PERIPHERAL NERVE INJURIES AND THEIR TREATMENT

By Andrew Russell Murray,
Orthopaedic Surgeon, Brisbane.

Structure and Functioning of Peripheral Nerves

In order to get a clear picture of what happens when a nerve is injured, and what is involved in the loss and subsequent restoration of function, the structure of a peripheral nerve should be described. It is composed essentially of an aggregation of axons in a neurolemmal sheath. It may contain axons of one type only, as in a purely motor or a purely sensory nerve, or of both types, as in a mixed nerve. Within the sheath the axons are grouped together in a varying number of funiculi separated by supporting connective tissue. In any one nerve the funiculi are constantly undergoing redistribution to such an extent that if a length as short as two millimetres is excised the funicular pattern of each end might be quite dissimilar. Accurate end-to-end apposition of the original funiculi and axons is therefore not possible when portion of a nerve is lost. Because of the repeated division and reunion of funiculi a nerve may be divided as much as a quarter of the way through without significant loss of function. Similarly, axons may also divide and two divisions of the same axon may even pass along different nerves. That fact explains some of the anomalous findings in peripheral nerve injuries. When a nerve is divided, anaesthesia may occur temporarily in an area not supplied by that nerve. For instance, when the median nerve is divided, anaesthesia may occur in the distribution of the medial cutaneous nerve of the forearm.

Normal functioning of a nerve depends on the integrity of the controlling centres in the central nervous system, the parent cell, its axon, and its end-organ in muscle or skin. Axonal damage may be manifested on the one hand by paralysis, anaesthesia, and trophic changes or, on the other, by signs of irritation such as paraesthesia, pain, and secondary tissue changes. The types of lesions that commonly occur are: complete division, partial division, loss of conduction in an intact nerve (neuropraxia), and irritation of a damaged nerve (causalgia).

Complete Division of Peripheral Nerves.

The presence or absence of palpable muscular contractions is the only infallible test in the diagnosis of section of a nerve; tests of sensation may be erroneous if the nerve supply is atypical or the patient is hysterical or is malingering. The fallacy of using the movements of joints as the test is that trick movements may be deceptive. Very many trick movements have been recorded, some of which are well worth remembering. An understanding of the various mechanisms of these movements—six in number—is important. (1) Contraction of a non-paralysed muscle which can perform the movement. This is exemplified by flexion at the wrist joint by abductor pollicis longus when the radiocarpal flexors are paralysed. (2) Contraction of accessory slips from a normal muscle to a paralysed one. An example is extension at the interphalangeal joint of the thumb by a slip from abductor pollicis brevis to extensor pollicis longus in a radial nerve lesion. Though the movement may be a weak one, it is usual in a radial nerve lesion to be able to obtain extension at the interphalangeal joint of the thumb. (3) By the passive pull of a paralysed muscle when the antagonist exerts its full range of movement. It is an anatomical rule that any muscle which crosses more than two joints is too short to allow of a full range of movement in the opposite direction at all of the joints simultaneously. The extensors of the fingers are too short to allow full flexion to occur at all of the joints of the wrist and the finger at the same time. If
full flexion is carried out at the wrist and fingers when complete paralysis of the extensors exists, the fingers extend quite well. (4) By "spring back" action. For instance, when an antagonist contracts strongly and is then suddenly relaxed, the paralysed prime mover appears to act.

(5) Anomalous nerve supply, such as is the case when the ulnar nerve supplies the thenar eminence. (6) By the effect of gravity.

After a nerve is divided complete degeneration of the axons occurs below the level of the section and for a variable distance above the level of the section. Return of function can occur only if motor and sensory axons grow down empty sheaths and make contact with appropriate and viable end-organs. That sounds simple enough, but unfortunately the ultimate results of peripheral nerve injuries is not good. There are many reasons. Fibrosis in the area of degeneration above the level of the lesion may prevent the growing axons from reaching the distal segment. At the level of the lesion fibrotic or other tissue changes may wall off the advancing axons. Axons have an extraordinary propensity for growing downwards. They will grow to great lengths, twisting and turning in all directions, trying to find empty sheaths. That may contribute to the formation of neuromata. In the distal segment of the nerve many things may happen to mar the restoration of function. Should a sensory fibre pass down a motor sheath, it will go on until it meets the muscle end-plate, but it will not make contact. Similarly, regenerated motor fibres growing down a sensory pathway are wasted. As mentioned above, the cross-sectional pattern of a nerve varies every few millimetres, so when the ends of a nerve are resected surgically dissimilar patterns are approximated; the greater the dissimilarity, the greater is the chance of wastage through fibres travelling down wrong pathways. A similar wastage arises from rotation of the nerve ends during suture. The time limit for suturing a divided nerve is approximately one year. The muscle end-plate also eventually degenerates and scars. The muscle itself always atrophies and, under certain circumstances, it may degenerate and be converted into fibrous tissue; then return of function is not possible even if reinnervation occurs. Persistent oedema, vascular stasis, and overstretched of the muscle by its antagonist or by gravity or by ill-advised splinting are all factors which adversely influence the degree of fibrosis of the affected muscle.

