PRINCIPLES OF NEUROLOGICAL SURGERY

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THE PRINCIPLES OF NEUROLOGICAL SURGERY

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PREFACE TO THE FOURTH EDITION

NEUROLOGICAL surgery has attained maturity as a surgical specialty in a comparatively short time. The first edition of this book was an attempt to make known to physicians and students the accomplishments possible in this field.

The wounded of World War II added a great deal to our knowledge of the traumatic aspects of neurological surgery by reason of the experiences of individual surgeons. It is to be hoped that organizational plans will be better laid to realize the utmost from combined and unified efforts in the event of a future catastrophe of such magnitude.

Again, I should say that this book does not include an exhaustive treatise upon each subject considered. I consider it unwise to include matters of surgical technique. If the student of medicine, regardless of age, is aided in obtaining a better concept of neurological surgery, the primary purpose will be achieved.

To the twelve men with whom I have been associated in their periods of training for neurological surgery, I wish to express my affection, respect and appreciation.

Dr. Hale Haven
Dr. John Martin
Dr. Joseph Tarkington
Dr. Moses Ashkenazy
Dr. Daniel Ruge
Dr. Max Ramirez
Dr. David Cleveland
Dr. George Perret
Dr. Frank Padberg
Dr. Stanton Goldstein
Dr. Robert Anderson
Dr. Nicholas Wetzel

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THE PRINCIPLES
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CHAPTER I

NEUROLOGICAL DIAGNOSIS

THE surgical treatment of diseases of the nervous system has reached the status of a mature surgical specialty. The teachings of a number of eminent neurologists, the wide sphere of experimental investigations and the contributions of many imaginative and ingenuous surgeons have made this possible in a relatively short period of time.

Many contributions have been made to the laboratory and surgical diagnosis of neurological diseases but collectively they cannot supplant the knowledge gained from physical and neurological examination.

Every doctor commonly encounters neurological cases in his practice, and the specialist in other diseases finds that many of his patients have neurological or neurosurgical complications. Invariably as a result of early teaching, the doctor may be confused, perplexed, bewildered and inarticulate when his patient presents symptoms of neurological significance. Often he concludes that the diagnosis of disorders of the nervous system is insurmountably difficult, that the prognosis is hopeless, and that treatment is hopeless.

This is not true provided one approaches the subject from the viewpoint of anatomy and physiology. If basic neuroanatomical and neurophysiological facts are applied, symptoms of diseases of the nervous system manifest themselves in a logical form. It should be remembered that it is impossible to memorize the signs, symptoms, and pathology of the various diseases of the nervous system without reference to those basic principles. The specialist must have a detailed knowledge of the nervous system, but the average physician need only remember a few of the long fiber tracts and some of their connections, the levels of reflexes, the levels of origin of cranial and spinal nerves, and the general form of the nervous system. These relatively simple, concise, practical facts may be readily acquired and mastered. They will provide an ample anatomical and physio-

logical approach to the understanding of the more common clinical symptoms which will be reflected in the accuracy and promptness with which the patient is referred to the neurologist or neurological surgeon for treatment.

An attempt will be made to contribute to the resources of the doctor to the end that he will be better able to advise his patients concerning the early existence of neurological and neurosurgical disease at a time when treatment is most effective. He will thus be equipped to give a more reasonable and logical prognosis and will be prepared to advise the patient about necessary and helpful therapeutic steps. It is obvious that since the clinical viewpoint of the average doctor is to be emphasized, a great mass of structural detail of importance to the neurologist will be omitted, and dogmatic statements may be made to which the neurologist will recognize exceptions.

One may begin to acquire a working knowledge of neurological diagnosis by directing attention to the *motor*, *sensory*, and *reflex* systems of the body.

MOTION

One of the most frequent symptoms encountered in neurological disorders is muscular weakness or paralysis. Paralysis of a single extremity is termed a *monoplegia;* of one-half of the body with the corresponding limbs, a *hemiplegia;* of both lower extremities, a *paraplegia;* and of both upper extremities, a *brachial paraplegia* or *diplegia*. There are two types of paralysis, *spastic* and *flaccid*.

Upon examination of the patient whose limbs are stiff and spastic, there will be found increased deep tendon reflexes, diminished or absent superficial reflexes, and certain pathological reflexes, such as the Babinski phenomenon. Atrophy of the muscles does not occur and if tested electrically, they will react normally to the faradic and galvanic currents. These symptoms occur as the result of an *upper motor neuron* lesion. In other words, the controlling influences of the higher lying cerebral centers have been removed from the lower reflex arcs. (Fig. 1.)

