been possible, it has been our experience that the prediction of the result has often been erroneous; in many cases the improvement was much greater than had been anticipated. Bailey (1951) states that of his 214 cases 41.6 per cent. were excellent, 32.7 per cent. improved and 13 per cent. unaltered after operation. Brock et al. (1952) after a careful follow up of their first 50 cases state that of the 42 survivors, the results were—excellent in 17; good in 15, four were fair only and five were poor. Our experience is closely similar to these two series and although there is necessarily a personal factor in assessing these results, which accounts for small differences, there is no doubt that the operation is of enormous value to many of these patients, who from chronic invalidism may be returned to an almost completely normal life.

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RHEUMATISM: THE PRESENT POSITION
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Not many years ago little interest was taken in the articular diseases. The field of 'rheumatism' was a loosely defined one and in this ill-defined area physician, orthopaedic surgeon, physiotherapist and others wandered rather at random. Therapy was and still is largely palliative and empirical. The rheumatoid in particular derived little help from any form of therapy except certain forms of physiotherapy, splinting, support and graded rest, measures which are still the backbone of treatment. Knowledge of the exact aetiological agents, without which therapy must continue to be empirical, is still lacking. Since the war more attention has been devoted to the study of this group of disorders and, when in 1949 Hench and his colleagues reported the dramatic effect of cortisone on rheumatoid arthritis, a great impetus was given to research into study of diseases of the locomotor system. Now, three years later, it is perhaps time to take stock of the present position.

Nomenclature
A glance at the history of medicine shows certain concertina-like features in the study of disease groups in which the aetiology is unknown. 'A,' a great man in his day, classifies all the diseases and points out their differences, splitting them into sub-groups on clinical and pathological grounds. 'B,' equally eminent, some years later draws attention to points of similarity throughout the whole group and unifies them as best he can. Later 'C,' a brilliant young worker, splits them up again and adds his name to two syndromes which emerge from the reshuffle. In the absence of exact knowledge this can go on indefinitely. A prolonged search for infective agents has, except in a few instances, proved fruitless in the field of rheumatology and there seems little to hope for in this direction with current methods. The pendulum is now over to host reaction rather than invading organism, to disease of soil rather than seed. The recent paper of Kellgren (1952) should be read in this context.

In this country our classification of the rheumatic disorders is in agreement with that used in the United States of America with only few exceptions. Only the more common disorders are discussed below.

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Rheumatoid Arthritis

This disorder is the most serious of those to be considered. While not as common as degenerative (osteo) arthritis it remains a great scourge and a major cause of loss of working time. It may affect anyone of either sex from three years of age onwards. While commonest in the female sex in middle age it is no respecter of persons and may occur out of the blue in a previously normal person of any age, even in the eighties. There is no evidence that physical or psychological trauma or any specific infection brings it on in most cases; in the vast majority no such factors can be found and the careful controlled study by the Empire Rheumatism Council (1950) failed to find any statistical evidence of such factors playing a part. Mental and physical stress and strain are an everyday part of our environment. Individual cases can always be found in which the onset of disease occurred shortly after some such upset, but there is no real evidence that stress plays a regular part in the onset of this disease in a statistically significant number of cases.

Rheumatoid arthritis may come on suddenly and dramatically and ape rheumatic fever; more frequently its onset is insidious and the disease remains undiagnosed for months. It is likely that mild abortive forms are frequent, for rheumatoid arthritis is a reversible disease and can cure itself by mechanisms as yet unknown. In the subgroup called palindromic rheumatism, most of which cases eventually turn out to be rheumatoid arthritis, the disease may cure itself every week for months, though eventually such cases usually develop irreversible changes and become established rheumatoids. Such cases have in high degree this ability to reverse the disease process, but study of them has not yet revealed the mechanism.

In jaundice and pregnancy the disease may virtually disappear as it may after operations or severe physical upset. This last is likely the manner in which shock or fever therapy in the past operated, but this remains largely speculative. Rheumatoid arthritis may, therefore, because of this self-reversing capacity, come on not only insidiously but also intermittently. Many early sufferers are considered to be cases of foot strain if the feet are early affected. Stiffness on waking is often an early symptom; weakness with stiffness, particularly in hands and wrists, may precede pain and swelling. Such early manifestations may be diagnosed as ‘fibrositis’ by the doctor, but are frequently called ‘rheumatism’ by the patients themselves. In general it may be said that any such early morning stiffness-cum-weakness is strongly suggestive of early rheumatoid arthritis if it involves more than one joint. The same applies to stiffness with swelling involving more than one joint. An elevated sedimentation rate with no other apparent cause for it in addition to one of the above symptoms is added evidence. Hydrarthrosis may be an early sign of rheumatoid arthritis or of ankylosing spondylitis. I have seen many cases of intermittent hydrarthrosis where remittent-relapsing swelling of the knees was present for months or years before other signs of rheumatoid arthritis or ankylosing spondylitis appeared.

