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Polyarthritis

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The scope of this talk is inflammatory polyarthritis excluding rheumatoid arthritis and gout. A print of 1809*, when rheumatism was a subject which was regarded very seriously (the golden age of gout) illustrates some of the primitive views on the aetiology of rheumatic diseases. It shows little gnomes sticking pins in a sufferer, and as far as our knowledge of the aetiology of rheumatoid arthritis goes, such a concept is probably as good as any that we have today. The primitive classification of the rheumatic diseases into " rheumatism, gout, and catarrh " has been improved upon slightly by even the most elementary student catechism nowadays which lists congenital, traumatic, inflammatory, neoplastic, metabolic, and degenerative types and those of unknown aetiology.

The rheumatic diseases still represent a great field of clinical opportunity, for to a certain extent they are unexplored. The gastroenterologists and cardiologists have more or less reached the limits of their diagnostic and investigational possibilities. They can explore their patients through every orifice conceivable, natural and man-made, and hardly any recess of the body is sacrosanct. In rheumatology, it is still possible by mere clinical observation, the use of hands, elementary methods of diagnosis and investigation, and with some understanding of the natural history of these diseases, to elucidate a great deal of clinical knowledge. If we proceed from what is known to conditions of unknown aetiology in a diagnostic classification, we shall commence with some of the problems liable to be encountered in practice, such as the infections. Here the aetiology is known, and it is a striking fact that the rheumatic diseases of known aetiology are generally those which are the most rewarding to treat. This is also true of gout, where even if the full aetiology is not known we know something about the pathogenesis. This suggests that instead of hopelessly and empirically trying remedy after remedy in clinical trial after clinical trial, it would be more profitable to divert a lot of this energy into a study of the basic origins and pathogenesis of these diseases, followed by a systematic and rational attack upon the disease process itself.

Fortunately now a rare form of arthritis, suppurative arthritis, is illustrated by a patient who sustained a crush injury with minor laceration and took little note of it but over a period of eight days developed progressive pain and swelling of the finger with progressive suppurative changes in the proximal interphalangeal joint. Not only was the joint damaged, but the infection also spread through the

*See frontispiece.

shaft of the phalanx towards the terminal interphalangeal joint. Accidents probably still represent the commonest cause of suppurative arthritis, and I see one or two patients a year with "rheumatism" which is unsuspected suppurative arthritis. Septic joint infection may also follow joint injections, but this is fortunately extremely rare. In my experience and in most of the published surveys it occurs only once in 7,000 occasions when an intra-articular steroid injection is given with proper aseptic precautions, but it explains why intra-articular injections of steroid are usually given in clinics appointed for the purpose.

Another rarity is gonococcal arthritis. Gonococci can be isolated from the joint fluid, and there is a large polymorphonuclear infiltration into the sub-synovial tissues. The majority of cases of so-called gonoccocal arthritis are in fact not gonococcal at all, but due to some co-incident infection or non-specific urethritis.

Another problem still with us is that of the tuberculous joint, particularly in the middle-aged and elderly. Very often the patient has an unsuspected pulmonary focus, and often the joint infection comes from a neighbouring bone. Tubercle bacilli may be grown from material obtained on joint aspiration. Syphilitic arthritis, with the gross mutilation and destruction seen in Charcot's joints, is a late phenomenon and there is no infective change present in the joint. This is probably a disease that is going to become extinct over the next few years, unless there is some late relapse of cases treated inadequately by antisyphilitic measures during the last war. Changes in Charcot's joint include softening of the bone ends and subluxation with gross attempts at repair, and overgrowth. These changes are far in excess of anything one would see with osteoarthritis and the patient is singularly free from pain in proportion to the extent of damage.

There remain a few other types of arthritis which are not clearly differentiated or understood but which are nevertheless important and usually seen in general practice; these are the acute arthritic processes associated with the exanthemata. The best recognized of these is rubella, and I am very glad that Dr Pickworth is here because some years ago he published by far the best account of rubella arthritis. Recent experience in the rubella epidemic of 1962 has re-emphasized the importance of this and its incidence (as high as 15 per cent). I have seen 5 patients referred to hospital for rheumatoid arthritis who had in fact subacute or chronic arthritis following rubella. Curiously enough, this complication appears to be almost restricted to females of child-bearing age. In addition to polyarthritis there appears to be diffuse swelling of the soft tissues, so that carpal tunnel syndrome with compression of the median nerve is a

not uncommon concomitant. In the survey of polyarthritis done by Dr Lawrence and Professor Kellgren and his colleagues, one feature was the discovery of numerous cases of so-called " benign polyarthritis " in patients who had had undoubted and well documented episodes of polyarthritis from which they had made a complete recovery. Very likely, many of these were associated with rubella or possibly with measles or infectious hepatitis, possibly even in subclinical attacks. It is quite clear that here is a field for profitable study in general practice; such studies would contribute greatly to our knowledge of both the geography and the epidemiology of many of these forms of arthritis.

