8th Edition

TEXTBOOK OF ORTHOPAEDIC MEDICINE

JAMES CYRIAX

VOLUME ONE
Diagnosis of
Soft Tissue Lesions

Baillière Tindall

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Diagnosis of Soft Tissue Lesions

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PREFACE

Orthopaedic medicine was born in 1929. Then, as an orthopaedic house surgeon, I saw a large number of patients whose radiographs revealed a variety of bony disorders. In these, a firm diagnosis was reached and crisp treatment was the rule. But I also saw many more in whom the X-ray appearances were negative or equivocal; about diagnosis and treatment in these cases there reigned a disturbing vagueness. After some months it dawned on me that no satisfactory method appeared to exist for testing the function of the radiotranslucent moving tissues. Realizing that here lay the crux of the clinician's dilemma, I set out to develop just such a system. This took me twelve years, though I can now explain the basic theory in as many minutes. I was then faced with scores of hitherto unrecognized disorders, for which treatment had to be found. It was not until twenty years ago that enough knowledge had been amassed for me to reach regularly a proper assessment in cases within the orthopaedic medical sphere.

The previous neglect of so large a section of human ills is remarkable, in that one of the commonest symptoms, ranking second only to neuroses and respiratory infection as a major cause of industrial disablement, is pain felt at a moving part of the body. Not all such symptoms stem from a local disorder, but a great many do, and everybody suffers from this kind of trouble at intervals throughout life. Thus, joints are sprained or become arthritic; muscles, tendons and ligaments are strained; bursae become inflamed. Nerve trunks, nerve roots and dura mater are liable to compression. Joints, especially spinal joints, are prone to internal derangement. These disorders of the moving parts of the body, so long neglected, deserve accurate diagnosis. Many require treatment by non-surgical orthopaedic measures, e.g. induction of local anaesthesia (which is also important diagnostically), infiltration with steroids, manipulation, traction or massage.

Who is to cater for this huge mass of patients? They wander from doctor to doctor, from one hospital department to another—finally visiting all sorts of lay healers—in the vain hope of finding the right man. Most of their disorders are not 'rheumatic' (although often misnamed so by the patient), since they are seldom concerned with rheumatic fever or rheumatoid arthritis, and they do not often call for surgery. Hence they are not the primary concern of the rheumatologist or the orthopaedic surgeon.

Orthopaedic medical disorders are the only major cause of human suffering and industrial sickness for which the National Health Service makes scarcely any provision. In consequence, many patients linger on in pain and off work (and, if they are athletes or sportsmen, off games) for indefinite periods, not for lack of the relevant medical knowledge, but for lack of doctors trained in the relevant discipline. This neglect has led to the eruption of numerous laymen into the void we have left gaping. Their number and success, together with the esteem in which the public holds them, serve to indicate the large numbers of people who have been compelled finally to look outside the ranks of the medical profession for relief, and have found it in lay hands.

But the picture has another side, for treatment without prior diagnosis entails great waste of time and money. Recourse to laymen, though it has its successes, involves many patients in repeated visits for futile treatment. Disorders easy to put right by the alternative measures of orthopaedic medicine are given routine manipulation in vain by enthusiastic laymen who, for lack of proper medical training, cannot know when or when not to apply their ministrations. This indefensible system is common knowledge; doctors and patients alike are aware that they must take their chance with unqualified people on their own initiative and at their own expense—all this at a time when the State has assumed responsibility for every type of medical care.

The hiatus must be closed on financial no less than on humanitarian grounds. If the Health Service can save itself money *and* help patients at the same time there seems little reason for delay.

In 1868 Sir James Paget gave a lecture on 'Cases that Bonesetters Cure', and his message was reinforced by Penny's criticisms on doctors' neglect in a paper 'On Bonesetting', published in the *British Medical Journal* in 1888. Yet the sad deficiency that they drew attention to persists little altered today. For the last forty years I have taken this hiatus seriously and the fruits of the work done have been set out in successive editions of this book.

The additions for the eighth edition include a review of the literature up to the end of 1981. Many of the facts that I had established clinically over the last thirty years have now been corroborated by recent and more objective studies. These are set out. Considerable trouble has been taken to establish when an observation was first made, so that research workers can be guided to the original record. It is remarkable how many discoveries, thought to be recent, were in fact first published during the nineteenth century. Chiropractice is discussed as comprehensively as in previous editions. It is clear that those who practise it are seeking to enlarge their sphere of action in the USA and Canada. A review of their assertions and advertising literature has therefore been added so that the medical profession becomes aware of the encroachment.

