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 γfibres, whereas the main muscles receive large or αfibres. 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 has 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 hyper tonus 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 m 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.

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, thejoint 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.

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. This can be shown by lifting the straight leg as far as it will go; in sciatica, this hurts. The patient is then asked to bend his head forwards, and the sciatic pain is often sharply increased. Whereas the nerve root can be stretched via the dura mater by neck flexion, the hamstring muscles cannot.

Though straight-leg raising may have remained limited for many years, no contracture of the hamstring muscles results. Even in chronic cases, epidural local anaesthesia often restores a full range of straight-leg raising within a few minutes, by abolishing the sensitivity of the nerve root whence the stimulus to the hamstrings to contract originates.

Spasm in Fracture and Dislocation

Spasm is constant about a recent fracture, immobilizing the broken ends, not necessarily in a good position. Reduction may prove impossible until the spasm is abolished. This can be accomplished by general anaesthesia, which inhibits the cerebral maintenance of muscle contraction, or by stopping the afferent impulses to which it is due, i.e. by local anaesthesia induced at the broken surfaces. Immobilization in a special position is often required, so that after reduction the broken piece is not pulled out of place again when muscle spasm returns after anaesthesia ceases.

Dislocation makes the muscles go into spasm and often prevents reduction, which has therefore to be carried out under general anaesthesia.

Spasm in Partial Rupture of Muscle Belly

Partial rupture of a muscle belly causes localized spasm, protecting the breach from tension. This spasm is localized; for example, when some fibres of the gastrocnemius muscle are torn, the muscle shortens centrally only. In consequence, the foot can be moved down and up by contraction and relaxation of the unaffected upper and lower parts of the muscle, but full dorsiflexion is limited by the contracture and the patient has to walk on tiptoe for the first few days. In partial rupture of the quadriceps and hamstring muscles, prone-lying knee flexion or straight-leg raising is often limited by the muscle shortening owing to localized muscle spasm

about the breach. This spasm does not hurt, but tension on the ruptured fibres, when exerted by passive stretching or resisted contraction of the damaged muscle, is painful.

When a tendon ruptures, the muscle belly does not go into spasm but, in due course, develops a contracture. No limitation of passive movement at the joint can result, although active movement may no longer be possible. Even if the belly shortens, since it is no longer attached to bone, the passive range at the joint remains unaltered.

Muscle Spasm protecting the Dura Mater

The dura mater is stretched in flexion of the neck and is at its shortest in full extension. An early sign in meningism is limitation of neck flexion and Kernig's sign is merely another way of eliciting limitation of straight-leg raising. In severe meningitis intense muscle spasm fixes the neck in full extension, thereby relaxing the dura mater to the maximum. This, of course, does not help therapeutically. A minor manifestation of this phenomenon is thoracic or lumbar pain on flexion of the neck when the mobility of the dura mater is impaired by a posterocentral disc protrusion.

In lumbago, muscle spasm also comes into play to protect the lower extent of the dura mater from being stretched. In a posterocentral disc protrusion of any size, straight-leg raising is bilaterally limited by the hamstring muscles springing into involuntary contraction. This restriction protects the theca from pull via the sciatic nerve roots, but only when its mobility is impaired at a low lumbar level.

Spasm in Sepsis

Sepsis in the region of a joint (e.g. staphylococcal olecranon bursitis) causes swelling and limited movement, the result of muscle spasm. Any inflammatory focus within the abdomen causes maintained spasm of all the anterior muscles. The board-like abdominal wall in peritonitis is the extreme example. Even a mere inflamed gland in the neck lying in contact with the scalene muscles may set up enough spasm to fix the neck in side flexion towards the painful side for a week or two. Such spasm has no virtue; the gland recovers at its own speed.

Spasm of unstriated muscle within the abdomen is of itself painful, as sufferers from biliary, renal or intestinal colic know well. Such intermittent contraction of the circular fibres provokes no secondary contraction of the abdominal muscles.

Arterial Spasm

Damage to an artery leads to spasm of the circular coat but, as in spasm of the bronchioles, no pain is caused; it is a beneficial phenomenon which arrests the bleeding when the artery is cut or torn. However, it is dangerous when the artery is badly enough bruised to go into spasm while it is still intact. At the elbow, ischaemic contracture in the flexor muscles of the forearm results

when the brachial artery is affected, usually after a supracondylar fracture of the humerus.

Should Muscle Spasm be Treated?

Except in cramp, no. In the lesions with which this book deals, muscle spasm is a secondary phenomenon and its treatment is that of the primary disorder. No one treats by relaxants the muscle spasm due to appendicitis. Similarly, if arthritis or a degree of internal derangement can be abated, the protection given to the joint by the muscles becomes unnecessary. Muscle spasm takes care of itself; all that is necessary is to treat the lesion. This is important, since the wide vogue for relaxant drugs for 'rheumatism', 'fibrositis', lumbago, etc., is based on the fallacy of painful muscle spasm.

Muscle Wasting

The bulk and strength of a muscle depends on three factors: use, nerve supply and the integrity of the joint it spans. The more the patient uses his muscles, the stronger they become. Disuse, especially immobilization in plaster, prevents a muscle working and quickly leads to wasting. If nerve conduction is impaired, a number of muscle fibres no longer contract since they receive no impulse; they waste. Use and a normal nerve supply do not suffice. For example, the gluteal and quadriceps muscles are given plenty of work by a man with a normal knee and osteoarthrosis of the hip, who walks about with a fairly good range of movement. Yet these muscles will have lost much of their bulk. The fact that the muscles waste unduly in rheumatoid arthritis has been noted for many years, and this wasting is much greater than the joint involvement warrants. Steinberg and Parry's (1961) electromyographic findings have demonstrated polymyositis in 85% of cases of established rheumatoid arthritis.

The integrity of the joint, even if the patient is unaware of any disease, is an important factor governing the state of the muscles. This is well illustrated by the following case. A man of 66 had spent six months in bed at the age of 40 with gonorrhoeal arthritis of the hip joint. The WR was negative. After apparent recovery he used the leg normally and stated that for 25 years he had walked as far as he liked without discomfort, apart from some feeling of tiredness in the thigh. He complained of some weeks' aching in the left thigh. Examination revealed gross wasting in quadriceps, gluteal and hamstring muscles, but, surprisingly, a full range of movement at the hip joint. However, his discomfort was produced by forcing rotation at the hip joint. X-ray examination revealed complete destruction of articular cartilage and large osteophytes (Plate XLV/2). A week later the pain ceased spontaneously.

This extreme instance of symptomless arthritis, accompanied by many years' full use of the muscles about the joint, shows how dependent muscle bulk is on the integrity of the joint as such. The wasting is not the result merely of disuse, because years of full function through the full range does not restore the muscle atrophy.