The *upper motor axons* conduct impulses from the large pyramidal cells of the cerebral motor cortex in the precentral gyrus to the motor nuclei of the cranial nerves, or to the cells of the anterior gray columns of the spinal cord upon the opposite sides, thereby forming the *corticobulbar* and *corticospinal* tracts. After arising from the giant pyramidal cells of the motor cortex, the corticospinal fibers occupy successively the rostral half of the posterior limb of the internal capsule, the intermediate three-fifths of the basis pedunculi, the basilar portion of the pons, and the pyramid of the medulla oblongata. At the caudal level of the medulla, the greater part of this tract crosses to the opposite side of the spinal cord where it descends in the lateral white columns and is known as the *lateral corticospinal* tract. The smaller uncrossed portion is continued directly downward upon the same side in the anterior white columns as

the *anterior corticospinal tract.* The fibers of the corticospinal tracts end about the motor cells of the anterior gray columns of the spinal cord. The corticobulbar fibers also arise from the large pyramidal cells of the motor cortex, pass through the knee of the internal capsule and the basis pedunculi, to end about the motor nuclei of the cranial nerves in the brain stem.

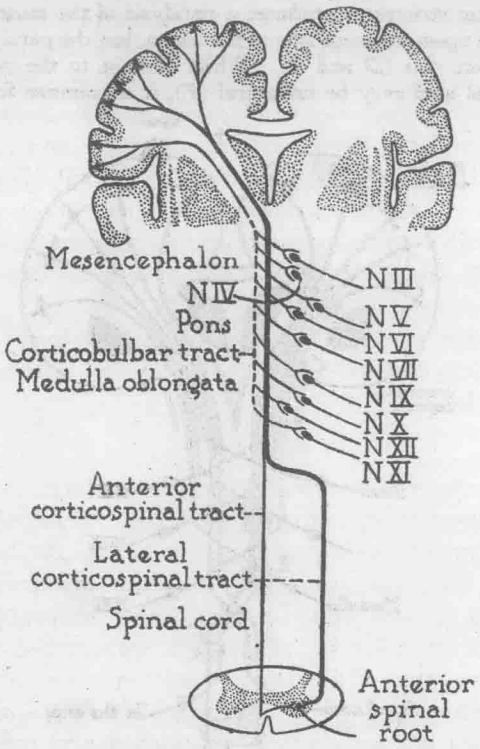

FIG. 1.—Diagram of the corticobulbar and corticospinal tracts which constitute the upper motor neuron.

The relations of the *upper motor neuron* pathway are shown in Figure 2. The entire motor cortex is not commonly destroyed by disease because of its rather widespread area. The more common minute lesions may give rise to a monoplegia (*A*). It will be remembered that the cortical representation of the motor centers are inverse in their position. If one pictures a man hanging over the side of the cerebral hemisphere with his toes hooked over the superior surface on to the medial aspect of the hemisphere and with his arms outstretched, there will be no difficulty in recalling the order of the motor centers. As the cortical fibers pass through the internal capsule, they are closely gathered together so that a lesion at that level may produce a paralysis of the opposite half of the body, including the lower portion of the face. The entire facial musculature would not be involved because the upper facial muscles receive fibers from the same side of the cortex, as well as from the opposite, and the former would remain intact (*B*). In the posterior limb of the internal capsule the sensory fibers to the opposite half of the body and the optic radiations are present in close association with the motor fibers. A lesion in such

a location would produce loss of cortical sensation over the opposite half of the body and blindness in the opposite half of the visual field in addition to paralysis of the opposite half of the body (*C*). Injury to the pyramidal fibers of the cerebral peduncles, pons, or upper part of the medulla produces a paralysis of the opposite half of the body and, in addition, may involve the corticobulbar fibers, the cranial nerve nuclei, or their emerging fibers. Involvement of the latter structures produces a paralysis of the muscles supplied by the cranial nerve upon the same side as the lesion, but the paralysis of the body is on the opposite side (*D* and *E*). While damage to the pyramidal tracts within the spinal cord may be unilateral (*F*), it is common for both sides to be injured (*G*).

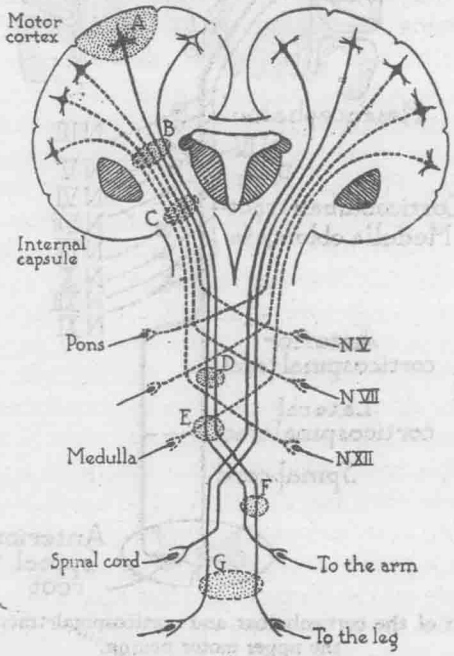

FIG. 2.—Diagram of the possible location of lesions which may affect the upper motor neuron.

