

Motor systems

Those portions of nervous systems that regulate and control the contractile activity of muscle and the secretory activity of glands. Muscles and glands are the two types of organ by which an organism reacts to its environment; together they constitute the machinery of behavior. Cardiac muscle and some smooth muscle and glandular structures can function independently of the nervous system but in a poorly coordinated fashion. Skeletal muscle activity, however, is entirely dependent on neural control. Destruction of the nerves supplying skeletal muscles results in paralysis, or inability to move. The somatic motor system includes those regions of the central nervous system involved in controlling the contraction of skeletal muscles in a manner appropriate to environmental conditions and internal states. This article discusses skeletal muscle innervation and contraction; the motor unit; and neural centers for motor control, particularly the motor cortex. *SEE GLAND; MUSCLE (ANATOMY); MUSCULAR SYSTEM.*

SKELETAL MUSCLE

A skeletal or striated muscle consists of a bundle of individual contractile elements called muscle fibers. The fibers are held together in the muscle by connective tissue, and their ends are attached via tendons to movable bones; thus, muscle shortening during active contraction results in movement. The nerve supply to skeletal muscles of the limbs and trunk is derived from large nerve cells called motoneurons, whose cell bodies are located in the ventral horn of the spinal cord (Fig. 1). Muscles of the face and head are innervated by motoneurons in the brainstem. The axons of the motoneurons traverse the ventral spinal roots (or the appropriate cranial nerve roots) and reach the muscles via peripheral nerve trunks. In the muscle, the axon of every motoneuron divides repeat-

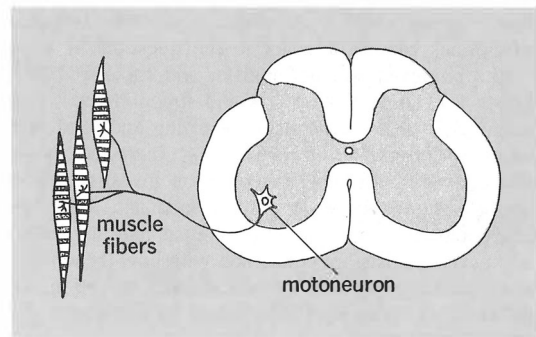


Fig. 1. Diagrammatic representation of a single motor unit. A motoneuron in the ventral horn of the spinal cord innervates multiple muscle fibers (typically more than the three shown).

edly into many terminal branches, each of which innervates a single muscle fiber. The region of innervation, called the neuromuscular junction, or motor end plate, is a secure synaptic contact between the motoneuron terminal and the muscle fiber membrane (Fig. 2).

Skeletal muscle contraction. A fixed sequence of events leads from motoneuron activity to contraction of skeletal muscle. An action potential initiated in a motoneuron propagates over the motoneuron axon into its many terminals. At each motor end plate, the action potential causes release of a neuromuscular transmitter agent, which depolarizes the postsynaptic membrane sufficiently to initiate an action potential in

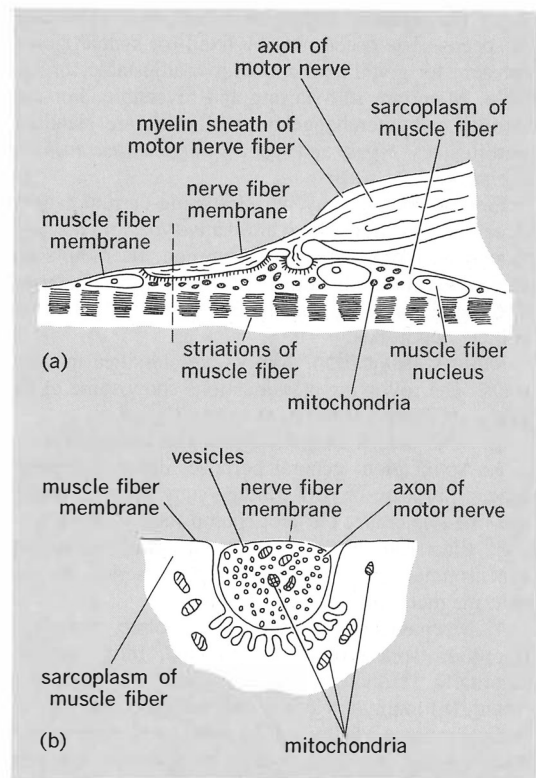


Fig. 2. Diagrams of the neuromuscular junction, or end plate. (a) Section cut parallel to long axis of muscle fiber, as seen by light microscopy. (b) Section cut perpendicular to long axis of muscle fiber (as through broken line in a), as seen by electron microscopy.

the muscle fiber membrane. This muscle fiber action potential propagates along the fiber at a speed of about 5 m/s and triggers the contractile process. *SEE BIOPOTENTIALS AND IONIC CURRENTS.*

