

Balneological and Climatological Section.

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Arthritis Deformans.

An Address Introductory to a Discussion on the Subject.

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WHILE deeply sensible of the honour, yet I must confess that it was with many misgivings that I acquiesced in our President's suggestion and consented to open a discussion before this Society upon so obscure and intricate a subject as arthritis deformans. However, having had the temerity to essay the task, I found myself forthwith confronted with the by-no-means trivial difficulty of deciding as to which aspect of this complex subject would most readily and most profitably lend itself to discussion. Upon reflection, it occurred to me that I could not do better than select as the theme of my remarks the classification and differential diagnosis of the various joint affections grouped generally under the term "arthritis deformans." Doubtless all of you who are familiar with the extensive literature will admit much remains to be done before we shall be in a position to effect such a differentiation as shall lie beyond the reach of cavil. Our tardy progress towards this much-to-be-desired achievement may legitimately be referred in part to the bewildering state of our nomenclature.

This question of the terminology of chronic joint diseases is, indeed, one that clamours for speedy and careful consideration, if only with the

object of insuring a greater measure of definitude in regard to the application of the various nosological terms. As matters stand, so numerous and so varied are the designations employed, and these withal are so vaguely defined, that not infrequently one finds the same term applied to denote wholly diverse joint affections and vice-versa identical arthritic conditions labelled by different appellations. Such indiscriminate application on our part of the various terms in use inevitably begets its own Nemesis in the shape of those mutual misunderstandings which so grievously hamper intelligent discussion, and for the same reason render perusal of the literature so laborious and but too often futile. Being therefore wishful to—as far as possible—obviate any confusion on this question of nomenclature, I would state that I use the term “*arthritis deformans*” in a generic sense, as covering all those various arthritic conditions to the discussion of which we are this evening committed.

Latterly the opinion has been steadily gaining ground that not one but several diseases have been included under one or other of the various terms used to designate collectively this class of joint disease. This view is, however, by no means as novel as certain writers on the subject are apparently inclined to consider, for they would appear to have overlooked the fact that even some years prior to the appearance of Charcot's classical monograph a German physician, von Volkmann, had clearly differentiated the anatomical lesions of rheumatoid arthritis and osteo-arthritis. Moreover, this contention of von Volkmann cannot be dismissed as mere speculative inference, for, as his writings demonstrate, he based his contention as to their fundamental distinctions upon the bedrock of ascertained pathology as revealed to him in his anatomical researches. Thus, he first established the fact that in rheumatoid arthritis the initial changes began in the synovial membrane, the cartilages being affected secondarily. In addition, he noted the primary thickening of the periarticular and synovial tissues, and the subsequent atrophy of the same; likewise the occurrence of fibrous and bony ankylosis. Furthermore, in his work he contrasted the features of this group with those of osteo-arthritis, of which he gave a classical description. It will be seen, therefore, from the foregoing details that the modern view as to the distinctness of rheumatoid arthritis and osteo-arthritis is, after all, but a recrudescence of that originally brought forward by von Volkmann. With the advent, however, of Charcot's monograph on the subject, the remarkable researches of von Volkmann underwent an almost total eclipse. The great French physician, as we know,

though he tacitly acknowledged the clinical distinctions of the two main varieties of arthritis deformans, nevertheless claimed that pathologically they were but different expressions or grades of the same underlying morbid process. So commanding was the personality of Charcot that this illustrious physician's conception as to the essential pathological unity of all forms of arthritis deformans dominated not only the French but most other schools of thought. Fortunately, however, the German school still retained their power of independent observation, and in the fullness of time von Volkmann's pioneer researches, as well as his deductions therefrom, were amply confirmed and reinforced by a series of anatomical investigations by different workers, among whom may be mentioned Hueter, Samdran, Waldmann, Hoffa, and Wollenberg.

Elsewhere¹ I have referred in detail to the researches of these observers on the morbid anatomy, but time does not admit of my alluding in detail to their post-mortem studies this evening, and I must content myself with stating that the results of these and other anatomical observations have largely determined me in regard to the classification I am about to submit to you of the various types of arthritis deformans. Under this title at least three distinct varieties of joint disease may be distinguished:—

- (1) Rheumatoid arthritis or atrophic arthritis;
- (2) Osteo-arthritis or hypertrophic arthritis;
- (3) Infective arthritis.

