PERIPHERAL NERVE SURGERY—DIAGNOSTIC CONSIDERATIONS*

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The diagnosis of peripheral nerve lesions depends primarily on a precise knowledge of the regional anatomy. The surgical anatomy of the extremities does not receive the attention in teaching of medical students and interns that its importance deserves. The writer has received the impression from years of teaching medical students and resident staff officers that there is a regrettable lack of knowledge not only as to the function of peripheral nerves but as to their physical appearance and anatomy. Interns assistants frequently express surprise that the median or ulnar nerve when exposed by operation at the wrist is so large. They have been under the impression that the nerves are more or less minute structures and difficult to recognize. Fortunately, there has been a trend in recent years toward better surgical training in conditions affecting the extremities but much remains to be done in this direction.

Study of nerve lesions by clinical and experimental methods shows great activity during times of war but if one may judge by the American literature in the interval between the First World War and the present conflict there is little evidence of a sustained interest in these important injuries. It is true that some valuable contributions, such as those of Stookey, Davis and Pollock, appeared in the interval between the two World Wars but on the whole the subject was rather generally neglected and this in spite of the fact that nerve lesions produced by industrial and transportation accidents in civilian life are by no means uncommon. They are easy to recognize but too often inadequately treated.

The clinical examination of the patient supplies the most important information as to peripheral nerve injuries. Clinical diagnosis, however, has received many accessions from careful experimental investigation. It is the purpose of this paper to discuss primarily the clinical diagnosis of peripheral nerve lesions, with brief reference to certain research methods as have proven helpful in advancing clinical diagnosis.

There are definite effects which follow a lesion of every peripheral nerve. The interpretation of these effects in a given case requires precise knowledge of the motor distribution of the nerve as well as its cutaneous sensory supply. While there are numerous effects of division of a large peripheral nerve, loss of motor power in the muscles supplied by the nerve and associated loss of sensation are by far the most serious results.

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In the diagnosis of nerve injuries, one must take into consideration the type of injury, the primary effects upon muscle function and sensation in the nerve field, the level of the lesion, and the interval between injury and examination. There are numerous other considerations but time permits discussion of only the more important ones.

Division of nerves in civilian practice most frequently results from cuts by glass or sharp instruments. One of the most common peripheral nerve injuries in civilian practice is that of the median or ulnar or both of these nerves at the wrist. With such injuries there is usually a severing of important tendons. Unfortunately the treatment of these very serious injuries is often delegated to an inexperienced interne, notwithstanding the fact that proper repair of such lesions requires the services of an expert surgeon. In the treatment of these injuries by the inexperienced operator, the median and ulnar nerves frequently are not identified, nerves are often confused with tendons and suture of the median to the palmaris longus tendon or the ulnar to the flexor carpi ulnaris tendon is not infrequent. I feel quite sure that this deplorable error has been discovered by many neurological surgeons at secondary operation.

The immediate loss of nerve function following a stab wound or penetration by fragments of glass in the region of the nerve is unequivocal proof that the nerve has been divided either partially or completely. A stab wound or glass cut about the parotid region resulting in immediate inability to close the eye on the injured side or inability to retract the corner of the mouth means that one of the main branches of the facial nerve has been severed. The diagnosis of nerve division in this type of injury is easy if one bears in mind this simple fact. The failure to make a correct diagnosis early and to carry out proper surgical treatment in any nerve injury due to sharp instruments is most unfortunate inasmuch as there is inevitable retraction of the nerve segments and formation of neuromas, making later direct suture more difficult and sometimes impossible. In median and ulnar lesions, delayed nerve repair allows for the development of advanced atrophy of the intrinsic hand muscles, periarthritic fibrosis and other crippling effects which permanently mar the results of later suture.

