapidly under modern clinical and bacteriological methods, until now we are familiar with the joint disorders of gonorrhoea, syphilis, tuberculosis, influenza, pneumonia, typhoid fever, dysentery, spinal meningitis, scarlet fever, as well as the more rare types in diphtheria, mumps, variola and measles.

Further, there are many affections of joints, usually monoarticular, called rheumatic which on careful examination prove to have nothing to do with rheumatism, but to result from injury, strain, or mechanical and skeletal defects, e.g., simple synovitis of the shoulder and the knee, and chronic villous arthritis of the knee.

When all these have been removed and classified, there still remains that large class in which, although no definitely causal organism has been found, yet the onset and clinical course present features so like those of arthritis of the specific infections, that there is no reason to doubt that the disease is microbic in origin. These cases constitute the large class going under the name of "infective arthritis of undifferentiated type".

Naturally, as chronic rheumatism was being broken up in this way into its component parts, the process was accompanied/

accompanied by changing views as to the causation of the disease, and it may be of interest to review these theories here.

Although, at first, rheumatism was simply a catarrh and synonymous with defluxions and humours of the mediaeval doctors, after acute rheumatism was defined as a clinical entity, Cullen (2) propounded the theory that all rheumatism was due to exposure of the body to cold and wet, causing a chill, wherein the surface blood-vessels were contracted locally; and the efforts of the body to overcome this, set up a febrile condition associated with effusion into the joints. Later, when the acidity and profuseness of the perspiration in acute rheumatism were noticed, the theory arose that it must be this abnormal amount of lactic acid that was causing the trouble. But still the idea of the effect of a chill could not be disposed of, and so the full theory stated that the chill, by causing the contraction of the surface blood-vessels, so interfered with the natural excretion of lactic acid that the accumulation thereof in the blood caused rheumatism. Unfortunately, the blood was found not to contain an excess of lactic acid and this theory collapsed, although, as is witnessed by the too numerous advertisements of quack remedies, the presence in the blood of a chemical poison as the cause of rheumatism is a belief commonly held by the public.

The/

The nervous theory was strongly held for a while: viz:- that the chill reacted on the central nervous system, and especially on the medulla oblongata where was supposed to lie the centre for the nutrition of joints, and that this reaction was caused by irritation of the cutaneous nerves in the area of skin chilled.

Lastly, with the advance of bacteriology, rheumatism was thought to be due to an infection, and this is the view generally held at present. That some other factors are at work is unquestionable, and again the action of a "chill" in lowering the resistance of the body to infection must be taken into account.

So then, the introductory and historical survey leads to these views, viz:- that the chief etiological factor in arthritis is a microbic infection, that many cases of arthritis have been definitely proved to have been caused by known organisms, but that there is a large number in which the identity of the causal organism or organisms has not yet been definitely ascertained and which have been grouped under the name "Infective Arthritis of Undifferentiated Type".

My interest in this subject arose from my being the victim of the general hazy conception of it, and my object in writing this thesis is to review the present position regarding infective arthritis of undifferentiated type, with special reference/



reference to the part played by foci of infection in causing the disease, and to give an account of my own experiences both as a patient, and as a physician who has had the opportunity of seeing and treating one or two cases in private practice.

### CLASSIFICATION.

Infective arthritis of undifferentiated type, from its very name postulates the existence of infective arthritis of a known type, and, at the onset of any such illness, the physician is presented with the problem of giving an exact diagnosis. His ability to do so will depend largely on his own mental classification of the diseases sometimes grouped under the comprehensive and non-committal term, chronic non-suppurative arthritis. I have avoided that term so far because I wish to deal particularly with the onset of the disease which is often far from being chronic, and in fact, which is so acute as to mislead the practitioner entirely as to its cause; but it is necessary meantime to embody all forms of arthritis under one name, for the references in literature to what may be termed now, infective arthritis, are so associated with those to rheumatoid and osteo-arthritis, that it is really necessary in dealing with classification to treat of the so-called rheumatic diseases as a whole, and show how gradually the existence of the class of infective arthritides came to be recognised.

There are various classifications given by various authorities, and no two of them agree in every respect. Since acute articular rheumatism (rheumatic fever) was first differentiated/

differentiated, it has remained definitely in a class by itself and may be defined as an acute infection affecting chiefly children and young adults, and characterised by an extremely painful poly-arthritis with a predilection for the large joints, and a marked tendency to flit from joint to joint leaving no residual thickening, by profuse sweating, by a tendency to hyperpyrexia, and to involve the pericardium and endocardium, and on which salicylates exert a specific action.

With the remaining types of arthritis lies the difficulty of adequate classification and until their etiology is definitely cleared up, this difficulty will remain. For example, Fuller (7) (1852) states that "however varying in the locality and intensity of their symptoms, all forms of the disease may and do constantly pass the one into the other": but practically he finds a classification useful and adopts the following:- 1. Acute Rheumatism or Rheumatic Fever, 2. Rheumatic Gout, 3. Chronic Rheumatism (muscular), 4. Neuralgic Rheumatism (sciatica etc.), but, in describing the symptoms of rheumatic gout, he includes cases which present features of rheumatoid arthritis and of osteo-arthritis, and also cases of what I believe to be infective arthritis. He states:- "Sometimes, however, the diagnosis between acute rheumatism and rheumatic gout is by no means easy. Indeed,

I entertain considerable doubt whether an attack which commences as acute rheumatism may not change its type under certain conditions of treatment or constitution, and terminate eventually in rheumatic gout. Certain it is, that I have seen several cases characterised at first by all the most striking features of acute rheumatism ..... in which, after the first intensity of the attack has been subdued, a different train of symptoms has arisen. The larger joints have gradually obtained immunity from pain, but the small joints of the hands have become painful; the articular inflammation, though less urgent in its character, has been extremely obstinate in its continuance, and has lost its distinctive migratory character ..... Thus I have repeatedly known patients crippled by unequivocal rheumatic gout which commenced, in the first instance, as a sequel of acute rheumatism." This, I think, is an excellent clinical description of the course of a case of infective arthritis, and is almost identical with my own history. Moreover, at the time when it was written the salicylic group of drugs was not available to apply a therapeutic test to the type of the acute arthritis.

Nearly all modern attempts at classification are based on that of Charcot (8) who, although declaring his belief that chronic articular rheumatism was one disease, divided/

divided his cases with regard to symptoms and to morbid anatomy into three fundamental types.

1. Progressive Chronic Articular Rheumatism - distinguished by its progressive nature, involvement of many joints, with preference for affecting and beginning in small joints, with severe muscular atrophy and contractures, and is subdivided into (a) Chronic Articular Rheumatism of rapid evolution, (b) Chronic Articular Rheumatism of slow evolution.
2. Partial Chronic Articular Rheumatism e.g. Morbus coxae senilis.
3. Heberden's nodes.

In referring to the sub-group, chronic articular rheumatism of rapid evolution, he states, "the general symptoms are those of acute or subacute articular rheumatism", but submits the view that, unlike other authorities who deem it acute rheumatism turning into chronic, he thinks it most frequently is really chronic rheumatism from the beginning, but that its course presents some of the features of the acute state. This seems to me to refer to such cases as may be called infective arthritis.

Charcot (8) also refers to the connection between articular affections and scarlatina and gonorrhoea, and arrives at the conclusion that there is scarlatinal and gonorrhoeal arthritis independent of rheumatism (a definite admission of the existence of infective arthritis); but then he qualifies that statement by declaring that frequently the articular/

articular affection which originates under these circumstances (scarlatina and gonorrhoea), is really rheumatism developing as a sequela of affections which also, in certain cases, have the power of acting directly on the joints on their own account.

Goldthwait (9) uses a classification which seems to me to be very commendable. He distinguishes three groups:-

1. Infectious Arthritis:- due to the presence within the body of some infectious organism, the symptoms being due to the presence in the joint of the organism or its toxins. Clinically its distinguishing features are its acute onset, involvement of many joints, more or less pyrexia etc.

2. Atrophic Arthritis in which the onset is gradual and insidious, there being no fever. At first, the joints are spindle-shaped, but later become much smaller as the result of atrophy of skin, periarticular tissues, cartilage and bone. This is the rheumatoid type.

3. Hypertrophic Arthritis, which corresponds to our osteo-arthritis.

Here then is one of the most eminent American authorities definitely grouping into one class all cases resembling, in their clinical course, arthritis in which the existence of a known organism has been proved. Most of these cases are included under the heading of acute rheumatoid arthritis/

Arthritis by British writers. Indeed, McCrae (10) objects to the use of the term infectious arthritis, except in the cases where known organisms have been found in the joints, on the ground that such a classification as Goldthwait's is faulty, in that, whereas the first class is differentiated according to its supposed etiology, in the other two classes the deciding factors are symptoms and morbid anatomy. His view is that an infective process is the cause of all types of Arthritis deformans, and he sees no necessity for using the term infectious arthritis to indicate any particular class, since the essential feature of all classes is a deforming arthritis. In his series of cases, McCrae found that he could group them into three types, viz:-

1. Those in which periarticular changes predominated, with occasional involvement of cartilage.
2. Those in which atrophy of all structures predominated.
3. Those in which bony overgrowth at the edges of articulating surfaces was the essential feature. Hypertrophic group.

The first group was by far the largest, and the third group was next, while only a few cases could be put into group 2. About one-eighth of his cases were unclassified owing to presenting features of more than one of the groups.

A.E.Garrod's (11) classification is as follows:-

1. Rheumatoid Arthritis.
- 2./

2. Osteo-arthritis.
3. Spondylitis Deformans.
4. Articular Lesions of Infective Diseases.

Under 1. he includes all cases commencing acutely like acute rheumatism in which there is no specific reaction to the use of salicylates, and in which he can find no organism. But he admits, inferentially, that they may be due to an infection, for he emphasises the importance of cleaning out any septic focus in the body, when it comes to treatment.

