THE PHYSIOLOGY AND PATHOLOGY OF MOTOR FUNCTION

By means of our motor system we move our bodies in space and the various parts of the body in relation to one another, maintain postures and attitudes in opposition to gravity and other external forces. All movements are effected by contractions of striated muscles through the control of the nervous system.

The examination of motor functions includes the determination of muscle power, an evaluation of muscle tone and bulk and the observation of abnormalities of movement. In order to understand the function of the motor system as a whole and to evaluate the changes in motor activity that occur in disease, one is justified in analyzing first the morphology and function of the constituent parts of the changes in each that result from pathologic processes. Then one can synthesize and correlate their functions. This does not mean that any one part ever acts individually; however, each has its own functions and its changes in disease processes, and an understanding of each part aids in comprehension of motor function as a whole.

THE LEVELS OF MOTOR FUNCTION

There are many varieties of movement; some of them are independent of consciousness, whereas others are directly under the influence of the will. Neurologists have referred to the various stages, or levels, of motor function, that are the following: 1) The spinomuscular level
2) The extrapyramidal level
3) The corticospinal (pyramidal) level
4) The cerebellar level
5) The psycho-motor level

These levels are components of the motor system as a whole. Disease of each is characterized by certain signs and symptoms.

t the time of birth simple spinal and brain stem reflexes have already been established, and more complex postural and righting reflexes appear during the first few weeks of life. Then, following the maturation of the cortex and commissural pathways, acts requiring associated sensory functions (grasping and groping) are possible. Still later volitional control of movement appears, and then the ability to perform skilled acts with a high degree of precision.

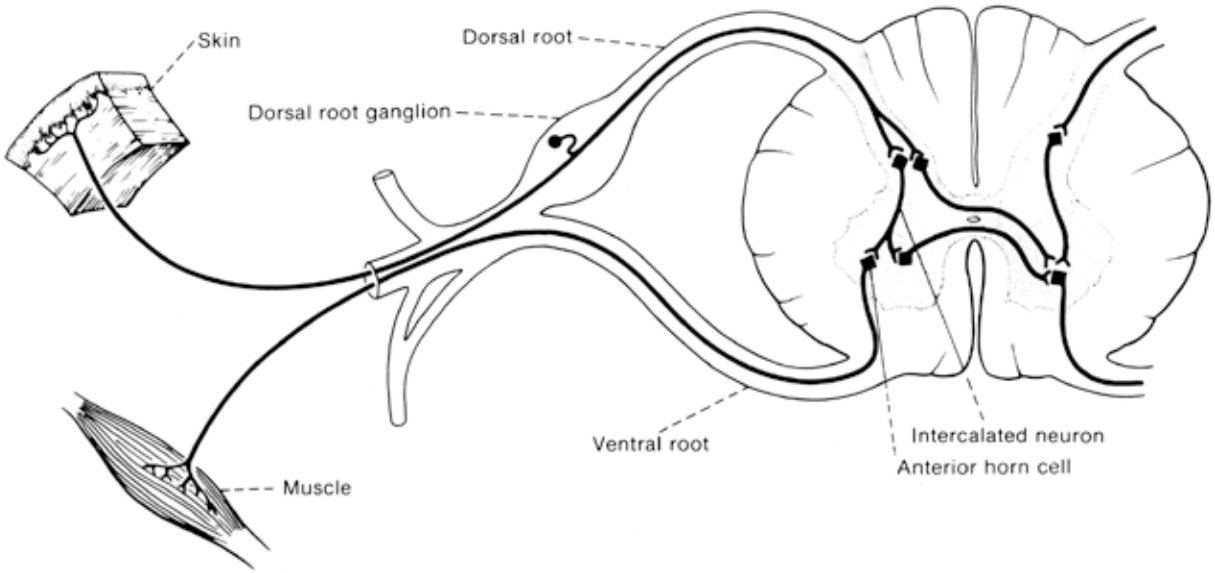
In this section the spinomuscular and corticospinal (pyramidal) levels will be discussed.

THE SPINOMUSCULAR LEVEL

The structure and function of striated muscles depend upon their connections with the central nervous system. The spinomuscular level of motor integration has, as its primary units, the motor nuclei in the anterior horn cells of the spinal cord and their homologues in the brain stem. Impulses which originate in these cells are carried through their neuraxes, the peripheral motor nerves, to the motor end-plates of the muscles. Sherrington, in 1904, described the anterior horn cell, from the point of view of function, as the “final common pathway”, through which all nervous impulses from higher centers must pass to reach the myoneural junction and influence striated muscle. The terms *lower motor neuron* and *primary motor neuron* are applied to this structural unit.