We are more concerned at the present time with nerve lesions produced by gunshot wounds of the extremities. The diagnosis of nerve lesions in such conditions must take into consideration certain concussional effects of missiles of high velocity which may cause a temporary interruption of nerve function without serious structural damage to the nerve itself. In other words the concussion from a projectile of high velocity passing near the nerve may cause a temporary physiological interruption of nerve function. However, the loss of function is not as complete as is the case when the nerve trunk itself is directly damaged. The nerve lesion produced by gunshot may not divide the nerve trunk but may cause a lateral defect or a neuroma in continuity from hemorrhage within the nerve trunk, and complete physiological interruption. In nerve wounds of warfare an effort must be made to differ-
entiate clinically between a lesion causing slight structural damage from which the patient may recover spontaneously, and one in which the nerve is severely damaged, requiring suture. To make this differentiation between the recoverable and nonrecoverable nerve lesions is not always easy. In the First World War the problem of diagnosis often was met by delaying exploration of the nerve to test spontaneous recoverability and, in some cases, because of persisting wound infection, this delay was required. However, if the nerve proved to be incapable of regeneration, much valuable time was lost and the delay resulted in further crippling of the motor apparatus from atrophy and other disabling effects of long continued denervation.

In the writer's opinion, a nerve showing complete physiological interruption at the time of complete healing of a wound from gunshot or other missile should be promptly explored to make as precise a diagnosis as possible and to suture those nerves that are found divided. It will generally be found in doubtful cases that the nerve lesion exposed is a more serious one than indicated by the clinical examination. It was the experience in the nerve reconstruction centers of the First World War that in many nerve injuries when there were thought to be signs of satisfactory nerve regeneration there was found complete division with wide separation of the nerve segments and no possibility of spontaneous regeneration.

The early recognition of nerve injuries in war wounds is of greatest importance. Because of the frequency of nerve injuries in war wounds of the extremities, it is essential that examination be made at the earliest possible moment in these cases to determine whether one of the main peripheral nerves has been injured. It is gratifying to know that this is the practice of the Medical Corps in the American Expeditionary Force in the present war. Such an examination should record as fully as practicable the essential facts of a nerve injury. In civilian practice it should be determined if possible in examination of a fractured extremity whether there has been an associated nerve injury or injury to one of the large vessels before treatment of the fracture is instituted. Musculospiral injury is a frequent accompaniment of fracture of the humerus and it should be detected promptly.

While the diagnosis of nerve lesions is primarily clinical, under certain conditions complete diagnosis can be made only by surgical exploration of the nerve. The surgical exposure for more exact diagnosis of peripheral nerve lesions is a fairly simple procedure in experienced hands, but expert surgical judgment may be required when the nerve is exposed to determine the potentiality of the nerve for spontaneous recovery when a fusiform neuroma and not division of the nerve is revealed. The surgical treatment of these lesions after diagnostic exploration will be discussed by Colonel Spurling.

The clinical examination of a patient with suspected nerve injury should be done carefully and systematically. One may well begin this examination by study of the motor function of the unaffected extremity. We find that patients do not always comprehend promptly what is expected of them if asked to perform movements in the paralyzed extremity but if one has the
patient execute first the desired movements of similar muscles on the un-
affected side, he will better understand what is expected when the involved
extremity is subsequently investigated.

In the study of median, musculospiral and ulnar lesions, the appearance
and posture of the hand give very definite information. Paralysis of the ulnar
and musculospiral nerves may be recognized at a glance. Certain character-
estic effects are wrist drop in musculospiral and griffe defect in ulnar lesions
with the inevitable deviation of the little finger from the ring finger. There
are certain simple well known tests which are useful in the study of all
median, ulnar and musculospiral nerves. These tests are based upon loss of
muscle power resulting from nerve interruption. The patient with a median
nerve lesion at any level is unable to hold the thumb at right angles to the
palm. In lesions above the origin of the branches to the long flexors of the
digits the patient is unable to flex the distal phalanx of the thumb and index
finger. He is unable to appose the pulp of the thumb to the pulp of the fingers
and efforts to do so result in the thumb crawling over the palm of the hand.
In ulnar lesions the patient can make only feeble lateral movement of the
fingers and in no case can he superimpose the little finger on the adjacent
ring finger. He may be able to hold with considerable force a sheet of paper
between the extended fingers of the unaffected side but he has practically no
power to do this on the injured side. In the study of nerve function of the
hand one thinks of the median as supplying the thumb primarily, whereas
the ulnar serves the refined movements of the fingers with some overlapping
in the function of both nerves (Figs. 1, 2 and 4).