With regard to the first-named division, rheumatoid arthritis, it may be mentioned that the synonymous term of atrophic arthritis was first introduced by Charcot, who noted in regard to one of his clinical types of arthritis deformans that secondary atrophic changes developed during the later stages of its life-history. This atrophy, he tells us, involved not only the skin, subcutaneous and muscular tissues, but even the bones: hence his suggestion that the name "atrophic arthritis" should be adopted as embodying the salient pathological characteristic of this variety. Recently, for the same reasons, the use of the term has been revived by Goldthwait, who would, however, restrict its application to the more chronic forms of rheumatoid arthritis. My clinical experience, however, accords with that of most English observers in that there is also an acute form of the malady to which the term under discussion is also applicable. Lastly, in addition to the foregoing acute and chronic types, I would also place under the same

¹ "Arthritis Deformans," Bristol, 1909.

heading of rheumatoid arthritis that group of cases known as Still's disease. I do this provisionally, inasmuch as I have not on Still's basis of differentiation found it possible to effect a satisfactory discrimination between examples of this variety and cases of rheumatoid arthritis occurring in children. Briefly stated, Still claimed that this disease could be clinically differentiated from rheumatoid arthritis by the absence of glandular and splenic enlargement in the latter, and by the presence in rheumatoid arthritis of bony thickening and lipping. It is not, however, obvious how a satisfactory differentiation can be achieved upon these lines. Thus rheumatoid arthritis, at any rate in its early stages, is not accompanied by bony outgrowths either in children or adults. Neither can any cleavage be upheld on the ground that lymphatic and splenic enlargement are absent in rheumatoid arthritis. For my own experience accords with that of others in that enlargement, at any rate, of the glands is frequent, while examples of splenic swelling are accumulating. To my mind too much stress has been laid on splenic and lymphatic enlargement as a differentiating factor, and, pending positive bacteriological findings, I am inclined to regard Still's disease as a form of rheumatoid arthritis in which the prominence of glandular swellings is to be accounted for as a peculiarity incidental to the early age of its victims, and that their presence cannot be construed as adequate evidence of its being a specific joint affection.

Passing now to consider the morbid anatomy of the above rheumatoid type, we are, I think, justified by recent anatomical researches in drawing the following conclusions:—

(1) The morbid process begins in the synovial and periarticular tissues, which undergo thickening and proliferation, but the cartilage, at any rate in the earlier stages, remains intact.

(2) The changes that ensue in the cartilaginous and bony structures are to be regarded as secondary, and are predominantly atrophic in character.

(3) In some cases proliferative changes take place in the bone and cartilages, but these occasional new bony outgrowths are inconsiderable in size.

(4) Lastly, in the terminal stages of the disease secondary atrophic changes ensue. Thus the cutaneous and capsular tissues undergo progressive shrinkage, the joint cavity becomes obliterated, with fibrous or bony ankylosis. This atrophic process involves also shafts of the bones, the cortex becoming attenuated and the cancellous tissue replaced by scanty, thin trabeculæ.

Before proceeding to consider briefly its clinical aspect, one may allude to the one salient characteristic of the ætiology of rheumatoid arthritis—i.e., the frequency with which the disease is associated with, or arises in sequence to, affections of avowedly infective or toxic character, hence the strongest presumption of the malady itself being also of like origin. From the clinical standpoint rheumatoid arthritis may be defined as a progressive polyarthritis, which runs a more or less acute or chronic course, diversified by remissions and exacerbations. The disease occurs chiefly in persons under forty years of age, and is far more common among women than men; but in children, boys, if anything, are more frequently attacked than girls. It begins in the small joints, spreading centripetally, with a tendency to symmetrical distribution. The more exposed joints become the seat of smooth, spindle-shaped swellings, due rather to periarticular thickening than to effusion, but no nodular outgrowths can be detected at the level of articulation. Eventually the enlarged joints undergo contraction or shrinkage, all the articular structures—skin, bone, and cartilage—participating in this secondary atrophic change. The temporo-maxillary and cervical articulations are almost invariably implicated. Coincidentally severe wasting of interossei and extensor groups of muscles ensues, with marked exaggeration of the deep tendon reflexes. Enlargement of the lymphatic glands is not infrequent, while pigmentation, localized sweatings, and vasomotor derangements are often striking collateral features. Cardiac troubles are rare. The temperature, when raised, is of low grade, when compared with other arthritic affections, especially so when we bear in mind the widespread involvement of joints. The pulse-rate is frequently quickened, and this feature, when accompanied by pyrexia of low grade and persistent swelling, especially of swollen joints, is very suggestive of rheumatoid arthritis. Considered briefly, the foregoing constitute the cardinal pathological and clinical features of rheumatoid arthritis, and we now pass on to consider the second group, osteo-arthritis or hypertrophic arthritis.

