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PRACTICAL CONSIDERATIONS IN THE DIAGNOSIS OF PERIPHERAL NERVE INJURIES, WITH SPECIAL REFERENCE TO COMPENSATORY MOVEMENTS

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I. INTRODUCTION.

II. FACTORS CONCERNED IN THE DIAGNOSIS OF PERIPHERAL NERVE LESIONS.

- A. Slight Motor Function Distinguished from Complete Paralysis.
- B. Relative Value of Various Observations.

- 1. Trophic Phenomena.
- 2. Electrical Reactions.
- 3. Sensory Changes.
- 4. Motor Loss.
  - (a) Tendon Palpation.
  - (b) Observations of the Muscle.
  - (c) Joint Action.

III. PRODUCTION OF COMPENSATORY MOVEMENTS.

- A. Compensatory Movements Produced by Momentum.
- B. Gravity as a Factor.
- C. Relaxation of Functioning Muscles.
- D. Contraction of Nonparalyzed Muscles.
- E. Elimination of Compensatory Movements by Proper Methods of Examination.

IV. LESIONS OF INDIVIDUAL NERVES CONSIDERED IN REFERENCE TO COMPENSATORY MOVEMENTS AND OTHER PHENOMENA.

- A. Circumflex Nerve Lesions.
- B. Musculocutaneous Nerve Lesions.
- C. Musculospiral Nerve Lesions.
- D. Median Nerve Lesions.
- E. Ulnar Nerve Lesions.
- F. Ulnar Combined with Median Nerve Lesions.
- G. Sciatic Nerve Lesions.
- H. External Popliteal Nerve Lesions.
- I. Internal Popliteal Nerve Lesions.

V. CONCLUSION.

I. INTRODUCTION

During the year of its active existence at Cape May, there passed through the U. S. Army General Hospital No. 11, at which the authors were stationed, 617 cases of peripheral nerve wounds. When the entire neurosurgical staff was transferred to Fox Hills, Staten Island, N. Y. (U. S. Army General Hospital No. 41), a large number of additional peripheral nerve cases were observed. These cases were studied with considerable care, and the findings fully recorded on forms especially prepared by the members of the Cape May neurosurgical staff. These forms were later adopted throughout the army for use in peripheral nerve hospitals. Shortly after admission each patient was examined by his ward surgeon and discussed at a conference of the neurosurgical staff. Re-examinations were made and the patients reviewed at conferences at intervals of from one to two months. In many instances patients have been operated on and the clinical diagnosis studied at this time in the light of the operative findings. By this procedure errors in diagnosis have been reduced to a minimum. The average case has remained under observation over three months so that ample opportunity for study has been afforded. The patients have all reached us some time after the occurrence of the wound, however, so that the observations which follow must not be taken to apply to early nerve wounds, but to injuries of over a month's standing.

As a result of these studies, some points have been noted which have not been sufficiently emphasized, or have escaped mention in descriptions of typical paralyses from nerve wounds. This paper will deal with this practical aspect of the subject rather than attempt a systematic discussion of nerve injuries, or the setting forth of debated theories.

II. FACTORS CONCERNED IN THE DIAGNOSIS OF PERIPHERAL NERVE LESIONS

A complete diagnosis should include the following points:

1. Identification of the nerve injured.
2. Description of the level at which the lesion occurs.
3. A statement of the degree of functional loss in the injured nerve.
4. Some note as to the status of recovery.
5. An estimate of the pathologic condition.

A. Slight Motor Function Distinguished from Complete Paralysis.—The first and second parts of the diagnosis generally present little difficulty. The fourth point is merely a matter of comparing the degree of function found at different times. The fifth (to determine the pathology existing in the nerve) is frequently impossible before operation. We are, therefore, particularly concerned with the third part of the diagnosis, for on this depends the whole question of

treatment. It is nearly always possible to distinguish between complete loss of function, and the first faint traces of functional return. For correct observation on this point the patient looks to his surgeon to shield him from months of delay on the one hand, and unnecessary operation on the other.