The neuromuscular transmitter agent is acetylcholine, which is synthesized by the nerve terminals and is contained in tiny vesicles seen in electron micrographs. When the terminal is depolarized, some vesicles rupture at the surface, releasing acetylcholine into the synaptic gap. The acetylcholine diffuses across the gap and combines with receptors in the postsynaptic membrane. This combination opens channels in the membrane, and they conduct ions across the membrane resulting in its depolarization. The presynaptic action potential liberates enough acetylcholine to depolarize the muscle membrane past threshold for initiating an action potential. The receptors in the specialized muscle membrane of the end plate make it about 1000 times more sensitive to acetylcholine than nonjunctional regions of the muscle membrane. *SEE ACETYLCHOLINE; SYNAPTIC TRANSMISSION.*

The contractile elements in a muscle fiber consist of two proteins, actin and myosin, present in a ratio of about 1:3. The combination, called acetomyosin, forms long molecular chains. In the presence of adenosinetriphosphate (ATP), actomyosin filaments contract. Each muscle fiber contains up to 10,000,000 such myofilaments of actomyosin. Electron microscopic studies of muscle show that the actin and myosin elements lie parallel to one another and that during contraction the interdigitated elements slide past each other, thus shortening the muscle. *SEE ADENOSINETRIPHOSPHATE (ATP); MUSCLE (BIOPHYSICS); MUSCLE PROTEINS.*

Summation of contraction. The mechanical tension generated by a single contraction of a muscle fiber is much more prolonged than the electrical action potential that initiates it. The muscle fiber action potential lasts only a few milliseconds, but the rise and fall of the resulting twitch tension can take over 100 ms (Fig. 3). The muscle action potential, like the action potential of neurons, is an all-or-none phenomenon; its amplitude cannot be modified by changing the strength of the stimulus eliciting it. The contractile mechanism, however, is graded in strength, and the tension produced by a single twitch contraction is less than the maximal possible tension of the fiber. Because the contractile process greatly outlasts the duration of the muscle action potential, the tension elicited by two action potentials briefly separated in time is greater than that produced by a single twitch. The twitch contraction produced by a second action potential sums with that persisting from the first. Several muscle action potentials initiated in rapid succession produce temporal summation of the successive contractile responses. However, the increment of tension of each contraction diminishes as the net tension increases, until a maximum tension, called the tetanic tension, is reached. In some muscles the tetanic tension is nearly four times greater than the twitch tension. The rate of discharge necessary to produce maximal tetanic tension varies in different muscles, from 30 discharges/s for the slow soleus muscle to 350 discharges/s for the rapidly contracting internal rectus muscle of the eye.

Motor units. Since synaptic transmission at the neuromuscular junction is so secure, an action potential in the motoneuron will produce contraction of every muscle fiber that it contacts. For this reason,

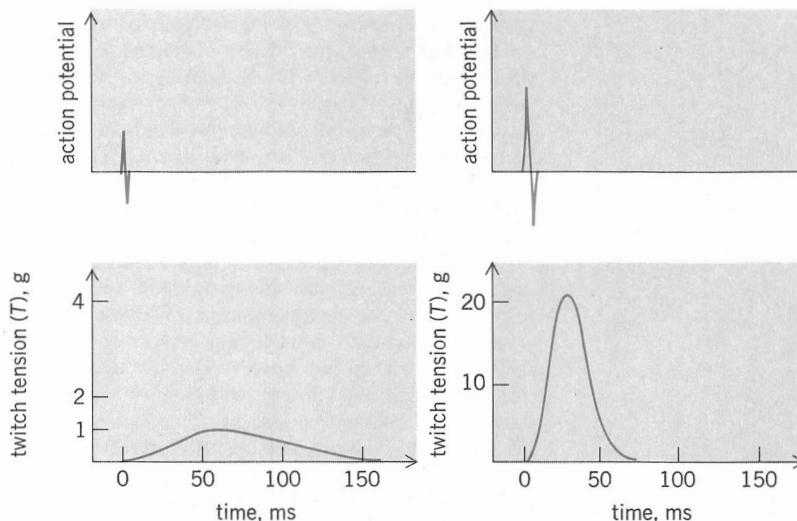


Fig. 3. Electromyographic action potentials and mechanical twitch tensions of two motor units. Type S (slow twitch) on left and type F (fast twitch) on right.

the motoneuron and all the fibers it innervates form a functional unit, called the motor unit. The number of muscle fibers in a single motor unit may be as small as six (for intrinsic eye muscles) or over 700 (for motor units of large limb muscles). For a given muscle the average number of muscle fibers per motor unit can be determined by dividing the total number of fibers in the muscle by the number of motoneurons in its pool. The typical innervation ratio for limb muscles is on the order of 120–150. In general, muscles involved in delicate rapid movements have fewer muscle fibers per motor unit than large muscles concerned with gross movements.

