DIAGNOSIS OF THE COMMON FORMS OF NERVE INJURY OF THE EXTREMITIES

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History-taking is the first step in diagnosis and it is useful to know how varied the causes of peripheral nerve injuries can be. Otherwise the true nature of a traumatic lesion sometimes may not be suspected.

The commoner ones are the result of:—

- (1) Cutting and laceration.
- (2) Stretching, which may be sudden (e.g. stretching of the sciatic by jumping upon the extended foot) causing fibre rupture and haemorrhage, or prolonged (e.g. lying with the arm extended for hours above the head) causing ischaemia.
- (3) Contusion.
- (4) Concussion (including that produced by a "near miss" when a missile passes through neighbouring tissues without touching the nerve).
- (5) Pressure (e.g. crutch and "Saturday-night" palsies).
- (6) Puncture (e.g. by an injection needle).
- (7) Local poisoning by injected substances; and
- (8) Freezing.

Loss or impairment of movement is usually the presenting symptom of a peripheral nerve injury. But such loss may be caused by fractures, joint lesions (dislocation, inflammation, adhesions, ankylosis), muscle or tendon injuries, local inflammatory or other swellings, and spasm, fibrosis or contracture of muscles. It may also be due to more remote causes such as Central Nervous System diseases, non-traumatic peripheral nervous lesions or hysteria. Care may sometimes be needed to exclude some of these conditions, but usually the obvious paralysis of muscles following upon trauma, with loss of sensation in the skin area supplied by the corresponding nerve points directly to peripheral nerve injury. Adequate diagnosis, however, involves first localising the lesion (anatomical diagnosis) and then an estimate of its kind and degree (pathological diagnosis). Localising the lesion is the simpler of the two tasks, although it requires a knowledge of a fairly large number of well-recognised facts. Many of the criteria of pathological diagnosis are also well established, but some are still being evolved.

The clearest guides to **localisation** are muscular weakness or paralysis, and wasting, with accom-

panying diminution or loss of reflexes. In the absence of an external wound or contusion near the nerve concerned these muscle changes may be the only guide.

Look first at the most peripheral muscles and particularly those which move the hands and feet. If these are normal (indicating an intact nerve supply) it is uncommon, although not impossible, for muscles to be involved whose supply leaves those same nerves at a more proximal level. And proximal involvement with normal peripheral muscles only occurs close to the actual spot where the nerve is injured. The state of innervation of the muscles moving the hands and feet gives no guide, however, to that of the limb girdle muscles, as they are supplied by comparatively short nerves arising directly from the two great plexuses.

Weakness or paralysis of a muscle having been found, one then follows the nerve proximally, muscle by muscle in the order in which their nerves of supply leave the parent trunk. The most proximally innervated of the muscles found affected is a useful pointer to the level of the lesion. Muscles should be tested by seeing and palpating the actual muscular contraction or the tensing of its tendon. Movement of a segment of a limb is not a safe test. Very similar movements can sometimes be produced by more than one muscle and they may not be supplied by the same nerve. There are also other varieties of "false movement." Furthermore, loss of the fixating action of certain paralysed muscles may give a false impression that other healthy muscles are weakened (e.g. the apparent weakness of the finger flexors when there is wrist drop).*

Impaired sensation can be useful in confirming the fact of involvement of a particular nerve, particularly if motor signs are slight. It is of little use, however, in locating the level at which the nerve is injured, particularly if the lesion is a complete one, for then overlapping adjacent nerves function to supply skin sensation in much of the area also supplied by the injured nerve. If the lesion is old enough even the residual anaesthetic area unaffected by the phenomenon of "overlap"

* A very useful pamphlet containing numerous illustrations of good methods of testing various muscles is Medical Research Council War Memorandum No. 7 entitled *Aids to the Investigation of Peripheral Nerve Injuries*, published by His Majesty's Stationery Office in 1942.

may show returning sensation due to the ingrowth of fibres from adjacent uninjured nerves.

So much for general principles in localisation.

Individual nerves can now be considered.