B. Relative Value of Various Observations.—Although we have studied this problem from almost every angle, in the hope of finding some sure guide in the diagnosis of nerve injuries, experience has taught us to depend largely on the simpler diagnostic signs as being the most reliable. In the order of importance these may be rated: (1) voluntary movement; (2) sensation; (3) electrical reactions, and (4) trophic phenomena.

Fig. 1.—The typical hand of ulnar paralysis. The interosii are wasted, and the little and ring fingers flexed.

1. *Trophic Phenomena.*—First, it has been interesting to note that the trophic disturbances—the least important diagnostic sign—often enable one to make the diagnosis the instant the patient enters the room. So characteristic is the hypothenar and interosseous muscle atrophy in the ulnar cases, and the thenar atrophy in median injuries, that by examining plaster casts of the hands, one can pick out the median or ulnar cases nine times out of ten, provided brachial plexus lesions are excluded (Fig. 1). The trophic condition of the hand is, however, of very little value in showing the early signs of returning function. Indeed there may be the most pronounced degree of atrophy of the interosii, and yet all movements be executed with the intrinsic hand muscles apparently as well as though the hand were normal (Fig. 2).

2. *Electrical Reactions.*—Electrical examination has proved a disappointment to surgeons as it has failed to determine conclusively the degree of nerve injury or of nerve recovery. In two ways however, it is of real value. The faradic current generally settles quickly and definitely the differential diagnosis between a functional and an organic nerve lesion. If there is any question of the existence of a functional basis for a paralysis, and faradic stimulation gives a response in the supposedly paralyzed muscles, it is most unlikely that the paralysis is due to organic changes. Another important use of the electric current is in determining whether a muscle which now contracts, was once paralyzed. It has been observed that a recovering muscle often gives a slow, wavy response to galvanic stimulation, with

Fig. 2.—Ulnar atrophy in a case of ulnar paralysis in which function was returned in all ulnar muscles.

a tendency toward a reversal of formula, even after it contracts voluntarily and responds to the faradic current. Therefore such a response as this, in a muscle which exhibits voluntary contraction, affords evidence that there was once paralysis. Attempts to determine the degree or nature of the injury by means of the electric current have, however, resulted in disappointment, since the familiar reactions of degeneration are frequently present in connection with spontaneous recovery, and may not be exhibited in their entirety in cases where the nerve has been completely severed.

3. *Sensory Changes.*—The sensory examination is of considerable value, but unless each examination is made by the same observer, or with standardized instruments, the margin of error is apt to be wide.

In routine examinations for impairment of sensation, all but three sensory tests have been discarded. Pressure, pain and tactile sensation are tested, the esthesiometers devised by one of the authors (S. D. I.) being found the most satisfactory (Fig. 3). These esthesiometers were adopted as part of the neurologic equipment of the army peripheral nerve hospitals. Standard charts are used, and loss of deep pressure sense is shown by solid black. Tests for this loss are made by an esthesiometer which exerts a pressure of 1,000 gm. over an area of 0.25 sq. cm. Loss of pain sense is recorded by means of stippling within the anesthetic area which has been outlined on the chart. For this test an esthesiometer is used by means of which a pressure of 15 gm. is exerted on a sharp needle. Superficial tactile sensation is tested by means of a soft artist's brush, so trimmed that pressure over the most delicate portion of the skin does not cause visible depression of the epidermis. Loss of superficial tactile sensa-

Fig. 3.—Esthesiometers devised by one of the authors, and used in the army peripheral nerve hospitals. The instrument above tests deep sensation, that below tests superficial pain sensation.

tion is charted by means of hatching within the outline of the anesthetic area. Even when thus reduced to their simplest terms, sensory examinations require more time than all the other examinations combined; moreover, the sensory is of less value than the motor examination except in paralyses of purely sensory nerves, and in certain cases where returning sensation is the first evidence of recovery or regeneration.

The test described by Tinel, consisting of distal tingling on percussion over a regenerating nerve trunk, has been found to be of confirmatory value.