The second group, osteo-arthritis or hypertrophic arthritis, now claims attention. From an ætiological standpoint it contrasts with rheumatoid arthritis in that infective conditions do not appear to be so intimately related to its genesis, while, on the other hand, the influence of trauma, so inconspicuous in rheumatoid arthritis, is very much in evidence in osteo-arthritis. Additional factors are to be found—senility, arterio-sclerosis, and in those cases which arise so frequently at or about the menopause. It is possible that its onset at this period may be

determined by some modification in the internal secretions either of the thyroid or ovary, or both. As to its morbid anatomy, in this variety of arthritis deformans the essential anatomical lesions are those produced in the cartilaginous and bony structures, which undergo enlargement or hypertrophy. Most authorities now agree with Virchow that the synovial tissues are only involved secondarily. Histologically, the ground substance of the cartilage undergoes a process of segmentation, while its cellular elements proliferate. Ultimately the cartilages by attrition become thinned, and their central portions, where pressure is greatest, being at length completely worn away, the denuded ends of the bones appear, and these subsequently become sclerosed and their surfaces eburnated. On the other hand, the peripheral portions of the cartilage, not being subject to such pressure, instead of being thinned become thickened, the proliferating elements forming irregular crests or nodules, which, primarily cartilaginous, ultimately become ossified. The osteophytic outgrowths thus developed are the source of the deformity of the affected joint, and are responsible also for the limitation of its mobility, but, although complete locking may occur, bony ankylosis does not ensue.

The secondary changes in the synovial membrane have been specially studied by Hoffa, who distinguished two stages in their production: (1) The first is characterized by villous overgrowth, proliferation of fixed connective-tissue cells, with extensive pigmentary deposit. (2) In the second phase, degenerative processes predominate, the synovial membrane undergoing numerous changes and ultimately complete transformation into structureless hyaline tissue, a metamorphosis said by Hoffa to be pathognomonic of the affection. The excessive deposit of pigment noted by Hoffa is of great interest, and may possibly possess some ætiological significance. Bowlby, as we know, has shown that intra-articular hæmorrhage can initiate osteo-arthritic changes in the subject of hæmophilia. Now it is quite possible in many instances of osteo-arthritis a long-forgotten trauma may have supplied the initial impulse to the morbid process. Godlee has recently adduced some striking instances in which a joint injured in early life has in later years become the seat of osteo-arthritis. Such injuries, it is conceivable, might readily determine the occurrence of a greater or less degree of intra-articular hæmorrhage, which by its presence might initiate what might be termed a reactive osteo-arthritis. It is easy to understand how, once started, the morbid process tends to perpetuate itself. Thus during the natural exercise of a joint so affected the roughened cartilaginous surfaces and bony asperities must be a source of continual irritation to the synovial

membrane, which in its turn responds by new formation of connective tissue and hypertrophy of its tufts. Again, these elongated synovial processes, either by becoming free in the cavity or from time to time becoming incarcerated between the joint surfaces, contribute to its irritability, and a vicious circle is thus established. This hypothesis as to the *modus operandi* of trauma may account for the presence of the abnormal degree of pigment noted by Hoffa.

It will be convenient here to institute a comparison between the anatomical features of rheumatoid arthritis as opposed to osteo-arthritis.

(1) In rheumatoid arthritis the initial changes are found in the cartilaginous tissues, and bony tissues are only affected secondarily.