Orthopaedic medicine and orthopaedic surgery must not be thought of as in any way opposed. It is the very reverse: they complement each other. The existence of a physician within the orthopaedic team relieves surgeons of much non-surgical work for which few have much liking and none much time. Moreover, the decision on whether or not to operate may rest on the likely outcome of non-surgical measures. Who is better placed to assess that probability than the consultant practising the conservative approach? I know that this collaboration works smoothly and well; for this was the situation during my many years as orthopaedic physician at St Thomas's Hospital. An orthopaedic team comprising surgeon and physician covers the whole field within one department and ensures that each patient comes under the care of the appropriate expert; as Evarts pointed out in 1975 in his chairman's address to the Orthopaedic Section of the American Medical Association. In England, seventy-five years ago, orthopaedic surgery was branching off from general surgery to the accompaniment of some scoffing. Robert Jones was appointed to the first lectureship in orthopaedic surgery in 1909 but E. H. Arnold became 'instructor in orthopaedic surgery' at Yale University ten years before that. Just as it seemed redundant to a past generation to make a separate speciality of bone and joint surgery, so will the suggestion of a medical colleague to deal with the non-surgical aspects of the locomotor disorders meet with some resistance. Yet this division already exists in several other sections of medicine, e.g. neurologist and neurosurgeon, gastroenterologist and abdominal surgeon. The birth of a separate province will not be without pangs, though in fact it relieves surgeons of so much unwelcome non-surgical work. Resistance to new ideas is to be expected; it delays but does not affect the eventual outcome, since the needs of the sick have always proved paramount in the end. Already the cost to industry and the insurance companies of avoidable invalidism, added to the sum of overt public frustration, is leading to mounting pressure for the creation of the relevant speciality. It is only a question of time now before hospitals realize that they cannot afford to do without a consultant in orthopaedic medicine.

It has been my life's work to devise, and as far as possible to perfect, a method of clinical examination which leads to accurate diagnosis in locomotor disorders, enabling the physician to ignore the ubiquitous misleading phenomena of referred pain and referred tenderness. It consists of assessing in turn the function of each moving tissue, the positive and negative responses to selective tension forming a pattern. This pattern is then interpreted on the basis of applied anatomy. Logical conclusions of incontestable validity are drawn (but have roused much controversy). Since doctors receive little or no undergraduate tuition in how to examine the soft moving parts, they have been apt to look askance at such simple deductions, regarding them as more clear-cut than such obscure clinical material warrants. However, now that the basic research has been carried out, the stage is set for immediate impact on contemporary medical thought, diagnosis (since it is purely clinical and requires none of the apparatus that only hospitals possess) coming within the scope of every interested medical practitioner. At present the number of doctors and physiotherapists trained in this discipline remain so small that the methods of orthopaedic medicine are available to only a tiny fraction of all patients who need them.

Since displacements within the spinal joints are so common, and one aspect of orthopaedic medicine involves their reduction, I have become known as that odd and scarcely respectable phenomenon: a doctor who manipulates and, worse still, teaches these techniques (together

with the indications and contra-indications) to physiotherapists. Nothing annoys me more; for, though true up to a point, it is a gross error in emphasis. I am a medical man who has spent his graduate days in elaborating clinical methods of examining the non-osseous moving parts (radiography takes care of the bones themselves). Based on these new concepts, I have gone on to as exact assessment as possible of the position, nature, size and stage of each soft tissue lesion. This has led to the discovery of scores of hitherto undescribed conditions within the sphere of orthopaedic medicine and of some outside it, e.g. irritation of the external aspect of the median nerve at the wrist (1942), and intermittent claudication in the buttock (1954). It has also led to a good deal of iconoclasm, 'sacroiliac strain' being debunked in 1941 and 'fibrositis' in 1948. The discal pathology of lumbago, regarded as a muscular affliction since 1904, was set out in 1945, together with the concept of pain arising from the dura mater. All these theories have been confirmed since by workers all over the world.

Logical extension of these clinical findings has led me to adapt, and where feasible improve upon, methods of treatment already in existence, but previously based either on empiricism or on false hypotheses. When no treatment existed, or the disorder had never been recognized, mere palliation was abandoned and methods of treatment were investigated in the light of our new-found diagnostic precision until, as far as possible, an effective measure was discovered. All successful manoeuvres were taught to our physiotherapists; for they were there to treat the patients, especially by the use of their hands. This is nothing new; the first record of the appointment of a teacher in bonesetting is contained in a ukase issued by the Tzar of Russia in 1655. Such delegation proved very satisfactory, since it enabled me to get on with my diagnostic work and carried the further advantage of affording physiotherapists a rewarding series of dramatic successes. On the one hand they were sent patients who had been found suitable for such procedures by a medical man; on the other patients were no longer asked to attend for ephemeral palliation that even today goes by the name of 'orthodox treatment'. (How could it ever be orthodox to treat a displacement by heat and exercises?) Neither was the patient left to the vagaries of fortune nor to the hits and misses of lay manipulators. Naturally, this policy enhanced students' interest in this part of their work. The good repute that manipulation by laymen enjoys from some people now began to be transferred to manual methods obtainable within the Health Service, with a corresponding increase in the esteem in which physiotherapists were held. Nevertheless manipulation, emotionally charged treatment though it is, has always provided only a minor part of the work, constituting merely one remedy called for by the major compulsion—an accurate diagnosis. Manipulation is easily learnt; diagnosis is not.

I did not invent massage, which has existed since time immemorial as an extension of the urge to rub a sore spot. Indeed, the first mention of a professor of physiotherapy dates from AD 585 when one was appointed under the Sui dynasty in China. I merely devised the method of giving deep massage penetrating to the lesion. I insisted that the structure at fault should alone be treated avoiding areas of normal tissue in the neighbourhood that happened to be the site of referred pain and tenderness. This turned out very fortunately; for, when the Medical Research Council allowed me hydrocortisone in 1952, the way to identify each lesion and the posture that made it easiest to palpate had already been established. It was thus merely a question of substituting the needle for the physiotherapist's finger. I did not invent manipulation or traction, both of which were practised by Hippocrates; a scamnum (bench for traction and reduction) made to his design and four hundred years old stands today in the Wellcome Historical Museum in London and a Turkish manuscript dated 1465 depicting spinal traction is preserved at the Louvre in Paris. My endeavour has been to codify the application of these measures, placing equal emphasis on 'when not' as on 'when', in an attempt to fit each into its due place in therapeutics.