Isolated lesions of the *Ulnar Nerve* are common, due to its fairly superficial course, particularly near the elbow. PLATE 1 shows a hand where there is a severe ulnar lesion. Atrophy of the first dorsal interosseous muscle and of the adductor pollicis is obvious from the hollowing of the first interspace and there is some indication of clawing of the hand with hyperextension at the metacarpo-phalangeal joints and flexion at the interphalangeal joints. Such a finding would lead one to test in order the remaining interossei and the medial two lumbricals, the opponens and the abductor digiti minimi, the slips of the flexor digitorum profundus to the ring and little fingers and finally the flexor carpi ulnaris. This search would give one the most peripheral possible site of the injury.*

Confirmatory sensory loss should be sought over the ulnar edge of the hand and the little finger. As in all nerve injuries, sensation of light touch will be found lost over a wider area than is pin-prick.

Isolated injury of the *Median Nerve* is not seen except as a result of penetrating wounds. Its position makes it less prone to injuries in general than either ulnar or radial. Trophic and vasomotor disturbances and causalgia are commoner in median nerve lesions than in any other. PLATE 2 shows a right hand where there is a severe lesion of the median nerve and the normal left hand of the same subject. Note the atrophy of the thenar muscles and of the pulp of the first finger. Note also the thumb lying back in the plane of the palm. It is this falling backwards of the thumb with hyperextended index and middle fingers which give rise to the term "ape-hand" in median nerve palsy. The middle finger in this case happens to be flexed due to the development of a contracture. Before this occurred there was the usual great difficulty in flexing its two distal phalanges. Characteristic and easily elicited signs in median paralysis are inability to oppose the tips of the thumb and little finger so as to make a circle (opponens, and short and long flexors pollicis), to abduct the thumb at right angles to the plane of the palm (abductor pollicis brevis) or to flex the two terminal phalanges of the index finger whilst the proximal one is held extended (flexor digitorum profundus—lateral slip) and (flexor digitorum sublimis).

Obvious median involvement having been diag-

nosed, the muscles it supplies should be tested in the following order—distalo-proximally:—the two lateral lumbricals, the opponens and short flexor of the thumb, the short abductor of the thumb, the lateral two heads of the flexor digitorum profundus, the long flexor of the thumb, the flexor digitorum sublimis, the flexor carpi radialis and the pronator teres.

Sensory loss is to be sought over the two terminal phalanges of the index and middle fingers. If the lesion is at all severe loss of tactile sensibility is more extensive than this and can be found over the radial half of the palm as well.

No picture of the appearance of the hand in lesions of the *Radial Nerve* has been included. This nerve, of course, supplies no muscles in the hand itself, and the reader can produce the characteristic wrist-drop simply by relaxing the extensors of the hand. With this as a usual initial sign, one can confirm the fact of a radial lesion by noting that the thumb cannot be extended in the plane of the palm (e.g. with the palm held flat on a table), nor can the proximal phalanges of the fingers be extended. Ascending the arm one then tests the supinator, extensor carpi radialis longus, brachioradialis and triceps. Sensory changes in radial lesions are limited to a small area on the back of the hand overlying the first and second metacarpals and perhaps the base of the thumb.

The radial nerve is apt to show extensive paralysis of good prognosis after relatively trivial injuries and it is from the radial that one gets the most striking examples of neurapraxia following pressure and stretching.

Musculo-Cutaneous palsy is very obvious with biceps paralysed and brachialis largely inactive and sensory impairment over the proximal part of the radial border of the forearm. (It should be remembered that the brachialis is also supplied by the radial). Damage to the musculo-cutaneous nerve usually occurs as part of a brachial plexus injury.

Deltoid paralysis is the outstanding sign of *Axillary Nerve* lesions. PLATE 3 shows the flattening of the outline of the shoulder produced when wasting of the deltoid supervenes. Palpation of the muscle is particularly important when testing the deltoid because compensatory action by the supraspinatus can abduct the arm and elevate it laterally to some extent, giving a false impression that the deltoid is recovering. The only other muscle the axillary nerve supplies is the teres minor. It is small and not readily tested as its functions are not easily distinguished from those of the infraspinatus which is supplied by the suprascapular nerve. Axillary nerve lesions produce a characteristic longitudinally oval area of sensory loss about the size of a hen's egg with its lower end over the deltoid insertion.

