BRITISH MEDICAL JOURNAL

LONDON SATURDAY AUGUST 20 1938

RHEUMATOID ARTHRITIS*

BY

WILFRID EDGEcombe, M.D., F.R.C.P., F.R.C.S.

Honorary Consulting Physician to the Harrogate and District General Hospital

The function of the opener of a discussion on the subject of "rheumatoid arthritis" is, I take it, first of all to give a concise summary of the present state of our knowledge; secondly, to indicate the gaps in that knowledge which require filling; and, thirdly, to stimulate discussion in a somewhat provocative way. The discussion has been wisely limited to rheumatoid arthritis, properly understood.

As a starting-point I propose to take the Report of the B.M.A. Committee on the Causation and Treatment of Arthritis and Allied Conditions, which appeared in the *British Medical Journal* of June 17, 1933. This report gives a summary of the state of our knowledge at that time, and it would seem profitable to consider that report and endeavour to assess what progress has been made during the five years that have elapsed since it was issued.

Nomenclature and Classification

The classification adopted by the Committee still stands as the most convenient clinical grouping. In it rheumatoid arthritis is divided into "primary," of hitherto undetermined causation, and "secondary," due to ascertained focal infection. Until it is definitely known whether primary rheumatoid arthritis is an infective disorder it must remain in a category of its own. The evidence in favour of its being infective is accumulating, and if and when it is finally established as of infective origin it will merge into the secondary group, having fundamentally a similar pathogenesis.

Clinical Features

The clinical features of the two diseases, if they be separate entities, are well known. The general resemblance of the two conditions is very striking. Nothing has appeared in published work during the last five years to occasion any fundamental changes in this comparison. Attention may be drawn to the extraordinary frequency of the primary disease among women as compared with men. The proportion given by various authors varies from nine to two females to one male. What is the explanation? Is it because they are women and therefore some female sexual disturbance may be at fault; or is it because women are more subject, from their habits and

environment, to the various causes said to predispose to the disease—emotional instability, sedentary lives, poor circulation, defective nutrition, and so forth? A definite answer to these questions would be illuminating.

Aetiology

The fundamental problem that remains unsolved is that of infection: is primary rheumatoid arthritis an infective disease, or are other causes competent to produce it without infection?

The B.M.A. report in its summary of the aetiological factors says categorically, "In primary rheumatoid arthritis, unknown; in secondary, infection"; and this apparently is the sole reason for distinction between them. If it be admitted that secondary rheumatoid arthritis is an infective disease the clinical picture of the two conditions is so extraordinarily similar and the points of difference so relatively unimportant that it is difficult to resist the overwhelming presumption that the primary disease is also infective; but because no constant infective organism has been found that will satisfy Koch's postulates the infective origin is regarded as not proven. If it be an infection it is certainly not clear-cut and straightforward, comparable to other proved infective diseases. Hence various theories have been put forward to explain the nature of the infection, which may be summarized as follows:

1. *Bacteraemia*.—Infective organisms gaining entry into the body settle in the joint tissues and there provoke the manifestations of arthritis. Against this is the rarity with which organisms are demonstrated in, and recovered from, the joint tissues or synovial fluid. In gonococcal arthritis, however, it is notoriously difficult to demonstrate the organisms in the joints, yet no one doubts that gonococcal arthritis is of infective origin.

2. *Bacterial Toxaemia*.—To overcome the difficulty mentioned it is assumed that the joint changes are provoked not by the actual presence of infective organisms but by the irritation of toxins elaborated by organisms situated at some distant and unascertained site in the body.

3. *Bacterial Allergy*.—This theory assumes that the joint tissues become sensitized to antigens derived from various organisms in the body, not necessarily frankly pathogenic but possibly also saprophytic. The group of organisms incriminated is the streptococcus, both haemolytic and

* Read in opening a discussion at a joint meeting of the Sections of Physical Medicine and Physical Education, and Orthopaedics and Fractures at the Annual Meeting of the British Medical Association, Plymouth, 1938.

viridans. The evidence against the staphylococcus is less convincing.