In particular, I have tried to steer manipulation away from the lay notion of a panacea—the chief factor delaying its acceptance today. My only important discovery, on which the whole of this work rests, is the method of systematic examination of the moving parts by selective tension. By this means, precise diagnoses can be achieved in disorders of the radiotranslucent moving tissues. If in years to come I am to be remembered as an original worker at all, it is with this fundamental study that I should like posterity to link my name.

I would like to take this opportunity to thank Feliks Topolski for allowing me to reproduce his painting of my giving him an epidural injection as the frontispiece to this volume.

August 1982 James Cyriax

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CHAPTER 1

GENERAL CONCEPTS

The disorders with which this book deals are universal. It is a rare individual indeed who does not suffer one or more lesions of his moving parts in the course of his life. Although diagnosis is considered difficult or impossible, it is in fact the reverse; it is merely a matter of applied anatomy. The function of every moving part has been established for years and clinical testing is no more than an informed, anatomical exercise. Function is assessed indirectly, like a series of simultaneous equations, and the pattern of movements—painful, painless; full range, limited range—elicited and interpreted in the light of the known behaviour of these tissues. Care is taken to avoid prejudice towards any particular hypothesis on the disorder likely to be present or on the causation of disease. The physical signs are paramount throughout. I have spent my life working out how best to ascertain the physical signs in soft tissue lesions and how to interpret the pattern thus brought to light. This devotion to physical signs is essential to the orthopaedic physician, for none of his patients dies in hospital and he is therefore denied the salutary discipline of the post-mortem room. Nor are X-rays of appreciable value when the radiotranslucent tissues are at fault, and in general other objective tests, e.g. on the blood, are of little assistance. Hence, he must take great trouble to be right, for contrary evidence is not often available to bring an error to his notice. Constant self-criticism is thus the hallmark of the orthopaedic physician, who has, with due humility, to approach the truth contained—better, perhaps, to say concealed—within each patient.

All pains have a source; the diagnosis names it. In visceral disease, abnormality is often difficult and sometimes impossible to demonstrate. With the moving parts the situation is reversed; function is obvious and easy to test clinically. A joint moves within certain known limits; a voluntary muscle contracts and relaxes to known effect. The examination of these structures thus presents little difficulty and interpretation of the findings is based on uncontroversial anatomical facts. The basis of this book is therefore a painstaking search for physical signs, positive and negative, and their interpretation on agreed grounds, unarguably valid. To my never-ending surprise this extreme simplicity has proved controversial and slow to gain acceptance.

'RHEUMATISM'

Nomenclature in medicine is important, for it is by words that we convey our meaning to others. 'Rheumatism' is a word often used by patients and doctors, but with many different meanings. To the layman it implies pain that he associates with the moving parts of the body, appearing for no clear reason. To some medical men it includes every disorder of the moving parts, whatever the cause—arthritis, tendinitis, tenosynovitis, ligamentous and muscle strain, post-traumatic adhesions and internal derangement, especially at the spinal joints. Others confine the term to the collagen diseases; yet others to chorea and rheumatic fever and its cardiac sequels. Hilton (1863) had already stated that the surgeon should not 'be satisfied, as is too frequently the case, with saying "Oh, this is rheumatism" (the favourite phantom)'.

The only useful way to employ 'rheumatism' is for the chorea–rheumatic fever group of diseases. Then it refers to well-defined clinical entities and has a clear aetiological significance. But when a variety of other disorders of diverse aetiologies is grouped together under this name the result is a logical morass. By common consent, arthritis is rheumatic; osteoarthrosis with a loose body, impaction of which is causing the symptoms, and neuropathic and pulmonary arthropathy are probably not; tuberculous and gouty arthritis are certainly not. Monarticular rheumatoid arthritis is rheumatic; the locally identical condition occurring in serum sickness is not,

because its allergic origin is obvious. Gonococcal and Reiter's arthritis are rheumatic only so long as their urethral origin remains undetected. Tabes, localized neuritis or displaced fragments of intervertebral disc cause pain felt in muscles and joints; these conditions are regarded as rheumatic only when the true nature of the condition is overlooked. A familiar example is lumbago; until recently it was regarded to be the result of fibrositis caused by rheumatic toxins settling in the lumbar muscles; now that it is known to be caused by internal derangement of a lumbar joint it has ceased to be rheumatic. Tennis elbow and supraspinatus tendinitis were thought of as rheumatism of the elbow and shoulder only so long as the traumatic cause of these two types of tendinitis was not realized. When the aetiology of rheumatoid and spondylitic arthritis is ultimately discovered, these disorders also will cease to be caused by 'rheumatism'. The medical use of the word can then cease (apart from rheumatic fever). Thereafter 'rheumatic' would remain a useful evasion, but it would no longer carry any medical significance.

The word 'rheumatism' has another disadvantage. Since it is applied to all sorts of painful conditions, it means quite different things to different patients. Thus one patient may be deeply relieved to know that his pain is 'only rheumatism'; another is appalled, because a relation of his is crippled by 'rheumatism' in every joint.

For a detailed discussion on medical semantics, the reader is referred to Asher (1972).