Physiological properties. Even within a given muscle, different motor units have significantly different physiological properties; some motor units are specialized for steady, prolonged contraction, while others are used for brief periods of high tension. The properties of motor units that underlie their functional specialization include the biochemical makeup of their muscle fibers, the speed and magnitude of their twitch tension, their response to repetitive activation, the size of their motoneuron, and physiological constraints on their relative recruitment. Detailed studies on hindlimb muscles of the cat indicate that these properties tend to vary systematically together. The spectrum of motor unit properties may be understood in terms of two contrasting types (Fig. 3). On one end of the functional spectrum are numerous “slow” type-S motor units, so called because their twitch tension takes longer to reach a peak (80–100 ms) and to decay than the more rapid twitch tension of “fast” type-F motor units at the opposite end of the spectrum. The maximum twitch tension of slow units (for example, 1 g for gastrocnemius muscle) is several times smaller than the peak tension of fast units (about 20 g). When stimulated repetitively for prolonged periods of time, the tetanic tension of the type-S units is sustained for much longer—on the order of hours—than the tetanic tension of the larger type-F units, which fatigue within minutes. These differences in mechanical performance are related to different metabolic properties of their muscle fibers. As a result, slow motor units are best at providing prolonged periods of tension, whereas fast units are spe-

cialized for brief bursts of intense activity.

Motoneuron recruitment. These functional differences are further correlated with differences in the recruitment of their motoneurons. The type-S motor units are generally recruited at lower thresholds in a variety of motor responses than the type-F units. The motoneurons supplying slow motor units tend to have smaller soma size than the large motoneurons of the type-F motor units. In most cases, the motoneurons of a pool are activated in a systematic sequence, in order of increasing size; this recruitment order is the same whether the motoneurons are activated by descending commands from higher centers or by input from peripheral muscle nerves. The fact that properties like twitch tension and recruitment order are related to the motoneuron size has been called the size principle. For example, in isometric contraction of the first dorsal interosseous muscle of the hand, the first motor units to be activated contribute twitch tensions of 0.1 g; as the net muscle force increases, ever larger motor units become active; near the maximum levels of force, the largest units are finally recruited, with a twitch tension of 10 g. Thus, the larger units are recruited at higher levels of net force and also contribute larger twitch tensions. In fact, the size of the motor units' twitch tension is directly proportional to their recruitment level over a wide range of tensions.

Muscle tension. Thus, during voluntary muscle contractions, progressively greater muscle tension is produced by two mechanisms: additional motor units are recruited into activity, in order of small to large; and the firing rates of active motor units increase with net force. In some muscles, the rate of motor unit discharge may increase from 8 to 50 action potentials per second, as the muscle contraction increases from light to maximal effort. At the lower firing rates, the successive twitch tensions of each motor unit produce a series of separate twitches. Since the different motor units of a muscle typically discharge asynchronously, the net tension in the muscle nevertheless varies smoothly. At higher frequencies of motor unit discharge, the twitch tensions of individual motor units begin to summate, producing still further increases in tension. Thus, increased temporal summation of twitches in individual units as well as increased spatial summation of twitches in different units both contribute to increased tension. Moreover, the contribution of the large units recruited at high tension is proportionately greater.

COMPONENTS OF SKELETAL MOTOR SYSTEM

Motoneurons are activated by nerve impulses arriving through many different neural pathways. Some of their neural input originates in peripheral receptor organs located in the muscles themselves, or in receptors in skin or joints. Many muscle receptors discharge in proportion to muscle length or tension; such receptors have relatively potent connections to motoneurons, either direct monosynaptic connections or relays via one or more interneurons. Similarly, stimulation of skin and joints, particularly painful stimulation, can strongly affect motoneurons. Such simple segmental pathways constitute the basis for spinal reflexes.

The other major source of input to motoneurons arises from supraspinal centers. **Figure 4** shows the main nervous system centers involved in controlling the input to motoneurons. Evidence that these regions play a role in control of motor activity comes from

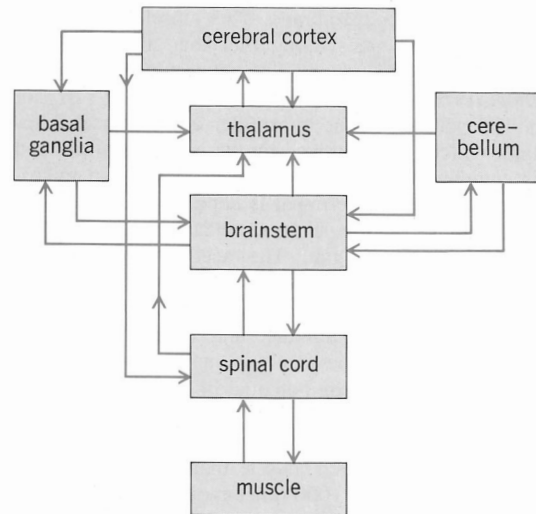


Fig. 4. Schematic diagram of the major components of the vertebrate motor systems. Arrows indicate the main neural connections between regions.

three types of observations: damage of cells in these areas by experimental or clinical lesions produces motor deficits; electrical stimulation in these regions evokes motor responses or interferes with ongoing movements; and activity of cells recorded in these areas in moving animals is clearly related to their motor activity. Figure 4 indicates the major interconnections between cells in these regions; as one might expect, they function together during voluntary movements. The representation of the primate motor system in Fig. 4 is greatly simplified to provide an overview of its major components.