4. *Virus Theory*.—Schlesinger, Signy, and Amies (1935), working on pericardial fluid from rheumatic pericarditis, claim to have discovered the elementary bodies of a virus agglutinated by the serum of patients with acute rheumatism. Eagles, Evans, Fisher, and Keith (1937) discovered similar elementary bodies in exudates from rheumatic fever, chorea, and rheumatoid arthritis, which agglutinate with sera of the corresponding disease. They regard their findings as indicating a common origin of rheumatic fever and rheumatoid arthritis. Goldie and Griffiths (1936) draw attention to the close relation between rheumatic fever and rheumatoid arthritis. They regard them both as infective, and consider that the clinical manifestations may be as well explained by changes in tissue sensitivity at different ages, and by variations in the reaction of patients to infection, as by assuming different infective agents.

To elaborate the arguments would be impossible within the limits of this opening address. A critical summary of the case for and against the infective theory has been given by Davidson and Goldie (1936). Poynton and Schlesinger (1937) discuss the whole subject in a temperate and judicial manner with full reference to the available literature. More recently Hench (1938) of the Mayo Clinic gave the most exhaustive account of the whole subject I have yet seen. His conclusions are: "As a clinical investigator I must conclude that the cause of rheumatoid arthritis is still unknown, and that the evidence for infection, though very impressive, is incomplete. As a practising physician I cannot evade the issue. I have committed myself, with reservation, to the microbic theory." With that conclusion, I think, most of us will agree.

Other theories advanced as causes, exciting or contributory, of rheumatoid arthritis are:

- (a) Metabolic abnormalities, from deranged carbohydrate, protein, or sulphur metabolism; or from food allergy or vitamin deficiency.
- (b) Circulatory deficiency, leading to "joint hunger" and consequent arthritis.
- (c) Endocrine abnormality.
- (d) Neurogenic disturbances of the sympathetic nervous system.

Hench (1936) states: "The last two or three theories are often combined to explain the pathogenesis of rheumatoid arthritis as follows: the endocrine imbalance affects the sympathetics, thereby impairing the articular and general circulation, general and articular metabolic abnormalities are fostered, and arthritis develops, with or without the aggravating influence of infections."

Therein lies the crux of the matter—with or without infection. The trend of modern research during the last five years is undoubtedly in the direction of the infective theory—that infection is the primary factor—the other factors being ancillary and subordinate, insufficient in themselves to give rise to the disease, but nevertheless necessary as providing a suitable soil for infection to take root. In short: seed present, soil unfavourable for its growth—no arthritis; soil favourable, no seed sown—no arthritis; seed sown in favourable soil—arthritis develops.

I suggest infection, yes or no, as the first and most important point for discussion.

Morbid Anatomy

There are no significant differences in the morbid anatomy of the two conditions. The resemblance is so strong as to strengthen further the presumption of an

aetiology common to both. Confirmation by Parker and Keefer (1935) has been made of the lymphocytic infiltration of the joint tissues described by Allison and Ghormley (1931) and regarded by them as specific to rheumatoid arthritis, contrasting with the polymorphocytic infiltration that occurs in frankly pyogenic infection. Fisher (1937) draws attention to the close resemblance in the microscopical appearances of the tissues in rheumatic fever and rheumatoid arthritis, both showing lymphocytes, histiocytes, and giant cells, but no leucocytes unless pyogenic organisms were present.

Biochemistry

Sedimentation Rate.—All observers are agreed that the sedimentation rate is increased in rheumatoid arthritis, both primary and secondary; in contradistinction to uncomplicated cases of osteo-arthritis, in which it remains practically normal. In some cases of osteo-arthritis a rise in rate is found, and in these it is suspected that an infective process has been superadded, resulting in a mixed type of case, combining the features of rheumatoid arthritis and osteo-arthritis. In rheumatic fever also the sedimentation rate is increased, but, curiously enough, no increase is found in cases of chorea (Poynton and Schlesinger, 1937).