The primary functional unit of the spinal level, or of any motor level, is the *reflex*, or response to a stimulus. The essential unit of the reflex is a center of adjustment, together with the conductors necessary to connect this center with the appropriate receptor and effector apparatus. The receptor is the peripheral sensory ending; the primary conductor is the afferent nerve fiber; the center of adjustment is the motor unit with its associated synapses; the secondary conductor is the efferent nerve fiber, the neuraxis of the motor neuron; the effector is the muscle stimulated. There may be intermediate or interposed association and commissural neurons.

Fig. 1. Diagrammatic section through the spinal cord, showing the anterior horn cell and its afferent and efferent connections that make up the simple reflex arc.



In the spinal level one of the most primitive reflex arcs is found. The impulse is carried from a receptor in the skin, subcutaneous tissue, tendon, or periosteum, through the dorsal root ganglion and into the dorsal horn of the spinal cord, where there is a synapse and the neuron of the second order, or the intercalated neuron, carries the impulse to the anterior horn cell. On stimulation of this center of adjustment the motor impulse is relayed through its neuraxis, which goes by way of the ventral root of the spinal cord and the α-subgroup of the A fibers in the peripheral motor nerve to the motor end-plate of striated muscle, where it stimulates the muscle and causes it to contract. The arrival of the motor impulse at the nerve ending is associated with the liberation of acetylcholine, which causes depolarization of the muscle membrane at the neighboring end-plate. This, in turn, starts a wave of excitation with resulting contraction of the muscle fibers.

The α-motor neuron in the anterior horn of the spinal cord is the terminal neuron of the voluntary motor system. It supplies the main large muscle fibers, and its neuraxis goes directly to the motor end-plate, or neuromuscular junction.

The γ-motor neurons in the anterior horn area are smaller in diameter than the α-motor neurons and are efferent to small, specialized fibers in the muscle spindles, *the intrafusal or fusiform fibers*. These latter in turn act as receptor organs, and stimulation of them causes afferent impulses to be sent to the anterior horns to initiate contraction of the muscle by reflex action of the α-motor neurons, thus producing the muscle stretch reflex. Special interneurons within the dorsal and ventral horns of the spinal cord, including the Renshaw cells which have an inhibitory action on the motor neurons of the anterior horn region, also play a part in the reflex activity of the cord, which is connected by means of synapses with interposed association and commissural neurons and with the higher levels of the nervous system.

All motor impulses, whether part of the spinomuscular reflex arc or a part of a higher, more complex arc, are relayed to the muscle through the neuraxes of the anterior horn cells, which are acted upon by the corticospinal, rubrospinal, tectospinal, olivospinal, vestibulospinal, reticulospinal, intersegmental, intra-segmental, and other reflex levels.

Within the anterior horns there is a somatotopic arrangement of the motor neurons, those innervating the trunk and neck being medial to those of the extremities, and cells innervating istal extremity muscles being dorsal to those of the proximal muscles.

The arrangement of motor cells in the anterior horns was originally metameric: those of one segment innervated the muscles of the corresponding myotome. However, one nerve may contain fibers from several roots, and individual muscles may receive nerve impulses from more than one segment of the spinal cord.

A physiologic classification of the various muscles divides these into the following groups:
_ *the agonists*, are the muscles which directly perform the desired movement.
_ *the antagonists*, are those which oppose the agonists;
_ *the synergists* assist the agonists and reduce unnecessary movement to a minimum.
Any movement is dependent upon the combined action of all groups. Loss of function of any of these will impair the motor response.

THE CORTICOSPINAL (PYRAMIDAL) LEVEL

The corticospinal level of motor integration is also referred to as the cortical level, and the upper motor neuron level. It is only one of many motor fiber systems that converge upon the anterior horn cell and the final common pathway, and therefore it is only one of many upper motor neuron levels.

The corticospinal level found fully developed only in mammals and reaches its highest development in apes and man. In the newborn infant the descending fibers of the corticospinal system have little or no myelin sheathing. The process of myelination starts immediately after birth and is usually complete by the age of two years. Walking and other skilled movements are learned as the pathway matures and myelination progresses.

The major motor units of the corticospinal level are situated in the posterior portions of the frontal lobes of the brain. It extends from the sylvian fissure up to and over the vertex and for some distance on the medial surface of the brain. This area coincides cytoarchitectonically with the area gigantopyramidalis.

The origin of the name of this pathway is linked with the medullary pyramids. It is probable that only about 3% of the pyramidal fibers arise from the Betz cells (giant pyramidal cells found in the fifth layer of this area); parietal lobe, areas 6 and 8 of the frontal lobe and other portions of the brain, including the temporal and occipital lobes and certain subcortical centers, make important contributions to the corticospinal tract.