Stockman (12) however, admits the existence of a class which he calls chronic infectious arthritis, and says of it "Under this name may provisionally be included a number of non-suppurative joint affections which are not merely part of a well recognised and definite infection such as gonorrhoea and tubercle". He distinguishes osteo-arthritis as a clinical entity and discusses chronic non-suppurative arthritis under the two headings (a) Rheumatoid Arthritis (b) Chronic Infectious Arthritis. His rheumatoid arthritis corresponds to Goldthwait's atrophic arthritis, and chronic infectious arthritis to Goldthwait's infectious arthritis.

Llewellyn and Jones (13) whose reasonings I have followed closely in my introduction, arrive at the existence of this type of infective arthritis of undifferentiated origin by a process of elimination. They state that this class gives rise to the bulk of our cripples. McCrae's (10) cases/



cases show that more than half belong to the "periarticular" class which corresponds more or less to the undifferentiated type of Llewellyn & Jones. Strangeways (14) in a recent classification of all types of rheumatoid arthritis grades them according to pathological findings into six classes, viz:-

(1) capsular, (2) dry, (3) adhesive, (4) rarefying, (5) villous, and (6) infective arthritis which presents the same pathological findings as the "adhesive" type. This classification seems too elaborate for clinical purposes, and it very obviously transgresses McCrae's idea of classification, but it shows the considered opinion of one who has, for many years, studied the subject exhaustively, that there is an infective type of arthritis.

I think I have justified my statement that there are as many classifications as there are authorities on the subject of arthritis, and that I have proved what I set out to do, that there is a definite class of case which may legitimately be called "infective arthritis of undifferentiated type", a class which is admitted, either openly or tacitly, by all authorities. In fact, the chief advance in the past half century in the treatment of chronic non-suppurative arthritis or chronic rheumatism, lies in the recognition of the existence of this type of infective arthritis.

That the dividing line between infective arthritis  
of/

of undifferentiated type and the more acute cases of rheumatoid arthritis may not be very distinct, is admitted, particularly if the trouble has progressed for some little time; and in the later stages it may be impossible to distinguish between them. Still, in the early stages, when the results of treatment along the right lines may be beneficial and may actually result in a permanent cure, I hold that the distinction is possible.

### ETIOLOGY.

The definite relationship established between gonorrhoea and gonorrhoeal arthritis is the best example of an infective arthritis. The one essential etiological feature of the complaint is the gonorrhoeal infection. It may not be easy to find an active infection. It may exist only as a small focus in the prostate or in the seminal vesicles, but it is there, and the cure of the arthritis is effected only when the infective focus is cleared up.

But, when we come to the infective arthritides of undifferentiated type, the one essential etiological factor is obscured, difficult to locate, and it may be even impossible to find. Indeed, Stockman (12) says that the original focus of infection has usually disappeared before the patient comes under observation.

Certain workers in this country e.g., Bannatyne (15), Hale White (16), and Poynton & Paine (17), and others both in Europe and in America have published accounts of the finding in the joints in cases of chronic arthritis of organisms, chiefly diplococci and streptococci.

Fayerweather (18) in a paper giving an account of his own work on the bacteriology of "Infectious Arthritis", carefully reviews the previous literature on the subject by Schüller/

Schüller (1892-1897), Bannatyne and Wohlmann and Blaxall (1896), Von Dungern & Schneider (1898), Chauffard and Ramond (1896), and Bouchard and Charrin (1891-1894). In a series of nine cases of arthritis, Fayerweather discovered organisms in four. Three of these cases were of the type he calls "infectious polyarthritis chronica villosa", and one was an acute articular rheumatism. A different organism was found in every case, and all the organisms were grown artificially, used in experiments on rabbits, and recovered from them in pure culture, sometimes having caused arthritis, and sometimes not. Comparing his findings with those of the previous observers he says:- "The findings in these cases differ from those of Schüller who found in his own cases the same organism constantly. Bannatyne, likewise, in the same type of cases, constantly found a single organism, but it was not Schüller's. It is impossible to explain the constancy of Schüller's results or of Bannatyne's. It is clear that they worked with the same type of cases as my own. From the reports of previous observers, then, we should be inclined to believe that these cases are undoubtedly of bacterial origin, but that a variety of organisms may be responsible for the mischief. My observations serve to confirm this belief".

Rosenow (19a) in a series of experiments in the production of infectious endocarditis used intravenous injections/

ions of pure cultures of cocci isolated from cases of chronic infectious endocarditis, both alone and variously mixed with pure cultures of streptococci, mixed aerobic and anaerobic cultures of cocci from the throat, streptococcus viridans etc., and found that there was a definite affinity of the endocarditic strains of cocci for the endocardium and of the streptococci for the joints.

He advances experimental proof of a theory held by some of the old physicians, notably, by Fuller, that all rheumatism is of the same nature and that the site of the incidence of the disease depends on the age of the patient. He found that, while experimenting on young and on mature rabbits with the same strain of streptococcus, in the young it constantly caused a typical endocarditis due to the arrest of the organisms in the capillaries of the valves which, in the young, are vascular, but that in the mature rabbit, myositis and arthritis resulted.

By examining the lesions of "rheumatic" myositis at different periods of their development he proved that they were due to the arrest in the muscles capillaries of streptococci of a particular grade of attenuation. The organisms multiply for a short time causing an embolism in the capillary and necrosis of the muscle cells. The organisms soon cease to multiply, they disappear, undergoing lysis, and, setting/

setting free their endotoxins, cause the destruction of the more highly specialised cells without pus formation, and a consequent proliferation of the fibrous tissue cells, except where, in favourable cases, there is a complete return to normal.

Rosenow (19b) has isolated from inflamed "rheumatic" joints organisms with which he has produced arthritis in rabbits, and which he has recovered from the rabbit's joints in pure culture. The organisms were of three types, viz:- streptococcus viridans, streptococcus haemolyticus, and a diplococcus practically indistinguishable from the pneumococcus. By suitably varying the culture media and conditions of growth he found that these three organisms were transmutable. Under certain conditions too, they were found to be non-pathogenic, e.g., in the oral cavity; but, if buried in tonsillar crypts, or in tooth sockets, they assumed pathogenic propensities. Summarizing this work Rosenow (19b) states that "the fact that variations in oxygen tensions and salt concentration, that growth in symbiosis with other bacteria, and that injections into cavities in animals commonly call forth mutational forms in streptococci, suggests strongly that similar changes might occur in various foci of infection where such conditions prevail. It would seem, therefore, that focal infections are no longer to be looked upon/

upon merely as a place of entrance of bacteria, but as a place where conditions are favorable for them to acquire the properties which give them a wider range of affinities for various structures".

This view of the transmutation of organisms is supported by Davis (20a) who, as a result of cultural experiments on the haemolytic streptococci, came to the conclusion that "transformation of one member into another within certain limits, appears to be not an uncommon phenomenon".

That these findings might account for the diversity of organisms hitherto found in chronic arthritis appears to be probable.

Although such evidence indicates that organisms of the streptococcic group are responsible for most cases of infective arthritis, and that such organisms acquire their particular affinity for the joints under certain conditions of growth in confined spaces, yet it does not demonstrate the manner in which the arthritis is caused.

In 1899 Adami (21) advanced the theory that, where there is a focus of inflammation, organisms are liable to be carried off into the system where their ultimate effect will depend on the virulence of the organisms and the reaction of the tissues. They may be overcome without causing any damage, but they can set up other foci of infection by a process which  
Adami/

Adami calls "subinfection". By this he means that the organisms undergo slight, if any, numerical increase wherever they settle, but they do not set up foci of suppuration. They undergo lysis; endotoxins are released and destroy the more highly specialized tissue cells, and appear to stimulate the proliferation of fibrous tissue cells. Writing in 1914, Adami (21b) emphasised the fact that his theory of "subinfection" had been amply substantiated by the accumulation of evidence in its favour.

Faber (22) found, in the experimental production of arthritis in rabbits, that the synovial membrane of a rabbit's joint may be sensitized by either

1. repeated intravenous injections of streptococci of attenuated virulence, or
2. injecting into the joint cavity a suspension of killed streptococci. After the reaction has disappeared, intravenous injection of minute doses of living streptococci of the same strain will cause an arthritis in that joint.

Faber therefore concludes that a relapsing arthritis is due to the effect of a virus upon a homologously sensitized joint and that such a virus might come from a small focus of infection.

Both Adami and Faber offer these reasonable explanations of the production of the chronic forms of arthritis from a small focus of infection from which the organisms have easy access/



access to the blood stream. Adami's theory of subinfection and Rosenow's work on the production of myositis of which Adami held a high opinion, offer an explanation of the failure of certain workers to find organisms in the joints, because from them, it is obvious that the organisms have died, and have probably undergone lysis, before their effects on the joints have become manifest clinically. Still, even those observers, like Stockman (12) and McCrae (10), who have failed to find organisms in the joints in their cases of chronic arthritis, are of the opinion that the disease has, in its onset and course, all the features of an infection; and the infective agent must come from somewhere.

Observation of the subjects of chronic arthritis has shewn that they are peculiarly liable to gastro-intestinal derangements, to tonsillitis, dental infections, post-nasal catarrh, and other such infections, and that acute attacks of arthritis are often preceded by such infections, recurrences of which often result in relapses or exacerbations of the arthritis. For a long time these conditions were thought to act as portals by which the "rheumatic" virus entered the system, but more recently, it has come to be recognised that they may function not only as simple "gateways", but as actual "factories" of the infective agent, and they are now called foci of infection.