There is general agreement as to the value of the sedimentation rate, first as an aid to diagnosis in differentiating rheumatoid arthritis from osteo-arthritis: if the sedimentation rate be raised in the absence of other causes tending to increase it—such as intercurrent febrile disorder, anaemia, etc.—suspicion is at once aroused of the presence of infection. In the second place, it is of value as a measure of progress in response to treatment. With clinical improvement the sedimentation rate diminishes, but commonly there is a lag between the two: clinical improvement outstrips the improvement in sedimentation rate, and until the latter is restored approximately to normal the disease cannot be regarded as arrested; an infective process is still at work. Further, in cases of apparent recovery a subsequent rise in sedimentation rate is often one of the first signs of impending relapse.

A discussion recently took place at the Royal Society of Medicine in the Section of Physical Medicine on the effect of physical treatment on the Arneth count and the sedimentation rate in rheumatic conditions (Gibson, Hill, and Collins, 1938), at which there was general agreement on the behaviour and value of the sedimentation rate. Observers, however, differed on the value of the Arneth count. Gibson found no parallel between it and the sedimentation rate. Hill observed a shift to the left in both rheumatoid arthritis and osteo-arthritis, and a parallelism between it and the sedimentation rate. Collins found the Arneth count stable during physical treatment, and the count does not reflect improvement as does the sedimentation rate. In osteo-arthritis he found a shift to the left, most marked in cases that showed cystic areas of rarefaction in the bones when radiographed.

One clear point emerged from the discussion, and that was the urgent necessity for an agreed standardization of the method of making the test. The same method used throughout in any group of cases is obviously valuable, but for the comparative measurement between different groups uniformity of method is essential.

Biochemical Changes.—No notable advance has been made in the biochemistry of rheumatoid arthritis. According to Race (1935) there is a diminution of plasma pigments and an alteration in the relative amount of albumin and globulin in the blood serum, the globulin

being increased by 2 per cent., which is equivalent to 100 grammes in 5 litres of blood. Assuming a similar alteration in the tissue fluid, there would be a calculated reduction of 300 grammes of albumin in the circulating blood and a large increase in globulin. Kahlmeter (1934) also finds an increase in blood fibrinogen and globulin and a decrease in the total blood protein of the rheumatoid subject. The significance of these changes is unknown. They may have a bearing on the changes in sedimentation rate.

Gastric Juice.—On the hypothesis that free hydrochloric acid acts as a germicidal barrier to the passage of organisms through the stomach to the intestine, many workers have examined the incidence of achlorhydria and hypochlorhydria in rheumatoid arthritis. Their findings vary within wide limits; some claim an incidence as high as 70 per cent., others a lower range of 20 and 30 per cent. After careful examination of all the data it has been concluded that the incidence of achlorhydria or hypochlorhydria in arthritis is five times higher than in normal subjects, but only slightly higher than in certain other diseases, the difference being too slight to warrant any definite conclusion being made as to a causal connexion.

Metabolism

No outstanding discoveries have been made during the past five years. No constant metabolic changes are found in rheumatoid arthritis, primary or secondary. Basal metabolism, nitrogen and fat, uric acid, phosphates, and lactic acid vary within the normal limits (Pemberton, 1929; Race, 1935). With regard to calcium, observers differ: some find a marked hypercalcaemia (Mark, 1923), others have failed to observe alterations from normal.

Sulphur metabolism (Cawadias, 1934) shows individual changes not characteristic of the disease. Carbohydrate metabolism (Pemberton, 1929) shows a lowered sugar tolerance ascribed to diminished utilization of sugar by the tissues, resulting presumably from defective circulation. No evidence of an acid or alkaline diathesis is deducible from the urine (Race, 1935).