(2) In osteo-arthritis, on the other hand, this sequence of pathological events is reversed, the primary alteration taking place in the bones and cartilages, the synovial membrane being only implicated secondarily.

(3) The character also of the changes in the bone and cartilage differs in the two affections. Those in rheumatoid arthritis are retrograde and passive, while those in osteo-arthritis are active and proliferative. In short, atrophy of bone and cartilage is the distinguishing feature in rheumatoid arthritis, hypertrophy in osteo-arthritis.

(4) Bony ankylosis occurs not infrequently in rheumatoid arthritis; rarely, if ever, in osteo-arthritis.

From the clinical aspect osteo-arthritis is essentially a disease of the middle and later period in life, being rarely met with under the age of 40. It affects both sexes equally. The mode of onset is usually insidious, and the course of the disease slow but progressive. Constitutional disturbance is slight, the temperature not raised, the pulse not quickened, while pigmentation and trophic disturbances are generally absent. Muscular atrophy is slight. The articular lesions are but rarely generalized, the tendency of the disease being to involve but a few joints, and these not symmetrically, while it shows a predilection for the larger articulations. In these respects it contrasts with rheumatoid arthritis, which is polyarticular, symmetrical in distribution, with a marked preference for the smaller joints. Moreover, when the smaller joints of the fingers are attacked by osteo-arthritis the terminal phalangeal joints are sites of election, as opposed to the marked affinity displayed by rheumatoid arthritis for the mid-phalangeal articulations. *Local characters* of the articular swellings will obviously be more easily studied in exposed articulations, by preference the mid-phalangeal; unlike rheumatoid arthritis when it affects the same articulations, the

skin over the joints presents no abnormal features, and at no period does it undergo atrophic change. Marked swelling, when present, is due to synovial effusion and not to periarticular thickening; hence we get no spindle-shaped appearance. On the contrary, the outline of the joints, instead of being smooth and rounded, is broken and irregular, owing to the presence of bony outgrowths studding the line of junction of the articular ends. Displacement of the bones, when it occurs, is usually lateral in direction, and due directly to the mechanical thrust of the bony excrescences, and not to muscular spasm. In rheumatoid arthritis, on the other hand, hyper-extensions and luxation are the rule, and for these muscular retractions are directly responsible. This diversity between the articular lesions of these two diseases becomes more pronounced in the later periods of their life-history. Thus in rheumatoid arthritis the one-time voluminous spindle-joints undergo progressive shrinkage—skin, bone, and cartilage tending to wither away. Eventually the articular structures dwindle into what Goldthwait has aptly termed the small-end joint of rheumatoid arthritis. On the contrary, in osteo-arthritis the more ancient the lesion, the more massive the bony outgrowths, the more marked the enlargement of the affected articulations.

In concluding this brief account of osteo-arthritis, one cannot help commenting on the paucity of its collateral phenomena as compared with rheumatoid arthritis. This, to our mind, constitutes the salient distinction between these two diseases. Osteo-arthritis is much more a disease of joints, and joints only, than rheumatoid arthritis, in which the arthritic changes form only one feature in a very varied clinical picture.

It now remains for us to consider the third division—i.e., infective arthritis—for it cannot be doubted that examples of this nature intrude themselves into any category of arthritis deformans, however scrupulously compiled. These cases of infective arthritis have to be considered chiefly in regard to rheumatoid arthritis, more especially its acute forms, inasmuch as it is in connexion with them that confusion is most likely to occur. Before proceeding further, it will, I think, be in the interests of lucidity if I preface my remarks by stating that I do not propose dealing with the differential diagnosis of those conditions reputed to be most frequently confounded with acute forms of rheumatoid arthritis. I refer, of course, to acute rheumatism, acute polyarticular gout, acute gonorrhœal rheumatism, and secondary syphilitic arthritis. Now, with regard to all these joint affections, if we except acute gout, their association with a