Proceeding from conditions of known aetiology and known relationship to those where the relationship is only partly understood, I would like to discuss rheumatic fever. There is presumptive evidence that a haemolytic streptococcus belonging to Lancefield group A is the consistent aetiological agent in rheumatic fever, usually as an acute streptococcal sore throat, although I have seen cases of rheumatic fever following streptococcal infections at other sites. Acute streptococcal sore throat due to a group A, β-haemolytic streptococcus is followed in a certain proportion of cases by an attack of rheumatic fever and then in a certain proportion by rheumatic heart disease. As part of the aetiology is known, it is possible to interrupt this chain at one point by giving penicillin prophylactically for many years to prevent recurrences. It is the recurrent rheumatic fever attacks that carry with them the extraordinary risk of rheumatic heart disease. A patient who has had one attack of rheumatic fever is more liable than the average person to have a second attack and it is the second attack and subsequent attacks which are liable to be accompanied by carditis and subsequent severe cardiac damage.

It has been a matter of dispute for some time whether there is a form of chronic rheumatic fever which results in the development of a polyarthritis similar to, but distinguishable both on clinical and serological grounds from, rheumatoid arthritis. There is a considerable amount of French literature on this point, suggesting that this is liable to be seen in subjects who have had two or more attacks of rheumatic fever. The syndrome has been called " Jaccoud's syndrome ", and again it would be a matter of some research over a period of years, probably by groups from several practices, to establish whether such an entity did exist and what its characteristics were.

Polymyalgia rheumatica has been isolated and identified within the last few years. It still does not warrant a status as an individual entity, but it has been separated from the morass of rheumatological

material. Most of these patients were previously considered to be suffering from an acute form of rheumatoid arthritis which atypically involved the shoulders, the hips, and occasionally the knees and elbows in people at the age of 60 and beyond. It has a relatively good prognosis after a stormy course, usually subsiding within two years and occasionally sooner, but it is liable to leave stiffness and some residual deformity. It is characterized by its very prompt response to small doses of steroids and/or phenylbutazone.

Reiter's syndrome also represents a departure from diseases of known aetiology to one where there is a certain amount of reason to suspect an infectious illness, associated with urethritis. The salient features of this disease are urethritis, conjunctivitis, and polyarthritis. A non-specific urethritis is usually followed fairly quickly by conjunctivitis, iritis or iridocyclitis, and a polyarthritis. The heels and the tarsal regions are particularly liable to involvement but no joint is immune, and mucocutaneous lesions of mouth or genitalia may occur. This disease has a striking predilection for males and is so rare in females that only a handful of cases have been described. While it may follow venereal infection with non-specific urethritis, cases have also been recorded after epidemics of bacillary dysentry. A rare condition associated with Reiter's syndrome is that of keratoderma blennorrhagica. There is a certain amount of evidence to suggest that Reiter's syndrome is due to a virus, but this is by no means proved. Some patients with Reiter's syndrome later develop a spondylitic syndrome, as in a case where the patient developed virtual obliteration of the left sacro-iliac joint, complete obliteration of his right sacro-iliac joint, some degree of bony bridging and lipping, calcification of ligaments, and later a complete bamboo spine with ankylosis of shoulders and hips. The aetiology of Reiter's syndrome is not yet perfectly understood but there is reasonable evidence to suggest that it has an infectious origin, perhaps occurring in people with certain allergic and sensitization reactions to infection.