In specific infective arthritis such as that following typhoid, dysentery, or pneumonia, it will be generally agreed that the metabolic changes observed—rise of temperature, loss of weight, anaemia, increased sedimentation rate, diminished glucose tolerance, etc.—are the result of reaction to the infective organism. In secondary rheumatoid arthritis due to focal sepsis the same applies. Why not to primary rheumatoid arthritis? Because so far no definite causal organism has been discovered that fulfils the criteria necessary to establish proof. And since no definite proof of an infective organism is forthcoming many writers have sought to incriminate metabolic and endocrine factors as the causal agents.

Endocrine Disturbances

In the B.M.A. report there is a reference to the endocrine glands as follows: "Endocrine imbalance between thyroid, pituitary, adrenal, ovarian, and corpus luteum secretions is very frequent. Hyperthyroidism is especially associated with primary rheumatoid arthritis, and hypothyroidism with chronic villous arthritis." Vague statements on "endocrine imbalance" as a contributory, if not a primary, cause frequently appear in the literature of primary rheumatoid arthritis, but little that is definite is said regarding the exact nature of that imbalance. And, indeed, when we consider the complexity of the relations

that exist between the endocrine glands and the paucity of our knowledge on the subject—greatly though it has increased during the last few years—it is not surprising that hypothesis tends to outrun fact. The *pituitary*, so far as is known, has no direct influence in the production of arthritis. In so far as it controls through its thyrotropic and gonadotropic hormones the functioning of the ovary and thyroid, it may be said to have an indirect influence on those forms of arthritis in which endocrine disturbance is prominent.

OVARY

Mention has been made of the extraordinary frequency in the incidence of primary rheumatoid arthritis in women as compared with men, and the suggestion made that this may be based on some primary ovarian disturbance. The amenorrhoea so common in the disease is a related phenomenon. Since the isolation of the ovarian follicular hormone as an active therapeutic agent many clinicians, especially among the Germans, have been using it, not only in climacteric arthritis, in which form there is stronger evidence of an underlying ovarian deficiency, but also in primary rheumatoid arthritis. Encouraging reports continue to appear in the German literature, but it is too soon yet to evaluate the results accurately.

THYROID

A study of the literature shows that rheumatoid arthritis may be associated with either hyper- or hypo-thyroidism, or with the two states alternating. The obvious inference is that there is no direct causal relation between the thyroid and the disease. The supporters of the endocrine theory regard the role of infection as bringing to light the underlying diathesis; the supporters of the infective theory regard the thyroid manifestations as the result of infection, and conceive the overactivity of the thyroid as an expression of a reactive attempt to control the infection by quickening up the normal metabolic processes of the body.

In a disease like rheumatoid arthritis, characterized by marked osteoporosis and loss of calcium salts in the bones, the *parathyroids* naturally come under suspicion. In spite of the work of Oppel and of Leriche the evidence for their incrimination is inconclusive, but further work is needed to establish the true relation between the parathyroids and rheumatoid arthritis.

Radiology

X-ray photographs of affected joints are of no help in distinguishing primary from secondary rheumatoid arthritis: the changes seen in both are alike—generalized osteoporosis, followed later by destruction of cartilage and articular bone and in the final stage by subluxation and ankylosis.

Treatment

In a disease the precise aetiology of which is as yet unknown, treatment must necessarily be largely empirical and symptomatic; hence the numerous methods recommended. The trend of recent years has been to stress, first, the importance of early recognition of the disease and the paramount need for early and sustained treatment. The sooner treatment is begun the better are the chances of arrest of or recovery from the disease. Secondly, the tendency is to regard the local manifestations of joint disorders as part of a general systemic disease, and hence to pay as much attention to the general health of the patient as to the local treatment of the