definite micro-organism has been more or less clearly established. Apart from these, however, there still remain a number of joint affections hitherto unaffiliated to any organism with which undoubtedly acute rheumatoid arthritis is very commonly confused. In this absence of any bacteriological findings, we have to rely upon those clinical features which apparently distinguish acute rheumatoid arthritis from these non-suppurative types of infective arthritis. Unfortunately, nothing can be gained from the age incidence of these two varieties of joint disorder, for both occur at all periods of life. But something may be gathered from a marked predilection of rheumatoid arthritis for the female sex. In approaching the study of such cases the previous history should be carefully elicited, especially as regards the recent occurrence of some of the infective maladies known to be followed occasionally by non-purulent forms of arthritis, such as dysentery, typhoid fever, and so forth. If such be forthcoming, it would constitute strong presumptive evidence that we are dealing with an infective arthritis following on other of these diseases rather than one of acute rheumatoid arthritis. Again, in the presence of local foci of infection, such as pyorrhœa alveolaris, otorrhœa, &c., it must not be too readily assumed that the joint condition is one of acute rheumatoid arthritis, for undoubtedly many of the instances of joint trouble occurring in such association are examples of infective arthritis. But when, as too frequently happens, no past or present source of infection can be detected, reliance must be placed on the following characteristics. Thus in infective arthritis the constitutional symptoms are often much more acute, the temperature is of higher grade, swelling of the lymphatic glands in association with the infected joints is almost invariable. The spleen is more commonly enlarged, while leucocytosis and a secondary anæmia are usually present. As regards the articular lesions, though a large number of joints may be involved in infective arthritis, in my experience such widespread invasion is not the rule. What happens in the majority of instances is that at the onset of the malady the disease attacks one, two, or three joints, and, having done so, shows no tendency to involve any other articulations, even though weeks or months may have elapsed during which the temperature may have been more or less continuously raised. Now, had the condition been one of acute rheumatoid arthritis, it would by that time have implicated all, or a large number of articulations. Again, the mode of distribution differs. In infective arthritis the large joints are by preference attacked, though I have known exceptions to this rule in the case of the small joints of the hand. In rheumatoid arthritis the disease almost invariably

begins in the smaller joints. Again, in infective arthritis the joint lesions show no tendency to symmetrical involvement, contrasting with rheumatoid arthritis.

With regard to the character of the articular lesions, nothing can be gleaned from their shape, for the spindle-shaped joints, so frequently claimed to be pathognomonic, of rheumatoid arthritis are exactly reproduced in cases of infective arthritis, when it attacks exposed joints such as the phalangeal. But the overlying skin in infective arthritis generally shows a definitive red blush, whereas in rheumatoid arthritis it is unnaturally white or bluish in colour. Again, widespread inflammatory œdema of high grade is not infrequently present in infective arthritis, and forms a very valuable aid in differentiation. Finally, apart from articular lesions, we find in acute rheumatoid arthritis, as opposed to infective arthritis, an abundance of collateral phenomena. Consequently, in those comparatively rare instances of rheumatoid arthritis in which the disease remains for some time confined to the small articulations of the hand, the following suggestion may prove helpful in distinguishing the case from one of infective arthritis: If the opposite hand, hitherto unaffected by joint lesions, is found to be the seat of vasomotor phenomena, neuralgic pains, or muscular cramps, the presence of such will be in favour of the condition being one of rheumatoid arthritis. While the foregoing, to my mind, constitute the chief points of distinction between these infective arthritides and the more acute forms of rheumatoid arthritis, it must be confessed that the dividing line is by no means sharply defined. In all probability a satisfactory differential diagnosis will not be available on purely clinical grounds, but on bacteriological data, which it is hoped will soon be forthcoming.

DISCUSSION.

Dr. FORTESCUE FOX was not disposed to attach great importance to the atrophic or hypertrophic character of arthritis, or to its commencement in the cartilages or synovial membranes, for purposes of classification. Arthritis was a degenerative joint affection, resulting from many causes, singly or in combination. In the early decades of life, when the tissues were most susceptible to infective diseases of all kinds, arthritis was commonly infective, acute, and generalized. It was part of an acute invasion characterized by rapid and progressive wasting and constitutional disturbance like that accompanying tuberculosis. At present it was often impossible to arrest this almost malignant arthritis of the young. A different clinical picture was presented by the arthritis of the middle period of life, in which infective changes were mixed with gout and the climacteric. Sometimes gout alone at this period of life set up a degenerative arthritis, usually hypertrophic, and with a favourable prognosis. The arthritis of middle life was remarkably amenable to treatment in the majority of cases. Lastly, the senile period of life had its own arthritis—"a disease of dissolution," in which infection and gouty elements had a certain place, but the main causative factor was traumatism. The arthritides of the basal joint of the thumb and of one hip or shoulder were common varieties of senile arthritis. He suggested that the anatomical distinctions upon which Dr. Jones had insisted were rather to be regarded as belonging to the *epoch* or the *phase* of the disease.