Segmental circuits. At the spinal level, the motoneurons and muscles have a close reciprocal connection. Afferent connections from receptors in the muscles return sensory feedback to the same motoneurons which contract the muscle. Connections to motoneurons of synergist and antagonist muscles are sufficiently potent and appropriately arranged to subservise a variety of reflexes. In animals with all higher centers removed, these segmental circuits may function by themselves to produce simple reflex responses. Under normal conditions, however, the activity of segmental circuits is largely controlled by supraspinal centers. Descending tracts arise from two major supraspinal centers: the cerebral cortex and the brainstem.

Brainstem. The brainstem, which includes medulla and pons, is a major and complex integrating center which combines signals descending from other higher centers, as well as afferent input arising from peripheral receptors. The descending output from brainstem neurons affects motor and sensory cells in the spinal cord. Brainstem centers considerably extend the motor capacity of an animal beyond the stereotyped reflex reactions mediated by the spinal cord. Even without any higher centers, animals with a brainstem can perform integrated activities such as standing, walking, and making appropriate postural adjustments. In contrast to segmental reflexes, these motor responses involve coordination of muscles over the whole body. In decerebrate cats, electrical stimulation of certain brainstem regions evokes a form of locomotion; the rate of walking is influenced by proprioceptive feed-

back from the limbs. Another major motor function of the brainstem is postural control, exerted via the vestibular nuclei. Sensory receptors in the vestibule of the ear, signaling the orientation of the head in space and changes in head position, have potent influence on the vestibular nuclei, which in turn effectively regulate limb and trunk muscles to respond in a manner that maintains postural stability.

Besides neurons controlling limb muscles, the brainstem also contains a number of important neural centers involved in regulating eye movements. These include motor neurons of the eye muscles and various types of interneurons that mediate the effects of vestibular and visual input on eye movement. For example, one important pathway mediates the vestibular ocular reflex; it transforms vestibular signals, initiated by head movements, to generate compensatory eye movements, with the result that eye position can remain fixed in space. Visual input can generate two different types of eye movements, each mediated by its own neural circuitry: rapid saccadic eye movements, which move the eye as quickly as possible from one visual target to another, and smooth-pursuit eye movements, which follow a slowly moving target. In contrast to limb muscles, which must deal with changing loads, the eye muscles control a fixed and predictable load. This, plus the need for maintaining accurate vision, has led to a highly developed oculomotor system, residing largely in the brainstem.

Cerebellum. Another important coordinating center in the motor system is the cerebellum, an intricately organized network of cells closely interconnected with the brainstem. The cerebellum receives a massive inflow of sensory signals from peripheral receptors in muscles, tendons, joints, and skin, as well as from visual, auditory, and vestibular receptors. Higher centers, particularly the cerebral cortex, also provide extensive input to the cerebellum via pontine brainstem relays. The integration of this massive amount of neural input in the cerebellum somehow serves to smooth out the intended movements and coordinate the activity of muscles. Without the cerebellum, voluntary movements become erratic, and the animal has difficulty accurately terminating and initiating responses. The output of the cerebellum affects primarily brainstem nuclei, but it also provides important signals to the cerebral cortex.

The function of the cerebellum has been likened to a servocontrol mechanism which detects and corrects errors during the course of a movement. Following injury to the cerebellum, movements are ataxic; that is, they suffer from errors in rate, range, force, and direction. Starting, stopping, and changing the direction of movement are especially disturbed; in reaching for an object the hand often misses the target. Voluntary movement is characterized by tremor which increases as the movement progresses. All these disturbances appear to be due to defects, not in generating movement, but in detecting errors and correcting deviations. Thus, if in reaching for an object the hand strays from the correct path, correction is begun too late and proceeds too far, so that the hand overshoots the direct path. A series of such overcorrections results in the oscillating tremor typical of cerebellar injury.

Injury to those portions of the cerebellum which receive afferent input from the vestibular nuclei produces marked disturbances of equilibrium and gait, so

that the victim, although showing little uncoordination of movement while lying in bed, is unable to maintain balance and to coordinate limb muscles in walking.

Basal ganglia. At another level of motor system are the basal ganglia. These massive subcortical nuclei receive descending input connections from all parts of the cerebral cortex. Their output projections send recurrent information to cerebral cortex via thalamus, and their other major output is to brainstem cells. Their substantial size suggests that the basal ganglia perform an important motor function, but the precise nature of the function remain unclear. Pathological lesions of the basal ganglia result in two types of motor disturbances, either decreased motor activity or generation of involuntary movements; such lesions involve no sensory deficits, nor do they impair mental capacities.

Decreased motor activity caused by disruption of basal ganglia, called negative signs, include akinesia (a disinclination to use the affected part of the body), delays and slowing of limb movements, absence of normal postural adjustments necessary for stable standing, and deficits in facial expression. In other cases, basal ganglia disorders produce excessive involuntary motor activity; such positive signs of basal ganglia pathology include muscle rigidity (resistance to passive stretch caused by tonic motor activity), rhythmic muscle tremor, characteristic of parkinsonism, and uncontrollable movements of the hands or feet (allegosis) or the entire limb (ballismus). These motor symptoms can sometimes be alleviated by surgical or chemical treatments affecting the basal ganglia. The fact that both the positive and negative signs of basal ganglia pathology represent a disorder in generation of movements suggests that basal ganglia play a role in the initiation of movements.