affected joints. And here I would stress the urgent necessity for the provision of institutional treatment for cases of rheumatoid arthritis on the same lines as is done for tuberculosis. Were it a lethal disease this would have been done long ago; but since it is merely intensely disabling and crippling little has been done about it. The prolonged treatment required, both general and local, can seldom be effected in a domiciliary way except for the very well-to-do. For the poorer classes treatment is all too frequently spasmodic, disjointed, and unsustained, with the inevitable result of permanent disablement. Rest, fresh air, warmth and sunlight, generous feeding, treatment to the joints by physical methods—dry or moist heat, massage, passive movements, splinting when necessary, diathermy, x rays, orthopaedic measures—together with specific drug treatment, as by vaccine or by gold therapy: where can all these be effectively combined other than in institutions, well equipped and staffed for the purpose? Much of our disappointment in results is due to lack of facilities for concerted, concentrated, and continued treatment. Professor Stanley Davidson at the Bath Conference on Rheumatic Diseases in April last forcibly stressed this aspect of the subject, maintaining that the voluntary hospitals under present conditions are quite inadequate to provide the necessary facilities for dealing with the large numbers of sufferers from the rheumatic diseases, and that municipal or State aid is required for the provision of centres for early investigation and prolonged treatment. The creation of rheumatic centres in various parts of the country, though belated, is a beginning of what must ultimately develop on a greatly extended scale if the rheumatic scourge is to be effectively combated.

VACCINES

The advocates of vaccine therapy in the treatment of rheumatoid arthritis, "primary" and "secondary," presumably accept unreservedly the infective theory of causation; otherwise the rationale of their treatment falls to the ground. The uncertainty as to the specific organism involved—beyond the general incrimination of the streptococcal group—casts doubt on the specificity of vaccine treatment. Multiple vaccines on the hit-or-miss principle are clearly unscientific, and any therapeutic benefit from them must result from general rather than specific stimulation of reaction against infection. My criticism of the published results of vaccine treatment in rheumatoid arthritis is that they are not subject to adequate control—first in diagnosis, and, secondly, by comparison with parallel cases untreated by vaccines. In secondary rheumatoid arthritis of proved infective origin, if the focus of infection has been removed, vaccines are usually unnecessary: recovery will take place without their aid. In primary rheumatoid arthritis where no infection is demonstrated vaccine treatment rests on no firm foundation, and is hypothetical in theory and disappointing in practice (Okell, 1937). I have seen too many cases of rheumatoid arthritis, treated by prolonged vaccine therapy alone to the exclusion of other measures, gradually drift into a helpless and hopeless condition when other means might have been usefully employed, to leave me in any doubt.

My considered opinion is that vaccine therapy has been frankly disappointing, and on inquiry from various workers in centres where adequate investigation and control of cases is undertaken, I find that vaccine therapy is being used less and less: in some centres it has been given up entirely. I suggest this as a fruitful topic for your discussion.

PHYSICAL TREATMENT

Perhaps the greatest advance in the physical treatment of rheumatoid arthritis has been the recognition of the importance and value of rest to the affected joints. This is obtained by temporary fixation in plaster, supplemented after a period of complete rest by daily passive movements of the joint through its normal range. By this means rest is assured to the inflamed joint, contractures into faulty positions are prevented, and subsequent deformity or disability is avoided. The more care taken in the early stages to prevent fixation in faulty positions, the less the need for drastic orthopaedic measures in the later stage. Active collaboration between physician and orthopaedic surgeon should take place throughout: prevention is better than cure (Kindersley *et al.*, 1936).

CHRYSOTHERAPY

Reference to chrysotherapy in the B.M.A. report is limited to a single sentence: "Gold preparations given by intramuscular injection have been used abroad for the treatment of arthritis, but beneficial results have not been confirmed." Since then much progress has been made. In my experience gold therapy represents the greatest advance in the drug treatment of arthritis made during the last thirty years. The almost dramatic results that are achieved in a relatively short space of time in selected cases are little short of marvellous. From no other form of treatment in former years have I seen such gratifying improvement even in advanced and seemingly hopeless cases. Careful choice of cases, however, should be made. The tendency is to use gold indiscriminately in all rheumatic affections and thereby to bring discredit on the method. Practically all observers are agreed that it is of no value in true osteo-arthritis and in non-articular rheumatic diseases: its use should be limited to primary and secondary rheumatoid arthritis—in short, to the presumably infective forms. The risks of untoward complications involved are not inconsiderable, but they are minimized by the use of a smaller dosage than was initially thought necessary, by careful test for idiosyncrasy to the drug and by a preliminary blood count and sedimentation rate before embarking on a course, and by the instant withdrawal on the first symptoms of intolerance. The results achieved by gold therapy are well worth the risks that may be run. Hartfall, Garland, and Goldie (1937) have done the most extensive work on gold treatment in this country. They have published a review of 900 cases, giving the clinical analyses and results in 750 cases of rheumatoid arthritis and in 150 cases of other forms of arthritis, with the toxic reactions that occurred in the whole series. Your collective experience of gold treatment would be instructive.