Even in the absence of all of these deterrents to restoration of gross function there are other factors affecting the end result less grossly. The new end-organs which replace degenerated ones are less efficient. If the length of a fibre was originally short and if perchance during regeneration it grows down the tube of a fibre which was originally long, it becomes less efficient. When a regenerating fibre branches and a branch makes contact with an end-organ, it will function but not as efficiently as an unbranched fibre would. Even under ideal circumstances the return of sensory function is usually less than perfect; sensations such as discrimination and joint sense are seldom restored. Proximal limb muscles recover more completely than distal ones because the axons of supply are larger and the arrangement within the nerve is more orderly. Besides, these muscles have a grosser action and they function more in combined movements than the more distal ones do; the latter function more individually. The higher the lesion is, the worse is the result in distal areas; this applies in quantity and in quality, and to motor and to sensory functions.

The rate of growth of a regenerating nerve varies considerably and it is influenced by a number of factors. The growth in a day varies between half a millimetre and three millimetres. The faster rate occurs in the more proximal part of the downgrowth; it slows as it reaches the more distal levels. Apart from actual rate of growth there is a period of latency before the fibres begin to grow into the distal segment, and there is also
A lag after the end-organ is reached before the return of function is detectable clinically. The combined duration of the two latent intervals varies but it is of the order of some two to four months.

The aim of active treatment is to maintain a state of good nutrition in all affected tissues, to prevent deformities, and to preserve full mobility of all the joints. Adequate nutrition, of muscles particularly, is important. The muscles have lost their pumping action, which assists the circulation; in consequence relative ischemia and stasis occur, tissue reaction is set up, and eventually there will be degeneration and fibrosis. It is the therapist's job to assist the circulation of blood and tissue fluids, and it matters little by what means. In the case of the upper limb, active usage may be all that is required; where possible early return to work should be encouraged. If that is not possible, physiotherapy is essential by means of massage, heat, passive movements, or electrical stimulation. Electrical stimulation may not have any virtue beyond that of the simple muscular contractions produced; it is not indispensable, but may be more effective than massage and passive movements. It may also have a psychological value; the morale of the patient is uplifted by seeing the paralysed muscles actually working. Deformities are created by the unopposed pull of antagonists and by the effects of gravity and habitual faulty posture. By a combination of accurate splinting and ordinary methods of physiotherapy, painful and crippling deformities of the foot and hand arising from intrinsic paralysis may be prevented.

Almost everyone who has had to treat peripheral nerve lesions has devised special splints for the common types. All the splints vary in external appearance, but the basic principles are the same. The ideal splint should allow a full range of movement of all the active muscles, but, when these muscles are at rest, the affected joints should assume the position of maximal function. In other words, the splint should replace the normal resting tone of the paralysed muscle and, at the same time, permit a full range of movement of the antagonist. That is the principle of the lively or dynamic splint in contrast with the static one. Where possible, splints in peripheral nerve injuries should be lively. Rigid splints may prevent deformity, but they encourage stiffness of joints and adhesions of tendons. Liveliness can be supplied by the use of elastic bands, coil springs, or spring wire. Stiffening of joints and adhesion of tendons may be lessened or even prevented by repeated small manipulations and daily passive movements. Where ambulation is possible in the case of affection of the feet, daily manipulation is considered essential to the maintenance of pliability of the feet.

Re-education begins when muscular contractions return. It is of importance to be aware of the trick movements. During the period of paralysis the general usefulness of the limb has probably been improved by the use of unaffected muscles in abnormal fashion in order to perform purposeful actions which would otherwise be impossible. Sometimes these abnormal actions become habitual, especially when recovery has been slow. It may then be difficult for the patient to revert to the normal use of the proper muscles. Objects can be picked up quite readily by cocking the thumb in median nerve paralysis or poliomyelitis in which the normal pincer action between thumb and finger tips has been lost. In that trick movement extensor pollicis longus pulls the thumb metacarpal into extension while the strong action of flexor pollicis longus brings the side of the terminal phalanx of the thumb against the side of the proximal phalanx of the index finger. When the thenar muscles are re-innervated or the means of normal apposition is restored by means of a transferred tendon the thumb-cocking habit may continue. Therapists must be on the watch for habit movements of that kind or they will be at a loss to explain why a strongly contracting muscle is incapable of producing the movements expected of it. Of course, the trick movement should be encouraged if recovery does not occur or the newly restored function is likely to be poorer than the result of the trick movement. In the re-education of muscles the importance of relaxation must be stressed. When a patient has not been able to perform a
movement for a long time he is desperately keen to succeed when the big day of the trial comes. He may try so hard that prime movers, antagonists, synergists, and fixators all contract together and thus prevent the occurrence of the desired movement. But if he is shown how to relax all of the muscles and to carry out the required movement very gently, and is assisted by a little rhythmical action, the patient can learn to perform the movement at will. When simple movements can be carried out without hesitation, purposeful movements, complex, and automatic movements, are best developed through occupational therapy.