Another phase of the sensory examination deserves mention, namely, spontaneous pain. When this pain is severe and burning in character and is increased by touching the skin over the painful area, it is known as *causalgia*. This symptom is considered as evidence of an irritative lesion involving the sympathetic nerves. The presence

of causalgia, however, by no means precludes the possibility of an anatomic division of the nerve. In two of our cases causalgia was observed shortly after suture of the entire sciatic nerve.

4. *Motor Loss*.—Most important is the examination of motor function, just as motor paralysis constitutes the greatest part of the patient's disability from the standpoint of utility. Three observations are possible in testing the motor function of muscles: the effect of muscular contraction on the tendon, the effect on the joint and the effect on the muscle itself.

(a). *Tendon Palpation*.—Observing the effect of muscle contraction on the tendon by palpation is the most accurate method when it can be utilized. The observer may, however, be misled by the tightening of the tendon due to a change in the position of the joint, the resulting passive tension on the tendon being mistaken for the effect of muscle action. To eliminate such errors it is well so to fix the joint that no movement can occur, and then to have the patient attempt to move the joint. A tendon can in this way be made to stand out so as to be readily perceived, provided its muscle contracts. The factor of passive pull has been eliminated because the joint is immobilized.

(b). *Observations of the Muscle*.—We may dismiss with a few words the subject of observation on muscles. It is generally most unsatisfactory to attempt to see or feel the contraction over the belly of a muscle. Muscles in the vicinity, if they contract, will give an erroneous impression of action in the paralyzed muscle, while a slight true contraction will pass unnoticed. Exceptions to this rule are found in the muscles of the thenar and hypothenar groups, the supinator longus and the deltoid. Palpation and inspection of these muscles furnish very valuable evidence of muscle function.

(c). *Joint Action*.—The observation of joint movements is of great value in testing the function of muscles, but the interpretation of this observation is apt to be difficult, and erroneous conclusions may easily be drawn. For example, we might conclude that a patient necessarily had musculospiral function because he could extend the wrist. This is by no means the case. Probably all who have had an opportunity to watch patients with musculospiral lesions have seen many who obtain a certain degree of wrist extension, the musculospiral nerve being completely severed (Fig. 4). Extension is here produced by the action of muscles ordinarily not concerned in this movement. Actions of this type are generally designated as compensatory movements, and are particularly prone to occur in patients who have had paralysis for a long time, patients who have received special training in the use of their muscles, or patients in whom muscle contractures have occurred. Compensatory movements may be due either to the

contraction of one set of muscles or to the relaxation of another. They may also be due either to the factors of gravity or momentum.

III. PRODUCTION OF COMPENSATORY MOVEMENTS

A. *Compensatory Movements Produced by Momentum.*—To understand the part which momentum plays in the production of compensatory movements, one may observe the patient with a fracture of the patella and separation of the fragments. He obtains extension of the knee by a swinging movement, thus substituting momentum of the leg for contraction of the quadriceps extensor muscle. The patient with musculospiral paralysis uses momentum in a similar manner, and frequently acquires considerable facility in extending the wrist by giving the hand a quick backward flip. For this reason joint action should not be taken as evidence of muscle contraction unless the movement is carefully analyzed by the observer, and the part so immobilized as to eliminate the momentum factor.

Fig. 4.—A case of complete musculospiral paralysis extending the wrist. This is accomplished by winding up the tendons of the extensor muscles (which have undergone contracture) by flexing the fingers.

B. *Gravity as a Factor.*—Gravity must also be considered as a means of producing compensatory movements. In musculospiral paralysis, the limp wrist which cannot be extended against gravity, falls into the position of extension if the hand is turned in supination. In order that the test may be perfectly reliable in showing all the motion that the patient is able to make, and at the same time rule out confusing movements due to gravity, the hand should be held midway between pronation and supination—the zero position. What is true of the wrist is also true of other joints; tests for muscle action should always be made with the joint in the zero position.