Monarticular disease is less likely to be rheumatoid, more likely to be traumatic or gouty in origin. However, early rheumatoid arthritis may start first in a traumatized joint and latent rheumatoid arthritis may first flare up in such a traumatized joint. Nevertheless, rheumatoid arthritis in the majority of cases is a symmetrical bilateral disease and in this respect differs from other disorders, notably gout.

Rheumatoid arthritis after onset may proceed in various ways. It may remit, occasionally permanently, it may remit and relapse, gradually progressing over the years to a measure of permanent change and crippling, or it may occasionally progress rapidly in fulminating form. The second method is by far the commonest and the patient learns gradually, in the words of the old coloured philosopher, ‘to co-operate with the inevitable.’ This last point should be underlined. There is no known curative therapy to date; even the best is suppressive or supportive. The patient will live with his disease, suppressed or unsuppressed, for many years, perhaps for his lifetime. He therefore comes to terms with it, evolves a philosophy to match his degree of crippling and a way of life to match his capabilities. Such adaptation should not be lightly discarded by instituting suppressive treatment such as cortisone, for if the latter has to be in turn discarded there may be a difficult period of readjustment to the old way of life after what has been in retrospect merely an excursion from symptoms and a holiday from the disease.

Quite apart from therapeutic advances, there have been several discoveries in the field of diagnosis and in the natural history of the disease which are worthy of note. The sensitized sheep cell agglutination test (Rose et al., 1948; Svartz and Schlossman, 1950) has proved its worth. While only positive in one-third to one-half of rheumatoids (Ball, 1950), it is negative in all other disease processes but for acute disseminated lupus erythematosus and some cases of scleroderma. False positives are rare. It is almost invariably negative in osteo-arthritis, gout and, interestingly enough, in ankylosing spondylitis. Muscle biopsies have shown in about the same percentage of
cases small round cell accumulations between muscle fibres alongside vessel and nerve, sometimes after the disease has completely abated. Although such cell foci have been reported in normals and in other diseases they seem vastly more common in rheumatoid arthritis than any other disease process and are almost always negative in gout, ankylosing spondylitis and osteoarthritis (Freund, 1945; Desmarais et al., 1948).

Although cardiac lesions are reported (Rosenberg, 1949) in rheumatoid arthritis they are essentially rarities of histological interest for there is little clinical evidence of rheumatoid disease of the heart in life. The same applies to rheumatoid disease of the lung though some possible cases are reported (Ellman, 1948; Hart, 1948).

Rheumatoid arthritis, as has been pointed out by many workers, is not just a disease of bones, joints and cartilage, but is characterized by generalized systemic upset with changes in muscles, tendons and other tissues. The alternative title, rheumatoid disease, has much to commend it.

As regards therapy, graded rest and graded exercises and exercise, with supportive measures and immobilization of the acutely inflamed joint remain, with general health measures, the backbone of treatment. Analgesics are essential for most cases and acetyl salicylic acid (aspirin) in one form or another, remains the most popular and the most widely taken. Cortisone (Compound E) and corticotropin (ACTH) are the most effective anti-inflammatory agents to date, for they not only reduce pain but also swelling and stiffness. In mild cases they may remove all signs of the disease in therapeutic doses; in more severe cases only partial suppression can be achieved in doses that will not cause undesirable side-effects. There is no doubt that these drugs will temporarily reverse, in part at least, the disease process, but the effect in most cases is only partial and in all cases is temporary. Even after a year's continuous therapy, relapse will occur within a few days or weeks of stopping treatment. As cortisone is given by mouth it is the drug of choice in rheumatoid arthritis, as corticotropin (ACTH) has to be given by injection. At present the majority of rheumatologists prefer to give the smallest maintenance dose that will maintain the patient approximately 70 per cent. free from symptoms and signs; total suppression is not now the aim. There is no doubt that effective temporary suppression can be achieved in the majority of moderate cases in such dosage and few people now use more than 75 mgm. a day, dosage being by mouth three to four times a day evenly spaced before meals. Continued dosage higher than this may prove dangerous and should not be used. More heavy dosage, in the region of 500 mgm. a day (Bayles, 1952), in the hope of giving more prolonged remission, has been tried, found unsatisfactory and has been given up. Most patients, then, can be promised partial temporary freedom from symptoms and signs. They cannot be promised a cure or even a lasting remission. There is no evidence that this treatment halves the disease or makes a natural remission more likely.