In the *flaccid* type of paralysis, smaller groups of muscles are affected, and there is a loss of muscle tone, absent deep tendon reflexes, muscular atrophy, and the muscles do not respond to the faradic current while upon galvanic stimulation, their reaction is slow and serpentine. These symptoms are produced by a lesion of the *lower motor neuron*.

Lower motor axons arise from the cells of the anterior gray column of the spinal cord and from the cells of the motor nuclei of the cranial nerves. The fibers from the spinal cord emerge through the anterior spinal roots and end in the skeletal muscles, while those from the brain stem are contained in the cranial nerves and end in the striated muscles.

Lesions of the *lower motor neuron* occur in a very few locations. These are the anterior motor horn cells or their emerging fibers in the anterior roots, the nerve plexuses, the peripheral nerves, and the cauda equina of the spinal cord.[1]

There are a few practical points in making a neurological examination which will allow the examiner to detect the disorders of muscular function more easily and with more interest.

When gross weakness occurs, the examination is a relatively simple matter, but often the patient is able to perform all movements apparently with equal strength. Pollock has emphasized the importance of observing the apparently trivial signs which are present when gross methods of examination fail to reveal any motor disturbance.

As one observes a patient lying upon his back, the palpebral fissure may be wider, and spontaneous winking less frequent or absent on the affected side. The corneal reflex may not be elicited as effectually. The upper eyelid upon the paretic side may be lifted upward by the examiner more easily than upon the unaffected side; and whereas, the normal lid quickly resumes its normal position, the affected lid slowly and incompletely covers the eyeball. The difference in muscle tone in the upper eyelids can be seen and felt very definitely. A voluntary attempt to close the eyelids forcibly will produce greater wrinkling of the skin about the lids on the unaffected side.

The aperture of the naris on the paretic side is smaller, and the nasolabial fold is not as deep. When the patient shows his teeth, the affected side may lag slightly or the nasolabial fold may not increase in depth as it does on the normal side. A strong attempt to show the teeth is always accompanied by a strong contraction of the platysma muscle on the normal side, but this is often absent on the weak side. The facial movements should be observed carefully upon voluntary contraction and in response to emotional expression. Often no weakness can be observed when the patient's face is in repose or when the facial muscles are contracted voluntarily, and yet when he talks, cries, or laughs, the difference in strength is easily detected.

In the absence of a demonstrable weakness of an upper extremity, some clumsiness, defect in associated movements, greater fatigability, or tremor will be found on the affected side. With the patient lying on his back and with both of his upper extremities outstretched before him, the affected extremity will fall downward slightly or

[1] There are, in addition, paralyses which are due neither to an upper nor lower motor neuron lesion. These are the recurrent and transient palsies, such as those which occur in myasthenia gravis, intentional hypertonia, family periodic paralysis and cataplexy. These disturbances are of greater interest to the neurologist.

deviate outward or inward after a short time. If the patient is asked to abduct and adduct his fingers and thumb, with the upper extremity held outstretched, the paretic arm may be seen to move outward and inward with each movement of the fingers. On the affected side only one or two of the fingers may be moved, and often they are deflected ulnarward and are flexed to various degrees at the metacarpophalangeal joints. At times the movements are tremulous, and often the response to the command is disorderly. When making a fist and opening the hand upon repeated commands, the affected hand is closed to differing degrees. Upon the unaffected side the hand is extended at the wrist when the fingers are completely flexed, but this is not so on the affected side. Throughout both of these examinations the affected extremity continues to fatigue and gradually to sink downward. Associated movements of elevation and sinking of the arm at the shoulder are seen on the affected side. The patient may look at the affected hand while performing these tests because he is in a way conscious of the greater effort necessary to perform the movements.

With the upper extremities extended forward, the index fingers pointing at the foot of the bed, the index finger of the affected side deviates downward and to the ulnar side and at times the entire hand deviates similarly. When the patient is asked to raise the extremity and bring it down to the examiner's finger, the affected extremity does not move smoothly. The extent of the movement upward and downward will vary. The forefinger will be brought down on the examiner's finger with varying degrees of strength; whereas, on the normal side the finger will be brought to rest with precision and accuracy. When the unaffected extremity is raised, the affected one is slightly lowered, and when the unaffected one is lowered, the affected one is raised. While performing repeated lifting and lowering of the unaffected extremity, the outstretched affected one begins to sink slowly. (Fig. 3.) Gradually it may sink almost to the bed without the patient's perception. Rapid drumming of the index and middle fingers of the affected side is difficult and clumsy, and repeated apposition of the palm and dorsum of the hand to the thigh is slow and awkward. Touching the finger to the nose or finger to finger may bring out a slight clumsiness, uncertainty, or tremor.

The passively elevated and slowly released unaffected lower extremity is held in position for a considerable time. The affected side fatigues, sways, and is tremulous. Passive dorsal flexion of the normal foot is accompanied by an active contraction of the tibialis anticus muscle. This may not occur at all on the affected side; and if it occurs, the contraction is much less marked. Babinski's

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