Generally/

Generally speaking, a focus of infection is not a condition that thrusts itself on to one's notice. Since Hunter's work on pernicious anaemia, great emphasis has been laid on pyorrhoea alveolaris as a cause of various systemic diseases. It must be remembered, however, that, although there may be an associated achylia, in many cases there is not, and the free hydrochloric acid of the gastric secretion acts as an efficient barrier to the passage of organisms. Moreover, pyorrhoea alveolaris is an infection in which the pus has a free outlet, which is readily seen, and which calls for and usually receives proper treatment. A focus of infection causes most trouble when there exists some obstruction to drainage. Watson-Williams (23a) contrasts the well-being of a patient whose nose is full of polypus and constantly streaming pus, with the obvious ill health due to systemic infection in a patient who has but a slight non-purulent nasal discharge that would escape attention under ordinary examination.

A typical focus of infection is one in which the bacteria exist in a crypt, pocket of tissue, or any circumscribed space in which their multiplication is not excessive and from which they may easily be absorbed into the blood stream. Such foci of infection are to be found in the tonsils, dental apical abscesses and granulomata, nasal sinusitis and chronic inflammatory conditions in any part of/

of the alimentary tract, and of the genito-urinary tract. Indeed, McCrae (24) states that any focus of suppuration may act as the cause of a general infective arthritis; thus, infected wounds or a suppurating joint may give rise to a non-suppurating general arthritis. Charcot (8) discussing the causes of articular rheumatism, mentions "traumatic influences," stating that there are several cases reported in which acute or chronic rheumatism has appeared after a blow, a fall, a boil, or a whitlow, and he himself instances three such cases.

That foci of infection harbour organisms of the streptococcic group, capable of producing arthritis in animals, has been demonstrated by various workers. Davis (20b) examined the tonsils enucleated from 113 cases with joint lesions, nephritis, heart disease, tonsillitis, and simple hypertrophy, and found that the haemolytic streptococci were the predominating organisms in the cases with arthritis, and that these organisms could cause arthritis in rabbits.

Poynton and Paine (17) quote a case in which cultures both from the tonsils and from the appendix gave a diplococcus identical in cultural characters. This organism produced arthritis in rabbits. The appendicitis, they concluded, was the result of a blood infection from the tonsils.

Rosenow's (19c) work on dental infection has proved that the bacteria in granulomata lie in very close contact with/

with the blood vessels, and that intravenous injection of cultures of these bacteria cause arthritis in rabbits.

A more conclusive proof of the part played by the tonsils in the production of arthritis is the work of Frank Billings (25) who reports cases of multiple arthritis in which the fluid withdrawn from the joint gave the same strain of streptococcus as was grown from pus removed from the tonsils, and in which enucleation of the tonsils cured the arthritis.

I have failed to find any record by other workers of results so successful or pursued to such a conclusive end as those of Billings. But, after all, the true test of this theory of the etiological relation of foci of infection to arthritis is the therapeutic one, and does not depend only on the finding of a causative organism. Going upon the assumption that there is an infective focus, and taking steps to have that focus eradicated, has had marvellous results. In many cases the sudden total cessation of all acute symptoms has seemed almost miraculous. In others, the improvement is steady but slower, depending largely on the nature of the infection, and the ease with which it can be thoroughly treated. In others again, it must be admitted that no improvement has resulted, but in these cases there is always the possibility of there being more than one focus of infection.

At/

At the Johns Hopkins Hospital (26), of 91 cases of infectious arthritis submitted to tonsillectomy on account of septic conditions of the tonsils, 31 cases were followed up. In 24, the joints had recovered their normal condition; 4 were improved; 2 showed no improvement; and one was worse, but this last had a chronic inflammatory condition of the ethmoidal sinuses, operative treatment for which he refused.

Lillie and Lyons (27) give their results in 200 consecutive cases of tonsillectomy in myositis and arthritis. Briefly, they found that 75% of the cases were cured or improved. They conclude that the operation should be done whether the tonsils are complained of or not, and that the earlier in the course of the disease they are enucleated, the better.

Similar conclusions are drawn by H.J. Starling (28) as a result of observations on tonsillar and rheumatic affections. He also emphasises the role of the septic tonsil in the production of the prolonged pyrexia.

All observers draw attention to the presence of an enlarged lymphatic gland under the angle of the jaw as a more constant proof of tonsillar infection than the appearance of the tonsils.

In view of the relapsing nature of infective arthritis it is interesting to note that Pilot and Davis (29), and later, Pilot (30) and other observers, as the result of examining/

examining a large series of cases, state that the frequency of the haemolytic streptococci is decidedly less in persons whose tonsils have been extirpated than in the throats of persons with normal tonsils.

Beddard (31) states that 90% of cases of so called rheumatoid arthritis are caused by infection of tooth sockets, that commonly the teeth and the gums appear healthy, and that the focus of infection is only to be demonstrated on X-ray examination. Bertram Watson also holds that dental apical infection is a common cause of chronic arthritis.

One of the earliest references in medical literature to focal infection and arthritis is by Benjamin Rush (32) who gave an account of the curing of a case of recurrent arthritis of the hip-joint by the extraction of decayed teeth.

American writers show none of the caution of the British in their statements of the effect of focal infections, and it may be on account of their enthusiastic claims that the general public in America have such a firm belief in the theory. Long before it was the practice here, they resorted to tonsillectomy and particularly to dental X-ray examination and treatment for even mild types of fibrositis. In a very popular American novel (Main Street, by Sinclair Lewis) published in 1920, I read the following:- One doctor, commenting on an older colleague, says:- Was he limping? If the/

the poor fish would have his teeth X-rayed, I'll bet nine and a half cents he'd find an abscess there. "Rheumatism" he calls it. Rheumatism hell! He's behind the times. Wonder he doesn't bleed himself!" That epitomizes the modern American's attitude to the vexed question of fibrositis in all its manifestations.

Watson Williams (23a) reports three cases of fibrositis of the joints, one more acute and the other two chronic, in which very marked improvement resulted from treating foci of infection in the accessory nasal sinuses. The organisms in all three foci were of the staphylococcus group, but in one case there was an associated infection with an unnamed bacillus and in another with a short streptococcus.

Syme (33) writes of the part played by accessory nasal sinus disease in the production of arthritis. He states that often the tonsils are infected secondarily from the nasal sinuses; and this view is upheld by Watson Williams (23b) and others.

Arbuthnot Lane's work on gastro-intestinal stasis and arthritis is well known, but his theory of auto-intoxication as the cause of the arthritis is not accepted everywhere. Adami (21b) protested vigorously against it, stating that "it is more rational to regard the evils of intestinal stasis the result of a condition favouring subinfection and

low forms of infection than as a result of chronic intoxication". Believing as I do that the presence of organisms is essential to the production of arthritis, I do not see how absorption of toxins from the intestinal tract can cause arthritis, but rather, I hold that, as a result of intestinal stasis, the bacterial flora of the intestinal canal may be so altered and the intestinal mucous membrane so damaged in places that the passage into the system of organisms capable of producing arthritis is facilitated.

Macalister (34) reported clinical histories of six cases of "protracted rheumatic fever resisting all treatment and gradually merging into a condition of arthritic deformity", in which the curing of local inflammatory conditions resulted in the cure of the arthritis. In three of his cases the seat of infection was in the nose, in two in the gastro intestinal tract, and in the third, a pustular condition of the scalp. These, then, are a selection, from the voluminous literature on the subject, of representative types of local inflammatory and suppurative conditions, on the curing of which the arthritic affection has been cured or improved, substantiating the theory that the local infection may act as a focus of infection in the systemic disease. Undoubtedly, such conditions may exist for a long time as only potential foci of infection, and, even when roused, do not always cause arthritis. They are blamed for causing endocarditis, nephritis, certain diseases of the skin, and/



and even mental derangements. There must be other factors at work to explain why only certain individuals develop arthritis. That something may depend on the type of organism inhabiting the focus of infection would seem to be indicated by Rosenow's (19a) work on the selective action of the various types of streptococci. But long before the infective nature of arthritis was recognised, there had been recorded a great number of facts relative to the incidence of the disease, such facts bearing on the influence of heredity, a rheumatic diathesis, age, sex, climate, soil, unhygienic surroundings and occupation; and various inferences had been drawn from them by various writers.

Still (35) has described an infectious arthritis in children. I have seen one case of a young lady of 20 years of age, a cripple who has to be wheeled about in a bath chair, in whom at the age of nine years, the disease started as an acute febrile polyarthritis running a course extending over almost two years, and gradually wearing itself out, and leaving her joints so deformed that her parents declare they have seen no change in them in the last nine years.

Summarizing the results of the clinical observations noted above, it may be stated that infective arthritis may occur at any age, but is most common during the active period of life, say between 20 and 40 years, when, probably owing to pressure/

pressure of work, least attention is paid to general health; that it is most common in those who, by reason of occupation, are exposed to various infections; or by reason of poverty are compelled to dwell in damp houses or under other unsanitary conditions, or are exposed to the effects of inclement weather which may reduce their vitality and render them more susceptible of infection; and especially in those who, exhibiting signs of the classic rheumatic diathesis, are most susceptible to these adverse influences. As an immediate cause of the onset of arthritis, a prominent place must be given to the effects of cold in lowering the resistance of the patient, and in this respect it is noteworthy that Rosenow found that cold actually increased the virulence of organisms grown under conditions similar to those in a focus of infection.

The incidence of infective arthritis in the two sexes depends on these general factors. The influences of heredity may be discounted, as, in this climate, I doubt if there are many of us who could not find evidence of some of our relatives and ancestors having suffered from so called "rheumatism". Indeed, Jonathan Hutchinson (36) held the opinion that the rheumatic diathesis was universal and shared by all.