Cerebral cortex. At the highest level of the nervous system is the cerebral cortex, which exerts control over the entire motor system. The cerebral cortex performs two kinds of motor function: certain motor areas exert relatively direct control over segmental motoneurons, via a direct corticospinal pathway, the pyramidal tract, and also through extrapyramidal connections via supraspinal motor centers. The second function, performed in various cortical association areas, involves the programming of movements appropriate in the context of sensory information, and the initiation of voluntary movements on the basis of central states. Cortical language areas, for example, contain the circuitry essential to generate the intricate motor patterns of speech. Limb movements to targets in extrapersonal space appear to be programmed in parietal association cortex. Such cortical areas involved in motor programming exert their effects via corticocortical connections to the motor cortex, and by descending connections to subcortical centers, principally basal ganglia and brainstem.

As indicated in Fig. 4, the motor centers are all heavily interconnected, so none really functions in isolation. In fact, some of these connections are so massive that they may form functional loops, acting as subsystems within the motor system. For example, most regions of the cerebral cortex have close reciprocal interconnections with underlying thalamic nuclei, and the corticothalamic system may be considered to form a functional unit. Another example is extensive connection from cerebral cortex to pontine regions of the brainstem, controlling cells that project

to the cerebellum, which in turn projects back via the thalamus to cerebral cortex. Such functional loops are at least as important in understanding motor coordination of the individual centers themselves. With this overview in mind, the role of motor cortex in controlling movement will not be considered. The anatomical and physiological information available on the organization of motor cortex also exemplifies the information required to understand other motor centers. *SEE BRAIN.*

MOTOR CORTEX

The cerebral cortex is an extensive network of nerve cells covering the forebrain. The axons of some cortical cells pass into the underlying white matter and descend through the brainstem to affect the motoneurons of the brainstem or spinal cord. Areas of cortex designated as motor cortical areas have many such corticofugal cells. Electrical stimulation of these motor areas elicits movement, and their excision produces either paralysis or paresis (weakness). Activity of cells recorded in these regions is closely related to limb movements.

Motor areas. Systematic stimulation of the exposed cerebral cortex with electrical pulses reveals several prominent motor areas.

Precentral area. In primates the major one is the precentral motor area, which lies anterior to the central fissure of Rolando. In the monkey it extends over the precentral gyrus (Fig. 5); in humans it is largely buried in the anterior wall of the central fissure. Repetitive electrical stimulation within this region can elicit discrete movement, such as flexion of the thumb or a retraction of the lip. The evoked movement is always on the side of the body contralateral (opposite) to the stimulated hemisphere, since the descending pathways cross the midline before reaching the spinal cord. Moreover, the particular muscles activated depend on the stimulated site within the precentral motor area. Beginning at the midline and proceeding laterally along the precentral gyrus, one finds an orderly array of motor points for leg, trunk, arm, and face

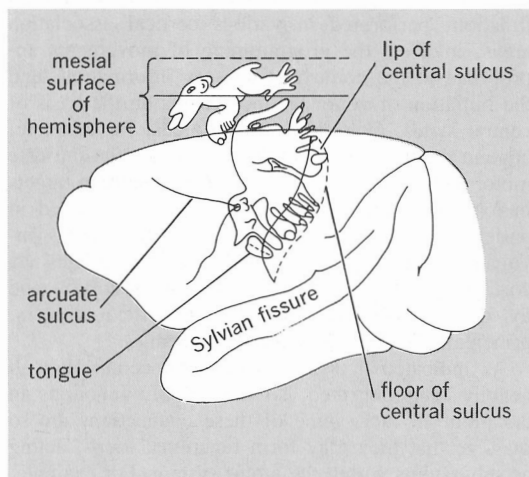


Fig. 5. Brain of monkey, with figurines showing motor representation of body in precentral motor cortex (lower figurine) and supplementary motor area (upper figurine). Parts of the body are drawn superimposed on the cortical regions that regulate their movement. The representation of toes and fingers buried in the precentral bank is shown with the mesial surface of the hemisphere folded out.

musculature. The topographical relations may be represented by displaying the body superimposed along the precentral gyrus as shown for the monkey in Fig. 5. In addition to this mediolateral representation of lower to upper body musculature, there is an anterior-posterior organization, with the musculature of the apical portions of the body (fingers, toes, lower lip, and tip of tongue) located posteriorly, mostly in the depths of the central fissure, and the musculature of trunk and back represented more anteriorly, on the free surface of the precentral gyrus. A striking feature of motor cortex organization is the disproportionate areas devoted to different muscles. The muscles of fingers, toes, lips, and tongue, which are involved in delicate, precise movements, have relatively large cortical areas devoted to their control. Comparatively smaller cortical areas are devoted to trunk musculature, explaining the distortion of the figurines in Fig. 5.