Primary Fibrositis

In this condition, pain and tenderness are experienced in the trunk. Since the trunk is covered by muscles, the patient complains of pain felt in the tissue he knows to lie there, i.e. the muscle. This provides no evidence that the pain arises from the muscle, and when resisted movement of the muscle alleged to be at fault proves strong and painless, the non-muscular origin of the pain becomes evident. In fact, primary fibrositis (the disorder, not the symptoms) is an imaginary disease. This has been amply borne out by post-mortem experience, for many pathologists have sought for evidence of 'fibrositis', and though almost every patient in the country has had this label applied to one or other of his symptoms, no evidence pointing to the real existence of primary fibrositis has ever come to light. Indeed, the conditions once ascribed to such inflammation in the soft structures of the body, e.g. acute torticollis, pleurodynia, lumbago, can be shown by proper interpretation of the physical signs to result from internal derangement of a spinal joint. 'Rheumatic' inflammation of the soft tissues was postulated as a pathological entity and the cause of lumbago by Sir William Gowers in 1904. He offered no evidence, but his bare statement was accepted for forty years until it was challenged for the first time in *The Lancet* and the *British Medical Journal* (Cyriax 1945, 1948, 1978). It had been my intention to omit this section from the present edition, since in England the battle against 'fibrositis' had been won. To my dismay, however, it has again reared its ugly head, this time on the other side of the Atlantic, figuring in titles of papers at various congresses in Canada and the USA. It has even been endowed with respectability by a review devoted to it in the *Bulletin on the Rheumatic Diseases* (1977). A further reason for debunking 'fibrositis' is the justification it offers for injections into the wrong spot, originally of a local anaesthetic solution, more recently of a steroid suspension. These are not wholly harmless; for Snell (1977) describes two cases of pneumothorax resulting from injections of hydrocortisone at a supposed trapezial lesion and Ritter and Tarala (1978) describe three more.

'Primary' fibrositis is in fact a secondary phenomenon. When the dura mater is compressed, usually via the posterior ligament by a protruding disc, pain is felt in the neighbourhood, but not necessarily at the site, of pressure. Within this painful area there is always a tender spot at a point where no lesion exists at all. It is a remarkable finding, but it does not mislead those who test the function of the tissue containing the tender spot. When such a spot is found in a structure, the function of which can be shown to be normal, its referred nature becomes evident. The irony of the situation lies in the fact that disc lesions, which do not result in inflammation of muscle but merely referred tenderness, are often called 'fibrositis', whereas when traumatic inflammation of fibrous tissue *is* present, e.g. in supraspinatus tendinitis, tennis elbow, a sprained ligament, this word is seldom used.

Various efforts have been made to relate referred tenderness to metabolites formed locally as the result of nervous impulses. That no such reaction occurs is clearly demonstrated by watching the changes in an area of referred tenderness during manipulative reduction of a displaced portion of cervical disc. At first, the patient has an area of tenderness which he fingers himself and regards as the source of his symptoms. As reduction of the displacement proceeds, this area