The characteristic feature of musculospiral paralysis is wrist drop. While
this nerve produces extension of the forearm, complete loss of this movement
is unusual due to the fact that the branches to the long head of the triceps
leave the nerve at a high level and these branches generally escape injury. If
the patient with musculospiral paralysis places the palm on a flat surface
he is able to elevate the tips of the fingers through ulnar interosseous action
but not the thumb, the extensors of which are supplied by the musculospiral
(Figs. 3 and 9).

The study of muscle function following nerve lesions in the lower extremi-
ties is by no means so complicated as that of the upper in view of the fact
that the muscles of the feet are not normally capable of the refined move-
ments required of the hand. Inability to extend the knee from a lesion of the
anterior crural nerve was rarely seen in the reconstruction hospitals of the
First World War, due to the fact that when this nerve was injured, the femo-
ral vessels were also divided and fatal hemorrhage occurred.

Flexion of the leg on the thigh is seldom abolished by division of the sci-
atic nerve inasmuch as some of the branches to the semitendinosus leave the
nerve at a high level and escape injury. The principal motor loss due to sci-
atic injury is inability to perform movements of the ankle joint and toes.
When the internal popliteal is paralyzed, plantar flexion is impossible. Dor-
siflexion of the foot or raising the toes from the floor when standing cannot
FIG. 1 (left). High combined lesion of the right ulnar and median nerves, showing typical appearance of hand. Note the deformity of the right hand as compared with the left.

FIG. 2 (right). Complete paralysis of the right median and ulnar nerves from division of the nerves in the upper arm by a fragment of glass. Note flexion of the wrist by substitutionary action of the extensor osseus metacarpi pollicis supplied by the musculospiral. The tendon of this muscle can be seen. The scar of incision is also shown. It was necessary to transpose the ulnar nerve to get satisfactory approximation. The neuromata of the median nerve were tied together by sutures with the forearm in flexion for four weeks. At the end of this time the nerve had been elongated sufficiently to permit satisfactory suture.

FIG. 3 (left). Beginning recovery of musculospiral paralysis from a lesion in the arm. The patient is able to accomplish slight elevation of the index finger.

FIG. 4 (right). Characteristic deformity of the hand following ulnar nerve lesion. Note the hyperextension of the proximal phalanges due to the unopposed action of the common extensors. Note also the flexion of the terminal phalanges from the pull of the deep flexors of the fingers.*

be performed when the external popliteal is divided. Partial lesions of the sciatic nerve are not uncommon and frequently one of its main constituents may be divided by a projectile while the other escapes injury.

In the study of loss of muscle function due to nerve paralysis, one must keep constantly in mind what is known as supplementary, substitutionary or "trick" movements performed by muscles supplied by the adjoining uninjured nerves when the muscles primarily responsible for the movement have been paralyzed. An interesting example of such a "trick" movement is elevation of the upper extremity to a right angle even though injury to the axillary nerve has produced deltoid paralysis. The writer recalls only one such case in the First World War but two cases have been seen in recent practice.

Substitution for the deltoid function in such cases is probably from the combined action of the pectoralis major, supraspinatus and trapezius muscles. In the examination of muscle function it should be determined whether the

*Loc. cit.*
ment for flexion of the wrist in high combined lesions of the median and ulnar nerves, in which case all the flexors of the forearm are paralyzed, is flexion of the wrist which may be accomplished by the extensor ossis metacarpi pollicis supplied by the musculospiral, and in some instances we have seen this muscle develop considerable compensatory hypertrophy (Figs. 10 and 11).

Reference should be made to another very important substitution of function in paralysis of the musculocutaneous, which supplies the biceps and brachialis anticus, the primary flexors of the forearm. Here the patient is capable of making rather forcible flexion by action of the brachioradialis, supplied by the musculospiral. Palpation of the paralyzed biceps in such case shows flabbiness and lack of contraction, whereas the brachioradialis will be seen and felt to contract with force.

It should be remembered that the long flexors of the fingers when acting tend to produce approximation, whereas the long extensors tend to separate the fingers. Both of these substitutionary actions may conceal a lesion of the ulnar nerve which is primarily responsible for lateral movements of the fingers. In musculospiral paralysis in which a cock-up splint has been used with resulting shortening of the extensors of the hand, making a fist tends to elevate the hand and conceals the musculospiral paralysis (Figs. 5 and 6).