Precise stimulation of precentral motor cortex sites can sometimes evoke contraction of individual muscles. However, while the threshold response to stimulation may be activation of a single muscle, in most cases such stimuli also evoke subthreshold effects in motoneurons of other muscles. This is due to the fact that the stimuli affect numerous cortical cells projecting to different motoneurons. In the primate, forelimb motoneurons receive direct input from a colony of corticomotoneuronal cells, whose cell bodies are distributed over several square millimeters of cortex. Careful analysis indicates that the cortical colonies converging on motoneurons of different muscles may overlap extensively. In fact, single corticomotoneuronal cells may send divergent terminal projections to motoneurons of several different muscles. This means that the activity of such cells would affect a group of muscles.

Besides having motor output affecting muscles, the cortical cells also receive sensory input from receptors in muscles and skin. The topographic map over precentral cortex in Fig. 5 represents not only the parts of the body that move when the cortex is stimulated, but also the regions of the body from which the cortical cells can be activated. In other words, the sensory map and motor map are superimposed in register: cortical cells receive sensory input from the same regions to which they send motor output. Such input-output relations indicate that normally the cells would be involved in a functional loop interconnecting the cortical site with a peripheral locus.

Second and supplementary areas. The precentral motor area is the most elaborate motor cortical area with the most direct output connections to motoneurons; in addition, three other motor areas for the body musculature have also been demonstrated. One of these, called the second motor area, is buried in the Sylvian fissure; its organization is represented by a figurine face to face with the precentral motor representation. A third complete motor representation, the supplementary motor area, lies on the medial surface of the hemisphere (Fig. 5). Threshold for evoking movements by electrical stimulation is higher for supplementary motor area than for precentral motor area; such responses often involve the musculature of both sides of the body.

Postcentral cortex. A fourth significant motor representation is in postcentral cortex. Its topographical organization would be represented by a figurine which is the mirror image of that shown on the precentral

cortex in Fig. 5. Like the precentral map, the postcentral map also represents both the sensory input from peripheral receptors as well as motor output to the relevant regions. The precentral and postcentral maps are heavily interconnected by cells making corticocortical connections. Nevertheless, some motor output from postcentral cortex is independent of its connections to precentral cells, since movements can be evoked by stimulating postcentral regions after motor cortex has been removed. The threshold for evoking motor effects from postcentral cortex is higher than from precentral cortex.

Eye and neck movements. Stimulation of the cortex in the region anterior to the arcuate fissure elicits movements of neck and eyes. The typical response consists of conjugate movement of the eyes and turning of the head to the side opposite the stimulated hemisphere. For this reason the area enclosed between the two limbs of the arcuate fissure is often called the frontal eye field. Since the major ascending pathways leading into the cortex cross the midline before reaching the cortex, stimuli applied to the right side of the body generate nerve impulses which reach the left hemisphere. Such impulses activating the motor eye fields on the left would cause movement of the head and eyes toward the right, that is, toward the stimulated side of the body. Such orientational movements may be part of the motor component of attention.

Effects of ablations. Removal of motor cortex within and just anterior to the central fissure causes a partial loss of voluntary movement in the contralateral muscles represented in the ablated cortex. Lesions confined to the leg area cause paralysis of the leg, leaving the arm and face musculature unaffected; thus the topographical organization of motor cortex revealed by stimulation is confirmed by ablation experiments. The paralyzed extremity is flaccid; that is, it hangs limply and displays no resistance to passive flexion or extension. Paralysis of voluntary movement is not permanent, however; the duration and severity of the deficit vary with species, being more severe in the human, ape, and monkey than in lower mammals such as dog and cat. This species difference reflects the increased dominance of the cerebral cortex in phylogenetically advanced forms. Following ablation of the arm motor cortex in monkeys and chimpanzees, movement at the shoulder reappears with a few days. Later, elbow and wrist movements reappear, and finally (8–12 weeks in chimpanzees) crude movements of fingers are restored. However, finger movements never regain the preoperative delicacy; thumb-finger approximation such as occurs in grooming remains permanently defective and awkward. This reflects the critical role of cortex in control of the finger muscles, which have an extensive cortical representation.

Following isolated unilateral ablation of the frontal eye fields in monkeys, the animal's head and eyes turn toward the side of the lesion; there is transient paralysis of conjugate deviation of the eyes toward the opposite side. In walking, the animal tends to turn toward the side of the lesion; that is, it tends to follow the eyes and head, so that locomotion is circular. Such circling gait may persist after paralysis of the eye muscles has disappeared.