* It is not possible in a short article like this, to go into detail which can be found readily in any text-book of neuro-anatomy, but it can be taken as a rule that branches of supply to muscles usually leave the parent nerve as it lies abreast of the muscle belly. There may be several such branches to an individual muscle, e.g. the flexor carpi ulnaris may receive as many as four.

PLATE 1.—Effects of an ulnar nerve lesion.
(Reproduced by kind permission of Dr. C. C. Worster-Drought.)

PLATE 2.—Effects of median nerve lesion (*right*) compared with normal left hand.
(Reproduced by kind permission of Dr. C. C. Worster-Drought.)

PLATE 3.—Effects of axillary circumflex nerve lesion (*right*) compared with normal left shoulder.

(Reproduced by kind permission of Dr. C. C. Worster-Drought.)

PLATE 4.—Effects of a complete sciatic nerve lesion (*right*) compared with a normal left leg.

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From the *Brachial Plexus* itself the more important of the short branches which may be injured and whose muscles can be tested easily, are:—

- (1) from the cords of the plexus
 - (a) the thoraco-dorsal nerve supplying the latissimus dorsi,
 - (b) the lateral and medial anterior thoracic nerves supplying the pectorals.
- (2) from the trunks
 - the suprascapular nerve supplying the supra and infra spinatus muscles.
- (3) from the rami or roots of the plexus
 - (a) the long thoracic nerve supplying the serratus anterior
 - (b) the dorsal scapular nerve supplying the rhomboids.

If these muscles are tested in the above order, they can give useful information about the level of a plexus lesion. If the injury involves the lowest cervical or first dorsal roots very near their origin there may be a cervical sympathetic paralysis with production of Horner's syndrome (miosis, enophthalmos, narrowing of the palpebral fissure and, much more rarely, vasodilatation and diminution of sweating over the face on the affected side).

The three classic varieties of brachial plexus lesion are mentioned here for completeness.

(1) Lower plexus type (Klumpke's paralysis) due to injury to the eighth cervical and first thoracic roots, caused usually by upward traction on the extended arm. This produces paralysis of all the intrinsic hand muscles and wrist and finger flexors with sensory impairment along the ulnar border of the hand and forearm. Occasionally there is a cervical sympathetic paralysis also.

(2) Upper plexus type (Erb's paralysis) due to injury to the fifth and sixth cervical roots, most commonly caused by strong downward traction on the arm or shoulder, particularly when combined with forcible flexion of the neck to the opposite side. In proximo-distal order the muscles found affected in this type are the spinati, the pectoralis major (partial), latissimus dorsi, teres major, deltoid, all three anterior upper arm muscles, and the brachio-radialis. The hand and forearm muscles escape. Sensory impairment may be found over the outer aspect of the whole of the upper limb.

(3) Middle plexus type. This involves the posterior cord and the picture is one of combined radial and axillary nerve paralysis with involvement of the nerve to the latissimus dorsi also. It is only caused by penetrating wounds and is uncommon.

Injuries of the nerves of the lower limb are much less common than those of the upper.

The *Sciatic* is the one most frequently involved and PLATE 4 shows the leg and foot in a right-sided sciatic lesion. Note the greatly wasted calf and the foot-drop. In complete lesions such as this, there is total paralysis below the knee and, if the lesion is sufficiently high in the buttock, a loss of flexion at the knee due to hamstring paralysis. The ankle jerk is lost but the knee jerk remains. Sensory impairment is to be sought over the whole of the foot except the medial malleolus and the area of the instep just in front of it, and over the lower two-thirds of the postero-lateral aspect of the calf.

The sciatic usually divides into tibial and common peroneal branches at the upper end of the popliteal fossa. It may do so, however, as high as the buttock, or the two parts occasionally may not be united into one trunk at any level. This is why a deep wound of the thigh may sometimes produce a lesion of one division of the sciatic only.