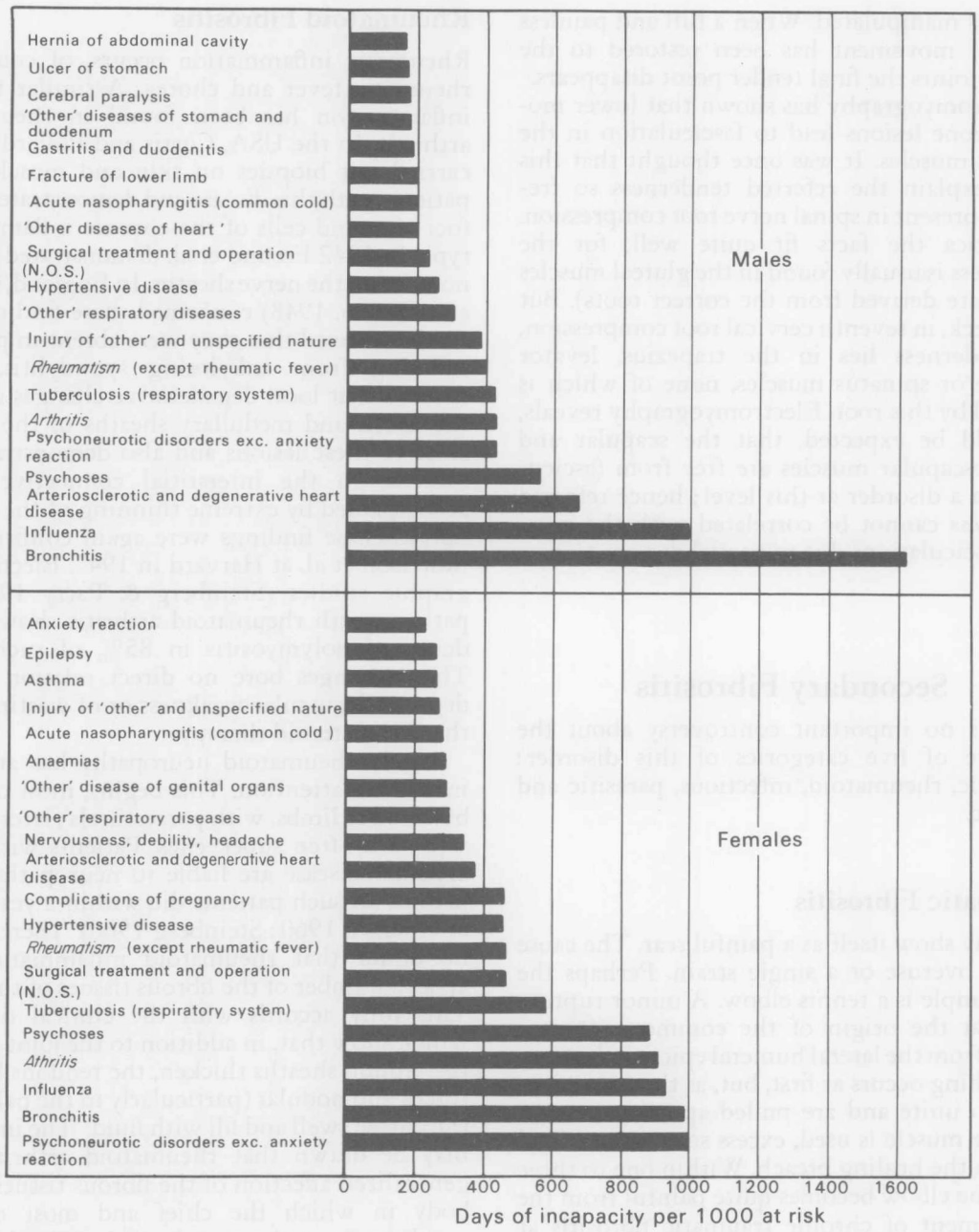

Fig. 1. Working days lost per thousand of the insured population (of equivalent 1951 age distribution) according to selected causes in Great Britain in 1961. Note that in man rheumatism and arthritis together are second only to respiratory disease, whereas in women they are the commonest cause of absence from work. The importance of back troubles has been emphasized by the Minister of Health (1967) who estimated that £100 million are lost on this account alone each year. Snider (1975) has already found that of all sums paid out in the USA for workmen's compensation, no less than 32% was for lumbar disorders. (*By courtesy of the Office of Health Economics*)

moves abruptly from place to place; as a rule, the pain and tenderness in the lower scapula area shift closer towards the midline and move upwards from the mid-thorax towards the lower neck. The tenderness follows the pain and, after a minor shift in the displaced fragment of disc, one tender spot disappears but is replaced by another in a fresh situation. Clearly, metabolites produced locally could not move from one muscle to another in a few seconds merely because the

neck was manipulated. When a full and painless range of movement has been restored to the cervical joints the final tender point disappears.

Electromyography has shown that lower motor neurone lesions lead to fasciculation in the relevant muscles. It was once thought that this might explain the referred tenderness so frequently present in spinal nerve root compression. In sciatica the facts fit quite well, for the tenderness is usually found in the gluteal muscles (which are derived from the correct roots). But at the neck, in seventh cervical root compression, the tenderness lies in the trapezius, levator scapulae or spinatus muscles, none of which is supplied by this root. Electromyography reveals, as would be expected, that the scapular and vertebroscapular muscles are free from fasciculation in a disorder at this level; hence referred tenderness cannot be correlated with the muscular fasciculations due to partial denervation.

Secondary Fibrositis

There is no important controversy about the existence of five categories of this disorder: traumatic, rheumatoid, infectious, parasitic and myositic.

Traumatic Fibrositis

This may show itself as a painful scar. The cause may be overuse or a single strain. Perhaps the best example is a tennis elbow. A minor rupture occurs at the origin of the common extensor tendon from the lateral humeral epicondyle. Very little aching occurs at first, but, as the torn edges begin to unite and are pulled apart again each time the muscle is used, excess scar tissue is laid down in the healing breach. Within one to three weeks the elbow becomes quite painful from the development of chronic traumatic fibrositis at the site of the tear.

Scarring in an intercostal or in the gastrocnemius muscle, golfer's elbow, tendinitis at the shoulder, adherence of a ligament after a sprain, periarticular adhesions after an injury, capsular contracture after immobilization, crepitating tenosynovitis caused by overuse, olecranon bursitis after a blow, ischaemic contracture—all these and a number of similar conditions could be regarded as caused by post-traumatic fibrositis. But they are better described under their proper names.

Rheumatoid Fibrositis

Rheumatic inflammation occurs, of course, in rheumatic fever and chorea. A similar type of inflammation has been found in rheumatoid arthritis. In the USA, Curtis and Pollard (1940) carried out biopsies on skin and muscle from patients with this disease and demonstrated small foci of round cells of the chronic inflammatory type. In 1942 Freund et al. demonstrated similar nodules on the nerve sheaths. In England, Gibson et al. (1946, 1948) confirmed these findings and further proved that they were absent in patients suffering from ankylosing spondylitis. They showed that local degenerative changes affected the axons and medullary sheaths of the nerves close to these lesions and also demonstrated an increase in the interstitial connective tissue accompanied by extreme thinning of the muscle fibres. These findings were again confirmed by Morrison et al. at Harvard in 1947. Electromyographic studies (Steinberg & Parry 1961) on patients with rheumatoid arthritis showed evidence of polymyositis in 85% of such cases. These changes bore no direct relation to the degree of muscular weakness or of wasting or to the use of steroid therapy.

Lately rheumatoid neuropathy has attracted increasing attention. This begins, most often in both lower limbs, with paraesthesia; later, motor weakness often supervenes. Patients with gross articular disease are liable to neuropathy; one-fifth of all such patients die within a year (Hart & Golding 1960; Steinberg 1960). There is thus no doubt that rheumatoid inflammation can affect a number of the fibrous tissues of the body. This fully accords with the clinical findings, which show that, in addition to the joint lesions, the tendon sheaths thicken, the tendons become rough and nodular (particularly in the palm) and the bursae swell and fill with fluid. 'The inference may be drawn that rheumatoid arthritis is a generalized affection of the fibrous tissues of the body in which the chief and most obvious incidence is on the capsule of the joints' (Cyriax 1947). Cox (1824) had already stated that 'it would appear that every texture of the body participates in the inflammation'.