A typical sufferer from ankylosing spondylitis is a man between the ages of 20 and 40, the disease being about five or six times more common in men than in women. It affects the spine, hips and the shoulders predominantly, although in about 25 per cent of patients peripheral joint changes may also occur. The most striking, significant, and early feature of the disease is the presence of inflammatory changes in the sacro-iliac joints and this is the certain means of diagnosis in the early stages. It has been stated that the average period taken to diagnose a patient suffering from ankylosing spondylitis is between four and seven years from the initial complaint of low back pain. Undoubtedly radiographs taken earlier in the illness would shorten this period considerably and, although there is no satisfactory cure for ankylosing spondylitis, unnecessary deformities

could be prevented. Not only is the disease rarer in females, but it usually runs a milder course and may be atypical. In the case demonstrated, a woman had long-standing damage to the sacro-iliac joints but some of the joint space was still preserved and there was surprisingly little else wrong; good mobility of the rest of her spine and the remainder of her joints was preserved over a period of many years and indeed she is still very mobile. Clearly, ankylosing spondylitis is a very different condition from rheumatoid arthritis clinically, radiologically, and serologically (the Rose-Waaler test is negative) and it seems strange that only a few years ago it was considered by many to be similar to or identical with rheumatoid. As our knowledge of these groups of diseases increases, we are faced more and more with diagnostic problems in rheumatology.

We now pass from the forms of polyarthritis in which something is known about aetiology to those where we know absolutely nothing of aetiology but we know just a little about the clinical relationship. An example is psoriatic arthritis. We now recognize that rheumatoid arthritis and psoriatic arthritis are separate disorders and although this may not help us very much in understanding the aetiology of either of them, it has some practical significance in treatment. In psoriatic arthritis the terminal interphalangeal joints are involved rather than the proximal interphalangeal joints, and sometimes there is related psoriasis of the nails. Radiographs show destruction of the terminal and some of the proximal phalanges of the digits with a characteristic whittling away of the bone, or osteolysis. This osteolysis, while not exclusive to psoriatic arthritis, is seen in this condition much more than in rheumatoid arthritis, so much so that we feel confident we could now base a diagnosis on radiological grounds in some cases. The distinction from rheumatoid arthritis is important in practice because, in the treatment of polyarthritis associated with psoriasis, chloroquine is contraindicated. This drug frequently leads to an exacerbation of psoriasis. Steroids are now considered disappointing in the treatment of psoriasis and indeed are often followed by a severe exacerbation, so that again there is a relative contraindication to treating psoriatic arthritis with them. Phenylbutazone used to be considered to be contraindicated in psoriasis because it might cause a rash but in fact it is not contraindicated, since the two rashes have nothing in common and a person with psoriasis is no more liable to get a dermatological complication of phenylbutazone therapy than is anybody else. Indeed phenylbutazone is probably the drug of choice in psoriatic arthritis. Aspirin is sometimes contraindicated because it may cause sweating, which is uncomfortable to patients with psoriasis.

Joints attacked by any inflammatory process obviously have a

limited repertoire of response. They show the basic features of an inflammatory change anywhere, with redness, swelling, heat and pain, to which are added effusion and limitation of movement. Irrespective of the cause therefore, one type of polyarthritis may superficially resemble another. Among the very rare causes of polyarthritis we may list neoplastic diseases. Polyarthritis is probably a much commoner feature of carcinoma than has been recognized hitherto. The relationship is probably better known in general practice where the end results of the carcinomatous process are often seen, but occasionally the polyarthritis associated with carcinoma may be an early or initial event. This is particularly liable to occur in cases of bronchogenic neoplasm, where the condition known as pseudohypertrophic pulmonary osteoarthropathy may occur, with nail clubbing, periosteal swelling and diffuse pain and tenderness of the joints of the extremities, but polyarthritis can also occur with other forms of carcinoma. This diffuse inflammatory type of polyarthritis is quite separate from secondary deposits in the joints. Occasionally the more severe blood disorders, such as Hodgkin's disease, leukaemia, and myelomatosis, may present with an arthritis or even a polyarthritis due to infiltrations of myelomatous, lymphogranulomatous, or leukaemoid tissue into the joints. There are a wide variety of other conditions which may present with arthritis, e.g., the shoulder-hand syndrome, sarcoidosis, and Henoch-Schoenlein purpura. It is very difficult to classify these odd, rare cases but one must always be aware of them.

Finally, we must mention and briefly describe some of the more serious connective tissue disorders, which, because they clearly involved the cardiovascular system in many cases, became known as the " collagen vascular diseases ". This term is not so popular now because there are doubts as to whether collagen is the tissue primarily at fault. This group includes systemic lupus erythematosus, polyarteritis nodosa, dermatomyositis, and scleroderma (progressive systemic sclerosis). All of these conditions may be characterized by skin eruptions so that these patients may be referred to a dermatologist, a general physician, rheumatologist, or other specialists, according to the clinical feature that is outstanding. These diseases give the lie to the jibe that rheumatic patients never die and never get better, because unfortunately in many cases the outcome is death. Although rare, they call for a considerable amount of effort and skill in their management.