Common Peroneal injury shows itself by loss of eversion of the foot (peroneus longus and brevis, supplied by its musculo-cutaneous (or superficial peroneal) branch) and also by loss of extension of the proximal phalanges of the toes (extensors digitorum brevis and longus and extensor hallucis longus) and of dorsiflexion at the ankle (tibialis anticus and long extensors of the toes), this second group of muscles being supplied by its anterior tibial (deep peroneal) branch. If the lesion is high in the thigh the supply from the common peroneal nerve to the short head of the biceps may be interrupted. Knee and ankle jerks are unaffected. The two branches of the common peroneal may be affected separately by penetrating wounds in the leg but this is not common. The area of sensory loss in common peroneal lesions extends in a band from the bases of the medial two toes up the middle of the top of the foot and immediately lateral to the ridge of the tibia up to a point two or three inches below the knee.

Lesions of the *Tibial* (medial popliteal) branch of the sciatic cause paralysis of all the intrinsic foot muscles (except the short extensor of the toes) of the long flexors of the toes and great toe and of the other three great calf muscles. If high in the thigh its supply to the semitendinosus and biceps may be involved. Sensory impairment is found on the sole and the outer border of the foot. The ankle jerk and the plantar reflex are lost.

The *Superior Gluteal* nerve supplying the tensor fasciae latae, the gluteus medius and the gluteus minimus, and the *Inferior Gluteal* nerve supplying the gluteus maximus are very occasionally injured by penetrating wounds.

The *Obturator Nerve* supplying the adductors of

the thigh, the obturator externus and the gracilis which bring about adduction and internal rotation at the hip, is rarely injured alone.

Femoral Nerve involvement produces quadriceps paralysis with loss of the knee jerk, paralysis of the sartorius, and if the lesion be high enough, paralysis of the iliacus. Its sensory area is the anterior and medial aspect of the lower two-thirds of the thigh, the anterior and medial aspect of the knee and the medial aspect of the leg, the medial malleolus and the medial part of the foot and the instep just in front of this malleolus.

Injury of the *Lateral Cutaneous Nerve of the Thigh* may cause meralgia paraesthetica, although the condition is more often of unknown origin. Its symptoms are spontaneous tingling, numbness, burning or neuralgic pain in the area supplied by the nerve. They are made worse by activity. On examination there may be tenderness of the nerve trunk near the point where it enters the thigh and impairment or distortion of sensation on testing the skin it supplies.

When diagnosing the **Nature of the Lesion** one must have in mind the possible varieties. These fall into three groups. First is complete severing of the nerve trunk which Henry Cohen has named *Neurotmesis*. It abolishes every function of the nerve.

The second is the "lesion in continuity" now named *Axonotmesis*, where there has been sufficient damage to the actual nerve fibres to cause them to degenerate peripherally to the lesion but the continuity of the nerve trunk is preserved by its connecting or supporting tissues. It is these connecting structures which act as guides to the damaged fibres, as they regenerate. The majority of axonotmeses recover spontaneously.

The third is a condition of temporary impairment of conduction in a nerve which is termed *Neurapraxia*. It recovers spontaneously at a rate far too rapid to be attributed to regeneration (usually in two to six weeks, but sometimes much less).

More than one of these types of lesion can exist simultaneously in the same nerve, but the pathological and clinical phenomena of such injuries can still be classified under these three headings.

It must be emphasised that unless the divided ends of the nerve can be seen it is not possible to diagnose with certainty a state of neurotmesis until several months after the injury. If, immediately following trauma, there is complete paralysis of all the muscles whose nerve supply arises distal to the lesion and loss of sensation of pin-prick and light touch in the area supplied exclusively by the nerve concerned, all one can say is that the lesion is probably severe, and usually either a neurotmesis or axonotmesis. Sometimes, however, even if all

these signs are present, it is not possible to estimate the severity of the lesion at all except after the passage of time. Slight injuries to the radial nerve, for example, can produce such signs with relative ease, but rapid recovery soon proves the condition to have been only one of neurapraxia. This state can often be surmised from the nature of the damaging force which is commonly pressure or stretching of brief duration, or a mild or moderate contusion.