There is no evidence either way to date that it speeds up or slows down radiological changes. The treatment carries certain risks that should constantly be kept in mind:

1. Mild side effects are of little import and are usually controlled by slight dose reduction. They include the round moon face, acne and striae on abdomen and shoulders. Electrolyte disturbances are usually slight and temporary; as there is in some cases sodium retention and/or increased potassium loss in the urine some physicians prefer to restrict salt intake while on therapy and to give potassium supplements (2 to 4 gm. potassium chloride a day by mouth).

2. Osteoporosis may occur. While in the average case this complication remains a remote risk only, in patients already suffering from postmenopausal or old-age osteoporosis this therapy may readily lead to crush fractures of the thoracic or lumbar vertebrae. The bedridden or only slightly active patient who cannot on treatment be adequately mobilized runs a very real risk of this complication and the drug is best given only to patients who can be adequately and reasonably rapidly mobilized on therapy.

3. Patients with irreversible bony change as the cause of their crippling will improve little and there will be no improvement on therapy in old fixed disease. Cortisone or corticotropin treatment in such cases can be used as a short-term measure to facilitate the instigation of other forms of treatment, but should not be continued as long-term therapy unless reasonably full mobilization can be achieved.

4. There is a real risk of gastric or duodenal ulceration. Haemorrhage and perforation has occurred. As the drug suppresses inflammation and therefore the manifestations of inflammation, peritonitis may be unrecognized. Fatalities have already occurred. If a past history of peptic ulceration is forthcoming cortisone treatment should not be instituted unless the patient is fully alive to the risk and is prepared to take it. Whether the physician is prepared to take it depends on the individual. Rheumatoid arthritis is not a killing disease and any therapy causing any risk at all to life should be only very carefully and cautiously administered.
568 POSTGRADUATE MEDICAL JOURNAL November 1952

(5) Infections may be masked. Fatalities have already occurred for this reason; patients have died of pneumonia of which there were no obvious signs. While such cases are rare, they have occurred. In treating patients the fact that cortisone suppresses the inflammatory response without removing the underlying disease should be borne in mind. Pulmonary and other forms of tuberculosis may be activated.

(6) Patients of unstable mentality may be upset by cortisone and a latent psychosis may become active. It is possible that the question of addiction may arise in the future.

(7) A latent diabetes may become frankly diabetic and a known diabetic worsen on therapy. Insulin dosage needs to be stepped up during cortisone treatment.

(8) Skin wound healing on therapeutic doses will show little change or rarely some slight delay. Healing of recent intestinal wounds such as intestinal anastomoses may be delayed on full therapeutic doses.

(9) Care should be taken with hypertensive patients and patients with congestive cardiac failure to maintain strict salt restriction and close observation throughout treatment. Cortisone is a safer drug than corticotropin in this group of patients.

(10) Feeling vastly better a patient on therapy may do too much; after prolonged periods of bed and chair rest, reablement must be gradual and exercise only gradually increased.

The above list looks forbidding and immediately raises the question: As rheumatoid arthritis is, per se, not a lethal condition but a crippling one it is legitimate to prescribe for it a purely suppressive palliative therapy which may, albeit rarely, cause disaster? Against this one has to oppose the prolonged pain, distress and crippling of this most unpleasant condition. Most patients are prepared to take all the risks for the chance of leading once more a near normal life and returning to work and normal duties. At present this decision is made easier for the doctor and patient in this country by restriction of supplies.