C. *Relaxation of Functioning Muscles.*—Far more confusing, however, than the movements produced by momentum or gravity, are those produced by other muscles which simulate the action of the paralyzed ones. Let us assume that we have a case of complete musculospiral paralysis with shortening in the extensor muscles, so that the hand naturally assumes the position of extension instead of wrist drop. If one were to grasp the hand and cause the wrist to flex, the hand would spring back into its extended position as soon as it was released. The patient can obtain the same result by the action of the flexors. These muscles pull the hand down, and when relaxation occurs, the hand again assumes the attitude of extension. When this movement is seen for the first time it gives a very misleading impression of extensor action, whereas the whole movement is really due to the flexors. Since the extension of the wrist is due to relaxation of the flexors instead of contraction of the extensors, it may be designated as a compensatory movement of relaxation. To eliminate this possible source of error, the flexors should be relaxed before the movement of extension is attempted. This is best accomplished by having the patient flex the wrist, slowly relax and then attempt slow, steady extension. No further movement of relaxation can occur under these conditions because the flexors are already completely relaxed. Any further movement of extension would therefore be evidence of musculospiral action, provided the relative position of the fingers and hand remains unchanged. The reason for this provision will be considered in the next paragraph.

D. *Contraction of Nonparalyzed Muscles.*—Let us again assume that we have a complete musculospiral paralysis, with contractures in the extensors of sufficient degree to prevent the hand from falling into the wrist drop position. If the examiner were to grasp the fingers and flex all of the phalanges until the hand assumed the position of a tightly closed fist, the extensor tendons, running over the summits of the knuckles, would be made more tense and would prevent the fingers from flexing unless some change in joint position could be secured which would lessen the tension. The wrist joint is the only one free to move which could release this tension. Extending the wrist joint tends to relax the extensors. This movement must therefore occur to allow the flexion of the fingers when the extensors have undergone contracture; and, indeed, this movement does occur when a patient with such contractures attempts to close the fist tightly, so that we have the curious spectacle of a man with musculospiral paralysis extending the wrist by flexing the fingers (Fig. 4). This type of movement may be characterized as a compensatory movement of contraction. On account of this movement of substitution, while testing

for wrist extension as an evidence of musculospiral action, the relative position of the fingers and the hand must remain unchanged.

E. Elimination of Compensatory Movements by Proper Methods of Examination.—It would therefore appear, from a study of the various factors concerned in compensatory movements of the wrist, that: (1) The forearms should be immobilized to eliminate the factor of momentum, and the movement should be made slowly. (2) The hand should be in a position midway between pronation and supination to eliminate the factor of gravity. (3) Before testing extension, complete relaxation of the opposing muscles should be secured. This is best accomplished by having the patient first flex the wrist, then relax and finally attempt extension. (4) The relative position of the fingers and the hand should remain constant.

What is true of the wrist is also true of other joints, and the same precautionary measures should be taken in all cases to eliminate compensatory movements. In general, the part should be immobilized proximal to the joint, and the movement made slowly. A zero position should be secured to eliminate the factor of gravity, and complete relaxation should be secured in the opposing muscles. No movement in the joints distal to the one under examination should be allowed. Failure to observe these measures when a well marked compensatory movement exists will almost certainly lead, sooner or later, to errors in diagnosis.

IV. LESIONS OF INDIVIDUAL NERVES CONSIDERED IN REFERENCE TO COMPENSATORY MOVEMENTS AND OTHER PHENOMENA

The general principles governing compensatory movements have been discussed. These movements will now be studied in relation to the nerve lesions which have most commonly presented themselves.

A. Circumflex Nerve Lesions.—The circumflex nerve is frequently involved in shoulder injuries. In such cases an anesthetic area is found over a portion of the deltoid muscle, and generally the arm can be only slightly abducted from the side unless the scapula is rotated. Occasionally, however, a patient, without apparent contraction in the deltoid muscle, and with everything including sensory and electrical tests indicating a complete lesion of the circumflex nerve, is able to raise the arm to a horizontal position before the scapula commences to rotate, and considerably higher when aided by rotation of this bone. In the cases we studied at Cape May, this movement was due to supraspinatus action. The supraspinatus muscle, however, is not situated advantageously for producing abduction, and without the deltoid the shoulder usually cannot be abducted to any marked degree.