Fuller (7), however, in explaining how, of certain persons living under the same conditions, only some are affected/

affected with rheumatism, states that these, although appearing "strong and equal to much bodily exertion, are peculiarly sensitive to atmospheric vicissitudes, are prone to perspire, and their perspiration has a sour disagreeable odour, whilst their urine, although usually clear when passed, not unfrequently deposits, on cooling, a red brick-dust sediment". These features, he says, indicate the existence of the "rheumatic diathesis". He instances the case of one man who was unable to carry any steel instrument in his pocket in consequence of its becoming rusty from the acidity of his perspiration. This recalled to me the fact that as a student, I was always surprised that my bunch of keys never would shine as others did, and that on adding a new key it quickly lost its brilliancy. Also, a gun-metal cased watch used to get very rusty, especially after I had been exercising myself vigorously. This state of affairs is not now so evident, but that may be because I do not exert myself so often or to such an extent.

Llewellyn and Jones (13) call attention to further signs and symptoms met with in the more marked cases of the rheumatic diathesis, which seem to them to indicate the existence of a toxic state of the blood plasma, e.g. sallow complexion, alteration in skin texture, appearance of papules, or pigment, headache, lassitude, mental irritability, cold hands/

hands and feet, subnormal temperature, sagging of the abdomen and signs of general muscular tone deficiency, and gastro-intestinal derangements.

Maclagan (37) states that "what constitutes the rheumatic constitution, and makes an individual liable to have rheumatism, is the presence in the fibrous textures of the motor apparatus of something which makes them a suitable nidus for the rheumatic poison". But, of course, he cannot explain what that "something" is.

Summarizing the pathological and bacteriological findings, it would appear that most cases of infective arthritis of undifferentiated type are marked by the presence of local foci of infection in the tonsils, the teeth, the nose or its accessory sinuses, or failing this, by gastro-intestinal disorders; that the arthritis is caused by a process of subinfection or repeated infection by organisms of the streptococcic group; that such organisms have entered the system at one or other of these local foci wherein they have grown and acquired the particular degree of virulence and the special properties that give them their affinities for the joint structures; and that the lesions are the result of the direct action of the organisms in situ.

MORBID ANATOMY.

It is generally admitted that the pathological characteristic of so called "chronic rheumatism" is an increase in the white fibrous tissue elements of the body, and, in view of our present knowledge of the etiology, we presume that that increase is due to a more or less chronic inflammatory process. The extent to which this fibrous tissue formation may proceed, is very variable, and the pathological findings in the different cases of infective arthritis, vary in degree, rather than in character. It may be said that each time a joint is affected there remains "a legacy of increased disability" (Maclagan (37) ) which may in favourable cases disappear to a great extent, if not entirely, but which most often leads to permanent fibrous thickening.

In the infective arthritides of unknown type, the periarticular and subsynovial tissues are most involved. The joint capsule, ligaments, synovial membrane, and even the attachments of the tendons are, at first, swollen with a serous exudate and leucocytic infiltration which, in favourable cases resolves, but in unfavourable cases, goes on to an active proliferation of the connective tissue cells, with the result that the various periarticular structures tend to become/

become thickened and matted together. Subsequent condensation and retraction of this new fibrous tissue holds the joint surfaces firmly together. This may, in time, result in the thinning and erosion of the articular cartilages, and where the tendinous attachments of the muscles have been affected, their retraction induces distortion and even subluxation of the joints, and, in fact, changes very similar to those of atrophic or rheumatoid arthritis.

Where the thickening of the synovial membrane has been extensive, there remains permanent villous proliferation of the fringes filling up the whole joint cavity, and producing a soft crackling swelling which gives such a characteristic sensation on palpation.

The muscles associated with the affected joints undergo marked atrophy due to disuse, but also probably due to some reflex neural influence from the joints.

### SYMPTOMATOLOGY.

Infective arthritis of undifferentiated type presents a great variety of pictures depending on the severity of the case. Probably in its simplest form, it is merely a fleeting arthralgia, coming on after a chill, exposure, or mild infection, associated with no objective symptoms, but merely the complaint of pain and stiffness related to one or more joints, and quickly passing off, leaving no apparent ill-effects. In its worst form, it is typified in Jaccoud's "le rhumatisme chronique fibreux", where, usually after several attacks of what has been looked upon as rheumatic fever, grave deformity results from the formation of dense fibrous bands.

Between these two extremes there exist cases showing mono-, oligo-, and poly-articular lesions. One patient may have suffered, at different times, from all three varieties, and so it is found that the extent to which the various joints are affected, varies considerably.

The onset of an average case of infective arthritis is more or less acute. Enquiry into the patient's history may elicit information of an attack of tonsillitis, a purulent or muco-purulent discharge from the nose, some gastro-intestinal dérangement, or perhaps even toothache, immediately preceding the onset of the arthritis. Generally, it may be for a few hours/

hours to two or three days, there is a feeling of malaise, accompanied by more or less fleeting pains in the back and the limbs. Then the pain settles down in one or more joints which soon become inflamed, swollen and exquisitely painful on movement. The temperature is found to be between  $100^{\circ}$  F to  $103^{\circ}$  F, and runs an irregular course; the pulse rate is proportionately increased. The glands in association with the affected joints may or may not be swollen, and the blood, if examined, will reveal a moderate leucocytosis.

Where only one joint is affected, it is usually one of the large joints, and there may be little or no fever. The joint, although acutely swollen, usually returns to a more or less normal condition in a week, but, where it has been neglected, may remain stiff, painful and weak for months.

In the polyarticular variety, the clinical picture at first may be exactly that of acute articular rheumatism. The swelling is not usually such a bright red as it is in rheumatic fever, but the pain and swelling flit from joint to joint and, in severe cases, practically every joint in the body may be affected, in some cases, swelling up many times. Although the large joints are affected, later on the disease shows a distinct predilection for the smaller joints. Usually in a moderately severe case, the affection, after a time, seems to limit itself to a few joints. Gradually the joints become more and more distorted, and, depending on their location, produce more



more or less crippling.

In the most severe cases, instead of limiting itself to a few joints, the condition is polyarticular throughout, and the resultant deformity and crippling is very severe. The types of deformity that may be produced, especially in the hands, have been excellently described by Charcot (8) whose description is more or less accurately copied in most text books.

When a joint is first affected, there is a more or less abundant exudate into the joint cavity, which may mask the involvement of the periarticular tissues, but with each successive swelling of the joint, it is noticed that, after the acute swelling subsides, there remains a soft thickening around the joint, which, as time goes on, gets worse and worse and gives the joint a spindle shape, especially noticeable in the interphalangeal joints. This thickening results from the infiltration of the periarticular fibrous tissues. In favourable cases, where an early arrest of the disease is effected, this thickening, if properly treated, may disappear, but more often some of it remains unresolved, and, in damp and cold weather, may cause pain and stiffness.

Associated with the joint lesions is muscular atrophy which may proceed to a marked extent and which does not result simply from disuse, but also from some reflex neural influence for it is/

is most evident in the muscles immediately in relation to the affected joints; where many of the small joints of the hands are affected the corresponding atrophy of the intrinsic muscles of the hand is extreme. The atrophy, not only of the muscles but of all the tissues, exaggerates the spindle shape of the joints.

Even in a moderately severe case the appetite may remain good, but usually the bowels require attention, and there may be flatulent dyspepsia on occasions. The urine is usually highly coloured, somewhat scanty, and deposits urates on standing. In some cases, there is an albuminuria which may be associated with dropsy.

Sweating is free and where the temperature is high and antipyretics and analgesics have been used, may be profuse and sour smelling.

Except where there is a history of a previous attack of acute rheumatism, there is, as a rule, no evidence of cardiac valvular deficiency. Any cardiac murmurs that may be heard are usually functional and result from weakness of the heart muscle due to the prolonged fever or from anaemia, and in favourable cases soon disappear.

In cases that have been associated with prolonged pyrexia, the blood picture is that of a secondary anaemia.

Besides the periarticular fibrositis, the course of/

of the illness is usually marked by affection of the fibrous tissues of the muscles, aponeuroses, panniculus adiposus and even of the eye. The frequency with which the eye is affected is not often referred to, except in relation to gonorrhoeal arthritis, but Fuller (7) (p.355) states "Affections of the eye, in connection with rheumatic gout are usually seen in those who are thoroughly out of health, or exhausted by previous attacks of the disease. The inflammatory process is not confined to any particular part or texture of the eye, but often attacks, either together or in succession, the various coats of which the eye is composed. ----- Very generally the inflammation commences in the sclerotic and spreads to the conjunctiva, and though not very violent, continues in spite of treatment for several days. Even when all symptoms of inflammation have been subdued, and the eye has regained its natural appearance, the morbid action is prone to recur without manifest external cause. Rarely, however, unless grossly neglected, does the inflammation leave permanent ill effects; nor does it require the same activity in its general treatment, nor same amount of topical applications which would be necessary for its relief in ordinary cases". Rosenow (38) was able to produce iritis and other ocular lesions in rabbits by intravenous injections of streptococci and states that they were due to the lodgment of the organisms in the capillaries of the iris.

Poynton/

Poynton and Paine (17) obtained similar results with the organisms found in cases of rheumatic fever.

The duration of the illness is as variable as are the different types and degrees of arthritis. It seems certain that, in a shorter or longer time, the infection burns itself out. Where this happens early, the joints may resume their natural shape and mobility, but relapses may occur if a similar infection is again contracted. Where the malady has run a course extending for many months or years, much permanent crippling results. Indeed, Llewellyn and Jones (13) state that the majority of our cripples are derived from this class. Hence the necessity for early diagnosis and treatment along correct lines.

## DIFFERENTIAL DIAGNOSIS.

The differential diagnosis in arthritis bristles with difficulties and where the condition has reached a more or less chronic stage, may be actually impossible so closely do the final states of all arthritides resemble one another. Much then depends on the history. In the acute stages, accurate diagnosis is possible and infective arthritis of undifferentiated type has then to be distinguished from 1. acute articular rheumatism, 2. acute rheumatoid arthritis, 3. infective arthritis of specific origin, 4. arthropathies of haemophilia and central nervous diseases.