To complete this story of peripheral nerves division reference must be made to the operation of tendon transfer. When a nerve has been irreparably damaged, a means of improving function is the transfer of the tendon of an active muscle to the site of a paralysed one. This type of surgery is fascinating and there are numerous examples of it. It involves a wealth of information on the action of muscles at joints, applied anatomy, and muscle pathology. The art of the physiotherapist is invaluable in the after-treatment.

Other Types of Injury to Peripheral Nerves.

We have been considering various aspects of the complete division of peripheral nerves but we must pass on to a general study of the other types of nerve injury and their complications. Partial division of a nerve may offer considerable difficulties in diagnosis and treatment. The actual arrangement of the axons at the point of injury will largely determine the effect of a partial division. If the motor and sensory fibres are freely mixed up one would expect partial anaesthesia and partial paralysis. If each type of fibre is, however, aggregated together and the aggregations are separated in the nerve bundles, anaesthesia or paralysis alone may be found. That type of lesion is often seen when either the median or the ulnar nerve is injured at the level of the wrist; at that level there is considerable separation of the two elements of those nerves. In the presence of an aberrant supply from another nerve difficulty arises in the differential diagnosis between partial division, partial neuropraxia, and complete division. Suppose we find anaesthesia of the thumb and index finger only together with loss of abduction but not of opposition of the thumb the lesion may be a partial division of the median nerve. But in an individual who has an aberrant ulnar sensory supply to the middle finger and motor supply to opponens pollicis the lesion present could be a complete division of the median nerve. When doubt of the kind exists, the physiotherapist may become the chief diagnostician; the presence or absence of contraction of opponens pollicis on faradic stimulation of the ulnar nerve usually clinches the diagnosis.

Neuropraxia, loss of conduction in an intact nerve, also produces diagnostic and therapeutic problems. The motor fibres suffer to a greater extent than do the sensory ones. That fact might be helpful, though, in the case of a purely motor nerve, such as the radial or the axillary nerve, the differential diagnosis may still be difficult. In some cases of neuropraxia complete paralysis may last for many months, but in most the recovery commences in a few days or a week and may be complete. In others there are varying degrees of permanent loss of function. The cause of a neuropraxial lesion is either a direct blow over the nerve or, more often, sustained pressure upon it. In the latter group many cases result from pressure of splints or maintenance of abnormal posture; they may be prevented by avoidance of these causes. The group is an important one for several reasons, of which the least is not the medico-legal implication. A few examples of pressure neuropraxia are: external popliteal nerve lesions from plasters and Thomas splints; crutch palsy; tourniquet paralysis; posterior interosseous nerve palsy from hanging casts; post-operative palsies of various types from abnormal posture under anaesthesia such as radial or ulnar nerve affections when an arm has been hanging over the side of the table, external popliteal nerve paralysis from pressure against leg supports, and
lesions of the brachial plexus from shoulder rests or excessive abduction of the arm. The treatment of these lesions from the physiotherapeutic angle is practically the same as that of complete division. Intense nerve irritation may occasionally be set up as a complication of partial division of a peripheral nerve. This causalgia is characterized by diffuse, intense burning pain which is subject to exacerbations induced by any form of stimulation, even including emotional stimuli. The limb becomes pink and clammy with long and tender nails and decalcified brittle bones. Sufferers from causalgia often exhibit neurotic signs, which is not surprising in view of the distressing nature of the complaint.

The treatment of causalgia is primarily physiotherapeutic, and is one of the greatest tests of the skill of the physiotherapist. Unless each day all the affected joints are put through a full range of passive movements, hopeless stiffness and deformity rapidly result. A temperature change of the limb before the commencement of the passive movements may be helpful; in any one case a particular temperature, cold or hot, may be found to ease the pain significantly. Such cooling agents as spirit or camphor and menthol may be useful. The stout-hearted will benefit considerably from expert physiotherapy, but it is useless to make the attempt for unstable patients; for the latter, radical surgery, such as complete division of the nerve, is the only rational form of treatment.

The subject of hysteria is so large that no more will be said on this occasion than is enough to remind you that hysterical causes of paralysis or paraesthesia are very common and must constantly be kept in mind. On many occasions the physiotherapist has the opportunity to clarify the diagnosis in doubtful cases by accurate observations.

The value of percussion in the treatment of painful amputation stumps and phantom limbs needs emphasis. The symptoms can be eased considerably by hammering the appropriate nerve or its neuroma. The patient may have a distressingly acute awareness of the missing limb, the toes or fingers may feel as if they were being screwed off, or the missing feet may constantly jump with stabs of pain. Explain carefully what you are going to do; start very gently and slowly; work up to heavy blows. At first the patient will have increased pain, then a pleasant sensation of glowing warmth, and, in the end, the harder you hit, the greater becomes the relief; and the longer you continue to hammer away, the longer will be the duration of the relief.

Acknowledgement.

I would like to thank Professor Sydney Sunderland from whose writings much of the technical details of this lecture has been taken.