Infectious Fibrositis

Epidemic myalgia (Bornholm disease) is an infectious disease due to a virus which has been identified. It is characterized by fever, severe pain in the abdominal and thoracic muscles and speedy recovery.

Parasitic Fibrositis

Infestation with *Trichinella spiralis* causes fever and painful swelling of the affected muscles; the overlying skin may become red; the tendons may also be invaded. The disease occurs about 10 days after eating infected pork. Active contraction of the affected muscle increases the pain. The symptoms and signs subside in some weeks, whereupon the patient becomes completely unaware of the foreign bodies in his muscles.

Myositis

This is a diffuse inflammatory disease of muscle. There is no pain; the muscle wastes progressively and marked weakness develops, which can be halted only by steroid therapy. The affection is often bilateral and symmetrical and is seldom distinguishable from myopathy except by biopsy.

Generalized Fibrositis

Rheumatoid arthritis is the only condition to which the term 'generalized fibrositis' properly applies. By contrast, the disorder to which this name is often given is disc lesions at several spinal levels. This may lead to considerable aching over part or the whole of the trunk—areas where muscular crepitus and fatty nodules are commonly detectable. Unrecognized osteitis deformans or ankylosing spondylitis is repeatedly called fibrositis. Snell (1977) describes two cases of pneumothorax created by injections of hydrocortisone intended to relieve 'fibrositis' of the trapezius.

Another disorder often called 'generalized fibrositis' is psychoneurotic pain. The idea of generalized fibrositis has led to such concepts as 'the psychological basis of rheumatism'—a notion in which the cart is put before the horse. Clearly, psychogenic pain is not rheumatism, and the discovery of the real cause should lead to revision of that ascription, not to an attempt to fuse two incompatible diagnoses.

MUSCLE TONE

Postural Tone

Feldberg (1951) points out that acetylcholine is released not only as a result of a nerve impulse, but also at a very low level when the muscle is at rest. So long as the mechanism for the destruction of acetylcholine is intact, the amount liberated is too small to cause muscular contraction and the electromyograph cannot therefore detect its presence. It is probable that this phenomenon is more marked in trained than untrained muscles; tone may well be affected by variation in the subliminal level of acetylcholine production. In mammals it appears that tone is served by what is now known as the small motor nerve fibre system. The anterior roots have long been known to contain a distinct group of small diameter fibres (Eccles & Sherrington 1930), as well as the large fibres. The function of these fibrils remained unknown until it was recently shown to serve the maintenance of sustained muscular contraction.

Kremer (1958) has summarized the results of Merton and his colleagues' work on the maintenance of postural tone, thus:

A muscle is brought into action by motor impulses, but the degree of that contraction is estimated by sensory receptors in the muscle, and in the light of this information, called the 'feedback', it modifies the rate of motor discharge. It is true that visual information may modify the motor discharge, as may cutaneous impulses, but it is the muscle sense organs which play the major part in assessing or monitoring the performance of the muscles themselves.

The muscle spindles are the sensory organs of muscles. They lie among the main muscle fibres, having the same attachments and therefore altering in size with contraction or relaxation of the muscle itself. It must be remembered that the poles of these muscle spindles are contractile and receive very fine-fibred efferent supply, the γ fibres, whereas the main muscles receive large or α fibres. The reflex connections of the muscles are such that impulses set up by stretching the spindles excite the muscles' own motor neurones. Thus extension of the muscle results in an augmented contraction which tends to resist the extension. This is the stretch reflex of Liddell and Sherrington (1924). This has the properties of a closed loop self-regulating mechanism using information from the spindles to maintain a constant muscle length. It is clear that this has enormous advantages over a straight-through system in which posture is maintained by a steady stream of motor impulses without sensory modification or feedback, in that it automatically compensates for changes in load or for fatigue.

This type of stretch reflex would maintain a fixed posture well, but it is clearly inflexible and needs modification for ease of changing muscle-lengths

while maintaining postural tone. This modification is carried out by means of the contractile poles of the muscle spindles. The sensory portion of the spindle lies between these poles, hence shortening the poles by impulses along the efferents will stretch the sensory spindle so that the stretch reflex will be activated just as if the muscle itself had been stretched. The muscle will then shorten reflexly until the increased rate of spindle discharge has been offset, and that will be when the muscle has shortened to the same extent as the contractile poles of the spindle.

Merton and his associates have named the loop mechanism of the simple stretch reflex the 'length-servo' mechanism and the modification next mentioned the 'follow-up servo'.

Joseph (1964) has shown that the maintenance of the upright position needs very little energy. The only muscles in constant action are the calf muscles and those over the maximum convexity of the trunk, i.e. mid-thorax; only slight activity can be detected in the lumbar and cervical regions. The knees are kept straight by the tautening of their posterior ligaments, not by quadriceps action. Provided the vertical dropped from the centre of gravity falls through the ankles, there is little difference between the energy consumption of a person erect or lying down, irrespective of different degrees of curvature of the spine.