1. Acute articular rheumatism has a more abrupt onset; the fever is high; chiefly the large joints are affected and the pain and swelling flit from joint to joint leaving no residual thickening; cardiac lesions are common. Salicylates exert a specific action.

2. Acute rheumatoid arthritis is most common in young women. Onset is slow and is preceded by premonitory symptoms such as pain, sensations of burning, tingling etc. Fever, if present, is of a low type. The disease tends to commence in the small joints of the hands and feet and to spread inwards more or less symmetrically. Cardiac lesions are not found as a rule. Various trophic disturbances such as glossy skin, sweating of palms, pigmentation of the skin are common.

Salicylates/

salicylates exert no specific action.

3. Infective arthritis of specific origin. Careful examination and enquiry into the history should reveal the presence of or history of a gonorrhoeal urethritis or vaginitis, the taint of syphilis or perhaps tuberculosis, an immediately antecedent pneumococcal infection, or one of the exanthemata. Microscopic examination of discharges or serological examination of the blood will reveal the presence of the causal organism.

4. (a) Arthritis of haemophilia is of very sudden onset, the joint swelling up rapidly as the result of some slight injury. There is usually a history of bleeding.

(b) Arthropathies of central nervous diseases such as Charcot's joints in tabes dorsalis, and in syringomyelia are characterized by a sudden, painless and spontaneous onset. The effusion is often massive and yet the mobility of the joint may not be at all impaired but may be exaggerated. There are general symptoms of some central nervous disease.

CASE I. My own History. I had measles in infancy. At age of 5 years, I had my tonsils out and adenoids removed. About the age of 7 years, I began to be troubled with attacks of acute follicular tonsillitis, having it every year as far as I can remember, and often one attack at the beginning of winter and another in the spring. I suffered from so called "growing pains". At the age of 17 years, I suffered from Sciatica, and was laid up for 3 weeks; and often thereafter the pain would recur, especially after dancing, and playing football. About the age of 20 years I had an attack of tonsillitis which developed into a quinsy which ruptured spontaneously, evacuating a large quantity of pus. There were no after effects. In January 1915, while I was a resident House Physician in the Royal Infirmary, and about a week after a mild attack of tonsillitis, I developed pains in both knees. The right knee especially swelled up and I was laid up in bed at once. With aspirin internally and tincture of Iodine strongly applied locally, the effusion disappeared in about 10 days, and quickly I recovered full use of the joint. Again in September 1915, I took a mild influenza, accompanied with sore throat. When I had recovered, as I thought, and was on duty again, my right knee swelled up. As I was medical Officer to an infantry battalion at home and was under orders to proceed overseas, I did not report sick, but eventually I had to do so, and it was only after about 3 months that I was able to move about at all freely, and/

and was able to proceed to France in January 1916. In April, 1916, I again suffered from a quinsy which, after I had vainly endeavoured to lance it myself, appeared to rupture during sleep, for on waking next morning, the pain and swelling had disappeared. There were no after effects and during the whole of my service in France up to November 1919, I never suffered from tonsillitis, a freedom I attribute to the open air life.

PRESENT HISTORY. Having been complaining of sore throat for a day or two, on 7th February 1921, I (aet. 29 yrs.) was laid up in bed suffering from an acute follicular tonsillitis affecting chiefly the left tonsil where it quickly developed into an acute parenchymatous tonsillitis or quinsy. It was lanced on two successive days, but the usual pocket of pus could not be found and, on waking on the morning after the second attempt to find pus, the throat felt distinctly easier, and the swelling had practically disappeared. In a fortnight from the commencement of the illness, I was back at work, but felt rather weak, and did not convalesce as quickly as usual. In a week, I began to complain of vague pains in my back and especially just above the knee joints. On 8th March 1921 the pains and the tiredness were much worse and were accompanied by shivering and a general feeling of malaise. I went to bed, and next morning, finding myself no better and with a temperature of  $99.4\frac{7}{10}$ , I remained in bed. The pains became localised to both knee joints, and by night my temperature had gone

up/



up over 102° F. Next day, both knee joints were hot, painful to touch and swollen, and the right ankle and left shoulder joints were painful when moved. On the assumption that it was an attack of acute rheumatism, treatment by local applications of menthol and wintergreen, and internally by large doses of sodium salicylate was instituted, and the diet was restricted to milk, with, later on, the addition of Benger's Food. A consulting physician was called in, but he merely acquiesced in the line of treatment being pursued, suggesting however, that, if the condition did not improve, a vaccine might be prepared from a swab from the throat if possible, otherwise mixed infection phylacogen might prove useful. Wrists, elbows, in fact all the large joints became affected, one swelling up, as in another the swelling faded away, and every one was affected several times.

The milk diet, regulation of the bowels with salts, continued administration of salicylates of various kinds or large doses of alkalis, local applications both soothing and counter-irritant, all failed to have any effect on the fever or on the rapidity with which joint after joint became affected, and so the disease progressed for about a month. Then the throat became painful again, but beyond looking somewhat inflamed, presented nothing abnormal as far as I know, and the pain quickly disappeared under treatment. Shortly/

Shortly after this, the meta tarso-phalangeal joint of the second toe of the right foot swelled up, and, although the large joints were occasionally affected, the small joints of the hands and feet and even the vertebral joints and the sternoclavicular and the temporo maxillary joints were the chief offenders. I cannot recall a single joint that was not at some time or other inflamed and painful. At my own request I had been allowed a more generous diet for about a week, but when the small joints became so markedly and rapidly affected, this was reduced again to milk and Benger's Food. By this time, I was miserably weak, could not turn myself in the bed, and yet, because of the pain in my back and limbs, was continually desirous of having my position changed. As the swelling of the smaller joints continued, the wasting of the muscles became a very marked feature of the case, especially the muscles of the limbs and most of all, the intrinsic muscles of the hands. The proximal interphalangeal joints began to shew a fusiform swelling which persisted even when the joints were not actively painful and acutely swollen.

After delaying, in the hope that the condition would burn itself out in about six weeks, treatment was commenced on April 20th with mixed infection phylacogen in addition to aspirin and local applications. A dose of the mixed infection phylacogen was given at intervals of two days/

days, starting with  $\frac{1}{2}$  cc. and rapidly increasing the dose until the maximum of 10 ccs. was reached when the dose was gradually tapered off. In all 40 ccs. were given. Every injection was followed within a few hours by an intense local reaction at the site of the injection, a rise of temperature, and marked gastro-intestinal disturbance causing severe colic and flatulence, but these effects all passed off in about twelve hours. Often the disturbance, especially the colic, was so severe that, when added to the pain in the joints which was aggravated by the restlessness, morphia had to be given to secure rest. During the course of these injections, all the large joints were affected but after about 11th May, the disease limited itself to the smaller joints, whether due to the use of the phylacogen or not, I am not prepared to say. One effect it did produce, I think, was a reduction of the fever to the extent that the morning temperature was normal but the evening temperature never fell below 99.6° F. Indeed, this evening temperature persisted so long that, in disgust, I ceased taking it after a while.

A curious incident happened during the course of the injections of mixed infection phylacogen which is claimed to be a "sterile aqueous solution of metabolic substances, or derivatives generated by bacteria grown in artificial media", and which probably contains toxins of a proteid nature. The injection/

injection, into the subcutaneous tissue of the abdominal wall, of the second last dose, 2 ccs., was attended with an exceedingly sharp pain. Within thirty seconds, I felt sick, attempted to vomit, but couldn't, and yet felt as if a ball were rising up in my oesophagus. This sensation of swelling spread to my throat and face which latter became curiously tightened so that it felt like parchment. I was propped up in bed, gasping for breath, and afterwards I was informed that my chest, neck and face were greatly swollen and cyanosed, tongue hanging out swollen, dry and blackish, skin drawn tight with every pore like a small pit, eyes glassy and protruded, and the pulse very rapid and feeble. This terrible condition gradually subsided in about half an hour, only cold water having been given me. Ten minutes later I began to shiver and soon was in a very vigorous rigor lasting fully twenty minutes before I began to feel warm and to break into a profuse perspiration. The temperature wasn't taken until after I had been sweating for some time, but it was then found to be 103.6° F, but it subsided to about normal by the next morning. The only explanation that I can give for this incident is that some portion at least, of the dose had been injected into a vein causing at first the neuro-vascular disturbance and profound collapse of an anaphylactic nature, and later a rigor similar to what is obtained in "protein-shock" or pyrogenic therapy/

therapy. Although it was shortly after this that I ceased taking my temperature, I cannot say that the "shock" was more effective in reducing my fever than the course of phylacogen itself.

During practically the whole course of the disease I seemed to be starving and had only submitted to the milk diet with a very bad grace, and about the middle of May, my grumbles were given in to and gradually I was allowed more food, but I always took more than my allowance. Although I was so miserably weak that I could not even feed myself from an invalid's cup, I craved for food, and improved, the more nourishing food I got, although the small joints continued to swell up, especially those of the thumb, first and second fingers of both hands and some of the joints of the feet. Occasionally I experienced slight attacks of pain and photophobia accompanied with some injection of blood vessels both deep and superficial, affecting now one eye and now the other, but never both at the same time. I first experienced this after I had been ill for about two months and when the affection had commenced to implicate chiefly the smaller joints. It seemed like a mild iritis and although painful, never lasted long, but ran a course similar to that of a swelling joint. Even without any treatment, the condition subsided in four days and has left no after effects.