Activity of cortical neurons. The activity of single neurons recorded in monkeys trained to make specific movements further confirms the role of cerebral cortex cells in initiating and controlling movement. The

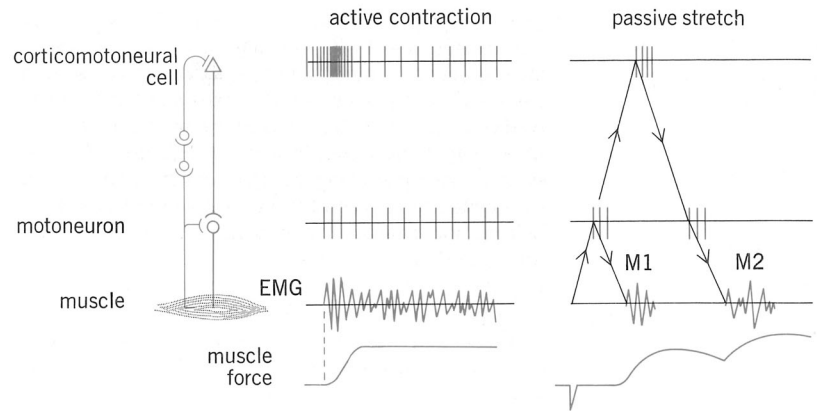


Fig. 6. Response of corticomotoneuronal cell and target muscle during active muscle contraction and in response to passive stretch of muscle. Diagram at left shows neural pathways: input from muscle stretch receptors affects motoneuron and corticomotoneuronal cell; the corticomotoneuronal cell also receives centrally originating input during active movement, and projects to motoneuron. Passive muscle stretch evokes impulses conducted to the motoneuron via segmental reflex, producing the M1 response, and to corticomotoneuronal cell, contributing to the M2 response via transcortical loop.

location of motor cortex neurons which discharge in relation to active movement of particular parts of the body is again represented by the topographic map in Fig. 5. When the monkey initiates a simple hand movement—for example, pressing a key when a light is illuminated—many cells in the precentral hand region become active well before the onset of agonist muscle activity; many neurons continue to fire as long as the related limb muscles are active.

In monkeys making wrist movements requiring different degrees of active force, and independent wrist displacement, experimenters found that motor cortex cell activity was more closely related to the active force of a movement than the resultant limb position. **Figure 6** illustrates the typical response pattern of a corticomotoneuronal cell during an active wrist movement. Such corticomotoneuronal cells facilitate the activity of one or more target muscles. At onset of movement, the cell discharges a burst of activity beginning well before onset of activity in its target muscles. During the static hold period most corticomotoneuronal cells fire tonically at a steady rate; this tonic firing rate was found to increase in proportion to the amount of active force exerted. Thus, the firing pattern of a corticomotoneuronal cell shows a contribution to maintaining steady force during the hold period, as well as an additional burst related to initiating a change in force.

Besides firing during active movement, corticomotoneuronal cells also respond to passive limb movements, typically passive joint movements that stretch their target muscles. Sensory input from stretch receptors in the same muscles which the corticomotoneuronal cell facilitates would make functional sense, since it would help compensate for load changes. A sudden increase in load during an active movement, for example, would stretch the agonist muscles, and would activate corticomotoneuronal cells whose output in turn would facilitate activity of those muscles, tending to overcome the increased load. Such a transcortical load compensation reflex is entirely analogous to the segmental stretch reflex mediated by direct input to motoneurons from stretch receptors.

Figure 6 also illustrates the responses evoked by a

sudden stretch of a muscle. The first, short-latency electromyogram (EMG) response (M1) is mediated by the segmental stretch reflex—the direct connection from stretch receptors to motoneurons. The second EMG response (M2) is mediated by long-loop reflexes, including those through motor cortex. In contrast to the spinal circuit, the gain of the transcortical loop could be changed by the state of higher centers. Indeed, the response of cortical cells to peripheral stimulation is enhanced when the animal is prepared to make a movement in which the cells are involved. Corticomotoneuronal cells with monosynaptic connections to motoneurons are an important component of the pyramidal system, which controls spinal cells directly.

Pyramidal tract. One of the most important corticofugal pathways is the pyramidal tract, or corticospinal tract, which originates from cortical cells and runs without interruption to the spinal cord. The human medullary pyramid contains about 1,000,000 fibers, mostly of small diameter. Almost half of the pyramidal tract originates from neurons in the precentral motor areas. Some of these are the giant pyramidal cells of Betz, large conical cells which histologically distinguish precentral motor cortex. In humans, the motor area of each hemisphere contains about 34,000 Betz cells, enough to account for about 2% of the pyramidal fibers, presumably those of large (11–20-micrometer) diameter. The supplementary motor area also contributes some fibers to the pyramidal tract. The rest of the pyramidal tract originates in the postcentral gyrus, posterior to the central fissure.

The axons of the pyramidal tract leave their cells of origin in cortical layer 5 to enter the subcortical white matter and then pass through the internal capsule, the cerebral peduncles, and the pons (Fig. 7). Some fibers terminate in the brainstem on motoneurons of cranial nerve nuclei. Below the pontomedullary junction the tract continues on the ventral surface of the medulla as a recognizable band of fibers called the pyramid. At the caudal border of the medulla