Long continued cortisone therapy is still sub judice; excellent accounts are to be found in the papers of Freyberg et al. (1951), Boland (1951) and Copeman et al. (1952). Controlled trials are now under way in this country. It is legitimate in the present state of knowledge to restrict the drug to those patients where risks of the above complications are slight, where the disease is not far advanced and there there is little or no irreversible destructive disease and crippling. The daily maintenance dose should not exceed 75 mgm. per diem, gradually reducing to the minimum necessary to maintain freedom from the more distressing symptoms and signs. When doses fall as low as 25 mgm. per diem it is likely that the disease has spontaneously settled under cover of the drug, which may then be discontinued for a trial period, permanently if there is no relapse. When treatment is discontinued it is best gradually to taper off the dosage; sudden cessation at high therapeutic level leads to the so-called rebound phenomenon, the disease returning with a rush to a state even worse than before treatment, usually with great mental upset and distress and occasionally with signs of partial adrenal insufficiency.

Short-term therapy, to cover short extremely painful bouts of activity and to precede and follow surgical or manipulative measures is entirely reasonable. Cortisone, though it can be given by one intramuscular injection a day, is now almost invariably administered by mouth at regular six- to eight-hourly intervals before meals.

Little has been said so far of corticotropin (ACTH). It is less useful in rheumatoid arthritis as it cannot be given orally, but intramuscularly six- or eight-hourly in doses varying from 20 to 80 mgm. a day it may be used for short-term therapy. The hazards are as with cortisone, but as it is rarely used for long-term therapy the complications of long-term suppressive treatment hardly arise. Abscesses and bruising occasionally give trouble at the site of injection. It may also be given intravenously and it appears that 20 mgm. given continuously intravenously over a period of eight hours gives approximately the adrenal cortical stimulation afforded by 80 to 100 mgm. or more intramuscularly by divided dosage. It has also been given subcutaneously with the spreading factor hyaluronidase. Here there is a risk of spreading infection unless aseptic technique is flawless.

Finally, hydrocortisone (Compound F) unlike cortisone (Compound E) has proved of value in local treatment and instillation into joints has given good results in the hands of Hollander (1952) and others. The effect appears to be entirely suppressive and not curative. Dosage depends on the size of the joint: 25 mgm. for a knee, 1 to 2 mgm. for a finger joint. Doses of more than 50 mgm. even for a large joint, are not recommended and even at this dosage they may cause local exacerbation of swelling and pain. Such injections may be given as often as required, usually every one to three weeks, until the affected joint subsides, the dosage being then spaced out with longer intervals between.

Hydrocortisone may be used locally in conjunction with general oral cortisone treatment. Although a useful addition to therapy, it remains palliative and needs to be repeated frequently. The patient must be warned not to over-exercise the joint when relief is obtained. The drug should
be given with strict aseptic technique or there is risk of infection, but this can be done in consulting room or surgery and without resort to full operating theatre conditions. It has at the time of writing received only scanty trial in the United Kingdom.

A host of other substances have been tried; with the exception of cortisolone, hydrocortisone and corticotropin all have been found wanting. Pregnenolone, deoxycortone and ascorbic acid and many others have now been generally discarded. 'Irgypyrin' (Geigy) has the disadvantage of containing amidopyrine which, because of the risk of causing agranulocytosis, has long been viewed with disfavour both in the United Kingdom and the U.S.A. Its solvent, butazolidine, orally or by injection, has good analgesic properties but probably no true anti-rheumatic ones. In my own series of cases no constant effect was noted in reduction of swelling, though pain relief did occur in most cases. Currie (1951), however, who made the original observation that butazolidine was as effective as 'Irgypyrin,' finds reduction of swelling in about one-third of his patients.

Finally, one of the most vital and often the most difficult thing to decide in rheumatoid arthritis is what measure of local and general exercise to allow. Uncontrolled rest is worse than no rest at all; an untreated patient who has kept active is perhaps crippled but is getting about, while the untreated one who has given up the struggle and taken to bed and chair has almost certainly developed deformities which may prevent her walking again. Every case in this respect is an individual problem; whatever programme is started in hospital the physician must see to it that this is continued religiously at home. All too often relapse occurs soon after returning home either because the patient has broken the rules or because the therapeutic programme was insufficiently detailed by the physician.

Ankylosing Spondylitis
(Rheumatoid Spondylitis)

This disorder is considered in the United Kingdom to be sufficiently distinct, clinically, radiologically and therapeutically, to be considered as a separate disease entity, though in the U.S.A. the latter title is used as it is considered to be a variant of rheumatoid arthritis. As the aetiology of neither of these conditions is known, it seems to me to be reasonable to keep it as a separate entity until such time as more is known of the aetiology of the rheumatic diseases as a whole. Clinically it is different in that the patient is usually a young male, and the disease largely—if not entirely—confined to spine, pelvis and thorax, peripheral involvement being more rare, with finger and wrist involvement rarer still.