A differentiation must be made between marked weakness and complete paralysis of muscles. A muscle or group of muscles recovering from paralysis due to nerve injury may be unable to execute a movement when such movement is opposed by the force of gravity. Therefore, in the examination it often becomes necessary in order to obtain a precise idea as to whether the nerve is recovering, to place the limb in such position that gravity will not materially interfere with the interpretation (Fig. 7).

Muscle atrophy is a constant effect of denervation (Fig. 8). This may not always be in proportion to the seriousness of the lesion. Certain irritative le-

**Fig. 10 (left).** Paralysis of the right deltoid muscle with marked atrophy. The patient is able to elevate the arm to a right angle by substitutionary action of the trapezius, pectoralis major and supraspinatus muscles.

**Fig. 11 (right).** Paralysis of the right deltoid muscle. The patient is able to elevate the arm to a right angle through substitutionary action, as is shown in Fig. 10. Such substitutionary action for the deltoid was rarely seen among the patients of the First World War, but it is perhaps more common than was formerly believed.
sions without serious direct structural damage, particularly in cases of slight blows to the ulnar at the internal condyle, may produce marked atrophy from a traumatic neuritis. Muscle atrophy may result in permanent residual disability, even though a satisfactory suture of a divided nerve has been carried out. This is particularly true in the intrinsic muscles of the hand and regardless of how early nerve suture is done, a certain degree of atrophy remains as a permanent stigma of ulnar nerve lesions.

In nerve wounds of warfare the diagnosis may be more complicated because of multiple lesions of a single nerve. To determine the presence of various lesions at different levels requires a long operative exposure of the nerve trunk for diagnostic purposes.

The diagnosis of traction lesions of a nerve trunk is particularly difficult insofar as determining the extent or level of the neural damage. A traction lesion is most common in the brachial plexus around the shoulder joint, in the external popliteal, and in the musculospiral from fracture of the humerus with wide dislocation of the fragments. In civilian practice, injury to the brachial plexus from blows or falling on the shoulder is a frequent lesion. These lesions, unfortunately, are generally avulsion of the cords of the plexus from the spinal cord. The lower cords of the plexus often remain intact, enabling the patient to make hand movements but without power of movement of any larger segment of the extremity. No form of surgical treatment can benefit avulsion injuries of the brachial plexus.

Investigation of sensory loss following peripheral nerve injuries forms an important part in the diagnosis but it is of secondary importance, in the writer's opinion, to the interpretation of disturbed motor function. There has been much discussion and difference of opinion among neurologists as to the sensory defects following peripheral nerve injuries. Important contributions on the subject have been made by Pollock, who has studied carefully the overlapping of uninjured nerves on the field primarily supplied by the injured nerve. There appears to be an attraction of the denervated skin field for nerve fibers from the surrounding uninjured nerves so that the original field of lost sensation may become very much reduced. The sensory examinations are time-consuming and require much patience on the part of both the examiner and the patient. In view of the fact that intelligent cooperation of the patient is essential to accurately chart sensory loss, the usefulness of this procedure is somewhat limited in many cases.

Reference should be made to studies in electrical skin resistance upon which valuable work has been done by Curt P. Richter of Baltimore. When the peripheral nerve is divided, sweating soon disappears from the area of denervation. The skin in this field then becomes an area of high electrical resistance which is determined by an ingenious apparatus. In order to carry out the test successfully the patient should not be cold and if the examination is conducted with the room temperature low, the patient should be given 1/10 grain of pilocarpine to produce sweating. It will then be possible, according to Richter, to map out within ½ inch a line of demarcation between
the denervated high resistance and the normal field in which the resistance is low. The advantages of this method are at once obvious. It is an objective test which may be carried out without the cooperation of the patient and this may be particularly important in patients with associated head injury. It will be useful in determining between functional or malingering cases and organic lesions. It may also be important in determining the recovery of function, since recovery of sensation and skin resistance coincide to a large extent. I am indebted to Colonel Spurling and Major D. L. Rose for permitting me to observe a demonstration of this test in ulnar nerve lesions at the Walter Reed Hospital. Unfortunately, the apparatus for carrying out these tests is not generally available.