SPA TREATMENT

In answer to the question, Is spa treatment helpful in the management of rheumatoid arthritis? the reply is undoubtedly, Yes. It should be premised that there is nothing specific in the waters of the spas in this or any other country as a cure for rheumatoid arthritis. The advantages of spa treatment are the removal from home surroundings to a new and healthy environment and the psychological element of hope; the availability at any well-equipped spa of a wide range of physical treatments, hydrological and balneological; access to medical advice from practitioners of special knowledge from long experience of the disease; facilities for thorough investigation, bacteriological and biochemical, necessary for diagnosis and control of treatment; and the use of natural mineral

waters for the correction of metabolic derangements and the improvement of general health, so necessary a part of a concerted attack on the disease.

The disadvantages are the expense entailed in a spa cure, which must necessarily be prolonged if good results are to be achieved. In so chronic a disease swift improvement cannot be effected in a short space of time. Nevertheless, even in a limited period a start may be made and a patient be put on the high road to recovery, which may be followed up at home.

REFERENCES

- Allison, N., and Ghormley, R. K. (1931). *Diagnosis in Joint Diseases*, Oxford Medical Publications, London.
- Cawadias, A. P. (1934). *Acta Rheum.*, 6, 26.
- Davidson, L. S. P., and Goldie, W. (1936). Reports of British Committee on Chronic Rheumatic Diseases, 2, 1, London.
- Eagles, G. H., Evans, P. R., Fisher, A. G. T., and Keith, J. D. (1937). *Lancet*, 2, 421.
- Fisher, A. G. Timbrell (1937). *Ibid.*, 1, 1162.
- Gibson, H. J., Hill, L. C., and Collins, D. H. (1938). *Proc. roy. Soc. Med.*, 31, 309.
- Goldie, W., and Griffiths, G. J. (1936). *British Medical Journal*, 2, 755.
- Hartfall, S. J., Garland, H. G., and Goldie, W. (1937). *Lancet*, 2, 784, 838.
- Hench, P. S. (1936). Reports of British Committee on Chronic Rheumatic Diseases, 2, 83, London.
- (1938). *A Survey of Chronic Rheumatic Diseases*, p. 35, Oxford Medical Publications, London.
- Kahlmeter, G. (1934). Conférence Scientifique Internationale du Rhumatisme Chronique Progressif Généralisé, Aix-les-Bains.
- Kindersley, C. E., et al. (1936). *Proc. roy. Soc. Med.*, 29, 237.
- Mark, R. (1923). *J. Metabolic Res.*, 4, 135.
- Okell, C. C. (1937). Reports of British Committee on Chronic Rheumatic Diseases, 3, 4, London.
- Parker, F., and Keefer, C. S. (1935). *Arch. Path.*, 20, 507.
- Pemberton, R. (1929). *Arthritis and Rheumatoid Conditions*, Baillière, Tindall and Cox, London.
- Poynton, F. J., and Schlesinger, B. (1937). *Recent Advances in the Study of Rheumatism*, 2nd ed., Churchill, London.
- Race, J. F. (1935). Reports of British Committee on Chronic Rheumatic Diseases 3, 4, London.
- Schlesinger, B., Signy, A. G., and Amies, C. R. (1935). *Lancet*, 1, 1145.