When it seemed as if nothing was having any effect  
on/

on the course of the illness, all drugs were stopped and only local applications were used to whatever joints happened to be affected. I was allowed light nourishing food in plenty and my general condition improved. My heart was very carefully examined and although the sounds were weak still they were pure, and so I was gradually allowed to get up. About the middle of June I was removed to a house where I could lie out in a garden all day. Every night my feet were slightly oedematous and it was found that a soft blowing v.S. murmur was present, best heard over the mitral area.

At this time I was never free from acute swelling of some joint or joints, either in the hands or feet. They would begin by being painful for the best part of a day, next day they would be acutely swollen and this would persist for two to three days, and then gradually the joint would subside and the pain depart. Five or six days was the length of the full course, and another joint would commence as one subsided. Here I was again seen by the consultant who had seen me previously and his opinion was that the arthritis was of the rheumatoid type, that I should undergo spa treatment at Harrogate or Buxton in an endeavour to effect some arrest of the condition, that I should think of some easier branch of the medical profession than general practice, or else decide to remove permanently to the South of England.

I wasn't fit to travel to Harrogate, I couldn't even dress myself properly; so I decided to take a month on the Ayrshire Coast with the idea of getting up my strength. While away, I had an attack of enteritis with fever of  $104^{\circ}$  F for two days. Even this febrile attack had no effect in the manner of the swelling of the joints which, even after the month's holiday when my general condition was much improved and I could with difficulty walk about a mile without requiring to take a long rest, continued to be affected on an average of two joints a week.

Many a time, during the course of the illness, I had voiced my belief in the tonsils being at the root of the trouble and had declared my intention of getting them enucleated as soon as I was fit to stand the operation, but I was advised to go to Harrogate first, and then to get my tonsils out if I liked, after I had improved under spa treatment. But, when I did get to Harrogate in the middle of August, I was told by the doctor whom I consulted there, that I should first find the seat of the infection, get it removed, and then if necessary, undergo spa treatment to cure the stiffness of the joints.

His opinion was that most of these cases arose from a bowel, bladder or urethral, or dental infection, and suggested looking for a streptococcal infection in these regions in spite of my advancing my view of the tonsillar infection. I remained in Harrogate/

Harrogate long enough to have a thorough bacteriological examination made of urine and faeces, and the results were negative. I then returned to Glasgow where I got my teeth examined and X-rayed and proved to be free from apical infection. Having eliminated these possible sources, I had my throat and nose examined and the possibility of the tonsils being septic was admitted, and I requested Dr. Brown Kelly to enucleate them, taking every care not to leave any particle of tonsillar tissue behind. This he did for me, under local anaesthesia, carefully dissecting out both tonsils. It was a long operation as the capsules were so difficult to define owing to the fibrous tissue proliferation caused by the many attacks of tonsillitis. There was very little haemorrhage either at or after the operation, and the throat healed up entirely in about three weeks.

Two days after the operation the proximal interphalangeal joint of the right index finger swelled up very acutely, and then gradually subsided. I was free of any swollen joint for two days and then the same joint swelled up, but not so acutely. By this time I was getting out again and was feeling wonderfully well, certainly better than I had been before the operation. Anxiously I waited, examining and testing my joints day by day, to feel if any one was particularly painful and likely to become swollen. A week passed without/



without anything happening, and by the time another week had elapsed I was convinced that I had got rid of the trouble altogether. My joints were becoming more supple, I could walk with more freedom, my skin looked healthier and my eyes brighter and clearer, and generally I made very rapid progress towards recovery, being fortunate in that that summer was an extraordinarily fine and warm one. I commenced gentle physical exercises to loosen all my joints and to restore the wasted muscles. The exercises were painful at first, especially as the tendinous attachments of muscles and other periarticular fibrous tissues were still swollen and painful. During the whole of the winter 1921-22, I suffered more or less from tenderness to pressure on any bony prominence, especially the internal condyles of the humeri, tips of the acromion processes, lower end of ulna, distal ends of phalanges etc., and sometimes they were painful even without being touched. This tenderness still makes itself felt at times, especially in cold wet weather.

The tonsils had been sent to a pathologist immediately after being extirpated. His report indicated that the right tonsil was larger in size than normal and shewed areas of fibrosis and chronic inflammatory change. The left tonsil was small, cartilagenous in character, and also shewed chronic inflammatory changes. Cultures showed the presence of a streptococcus/

streptococcus, staphylococcus, and micrococcus catarrhalis. These organisms were separated and a combined vaccine prepared of 200 millions m.catarrhalis and staphylococcus, and 50 millions streptococcus per c.c. Examination of sections of the tonsils by low and high power magnification confirmed the naked eye appearances. Sections were stained for organisms by simple stain, by Leishman's stain, and by Gram's method, and the presence of a streptococcus was definitely ascertained.

Unfortunately, no endeavour was made to identify the streptococcus culturally. I commenced using the vaccine about a month after the operation. I started with a dose of 9 minims and increased it by a minim every seven days. The smallness of the dose was decided by a desire to avoid a too vigorous reaction, with the possibility of a recurrence of effusion into the joints. I continued with the vaccine for about five months, but, beyond a slight local reaction, I never felt any effect from any dose. The improvement in my condition was most marked before I started vaccine treatment, and I am convinced that the cure was effected by the enucleation of the tonsils, and by that alone.

CASE II. Miss W.M. aet.33 yrs. Clerkess, had acute rheumatism 14 years ago and influenza 4 years ago, since when she has suffered from pain and stiffness of a general character, especially in cold weather. In February 1923, she developed acute tonsillitis which lasted about fourteen days. Then she resumed work. In a week she began to feel severe pains in large joints, and throat became painful again. She was highly fevered for a day or two, the pains flitted about from joint to joint but, although the joints were stiff, yet the swelling was practically negligible. This went on for seven weeks. Then, on 13th April, the tonsils were enucleated by guillotine under local anaesthetic. Two days later, joint swelling became obvious, first in the right knee, then right ankle, elbows, wrists, shoulders, all in turn. The swelling lasted only a day or two in each joint. The finger joints, especially the proximal interphalangeal joint of the left middle finger and the metacarpo-phalangeal joint of index finger of the right hand, became swollen and painful. As the throat healed, the swelling ceased, but thickening remained in the smaller joints, which may have been exaggerated by the muscular atrophy. The fever which was present for a week after the operation gradually subsided. No cardiac lesion was found. In four weeks, the patient was up and going about, although the right thumb and forefinger were slightly swollen/

swollen. No recurrence occurred after patient got up, and gentle exercises soon restored full movement to all the joints. After a short holiday, she went back to work at the end of June and has remained well since then.

Here also in the early stages, salicylates were of no avail in controlling the pain and the rapidity with which joint after joint became affected.

The milk diet of the acute febrile stage was dispensed with as soon as possible and the patient improved and felt better herself when a light nourishing diet was allowed.

CASE III. Miss J.R. aet. 21 yrs. student. In childhood had measles, mumps, scarlet fever and repeated attacks of tonsillitis. Tonsils were cut. Had at one time a bluish eruption on front of the legs, diagnosed rheumatic purpura. At age of eleven had rheumatic fever, and also pleurisy. Six years ago, began to complain of pains in both knees which steadily became more painful, stiff and swollen. In Oct. 1921 the right knee was punctured and a cupful of fluid removed. The knee was subsequently treated with massage, Iodex, heat and pressure, but both in it and in the left knee, the swelling came and went repeatedly. Her general condition was not good, but she did not lie up in bed. The wrists became painful and swollen, especially the right. In June 1922, knees were examined by X-ray but no change in the joint structures was reported. Blood examination revealed a negative Wassermann. The right knee was put up in plaster of paris and the right hand in a splint, and kept so for three months, removed and re-applied for a further period of three months. During this time the left knee, right shoulder and left temporo-maxillary joints gave trouble. She was taking cod liver oil and syr. ferri iodidi and her general condition improved. With the involvement of more joints, she was given sodium salicylate, but without shewing any improvement. Went to Harrogate at Easter time 1923, and improved under treatment with baths and waters/

waters. A septic condition of her tonsils was diagnosed and their removal advised. She returned to Glasgow and had her nose and throat thoroughly examined, and was found to have a septic condition of both maxillary sinuses as well as septic tonsils. A history of occasional foul discharge from the nose for many years was elicited. From April to June 1923 she had a series of operations for draining the sinuses and enucleating the tonsils. Her general condition improved. Nasal discharge cleared up almost entirely and the joint condition which had begun to affect some of the fingers, was distinctly easier. She has persevered with douching out the nose and when last seen on 4th. February 1924 was in excellent health and walked without limping. The carpo-metacarpal joint of the right thumb was slightly swollen. The patient blamed writing too much for this. Right elbow was slightly puffy in appearance as if from thickened synovial membrane. No fluid could be detected. Extension was slightly limited. The right knee was similarly affected but movement was full in range. She reported that for several months now there has been no acute swelling in any of the joints. The hands and fingers appear normal. The right wrist movements are not quite as extensive as they might be, but this is scarcely noticeable. The patient is attending her classes, and her joints now permit of her playing golf.

She has a very short and soft V.S. murmur best heard at the apex, probably a relic of the rheumatic fever in childhood.

CASE IV. Mrs.E.M. aet. 30 years. At the age of 7 she had rheumatic fever. Later, she took a severe illness diagnosed cerebro-spinal meningitis, at which time a V.S. mitral murmur was detected and stated to have been caused by the previous acute rheumatism. Otherwise her health was good until the spring of 1921 when, after a confinement, she complained of pains in the knees. Then left hand, elbow and shoulder became painful and stiff without obvious swelling of the joints. Occasionally the hand if kept flexed for long, refused to open, and later, the middle inter-phalangeal joints of left hand became swollen.