Radiologically, the sacro-iliac joints, almost invariably normal in rheumatoid arthritis, are invariably abnormal in ankylosing spondylitis. Therapeutically deep X-ray therapy, of little or no help in the treatment of rheumatoid arthritis, is the therapy of choice in ankylosing spondylitis, together with suitable spinal and breathing exercises and measures designed to avoid kyphotic deformity. As immobilization tends to increase and accelerate stiffness and fusion, much more activity is allowed the spondylitic than the rheumatoid and unless the disease is in acute exacerbation he is not confined to bed. Deep X-ray therapy is now administered locally to the spine and sacro-iliac joints; the wide-field 'X-ray bath' of Gilbert Scott is no longer given. While not curative, prolonged remissions frequently occur lasting several months or in some cases years. Almost all patients experience symptomatic improvement. Cortisone and corticotropin are of great help in the acute phases; relapse tends to occur as soon as therapy is discontinued unless natural remission has set in (Hart, 1952). Few spondylitics are so bad that continuous therapy need be considered, though such cases do occur. Plaster shells and prolonged immobilization in any form are now considered an incorrect form of therapy; light supporting jackets to maintain good position are advocated by some (Swaim, 1939), though others consider the benefit slight or non-existent. Certainly most jackets I have seen on patients have been too heavy and cumbersome and have restricted full respiratory movements without controlling posture adequately. As the thorax is involved in this disease, costovertebral and costo-transverse joints being affected so that rib movements are restricted and vital capacity reduced by reduction of supplemental and complementary air, breathing exercises are usually advocated as a routine measure; supports that restrict respiration are therefore contraindicated. This does not apply to the light supporting jacket advocated by Swaim, which does not restrict thoracic expansion and is constructed to throw the spine into slight extension on full inspiration. For marked forward curvature of the spine, wedge osteotomy may be performed (Smith-Petersen, 1945; Law, 1949), the upper portion of the spine above the bone wedge removed being 'rocked back' on the lower.

Variants of Rheumatoid Arthritis

Psoriatic Arthropathy. In many cases rheumatoid arthritis occurring in conjunction with psoriasis presents no different features from the usual picture. In others the 'sauce' finger with
involvement of the terminal interphalangeal joint is seen in addition to the more common proximal joint and metacarpo-phalangeal joint swelling in the same finger. The terminal interphalangeal joint only may be affected in some fingers, often where psoriasis exists in the nail itself, though not invariably. It is usual to see this terminal joint spared in rheumatoid arthritis but involved in osteo-arthritis with formation of Heberden’s nodes. Psoriatic arthropathy is the exception. Therapeutically it is exactly the same problem as ordinary rheumatoid arthritis with, in addition, the skin condition. Corticotropin and cortisone have their usual effect on the arthritis; the psoriasis behaves variably, sometimes improving dramatically, sometimes showing no improvement at all, occasionally worsening slightly. Any beneficial effects noted are transient in almost all cases.

Still’s Disease. What has been said of rheumatoid arthritis in general applies also in Still’s disease and the same general lines of therapy hold. Opinions regarding cortisone are mixed; its beneficial effects last only so long as therapy is continued. Schlesinger (1951) is of the opinion that it should not be regarded as orthodox therapy as the disease appears to progress in spite of suppression of symptoms.

Felty’s syndrome is that variant of rheumatoid arthritis with features of Still’s disease occurring in the adult. Splenomegaly, lymphadenopathy and leukopenia, sometimes with pigmentation of the skin, occur in a small percentage of rheumatoids, often the more severe cases, but there is no need to make this a separate group as far as therapy is concerned. Some of the above features occur commonly in rheumatoid arthritis; occasionally all four features are present. Splenomegaly has been performed in this condition and in simple rheumatoid arthritis, but with mixed results. Like so many forms of therapy it remains unproved. Cases have been known to worsen after operation; any operation occasionally benefits the rheumatoid sufferer temporarily, possibly by causing adrenocortical stimulation and increased output of cortical steroids. A percentage of cases of rheumatoid arthritis (‘the inevitable 30 per cent.’) will improve on any form of therapy; this simple fact has been the cause of literally hundreds of vaunted cures in the past.