Since the majority of cases of axonotmesis recover spontaneously the paralysed muscles must be suitably cared for and watched, but one should never wait until a diagnosis of neurotmesis is established beyond all doubt before advising exploration of the lesion. It becomes a highly probable diagnosis if at the end of three months there is no recovery of motor function in any of the muscles originally paralysed and these show the characteristic reactions of degeneration on electrical testing. It is then that exploration is advisable. Neurotmesis can only be diagnosed with certainty when the full time necessary for regeneration has elapsed and it is found that paralysis and the reaction of degeneration still exist. By then the chances of surgery being beneficial will have been lessened.

Some facts about sensory findings are to be noted.

There is much overlapping of the sensory areas supplied by adjacent nerves and although it is probable that such overlap does not fully function except when there is complete nerve section or block, it is precisely in such cases that sensory tests are most important. So for practical purposes it is best to confine one's tests to those areas not overlapped by adjacent nerves.

But tests even in these areas are only reliable shortly after total nerve lesions, because ingrowth of fibres from adjacent uninjured nerves occurs, and may give a false impression of recovery.

If immediately following trauma only some of the muscles distal to the lesion are paralysed, if there is not complete loss of sensation in the skin area exclusively supplied by the nerve or if not all kinds of sensation are lost in that area, then the lesion is incomplete and must be either an axonotmesis or a neurapraxia. The rate of recovery in this group is a cardinal point of distinction between the two varieties. Neurapraxia is also strongly suggested if normal electrical responses are preserved in the muscles for more than about eight to ten days following the lesion.

The most reliable *Signs of Recovery* in nerve lesions again are motor and both the active movements and the electrical reactions of the muscles serve as guides. Where regeneration occurs the muscles lose their paralysis in the anatomical

order of origin of their nerve supply from the trunk. The rate of regeneration in motor nerves is about 1 cm. per week, slowing down as the fibre nears the end of its journey. About forty days elapses following an injury before this process begins. The gap is rather longer in cases of nerve section. Thus, if the site of the lesion is known, one can calculate the time at which a particular muscle should show signs of recovery if regeneration is occurring. In cases of neurapraxia, however, there is no particular order of muscular recovery and all may regain power almost simultaneously. Only return of sensation in the exclusive area of the nerve concerned is of significance in estimating recovery and then only if it occurs too early for ingrowth from adjacent nerves to have taken place. Pain sensibility reappears before tactile, but in cases of neurapraxia tactile and pin-prick sensation may be both absent and return together. Sensory and motor recovery are not necessarily co-eval. In tibial and peroneal nerve lesions, for example, sensory recovery is often much earlier and in ulnar and median lesions often much later than motor recovery.

The degree of muscular atrophy, the state of muscle tone, trophic disturbances and the sensations produced by pressure on nerve trunks and muscles are not reliable guides to the state of recovery.

Finally, a few facts ought to be remembered about electrical tests. If, thirty or forty days after the injury, bearable faradic stimulation causes a response from paralysed muscles, then the nerve is not severed. Faradic current acts through the nerve because the duration of the effective portion

of its wave is .001 of a second which is too short a time of action to evoke a response from muscle. The loss of response to faradism of a muscle-nerve complex is part of the Reaction of Degeneration. The other parts concern the response to galvanism and they are:—

(1) Hyperirritability of the muscle which responds to half or a smaller fraction of the normal minimum current required. Instead of responding by the normal sudden twitch, however, it responds with a slow contraction.

(2) The amount of galvanic current required to throw the severely degenerated muscle into tetanus is only 1.5 to 1.2 times the amount which just produces a contraction. In healthy muscle the ratio is 4 or 4.5 to 1.

(3) To elicit contraction from denervated muscle the amperage required for cathode-closing stimuli has to be as great or at times even greater than that required for anode-closing stimuli. In health, of course, a smaller amperage suffices to elicit contraction with a cathode-closing stimulus.

This reaction of degeneration requires at very least a week to develop after the severance of a muscle from its nerve. It may take from one to two years for the degeneration of muscle to become so complete that all response to galvanism is lost. A rise in the amount of galvanic current necessary to produce muscular contraction is one of the earliest signs of regeneration of the nerve, provided that increased local tissue resistance due to such changes as inflammatory infiltration can be excluded.

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