This condition, involving other joints as well, persisted more or less, depending on treatment and her general health, until Nov.1922 when she removed to a house on a damp clay soil. Within a fortnight her knees were so swollen that she had to lie up for a week. She noticed that if she walked too far, or jerked her knees in any way they were liable to swell up. During the winter of 1922-23, the wrists and some of the finger joints were also troublesome. In February 1923, she was complaining of toothache located to one tooth that had been crowned some years ago. It was decided to have the tooth extracted and on such being done, an abscess was found at the root. The tooth alongside was also extracted as it was suspected of being infected, as turned out to be the case.

The/

The arthritic condition improved considerably, and she was able to get about with much greater freedom. Twice, however, since the summer of 1923 she had had to lie up for a day or two on account of sudden acute swelling of the knees, but the day or two in bed was quite sufficient. She removed to a house, more pleasantly situated, and since Oct. 1923 has been comparatively free from pain.

When last examined, 28th January 1924, she was able to move about freely and do all house duties. The wrists especially over the lower end of each ulna were swollen but movements were not limited. Knees were both somewhat puffy but no fluid could be detected. Feet and finger joints appeared practically normal, but the wrists were stated to swell occasionally if extra hard housework is undertaken.

She has other crowned teeth

#### COMMENT ON CASES.

CASE I. Here is a remarkable liability to tonsillar infection, with noted examples of conditions long recognised to be of a rheumatic nature, following an attack of tonsillitis.

"Growing pains" have been stated to be akin to acute rheumatism, but it seems as if it would be more correct to call them the mildest form of infective arthritis viz. an arth<sup>r</sup>algia of infective origin.

Similarly/



Similarly, the attack of sciatica was probably preceded by a tonsillitis although I cannot definitely recall such, but then, I often suffered from a mild tonsillitis which although calling for treatment, did not make me lie up in bed. Sciatica, an inflammation of the fibrous tissue structures of the nerve, is probably infective in origin.

But it is the more recent history that is most conclusive. The connection between the invasion of the tonsils and the two attacks of "synovitis" is so close that it leaves no doubt as to the portal by which the infective agent gained its entry into the system. Unfortunately the fact that the so called "synovitis" was an infective arthritis due to the tonsillar infection, was not impressed upon me early enough to cause me to take steps to prevent the risk of a recurrence. Even when the generalised arthritis developed, the suggestion of removing the tonsils was made with the idea of minimising the risk of future trouble. It was not until the chronic element of the illness became manifest, and the idea of focal infection engendered, that there grew up the conception that enucleation of the tonsils would remove not only the entrance ground and starting place for future trouble, but also and more especially, the seat of manufacture of the agent that was causing the present illness. The almost dramatic cessation of the arthritis and the returning sense of well-being after/

after the enucleation of the tonsils seem to me to prove conclusively the relationship between the tonsillar infection and the arthritis. Although the presence of streptococci was never even looked for in the joints and so one cannot dogmatically affirm that it was a streptococcal infection that caused the arthritis, still the pathological and bacteriological examination of the enucleated tonsils revealed just such a condition as Rosenow's (19b) experiments shewed was necessary for streptococci to develop and assume their peculiar affinity for joint structures.

CASE 2. This presents many features in common with the first case, but is of a much milder type. It especially demonstrates one of the risks of sudden total extirpation of a focus of infection. The raw surface left on enucleation of the tonsils may, as was probable in this case, be infected with the organism causing the arthritis, and the result is an exacerbation of the arthritis. In this case, the resultant arthritis was worse than that which called for the operative treatment, but it quickly subsided entirely.

CASE 3. Shews that there may be more than one focus of infection and that no improvement will result until all are cleared. Tonsils, from the ease of access to them, are very often blamed when the infection is not so obvious. The complete curing of an infection of the Nasal sinuses is a very slow process compared with enucleation of the tonsils and so the/

the improvement on the arthritis is correspondingly slow.

CASE 4. In this case the steady improvement after the removal of the infected teeth seems to point to their being the true culprits. The fact that the tooth next to the painful one was found to be infected and that other teeth have artificial crowns and presumably dead fangs, suggests that there is still an infection there that may, even in her healthier condition and surroundings, be preventing a complete cure. I have repeatedly urged her to have the teeth X rayed, but so far, this has not been done.

Generally it is to be noted that all four cases present signs of a rheumatic diathesis, demonstrated by their histories of previous attacks of arthritis or of acute rheumatism,

I had advised Case III also to have her teeth examined and while this was being typed, I received the following report"- "The X-ray examination shows evidence of generalised absorption of bone round the necks of the teeth varying from one sixteenth to one eighth of an inch in extent. This is indicative of a chronic marginal rarefying osteitis associated with chronic pyorrhoea alveolaris.

The only tooth showing evidence of apical infection is the lower left lateral incisor which shows an extensive chronic apical abscess"

### TREATMENT.

The treatment of arthritis is passed over as quietly as possible in most text books, which proves how unsatisfactory it has hitherto been. That drug treatment is particularly so, is evidenced by the number of drugs advocated. Analgaesics and antipyretics of all kinds, singly and in all sorts of combinations have been used in the more acute phases to relieve pain and reduce fever, all more or less unsuccessfully; local measures such as hot and cold applications, anodyne lotions and liniments, and counterirritants to allay pain and reduce swelling; purgatives and intestinal antiseptics of all kinds, including lactic acid bacilli, to check intestinal fermentation; tonics like cod liver oil and haematinics like arsenic and iron to improve the general condition of the patient; and, in the later stages, iodine and iodides in all manner of preparations to aid in absorption of fibrous tissue. Charcot (8), after mentioning a few of the drugs that would come under such headings, remarks that "it must be admitted, chronic rheumatism is an affection which in the majority of cases not all the resources of medicine can succeed in curing". Garrod (11) takes a more hopeful outlook, and, while admitting the general failure of drug treatment, holds that much may be done to alleviate the suffering that the disease entails, and that/

that an arrest in the earlier stages may be possible. That some cases yield to no measures and that the physician feels absolutely helpless when faced by their progressive nature, is the reason why the lesson must be enforced that this disease must be combatted by very prompt and appropriate measures at its earliest appearance.

In view of such opinions as these, and of the cases here cited, and of the results obtained by others, it seems reasonable that, when confronted with a more or less acute arthritis which does not respond to treatment with salicylates, every endeavour should be made without loss of time to find a focus of infection that might be the possible cause. The general practitioner probably will not be able to do this himself. He will have to have the assistance of the bacteriologist, dental radiologist, of those who specialise in the treatment of diseases of the ear, nose, and throat, and perhaps of the gynaecologist, but he should not delay, either on the plea of the weakness of the patient or in the hope that under expectant treatment the condition will clear up. Undoubtedly in some cases, after months of treatment on such lines, a spontaneous arrest of the disease is effected, but, by that time, more or less crippling will have occurred.

Where a probable focus of infection has been found treatment must be instituted at once with a view to eradicating it, on the assumption that the infection is the cause of the/  
the/

the arthritis. Until the bacteriology is completely worked out we can do no more than assume this relationship of cause and effect, and leave it to the results of treatment to prove the correctness of the assumption.

Logan Turner (39) emphasises the necessity for early removal of the infective focus, believing that, if this is delayed too long, the generalised infection may have had time to perpetuate itself in various other sites in the body, with the result that no improvement is obtained even when the original focus is removed.

In treating the focus of infection, it is best to bear in mind the possible results of its sudden removal in precipitating an exacerbation of the arthritis. It is possible for the causative organism to infect the operation wound. Bertram Watson (40) and Kerr Pringle (41) both advise that an autogenous vaccine should be prepared first, and that two or three doses of it should have been administered before operative treatment is commenced, to increase the patient's power of resistance and to minimise the risk of a too acute and wide spread implication of the joints. Whether this be done or not, it must be insisted upon that treatment of the focus of infection must be the most thorough possible. Where the tonsils are the focus of infection, all authorities emphasise the necessity for complete enucleation. If the tonsils/

tonsils are only cut, the scar formed on the surface may occlude the crypts, and enclose the infection more securely, and this is likely to occur where even a small piece of tonsillar tissue is inadvertently left behind.

Where no focus of infection can be found, treatment has to be conducted on the general lines applicable to all febrile debilitating diseases. It is harmful to continue giving salicylates which will only have the effect of producing a worse anaemia than results from the prolonged illness itself. They may be required if it is found that something in addition to local application is necessary to leave pain, but if so, they should be used sparingly and preferable combined with quinine. The most important thing in the treatment of these cases is to aid the natural powers of the patient to overcome the infection. Even when there is a moderate fever, the diet should be as abundant, although light and nourishing, as the patient feels able for. The benefits of fresh air and sunlight cannot be too strongly insisted upon. To be cooped up in a sick room day and night, does not help to keep up the patient's strength, nor does it allow him to make the best use of the nourishing diet he should have. From my own experience I can testify to the depressing effect, both physical and mental, of a prolonged course of milk diet and salicylates. When I got more food, I felt better. That did not hinder at first/

first the swelling of the joints, but I was better able to bear the pain and certainly the extra nourishment never made the condition worse. When, later, I discarded the thermometer and with it the use of salicylates, I improved steadily in general health, and was able, in spite of painful and swollen joints to take a more hopeful outlook. Fortunately for me, the summer of 1921 was exceptionally dry and warm, and almost as soon as I was allowed out of bed I was able to lie outside in the sun, and there is no doubt in my mind that this circumstance aided me greatly.

While it is of great advantage to the patient to be able to live in a dry and warm climate, I think that this should be considered along with the many other circumstances of the patient. To banish a patient to such a place, away from intimate friends, might actually have a bad effect. If the locality in which the patient lives is reasonably healthy, i.e., is not damp, marshy or low lying, then, probably, the added comforts of home life will make up for a slightly inferior quality of weather. Regarding the relative merits of coast or country life, one may say that these vary with different patients and that the place at which the patient feels best, is the best place for him.