Dr. PRESTON KING could not follow Dr. Jones in his fine distinctions between rheumatoid arthritis and infective arthritis. He believed rheumatoid arthritis not to be a distinct disease so much as a train of symptoms and changes following from infections from various centres. The "soil" factor was of more importance than was apt to be recognized. The quick pulse, sweating, and pigmentation of the skin were not, in his experience, confined, as Dr. Jones suggested, to the later osteo-arthritic or hypertrophic forms of the disease, nor were hyper-extensions and ankyloses confined to, or even more common in, the earlier rheumatoid cases.

Dr. BUCKLEY (Buxton) said he recognized the existence of the three forms of arthritis deformans, but that there were many intermediate types. He believed in the infective theory, but considered that very different forms might arise from the same infection, the modifying factors being especially such constitutional states as goutiness in its widest sense, impoverished conditions of the blood such as might arise from anæmia, habitual constipation, &c., and from external causes such as trauma and general wear and tear. The possible combinations of these factors could produce many apparently differing forms of arthritis. Hypertrophy of the structures in the neighbourhood of the affected joints sometimes occurred in the later stages of cases beginning as atrophic arthritis, and this, in his opinion, indicated an attempt at repair of the damage

and was accompanied by the disappearance of the more acute symptoms. It was therefore possible that some cases which should apparently be classified as hypertrophic arthritis were due to the action of the same infective agent as the atrophic form, but that the powers of resistance in the individual were much greater. It had recently been suggested that Heberden's nodes were due to attempts to compensate for excessive wear and tear, or to repair damaged structures—a view which deserved careful consideration.

Dr. HALE WHITE thought that the previous speakers had not laid enough stress upon the extreme chronicity of the slight joint changes to be met with in some cases of infective arthritis, nor to the frequency with which these changes were due to gonorrhœa. He was of opinion that there was a definite disease to which the name "rheumatoid arthritis" should be applied. He detailed the symptoms of a typical case, and his description tallied with that given by the reader of the paper. He admitted that some cases of arthritis, in which the source of infection could be clearly traced, closely resembled those of rheumatoid arthritis, and he thought it would conduce to clearness of thinking if such cases were called rheumatoid arthritis with a prefix indicating the micro-organism which was the cause of the trouble; but in the present state of our knowledge there remained a considerable number of cases of rheumatoid arthritis, chiefly occurring in young women, the micro-organism causing which was at present unknown, but that a micro-organism was the cause he had no doubt. He had been fortunate in obtaining a post-mortem examination of such a case, and his description of the microscopic changes in the joints agreed with that given by Dr. Llewelyn Jones. The principal feature was that the disease began in the synovial membrane and in the tissues around the joint, the cartilage often not being affected.

Dr. T. FRED GARDNER (Bournemouth) drew attention to a monograph issued by Mr. Hugh Lane, of Bath, nearly twenty years ago, where the connexion between rheumatoid arthritis and tuberculous disease was well shown. The results of treatment, on the whole so disappointing, showed that remedies like cod-liver oil, arsenic, cream, fats, &c., had a beneficial effect on some cases. A history of arrested tuberculous lymphadenitis was of very common occurrence in rheumatoid disease. He also pointed out the striking fact that in advanced cases of rheumatoid disease changes occurred in the spinal cord indistinguishable from bulbar disease, pointing to an ascending degeneration. One such case in the speaker's experience exhibited the classical symptoms of bulbar paralysis.