Within the motor cortex there is a definite localization of function. Skeletal musculature is represented in reverse order upon the contralateral motor area. The lower extremities are represented on the medial and the upper surface of the hemisphere while the motor centers for the upper extremity extend upward on the lateral and the middle surface of the brain (fig. 2). Areas for the tongue, face, and digits are exceptionally large and are out of proportion with those that control the proximal musculature.

The neuraxes of the motor units in the precentral convolution, or the pyramidal fibers, pass through the *corona radiata* into the *genu* and the anterior two thirds of the posterior limb of the internal capsule. At the brain stem level the portion of the pyramidal fibers so-called the *corticonuclear tract* contact with the cells of the motor cranial nerves. The majority of them partially decussate before synapsing. The neuraxes that convey impulses to the spinal cord are the *corticospinal fibers*. In the caudal portion of the medulla the majority of them (80% -90%) decussate and descend through the lateral funiculus of the spinal cord in the lateral corticospinal pathway to supply the muscles of the opposite side of the body. The smaller ventral corticospinal tract descends uncrossed in the ipsilateral ventral funiculus and usually does not extend below the midthoracic region; these fibers, too, usually cross before terminating.

The neuraxes carried through these pathways terminate at appropriate levels to supply the motor nuclei of the cranial nerves and the anterior horn cells of the spinal cord. Those to the cord end in the *zona intermedia* between the anterior and posterior horns, and there is an intercalated neuron between the neuraxis of the corticospinal cell and the anterior horn cell for the majority; a few fibers end directly on the anterior horn cells. Impulses then travel from the motor nuclei and anterior horn cells, which together with their neuraxes are the final common pathway, to the neuromuscular junction of striated muscles. A single corticospinal fiber innervates more than one neuron in the spinal cord, and probably some innervate many.

The corticospinal tract deals with discrete, isolated motor responses, especially with the liner adjustments of voluntary movement of the digits, and that disease of the pyramidal system *per se* results in hypotonia, areflexia, and possibly atrophy. Hemiplegia is generally produced by

combined destruction of the motor and premotor components of the upper motor neuron, and there are combinations of the foregoing symptoms, often with severe motor paralysis. In all probability the more violent reactions, such as spasticity and hyperreflexia, overshadow the flaccidity and hyporeflexia.