Gout. This classical disease has come in recently for further investigation and the use of isotopic nitrogen (N 15) to measure the ‘miscible pool’ of uric acid has been reported (De Witt Stetten, 1950).

In therapy there is little new and colchicine, the hermodactyl or ‘Finger of Hermes’ of the Greeks, continues to be the usual standby. Colchicine in an acute attack is best given hourly by mouth in $\frac{1}{2}$ mgm. (gr. 1/120) dosage until relief is obtained, gastro-intestinal irritation has occurred or until 10 to 12 doses have been given. After relief is obtained it is best to continue with suppressive doses of $\frac{1}{2}$ mgm. three or four times a day for several days, gradually reducing the dose. Corticotropin (ACTH) will abort an acute bout rather more quickly in most cases in doses of 50 mgm. six-hourly. Usually no more than three or four injections are necessary; colchicine should be given over the same period and for some two weeks subsequently in dosage of $\frac{1}{2}$ mgm. thrice daily to prevent a ‘rebound’ attack which may otherwise occur.

Colchicine is given in some parts of the world in severe attacks intravenously in dosage of 1 to 3 mgm.; this is not without its dangers and generally speaking the oral route is to be preferred. Recently Freyberg (1952) reported administration of butazolidine (phenyl butazone) as effective in rapidly aborting acute gout.

In ‘interval’ gout between acute attacks it is usual to give no treatment, but if attacks are severe and frequent, various forms of therapy may be tried. Gutman (1951) has shown that tophi can be reduced and the output of urates in the urine consistently increased by prolonged administration of salicylates or ‘Benemid’ (di-n-propylsulfonyl benzoic acid). The latter may precipitate acute attacks. Colchicine can be given for prolonged periods orally, $\frac{1}{2}$ mgm. twice or thrice daily; it has no effect on plasma uric acid or urinary excretion of urates and there is no evidence of adreno-cortical stimulation; its mode of action remains unknown.

Oral cortisone is more variable than corticotropin in its action and on the whole has proved a disappointment. Recently hydrocortisone (Compound F) has been injected into the inflamed joint and success has been claimed.

Rigid dietetic restrictions are now rarely imposed; certain foodstuffs and drinks may be forbidden, but it is impracticable to impose too rigid a regimen on a patient whose disease lasts for the duration of life. Surgery may be used to improve local deformities; local amputations of fingers or toes may occasionally be necessary.

Osteo-Arthritis

This degenerative condition is the commonest of the articular diseases; it is present in varying degree after middle age in most people and appears to be part of the ageing process. Nevertheless it can be quite as crippling and as productive of pain as rheumatoid arthritis.

In a condition notoriously resistant to therapy the following are the more useful practical measures:
Weight reduction. Patients with osceo-
arthritis are frequently obese; the joints affected are weight-bearing (hips and knees) in most instances.

(2) Simple, non-habit-forming analgesics (e.g. acetylsalicyclic acid or compound aspirin tablets).

(3) Graded exercise with rest.

(4) Local physiotherapy or light dosage deep X-ray therapy.

(5) Local injections of procaine or, more recently, hydrocortisone. Unfortunately the hip is the most difficult of joints to inject and one of the commonest to be involved.

(6) Surgery—arthrodesis, cup arthroplasty or, more popular at the moment, an acrylic prosthesis, the diseased femoral head being replaced by an acrylic cup and stem. Vitallium and stainless steel is also used.

Kellgren and Moore (1952) have pointed out that osceo-arthritis can come on acutely and painfully and in its generalized form is not infrequently mistaken for rheumatoid arthritis. Heberden's nodes may, in the early stage be hot, painful and throbbing; only later on are they the classical painless bony nodularities. In most cases, however, the two diseases are sufficiently distinct to allow of accurate firm diagnosis.

Conclusions

It is impossible to summarize a summary. The most interesting event since the war in the field of rheumatism has been the discovery of the effects of some of the adreno-cortical hormones on rheumatoid disease. This has intensified and concentrated research in a previously rather neglected field of medicine. Rheumatism is responsible for more suffering and more loss of working time than any other organic disease; because it holds no immediate risk to life it has not the dramatic appeal of other disorders to therapist, research worker or generous donor. Nevertheless, it is of prime importance to the community to attack this group of diseases with all methods available. This country has in the past made many major therapeutic discoveries; let us hope its next will be in the field of rheumatic disease. There is no shortage of clinical material.

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