B. *Musculocutaneous Nerve Lesions*.—Musculocutaneous paralysis results in loss of sensation over a portion of the forearm, and weakness in supination and elbow flexion. Although the brachialis anticus and biceps are completely paralyzed, there is not complete loss of any movement of the elbow because the supinator longus, a powerful flexor of the elbow which is innervated by the musculospiral, partially substitutes for the muscles supplied by the musculocutaneous

Fig. 5.—Patient with musculospiral and musculocutaneous paralysis with muscles relaxed.

when it is paralyzed. When both the musculocutaneous and the musculospiral are injured, one would expect to find flexion of the elbow and supination abolished. This is often the case. Supination was abolished in all of our cases, but in a few instances flexion of the elbow was obtained through a compensatory movement of contrac-

tion by the pronator radii teres, which is supplied by the median nerve. This muscle, having its origin at the internal condyle of the humerus, and its insertion in the shaft of the radius, is so situated that it does not act with sufficient mechanical advantage to produce strong flexion; but when a patient has trained his power of flexion by exercise, a sufficiently strong contraction may be obtained to flex the elbow to 90 degrees (Fig. 5), and to lead the patient (and perhaps also the physician) to believe that function is returning in the injured nerves. Palpation over the supinator longus and biceps and all other tests at our disposal will, however, show these muscles to be completely paralyzed, whereas the pronator radii teres will be found to contract strongly while the elbow is being flexed. Pronation accompanies the movement. Thus, we find that the musculospiral nerve always partially compensates for the loss of function of the musculocutaneous, and that even when both of these muscles are paralyzed slight compensation is occasionally secured through the pronator radii teres.

C. *Musculospiral Nerve Lesions*.—Loss of musculospiral function results in inability to extend the elbow, wrist and thumb. The metacarpophalangeal joints of the fingers cannot be extended. Besides this there is found, as in the case of musculocutaneous paralysis, weakness of supination and elbow flexion, without complete loss of either, the biceps supplementing the supinator longus and brevis in these movements. It is sometimes difficult to tell whether action in the supinator longus is absent or merely weak, since contraction of the biceps tends to pull on the fascia of the antecubital space, giving an erroneous impression of muscle contraction in the supinator longus. In such cases it is well to fix the forearm by holding the wrist, and then to have the patient attempt to flex the elbow while the palpating finger is applied on the supinator longus, over the shaft of the radius, several centimeters below the insertion of the biceps tendon. At this point any contraction of the supinator longus may readily be discerned.

It is to be noted that in musculospiral paralysis, extension of the interphalangeal joints of the fingers is possible, being accomplished by the interosii and lumbricales (Fig. 6). This movement in some cases is so marked as to simulate musculospiral action. Errors of interpretation from this source may be avoided by carefully observing the metacarpophalangeal joints. No true extension will be found if the case is a complete musculospiral injury.

Low lesions of the musculospiral may be overlooked because of their failure to produce wrist drop. Extension of the wrist is secured by the action of the extensor carpi radialis longior. The branch from the musculospiral to this muscle comes off well above the elbow; therefore a lesion below this point cannot produce wrist drop.

Compensatory movements in musculospiral paralysis in their bearing on the wrist joint have already been discussed, and nothing further need be added. If the examiner is on the alert for these movements and takes the precautions which have been suggested to eliminate compensatory movements of contraction and relaxation, and the factors of gravity and momentum, he should have little difficulty in securing an accurate estimate of the degree of musculospiral loss.

Fig. 6.—Same patient with flexion of the elbow obtained by action of the pronator radii teres.