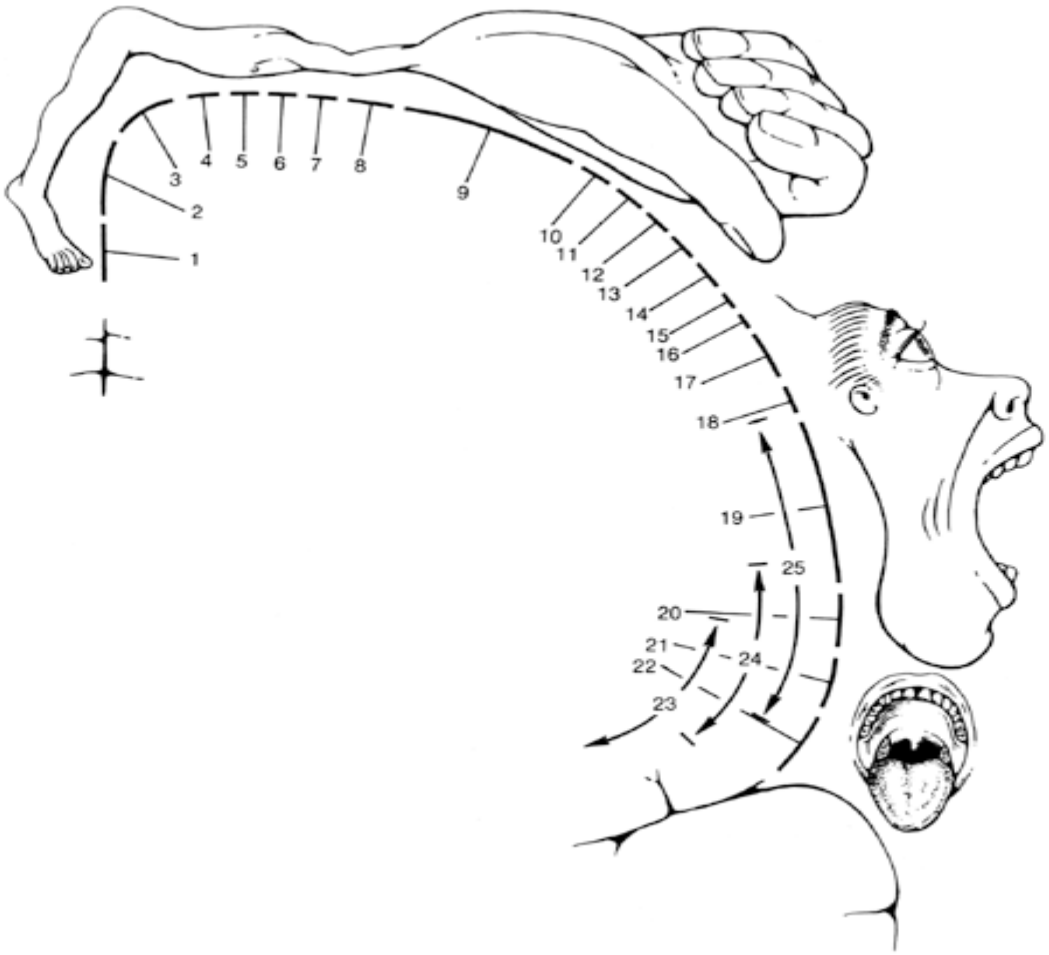


Fig. 2. Motor homunculus, showing the relationship of the motor centers to cortical representation.

CLINICAL MANIFESTATIONS OF DISEASE OF THE SPINOMUSCULAR LEVEL

Certain specific clinical findings become manifest in disease of the anterior horn cell, its neuraxis (which includes the nerve root, plexus, and peripheral nerve), the myoneural junction, or the muscle itself. In all, loss of motor power is evident. The weakness is focal, or restricted, and is segmental, affecting only the muscles or muscle groups that are supplied by the involved cells or nerves. The paralysis is characterized by a loss of tone and atrophy of the involved muscles; this is called flaccidity, or hypotonicity. Denervated muscle fibers undergo spontaneous contractions, known as fibrillations or fasciculations; these are too fine and rapid to be seen with the naked eye but can be demonstrated electromyographically. No pathologic reflexes are found.

CLINICAL MANIFESTATIONS OF DISEASE OF THE CORTICOSPINAL LEVEL

Destruction of the neuron or of its neuraxis, whether resulting from involvement of the gigantopyramidal cells themselves, the internal capsule, the cerebral peduncle, the fibers as they pass through the pons or medulla, or the corticospinal tract, produces a definite syndrome. Inasmuch as the corticospinal pathway has an integrating and inhibiting effect upon the lower centers, the essential manifestations of such a lesion consist of a loss of skilled voluntary movements, or impairment of integration of movements, and an overactivity or exaggeration of response of the lower centers. There is a loss of voluntary movement. This loss of power, being associated with is hypertonic, or spastic.

The paresis is generalized rather than focal; it involves either entire extremities or specific movements, rather than specific muscles. There is usually no localized atrophy. There are no fasciculations. The muscle stretch reflexes are exaggerated, and clonus may be elicited. The superficial reflexes are diminished or absent. Finally, there are various pathologic reflexes, such as the Babinski sign, which are often termed pyramidal reflexes or upper motor neuron signs. Some of the normal associated movements may be lost, but abnormal associated movements are present. Trophic changes are usually absent. The gemiparesis has a characteristic distribution. In the upper extremity the extensor and external rotation muscles are primarily affected; in the lower extremity weakness is most marked in the flexors and internal rotators, with relative sparing of the extensors and external rotators. The patient circumducts his leg as he begins walking.

There are several possibilities of damage of the corticospinal tract in different levels: in affecting the cortex motor area; internal capsule; corona radiata; corticospinal pathway in the brain stem and in the spinal cord.

In the capsular hemiplegia both the upper and the lower extremities are equally affected. In brain stem lesions involving the pyramidal pathways, unilateral or bilateral hemiparesis may result, it may even be crossed. In spinal cord lesions the paralysis observes below the level of the lesion. It may be mono-, para-, hemi-, and tetraplegia (paresis) depending on the level and extent of lesion.

Irritation of the corticospinal system, especially stimulation of the pyramidal cortex, causes an increased motor response with involuntary movements on the opposite side of the body. This may result in jacksonian convulsive seizures.

Table 3. ***Changes in motor function in disorders of the spinomuscular and corticospinal levels***

Level of disorder	Loss of Power	Tone	Atrophy	Fasciculations
Spinomuscular level				
1. Anterior horn cell	Segmental	Flaccid	Present	Present
2. Nerve root, plexus, peripheral nerve	Focal	Flaccid	Present	Occasionally present
3. Muscle	Diffuse	Flaccid	Present	Absent
4. Neuromuscular junction	Diffuse	Normal	Usually absent	Absent
Corticospinal tract lesions	Generalized. Incomplete.	Spastic	Absent	Absent