Mr. GORDON WATSON believed that Dr. Jones divided arthritis deformans into two main classes—the synovial (rheumatoid) and the bony (osteo) arthritis. Broadly speaking, he would like to argue that these two main classes were quite distinct in their pathology—the former infective, the latter traumatic. Of the latter class he had seen many examples, following definitely on injury to a healthy single joint in young people. During the past week he had operated in a case exhibiting the classical changes associated with osteo-arthritis. The knee had been injured, and the internal semilunar cartilage torn. Recurrent

attacks of "locking" and subsequent synovitis had resulted in well-marked osteo-arthritis, with fibrillation of the articular cartilage of the femur and osteophytic outgrowths from the margins of the tibia. These changes, it seemed clear, had resulted from the chronic irritation which the internal derangement produced. He would like to see a sub-committee of the Balneological Section appointed for the purpose of defining, on a pathological basis, the many varieties of arthritis deformans.

Dr. F. H. HUMPHRIS, while admitting that there are some cases in which treatment seems unsatisfactory—he would not say useless—thought some attention should be drawn to two therapeutic agents which at any rate afford relief from the pain and stiffness. He had had six cases of rheumatoid arthritis referred to him during the last three months, and in none of them did he fail to get some measure of relief. They were treated with high candle-power electric light placed in a suitable reflector, followed by an administration of static electricity.

Mr. MIDELTON said he was quite sure Dr. Llewelyn Jones had excellent reasons for mapping out the types of arthritis he had submitted for criticism. He (Mr. Midelton) believed thoroughly in continuous counter-irritation to the spine and joints, and, if necessary, to other parts of the body, in the treatment of arthritis deformans, as suggested by Dr. P. W. Latham and others. It mattered not how long the disease had existed, or its variety; he approached it with confidence provided there was no serious complication, such as tuberculosis or cancer. He had twenty-seven cases under his care, of one sort and another, being treated by means of continuous counter-irritation. He would welcome any member of the Section at Bournemouth, and would show them as many cases as possible.

Dr. SOLLY said that he believed that all cases described as rheumatoid or osteo-arthritis, atrophic or hypertrophic, were due to infection. The differences were due to variations of degree rather than kind, and while in some cases it was comparatively easy to recognize the particular form of infection, as, for instance, in gonorrhœal arthritis, in others the actual organism could not be ascertained; but all the evidence gradually accumulating seemed to favour the view that all arthritis was primarily infective. A mixed infection was probably always present, and the combined action of various organisms, mixed in different proportions, would explain many of the variations in the classes of symptoms. Many forms of arthritis cleared up rapidly after the removal of some apparently trivial but constant source of septic infection, such as pyorrhœa alveolaris, or chronic nasal or tonsillar catarrh. Chronic colitis and chronic leucorrhœa seemed to be responsible in cases which had come under his observation; and, without going into the question of "appendix dyspepsia," it was quite certain that there were a great many people going about the world affected with chronic catarrh or even definite ulceration of some of their mucous membranes sufficient to provide a constant source of insidious septic infection, without being actually aware of the fact. It would be in everyone's range of

experience that persons have become suddenly ill and died from hæmorrhage from a duodenal ulcer of which there had been no previous indications recognizable. That was only one illustration, and, while arthritis of the "rheumatoid" type could be *proved* to be due to septic infection in a considerable number of cases, he considered it absolutely impossible for anyone at present to disprove the existence of some infection in any case. It should also be remembered that the early stages of the disease, as in the case of infantile paralysis, are often overlooked, and it is the track of the storm and not the storm itself which attracts the most attention.

Dr. ACKERLEY, though he acknowledged that there was some convenience in using a comprehensive term, thought that it would be wiser if, when a case of arthritis was seen, it was called simply an arthritis, and an endeavour were made to discover the cause or causes in that particular case. The arthritis was only one manifestation of some disease. But what was much needed was a more thorough study of the soil. Recognizing the ætiological factor of gonorrhœa, pyorrhœa alveolaris, and other forms of sepsis, what was it that determined an arthritis in one man and not in another? He doubted whether there was a real distinction between acute and chronic cases. Was it not possible that the chronic forms simply depended on a smaller daily dose of the particular toxin, or greater personal resistance to its effects?

The discussion was adjourned until the next meeting.