D. Median Nerve Lesions.—Loss of function in the median nerve, besides resulting in loss of sensation over a portion of the hand and fingers, produces loss of thumb flexion and thumb abduction. Next in importance is the marked weakness in flexion of the index finger; there is also weakness in pronation, wrist flexion, apposition of the thumb against any other finger and in flexion of the fingers. No commonly seen peripheral nerve injury affords greater difficulty in diagnosis than does the median, partly on account of the tendency of the

musculospiral and ulnar sensory functions to overlap the median, but particularly on account of the large number of compensatory movements that patients with median paralyses display. When the median nerve is severed, pronation may be begun by the action of the supinator longus and finished by the action of gravity so that an almost normal range of movement is secured without the action of the pronator radii teres and pronator quadratus muscles. Wrist flexion is likewise secured by the synergic action of the flexor carpi ulnaris, extensor ossis metacarpi pollicis and that portion of the flexor profundus digitorum which is supplied by the ulnar nerve. Because of this partial ulnar supply to the flexor profundus digitorum, flexion of the fingers is not lost in median lesions.

Flexion of the thumb may occur through a mechanism of relaxation to which we have already referred, this movement being dependent on the pull of the shortened flexor longus pollicis in the relaxation period following the action of the extensors. Flexion may also be secured by wrist extension when the flexors are shortened. This is another example of a compensatory movement of contraction. Apposition of the thumb to the fingers may be secured by the synergic action of the adductor pollicis, the deep head of the flexor brevis pollicis and the extensors of the thumb.

Abduction of the thumb may be secured in the absence of the abductor pollicis by the combined action of the deep head of the flexor brevis pollicis, which is supplied by the ulnar nerve, and the extensors of the thumb. This movement of substitution is not common, and is generally incomplete, so that loss of thumb abduction is one of the best signs of the absence of median motor function in the hand. A convenient and practical test for this function is to have the patient lay the hand, back down, on a table and attempt to point the thumb straight upward; or the examiner may place the patient's thumb in this position. Inability on the part of the patient to maintain this position of the thumb is evidence of loss of median action.

E. Ulnar Nerve Lesions.—Ulnar lesions produce the well-known anesthetic area with a fair degree of constancy. This furnishes an important diagnostic sign, and constitutes a leading factor in the production of the disability which results from such injuries. Destruction of the ulnar nerve results in some cases in surprisingly little motor loss which cannot be compensated for by muscles supplied by other nerves. Generally, the only motion completely absent is abduction and adduction of the fingers.

Flexion of the fingers, with the exception of the thumb, is provided for by the combined action of the median and ulnar nerves. Finger flexion is present, though perhaps weak, in paralysis involving

either one or the other of these nerves. Bearing in mind that the portion of the flexor profundus digitorum, which has its insertion into the little and ring fingers, is supplied by the ulnar nerve alone, one would expect that in ulnar paralysis the terminal phalanges of the little and ring fingers could not be flexed. This is not the case as a rule, because apparently all of the tendons of the deep flexor muscle are acted on to some degree when either the median or the ulnar portion contracts; hence the little and ring fingers are usually flexed in their terminal portions when the index and middle fingers flex. A fairly satisfactory test for function in the ulnar portion of the flexor profundus digitorum is obtained as follows: Completely immobilize the index and middle fingers, and immobilize the little and ring fingers up to the terminal phalanx; then have the patient attempt flexion of the fingers. In the absence of ulnar function, the little and ring fingers cannot be flexed in their terminal phalanges.

In ulnar paralysis the loss of the flexor carpi ulnaris may be compensated for by the action of the extensor carpi ulnaris. This muscle pulls the hand toward the ulnar side, while flexion of the entire wrist is attained through the action of the median flexors of the wrist.

Loss of the lateral movements of the fingers is probably the most distinctive sign of ulnar paralysis. Strangely enough, however, the extensors may be trained to produce lateral movements of the fingers, probably because the extensor tendons do not usually run directly across the apices of the knuckles, or if they do, they are inserted laterally into the phalanges. The action of the lumbricales also tends to produce lateral movement of the fingers. The lateral action which is sometimes found in the little and ring fingers in ulnar paralysis cannot, however, be explained in this manner. A useful method for overcoming this compensatory movement is to put the extensor tendons under tension by flexing the wrist and fingers before attempting the movement of spreading the fingers. Another useful procedure is to palpate the hypothenar eminence while the little finger is abducted. The abductor minimi digiti contracts if ulnar action is present to any marked degree, and the skin over the hypothenar eminence wrinkles and dimples inward as the muscle contracts. The latter action is due to the contraction of the palmaris brevis which is supplied by the ulnar nerve and inserts into the palmar fascia.