Athletic Tone

Electromyography has demonstrated that the concept of muscle tone as a state of slight neurogenic sustained muscular contraction is false. This is not surprising, for training increases what used to be called tone. Obviously, if use of a muscle caused it to relax less readily than before, training would defeat its own object and a highly trained runner would have to walk on tiptoe. Training clearly enhances the function of muscle, i.e. it contracts *and* relaxes more efficiently. Joseph (1964) states that it is difficult to eradicate the idea that a relaxed muscle still possesses tone. This idea was first put forward by Müller in 1838 and had proved most tenacious, in spite of clear demonstration by even the most delicate electromyography that no contracting motor units exist in relaxed muscle. Joseph suggests that the term 'muscle tone' should be abandoned and 'response to stretch' substituted. Hypertonic and hypotonic states would then refer to excessive or reduced stretch response respectively. He states that muscles which cannot be completely relaxed are contracting and should not be regarded as hypertonic. A spastic muscle is not just hypertonic; it is a muscle undergoing a continuous contraction easily demonstrated electromyographically.

This fact has an important practical bearing. For example, if a patient suffering from the thoracic outlet syndrome is given exercises to the elevator muscles of the scapulae, no advantage accrues; for however strengthened these muscles become, they relax perfectly as soon as voluntary contraction ceases and the scapulae then occupy the same position as before.

Neurogenic Hypertonus

Muscular spasm secondary to painful lesions is unconnected with the hypertonus that accompanies neurological disease. In the former, when movement is limited at an arthritic joint a certain amount of mobility is painless, but at a constant point muscular spasm brings it to an abrupt stop and no forcing without anaesthesia can take it beyond this point. By contrast, neurogenic hypertonus results in an early resistance to passive stretching until, suddenly, the resistance of the muscles is overcome and a full range of painless movement is revealed. Initial resistance, later giving way, also occurs in hysteria.

Cramp

This may result from hyperventilation, hypocalcaemia, tetanus, strychnine poisoning, salt deprivation or pyramidal lesions, and can be very painful. It is also common in healthy people, usually occurring only at night. The pain is in the calf, possibly in the foot also, the foot and toes becoming fixed in full flexion or full extension. The disorder is unconnected with tetany, but it is apt to affect the calf muscles on the same side as a past attack of sciatica and is a common sequel to a posterior radicotomy at the fifth lumbar or first sacral level. The fact that several muscles of one limb are affected in a coordinated way suggests a nervous aetiology; it may be due to a discharge of impulses from the spinal cord, analogous to the mechanism of epilepsy—a concept supported by the electromyographic studies of Norris et al. (1957) who regard the cramp as being initiated in the central nervous system. Certainly, in cramp, it is the muscles that hurt. Cramp does not spontaneously affect a muscle; it is brought on by a voluntary contraction. Hence patients soon discover that it is most quickly abolished by passively stretching the affected muscle.

Muscle Spasm

The notion of 'fibrositis', with its emphasis on alleged primary disease of muscle, has led to further misconceptions. One is painful muscle spasm fixing a joint (Brown 1828). The spasm is thought to be primary, but it is merely called into being by a protective reflex originating elsewhere. Capener (1961) has lent his authority to the idea of painful muscle spasm in 'acute derangements of the lower spine'. In his view, the muscle spasm overshadows everything else and as soon as it is controlled the trouble begins to subside. The converse is the case, as can be proved by epidural local anaesthesia which cannot reach the lumbar muscles. When the disc displacement recedes, the pain, felt in the muscles but not originating from them, and muscle guarding abate together. This concept was finally proven by Conesa in 1976. He administered a muscle-relaxant (baclofen) to two hemiplegic patients with stiff and painful shoulders. The muscles relaxed but he found the range of passive movement and the pain on stretching the joint unchanged.

In orthopaedic disorders, the muscle spasm is secondary and is the result of, not the cause of, pain; it causes no symptoms of itself. It is only cramp and neurogenic spasms that hurt muscles. Muscle spasm is thought to require treatment, as evidenced by the many muscle relaxants that are advertised for the cure of lumbago, for example. Osteopaths attribute all sorts of dire diseases to vertebral muscle spasm. The treatment of muscle spasm is of the lesion to which it is secondary; it never of itself requires treatment in lesions of the moving parts.

The main function of muscle is to contract. This function is evoked by any important lesion in the vicinity of the muscle, whether it involves a moving tissue or not. For example, appendicitis or a perforated ulcer leads to spasm of the abdominal muscles, although this has no effect on the mobility of the viscus at fault. It is true that muscles spring readily into spasm to protect a moving part, but they also contract about lesions whose behaviour they cannot influence. Spasm is thus the reaction, indeed the only reaction of which a contractile structure is capable, to any lesion of sufficient severity in its neighbourhood. Although spasm (neurogenic apart) originally evolved as a protective mechanism, it is not always beneficial. It is clearly useful in acute arthritis, preventing movement at the joint; it is equally obviously harmful after the disorder has become chronic. If manipulation under anaesthesia does good, the spasm was clearly militating against recovery.