I have been surprised at the amount of movement that sufferers from chronic arthritis with much deformity may have in certain joints compared with the absolute fixation  
or/



or relative immobility of others. I cannot see that it is always due to a difference in the incidence of the infection, or in the extent of the involvement of the various joints. I think that these joints which retain most movement are those which the patient has used most, either from compulsion or from pleasure. This is very evident in how a crippled lady will be able to knit, when she is unable to put up her own hair. Knitting has been a pastime which has filled up long weary hours and the joints quickly permitted of the necessary range of movement. But the shoulder movements necessary to raise the hands to the back of the head were probably neglected for months until they were impossible of accomplishment, and that too, although the patient will admit that the shoulder joints were neither any more often affected nor more painful than any other. In this connection my own experience is interesting. During the whole course of my illness, I rebelled against the use of a bed-pan, and so I had to get out of bed at least once, and sometimes more often, every day to use a commode. I contend that even this served a good purpose for when I was permitted to rise for an hour or so, I was very soon fit to move about, and did so long before I was able to use my arms well enough to dress myself. As soon as possible, too, I started to loosen my finger and hand joints by playing the piano. Some days they were too painful/

painful to move freely, but as soon as the acute pain left them, I would be using them again. Often I would be seen playing the piano with several finger joints encircled with a brown band the result of painting them with tincture of iodine. This exercise helped greatly towards restoring and preserving movement in the fingers and hands, and in preventing further wasting of the hand muscles, and stiffening and crippling of the fingers. I was doing this two months before I had the focus of infection removed. It was some months after that, before I could put my arms straight above my head, yet in the last two months of my illness, none of the large joints and certainly not the shoulder joints had been affected.

I am convinced then, that massage, passive movements and even active movements should be commenced as soon as possible and that the movements should be of the fullest possible range. I don't think that there is any fear of damaging the joint structures by such treatment which is so advantageous to the patient. Prolonged rest and immobilization of the joints, even when it does not result in actual deformity or fixation, or permanent restriction in the range of movement, always causes such stiffness that only prolonged, patient, and often painful exercises can overcome.

Treatment by vaccines has been strongly advocated. When used, they should be autogenous and should not be relied upon/

upon alone, but only as an adjunct to the removal of the focus of infection from which the vaccine has been obtained. They could be used before, during, and after the radical treatment of the focus of infection, but to be of any service in increasing the resistance of the patient, each dose must be so carefully graduated and timed that a slight general reaction is obtained. Mollison<sup>(42)</sup> advises that, as a routine measure, a vaccine should be prepared from extirpated tonsils in cases of chronic arthritis, but admits that it may not be necessary to use it.

The injection of a foreign protein, or of other non-protein substances - so called protein-shock or pyrogenic therapy - has been successfully used with the object of bringing about an acute febrile reaction and of stimulating and increasing the defensive resources of the body to deal more actively with the subacute or chronic infection whenever it may be. This treatment is often very exhausting, is not without risks, and therefore has to be very carefully supervised.

In the later stages, when the infection seems to have burned itself out and the disease has ceased to progress, or when the focus of infection has been removed, treatment at a spa may be advisable, as it is at such places where all the types of baths, etc. are conveniently congregated, that most good can be obtained in the way of promoting the absorption of the/

the newly formed fibrous tissue. I do not intend to review the different types of baths, but would only emphasise the necessity for avoiding lowering the strength of the patient by overdoing the treatment.

Electricity, especially in the form of Ionic medication, has in some hands proved very successful, but other workers put no faith in it.

It often happens that, especially in the very late stages, surgical measures may have to be undertaken to restore function to crippled joints. Such should not be lightly undertaken, and should always be left in the hands of a competent orthopaedic surgeon.

Such then is an outline of the multiplicity of methods of treating this intractable disease. They all aim to control the infective process or to alleviate the suffering and deformity caused by the sequelae, and individual workers praise or condemn the various methods according to their own opinions and prejudices. The truth probably lies in this, that no one method always succeeds, but that the best results are obtained by the most careful adaptation of the various methods to the individual needs of each case.

One last thing in treatment! Everything possible must be done to occupy the mind of the patient and to prevent him brooding over his trouble. Any hobby should be encouraged/

encouraged, especially if it is one that calls for exercise of the joints, particularly the small joints of the hands and fingers. Unless the patient is prepared to put forth every effort to avoid crippling no treatment will do it for him. A depressing or hopeless outlook by the physician is sure to be reflected by the patient and to avoid this, I would suggest that in such cases as I have illustrated here, and indeed in all cases of chronic arthritis, the name "rheumatoid" should not be used. The general public have a horror of that name and associate it with an incurable and progressively crippling disease, and to pronounce that diagnosis over any man is to depress his spirits to the uttermost. By using the term infective arthritis, we can stimulate within the patient the hope that, even where we cannot find any focus of infection on which to make a frontal attack, still by a series of flanking attacks, or even sallies into the unknown, we may be successful in overcoming the infection and arresting the ravages of the disease.

SUMMARY AND CONCLUSIONS.

1. Infective arthritis of undifferentiated type is a distinctive sub.group of the large class of non-suppurative arthritis.
2. Cases of infective arthritis of undifferentiated type are characterised and caused by the presence somewhere in the body of a focus (or foci) of infection.
3. Individuals who present the classical signs of the rheumatic diathesis are more prone to develop arthritis when exposed to infection.
4. Treatment, to be effective, consists in the early and complete removal of the focus (or foci) of infection; in default of this, treatment must be along the lines of that suitable for a general infection.
5. Diet should be light, but abundant and nourishing. Fresh air and sunlight are essential.
6. Massage, passive movements, and active movements of full range are to be commenced as soon as possible and to be persisted in, if deformity is to be prevented.

BIBLIOGRAPHY.

1. Baillou. De Rheumatismo 1635. Vide "A Treatise on Rheumatism" by A.E.Garrood.
2. Cullen. Practice of Physic 1784.
3. Landre Beauvais. These de Paris 1800. Vide "Arthritis Deformans" by R.Ll.Jones.
4. Heberden. Commentaries 1804. Vide A.E.Garrood & R.Ll.Jones.
5. Haygarth. On Nodosity of the Joints 1805. Vide Garrod; & Jones.
6. Bouchard. 1881. Quoted in "Fibrositis" by Llewellyn & Jones.
7. Fuller. "Rheumatism, Rheumatic Gout, and Sciatica" 1852.
8. Charcot. Lectures on Senile Diseases. (New Sydenham Society Translation 1881).
9. Goldthwait. "Infectious Arthritis". Boston Med.& Surg. Jour. 1904.
10. Osler and McCrae. A System of Medicine. 1915. Vol.V.
11. A.E.Garrood. Allbutt's System of Medicine. 2nd Ed.Vol.III and "A Treatise on Rheumatism" 1890.
12. Ralph Stockman. Rheumatism and Arthritis. 1920.
13. Llewellyn and Jones. Fibrositis. 1915.
14. T.S.P.Strangeways. Report of Royal Society of Medicine B.M.J. May 5th.1923.
15. Bannatyne. Rheumatoid Arthritis. 1896.
16. Hale White. Guy's Hospital Reports. Vol.LVII. 1902.
17. Poynton & Paine. Researches in Rheumatism. 1913.
18. Fayerweather. American Journal of the Medical Sciences Vol.CXXX. 1905.
- 19./

- 19 (a) Rosenow. Journal of Experimental Medicine. Vol.XI. Sept.1912.  
 (b) Ditto. Vol.XIV.Jan.1914.  
 Also quoted by Jones Llewellyn (Fibrositis).  
 (c) " " Bertram Watson. The Practitioner  
 June 1922.  
 and " " Martin Beattie. B.M.J. Nov.12th.1921.
- 20 (a) Davis D.J. Journal of Infectious Diseases. Vol.XII, page  
 386. 1913.  
 (b) Ditto Vol.X. March 1912.
- 21 (a) Adami Journal of American Medical Association Dec.16 & 23  
 1899.  
 (b) British Medical Journal. 1914. Vol.I.
22. Faber, H.K. Journal Experimental Medicine. 1915. Vol.XXII.
- 23 (a) Watson-Williams. B.M.J. Jan.21st.1922.  
 (b) Proceedings of Royal Society of Medicine.  
 1919 Vol.XII. p.220.
24. McCrae. Quoted by Willcox. B.M.J. June 4th.1921.
25. Billings, Frank. Ill.Med.Jour. March 1912, 261-274, quoted  
 by C.H.Mayo. Collected Papers.  
 Mayo Clinic 1913.
26. Johns Hopkins Hospital Bulletin, January 1917.
27. Lillie & Lyons. Jour.Amer.Med.Assoc. 1919. 1214-1216.
28. Starling, H.J., Guy's Hospital Report. October 1923.
29. Pilot and D.J.Davis, Jour.Exp.Med. Vol. XXIV. 1919.
30. Meyer, Pilot and Pearlman, Jour.Exp.Med. Vol.XXIX. 1921.
31. Beddard. Transactions, Medical Society of London. Oct.1918.
32. Benjamin Rush. Quoted in Jour.Amer.Med.Assoc. 1919 page 817.
- 33./



33. W.S.Syme. The Practitioner. Vol.CX. May 1923.
34. C.J.Macalister. Medical Press & Circular, Dec.1902.
35. Still. "Allbutt's System of Medicine". 2nd.Ed.Vol.III.
36. Jonathan Hutchinson. Quoted by Garrod in "A Treatise on Rheumatism".
37. T.J.MacLagan. 1896 "Rheumatism".
38. Rosenow. Jour.Inf.Diseases. Vol.XVII. 1915. Page 403.
39. Logan Turner. Trans.Med.Chir.Soc.Edin.Med.Jour. Nov.1923.
40. Bertram Watson. The Practitioner. June 1922.
41. Kerr Pringle. The Practitioner. October 1921.
42. Mollison. Jour.of Laryng. & Otology. April 1921.