F. Ulnar Combined with Median Nerve Lesions.—When ulnar and median lesions coexist, flexion of the wrist and fingers is no longer possible except when compensation is secured by means of the factors already discussed. The extensor ossis metacarpi pollicis furnishes the most important action in this respect; a surprising degree of flexion is often obtained by contraction of this muscle (Fig. 7).

Fig. 7.—A patient with complete musculospiral paralysis extending the interphalangeal joints by means of the interossei and lumbricales.

Fig. 8.—Flexion of the wrist by means of the extensor ossis metacarpi pollicis. The case is one of complete ulnar and median paralysis.

G. *Sciatic Nerve Lesions*.—Lesions of the sciatic nerve when complete usually cause loss of all movements below the knee. A striking fact in this connection is that even when the lesion is found to be high on the sciatic nerve, frequently no paralysis of the hamstrings occurs. Another peculiarity of sciatic lesions is the tendency toward dissociation which they display. Lesions high in the thigh will often produce paralysis only in the internal or external popliteal supply, usually in the latter.

H. *External Popliteal Nerve Lesions*.—In lesions of the external popliteal nerve one finds loss of sensation over the lateral aspect of the foot and leg, inability to evert or extend the foot, and loss of extension in the metacarpophalangeal joints. As in the hand, the distal phalanges can be extended even in the presence of a paralysis of the extensor muscle, the extension being accomplished through the agency of the interosii and lumbricales. All of the types of movements of compensation found in the upper extremity may occur, and this source of error may be overcome by the means already described. Because movements of the toe and ankle joints tend to be more restricted than finger and wrist movements, tendon palpation is a more valuable guide to muscle contraction in the lower extremity than joint action. Before the toes or ankle commence to extend, it is often possible to feel the extensor tendons become taut where they run beneath the anterior annular ligaments of the ankle.

I. *Internal Popliteal Nerve Lesions*.—Internal popliteal lesions are accompanied by anesthesia on the sole of the foot. The motor disturbance consists in loss of plantar flexion of the foot and toes, absence of lateral movements of the toes and inability to evert the foot. A fairly common and somewhat confusing compensatory movement may occur. Some patients obtain plantar flexion of the foot by use of the peronei, which are supplied by the external popliteal nerve. When these muscles contract even when the internal popliteal nerve is completely paralyzed, there may result a diagonal tilting of the foot which may be mistaken for plantar flexion; true plantar flexion may even occur in cases where the peronei insert well back of the axis of rotation of the ankle. Other types of compensatory movements are found to some degree in connection with internal popliteal lesions, but if proper precautions are taken none will be likely to lead to an error in diagnosis with the exception of that to which we have just alluded. This confusing compensatory movement should be suspected if, in the presence of other evidences of internal popliteal paralysis, the patient obtains feeble plantar flexion of the foot without evidence of gastrocnemius contraction. In such cases palpation of the peronei will demonstrate contraction of these muscles, but the Achilles' tendon will be found

to relax instead of tighten with each downward movement of the foot. When these conditions exist, whatever plantar flexion is obtained is the result of a compensatory movement of contraction by the muscles supplied by the external popliteal nerve.

V. CONCLUSION

The list of compensatory and substituted movements might be prolonged indefinitely, but enough have been considered to emphasize the importance of careful observation to avoid errors in diagnosis. In examining the results of a large number of peripheral nerve lesions, the characteristic and classic symptoms will commonly be found; however, a certain number of cases will exhibit unusual phenomena. These atypical cases are the ones that offer the greatest difficulties in neurosurgical diagnosis.

It is hoped that sufficient has been said to show the inadequacy of simply learning a list of the classic symptoms as signs of nerve injuries. The fundamental requisites for accurate diagnosis of such cases include a thorough anatomic knowledge, a mastery of the mechanics of joint action and discriminating observation. With the application of these broad principles to neurosurgical diagnosis, the difficulties are minimized and the proper treatment can be confidently instituted.