A. Dubois (*Thèse Paris*, 1938, No. 140) states that there are numerous cases on record indicating the possibility of tumours developing after trauma. In the absence of any knowledge as to the mechanism of the formation of new growths it is impossible either to affirm or to deny the relation of cause and effect between the trauma and the tumour. Experimental work does not yet appear to have provided sufficient data to prove or disprove this connexion. Most discussion has taken place about the development of tumours after a single trauma, whereas the possibility of the formation of new growths after repeated injuries is readily granted. There are numerous cases on record of the occurrence of sarcoma after injury to bone or muscle, and a large number of cases have been reported in which tumours of the lung, stomach, intestine, or kidney have developed after a trauma and in which the most reliable methods of investigation—such as radiography or surgical operation—have shown the absence of any tumour before the trauma. Tumours of the testis or brain are remarkably frequent after trauma. Numerous cases are also on record in which aggravation of the tumour following trauma is shown by increase in its size and the formation of metastases, accompanied by deterioration of the general condition. In order to affirm that a tumour is of traumatic origin a number of conditions must be present—namely, a latent period varying with the nature of the tumour, identity of the site of the trauma and of the tumour, absence of any tumour before the trauma, and histological confirmation of the presence of the new growth.

ORTHOPAEDICS IN RHEUMATOID ARTHRITIS*

BY

NORMAN CAPENER, F.R.C.S.

*Orthopaedic Surgeon, Princess Elizabeth Orthopaedic Hospital, Exeter, and Mount Gold Orthopaedic Hospital, Plymouth ;
Consultant Orthopaedic Surgeon, Royal Devon and Exeter Hospital, Exeter*

As a simple orthopaedic surgeon I feel very diffident in contributing to the opening discussion on this subject, which has such wide general medical aspects. Dr. Edgecombe, however, in his excellent exposition has prepared the ground very well for me. He has drawn attention to the need for special centres for the treatment of this crippling disease. It is not generally realized, however, that the orthopaedic hospitals of the country are being asked to do an increasing amount of work in this field. This is, for example, true of the orthopaedic hospitals of Devon, where it has been found that the facilities we have for the treatment of tuberculosis of joints, infantile paralysis, and fractures are applicable to the prolonged general and local treatment that Dr. Edgecombe has stressed.

To take bone and joint tuberculosis as an example, we have to deal with much else besides the purely orthopaedic problems of immobilization and surgery. Opportunities are provided for the more important general treatment of the disease, combined with facilities for physical treatment and with occupational and educational interests. These facilities render an orthopaedic hospital almost an ideal centre for the treatment of rheumatoid arthritis.

In orthopaedics our great concerns are to preserve as well as to restore function; to prevent just as much as to correct deformity. In rheumatoid arthritis, as to a large extent in bone and joint tuberculosis, we stress from the outset of the disease the importance of the natural recuperative powers of the body if placed under proper conditions—namely, the protection from painful stimuli, the utilization of the beneficial effects of sunlight, fresh air, warmth, cleanliness, discipline, and what is well called "physiological rest."

Physiological Rest

This involves immobilization of an affected joint in the best physiological position for the relief of pain; for the arrest of the inflammatory process; for rest to over-active muscles and the retardation of muscle atrophy. The position should be the best for function should ankylosis occur. This immobilization is combined with periodic movement and with other measures to improve joint and muscle function of a type and quantity suitable for the particular state of the disease. In the active stage the amount of the permitted movement and physical therapy may be *nil*, and may remain so for two weeks or more. After the acute stage has subsided function is graduated according to the progress of the individual joint. While affected joints are being rested those unaffected are given greater opportunities and encouragement for movement.

Having generalized in this manner I feel that I should proceed with a detailed statement of what are these optimum positions for physiological rest. Before doing

* Read in opening a discussion at a joint meeting of the Sections of Orthopaedics and Fractures, and Physical Medicine and Physical Education at the Annual Meeting of the British Medical Association, Plymouth, 1938.