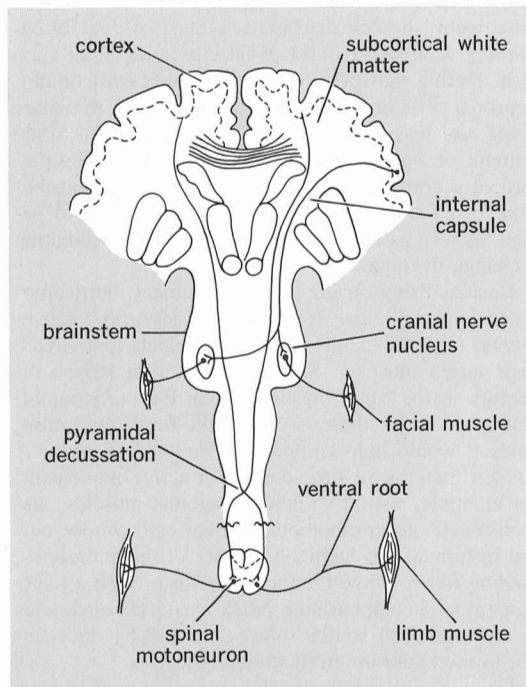


Fig. 7. Diagram of pyramidal system, showing origin and course of corticospinal (pyramidal) tract. In a similar manner, corticobulbar fibers affect cranial motoneurons of facial muscles.

most of the fibers cross to the opposite side of the neuraxis to descend in the dorsolateral column of the spinal cord. Some pyramidal fibers do not decussate but continue either in the ventral white funiculi or in the lateral column of the same side. The spinal portion of the corticospinal tract extends throughout the full extent of the spinal cord but progressively diminishes in size as it passes from cervical to lumbar segments. Terminations are particularly numerous in the cervical and lumbar enlargements, which contain the motoneurons supplying the musculature of upper and lower limbs.

The role of the pyramidal tract in movement in monkeys and chimpanzees has been studied by sectioning the medullary pyramid and observing the resulting changes in motor performance. The most prominent defect following unilateral pyramidotomy is contralateral paresis involving the musculature from the neck down. The affliction is more severe in chimpanzees than in monkeys; in the former, even stereotyped movements of progression are impaired but not abolished. In neither animal is paralysis so grave as to render the affected parts useless, but there is severe impairment of voluntary movement and loss of such fine movements as opposition of thumb and index finger in grooming and individual movements of the fingers in manual exploration. This type of deficit has been observed to persist up to 4 years after operation, and thus may be considered permanent. Associated with the paresis is flaccidity like that seen following precentral cortical ablations; the extremities are limp and offer no resistance to movement at the joints.

Extrapyramidal systems. Isolated destruction of the corticospinal tract does not cause complete loss of voluntary movement. Moreover, electrical stimulation of the motor cortical areas after pyramidotomy still elicits muscular contraction. Therefore, the cortex must give rise to motor pathways other than the pyramidal tract. These other pathways are often referred to collectively as cortically originating extrapyramidal pathways. Although extrapyramidal projections probably arise from nearly all portions of the cortex, the precentral motor area appears to give rise to particularly large contributions. Figure 8 illustrates the anatomic relation of some of the better-known extrapyramidal systems:

Corticostriatal and corticopallidal systems. These two systems originate from rostral precentral cortex and project to the caudate nucleus and the putamen, which are portions of the basal ganglia. From the basal ganglia, impulses are relayed to the brainstem, which in turn affects the spinal levels.

Corticoreticular systems. These systems originate from the cortex around the central fissure, especially from the motor area. The axons terminate in the region of the pons and medulla on the diffusely organized neurons constituting the brainstem reticulum. The projection is bilateral and poorly, if at all, organized somatotopically. Impulses are presumably relayed to the spinal cord via the reticulospinal tracts which traverse the ventral and lateral white columns of the cord. *SEE RETICULAR FORMATION.*

Corticopontine systems. These systems arise from the cortex of each of the four lobes of the brain, although the contribution from the rostral precentral gyrus is most prominent. A significant contribution also comes from the supplementary motor cortex on the mesial surface of the hemispheres. The fibers terminate in the pontine nuclei, which in turn project to the

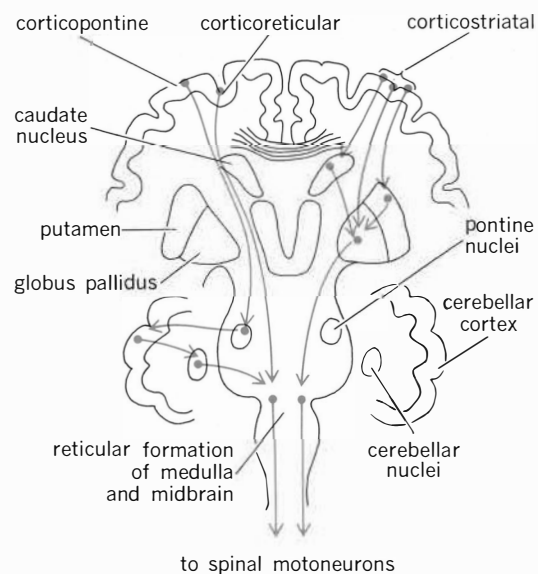


Fig. 8. Diagram of extrapyramidal systems, showing major descending connections from cerebral cortex.

cerebellum. From the cerebellum, projection systems feed impulses into the medullary reticulum, which in turn projects through the reticulospinal tracts to the spinal cord as described above. *SEE BRAIN; NERVOUS SYSTEM (VERTEBRATE).*

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