Spasm in Arthritis

The muscles are *not* in constant spasm about an arthritic joint. When the joint is at rest in a neutral position, spasm is absent. It springs into being to prevent movement beyond a certain point and even then only one group of muscles contracts. When the capsule of the joint is stretched to a certain limit, involuntary spasm of the muscles that oppose that movement is elicited; the movement stops instantly. However often this movement is repeated, it always ceases at exactly the same point. If movement in a different direction is attempted, that too is restricted by spasm of another group of muscles. Such contraction of muscle is no more painful nor greater than if the patient had voluntarily used his muscles to arrest movement at that same point. For example, the muscle spasm that limits movement at the wrist in carpal fracture is no more intense than if the movement were stopped voluntarily. Moreover, at the extreme of the possible range, the pain is felt at the wrist, not in the upper forearm where the contracting bellies lie. It would have been reasonable to suppose that this muscle guarding would give them more to do; yet muscles waste about a damaged joint.

Another way to prove that it is not muscle spasm that hurts is to consider those joints that no muscle spans, e.g. acromio- and sternoclavicular and sacroiliac. Limitation of movement due to muscle spasm is now impossible; there is no muscle that can control movement. Yet stretching the joint hurts, as it does at any other arthritic joint. However, pain is not a necessary accompaniment of limitation of movement at a damaged joint. This is quite often the situation at an early osteoarthritic hip joint. Movement is restricted quite painlessly. Presumably, though the patient is unaware of it, the nociceptor system is sufficiently stimulated to provoke reflex muscle spasm.

Though muscle spasm in arthritis is protective, and in bacterial arthritis most beneficial, it is excessive in less grave articular disorders. For example, the marked traumatic arthritis in the knee after sprain of a ligament causes far more limitation of joint movement than is required merely to prevent further overstretching of the ligament. Indeed, there is no muscle at the knee which can limit the valgus mobility that would result in further stretching in medial ligament strain. The prompt abatement of the arthritis by

a steroid applied at the point where the ligament is torn greatly hastens recovery. It is clear, therefore, that the arthritic reaction to the injury, and the consequent restriction of movement by muscle spasm, serve no useful purpose. The same may or may not apply to a chronic articular lesion. An adhesion may have formed and may prove incapable of rupture because of muscle spasm limiting the therapeutic movement. After rupture under anaesthesia, the joint remains mobile and painless. In this instance, the spasm is harmful. Yet in rheumatoid arthritis the same joint with the same degree of limitation of movement would flare up severely if anaesthesia were employed to abolish spasm and to permit manipulation. In this case the spasm is beneficial. When an abscess forms in the bone near a joint, arthritis with limited movement maintained by muscle spasm results. Such sympathetic arthritis serves no purpose, for no lesion of the joint exists at all. Immobility of the temporomandibular joint does not hasten the healing of a septic tooth socket. A similar situation exists in the lung, where commencing erosion of the ribs by a neoplasm may set up spasm of the pectoralis major muscle, such that the arm cannot be raised above the horizontal.

It is clear that the defences of the body cannot distinguish between lesions in which spasm is beneficial (e.g. bacterial and rheumatoid), in which it is useless (e.g. visceral) and in which it is harmful (e.g. post-traumatic adhesions). The lesion, whatever type it is, merely engenders spasm in neighbouring muscles, as a uniform reaction to various stimuli.

Spasm in Bursitis

In bursitis, although limitation of movement occurs, involuntary muscle spasm is absent. For example, when the subdeltoid bursa is acutely inflamed, movement of the arm is so painful that the patient brings it to a halt by voluntarily contracting the relevant group of muscles. If he is asked to allow a little more movement disregarding pain, he can do so. This is a situation quite different from arthritis where the patient cannot be cajoled into permitting greater range, since this is limited by involuntary muscle spasm.

Spasm in Internal Derangement

Internal derangement blocks a joint, partly mechanically, partly as a result of protective muscle spasm. This is beneficial when it prevents the ligamentous overstretching which would result if the blocked movement were forced, but a disadvantage when it impedes reduction of the displacement. When the meniscus is displaced at the knee, both mechanisms arise. The hamstrings go into beneficial spasm to prevent the ligamentous overstretching that full extension of the joint would produce; but this militates against manipulative reduction, which therefore has often to be carried out after the spasm has been abolished by general anaesthesia. The same applies in lumbago with considerable lateral deviation at the deranged spinal joint; side flexion towards the convex side is prevented by muscle guarding. Contraction is often on the painless side, thus proving that it is not the muscle that hurts. Lying down diminishes the compression strain on the lumbar joint and consequently the degree of protrusion. The list to one side visible on standing may therefore disappear so long as the patient remains recumbent. Manipulative reduction abolishes the pain and the deviation *pari passu*. This is quite a different situation from arthritis where, for example, the amount of limitation of movement at the knee or a tarsal joint is the same whether the patient bears weight on the joint or not. The patient whose lumbar spine tilts sideways may be told of his awkward posture and see it in a mirror, but he does not feel asymmetrical. The position which his lumbar spine adopts because of muscle contraction is involuntary and painless. In general, muscle spasm precludes treatment by forcing movement, and a safe principle is 'never manipulate against muscle spasm'. The exception is internal derangement when, despite muscle contraction, reduction is the doctor's first thought. The attempt, at least to start with, never consists of just forcing the joint in the limited direction.

Spasm in Nerve Root Compression

Muscle spasm comes into play to protect the nerve roots from the third lumbar to the second sacral from painful stretching. This occurs only when the mobility of the dural sleeve of these five nerve roots is impaired. When the third lumbar nerve root loses its mobility, prone-lying knee flexion may be limited. When the other nerve roots are compressed, straight-leg raising is nearly always restricted. Spasm of the quadriceps or hamstring muscles is responsible; it is involuntary and painless. The pain on stretching originates from the nerve root, not the muscle.