A simple test which was thought to have a considerable degree of infallibility in the early stages of nerve reconstruction in the First World War was called the Tinel's sign. By starting some distance peripherally from the site of the injury and gently tapping the skin upward over the course of the nerve toward the injury, a tingling or formication was produced as soon as the down-growing regenerating fibers were percussed. It was thought by this method one could determine whether the lesion was recovering spontaneously and, if so, the level reached by regenerating axones from the central stump. However, the value of the test was considerably discounted in many cases in which the Tinel's sign showed favorable progress of the nerve and later exploration disclosed the nerve completely separated without the slightest chance of spontaneous regeneration. Perhaps the impact from the stimulated area went through scar tissue to the proximal segment and was responsible for the misleading information. It may be that the Tinel's sign would be of some value after suture of the nerve to determine whether regeneration is proceeding satisfactorily. On the whole, however, the writer's experience with the Tinel's sign is not productive of any great amount of confidence in the test.

Reference should be made to electrodiagnosis. The injuries that present the greatest problems as to treatment are those in which there is complete reaction of degeneration. Electrodiagnosis in its usual application is merely confirmatory of the more important clinical history and findings. Study of the nerve at exploration by the faradic current applied to the nerve above the lesion may give important diagnostic information. If application of the electrode above the injury produces contraction of the muscle supplied by the nerve, obviously one would not remove a neuroma in continuity. However, if there is no response in the muscle, the neuroma is a hard resistant one and there is complete sensory and motor loss in the domain of the nerve, the nerve should be regarded as incapable of spontaneous recovery.

Division of peripheral nerves is rarely followed by pain due to the nerve lesion. Patients with severe partial lesions, however, often develop intractable pain. This condition was described in the studies of patients in the War between the States by Mitchell, Morehouse and Keen and is called causalgia. Patients with this pain may sweat profusely in the field supplied by the in-
jured nerve. The pain is constant and burning. Causalgia is likely to be found more often in the median and the internal popliteal. The diagnosis of causalgia due to vasomotor disturbance may be made by injection of the appropriate sympathetic ganglia with novocaine. This should temporarily abolish the sweating and relieve the pain.

Some confusion in diagnosis of peripheral nerve lesions may result from the presence of ischemic myositis or Volkmann’s paralysis. The patient has lost sensory and motor power but fibrosis of the muscles is at once apparent on palpation and loss of sensation does not follow nerve distribution. The differentiation between nerve lesions and ischemic myositis should not be difficult but the two conditions may be associated.

A voluminous literature has accumulated on the study of nerve regeneration. There is general agreement as to the more important aspects of regeneration of divided nerves with almost unanimous endorsement of the Wallerian theory. When a nerve is divided the distal segment, through disconnection with the cell body, degenerates and in this process the axis cylinders disappear. Conducting tubes are formed by the Schwann cells in the distal segment and these cells attempt to bridge the gap between the divided segments of the trunk. Efforts of these cells, however, are obstructed by scar tissue formation between the segments so that the gap is filled by scar and is an impermeable barrier to regeneration. The dominant process of regeneration is inherent in the proximal segment of the divided nerve. This segment degenerates proximally for a few millimeters. The axis cylinders of the proximal segment by retaining their connection with the cell body, under favorable circumstances grow into the pathways provided by the process of degeneration in the distal segment. The down-growing axis cylinders, through the distal segment, ultimately reach the peripheral field if there has been accurate and careful coaptation of the divided nerve ends with minimal amount of scar tissue at the point of nerve suture.

There are inherent defects of regeneration which should be mentioned. Regardless of the care taken in approximating nerve segments and how successful the surgeon may be in preventing rotation of the nerve, certain fibers originally intended for one domain of the nerve may pass into another, and motor fibers may enter sensory pathways and vice versa. The fact that the number of down-growing axis cylinders from the proximal stump in the process of regeneration is greatly increased, enables many of the axis cylinders to find their proper terminals. There is believed to be branching of axones in nerve regeneration so that one axone originally intended for one part of the nerve field may supply several areas. This axonal branching, together with the misdirection of fibers, imposes certain apparently insurmountable limitations on the results of surgical treatment of divided nerves.