

MINISTRY OF HEALTH OF UKRAINE
ZAPORIZHZHIA STATE MEDICAL UNIVERSITY
Department of Normal Physiology

MOTOR FUNCTION OF CENTRAL NERVOUS SYSTEM

educational manual for independent work at home and in class
for student for licensing examination “KROK 1”

for students of International Faculty
(the second year of study)

Zaporizhzhia

2018

UDC 612.81(072)

M 89

*Ratified on meeting of the Central methodical committee
of Zaporizhzhia State Medical University
(protocol N ___ from _____)
and it is recommended for the use in educational process for foreign students.*

Reviewers:

A. V. Abramov - profesor of the Pathophysiology Department, PhD, professor.

A. M. Kamyshny – head of the Microbiology, Virology and Immunology Department, PhD, profesor.

Authors:

M. A. Tykhonovska, I. Ye. Sukhomlinova, D. G. Ivanchenko, O. V. Tykhonovsky.

Motor function of central nervous system: a manual for independent work at home and in class preparation for licensing examination "KROK 1 for students of International Faculty (2nd year of study) / M. A. Tykhonovska, I. Ye. Sukhomlinova, D. G. Ivanchenko, O. V. Tykhonovskyi. - Zaporizhzhia : ZSMU, 2018. – 77 p.

Methodical recommendations compiled in accordance with the program of "normal physiology". Guidelines are intended to help students prepare for practical classes and learn the material. Can be used for training of 2th-years students of international faculty, discipline "normal physiology".

This manual is recommended for II year students of International Faculty of specialty "General medicine" studying biological chemistry, as additional material to prepare for practical training and licensing exam "KROK 1: General medical training".

Моторні функції центральної нервової системи: навч.-метод. посіб. для самостійної роботи при підготовці до ліцензійного іспиту "КРОК 1" для студентів 2 курсу міжнародного ф-ту / М. А. Тихоновська, І. Є. Сухомлінова, Д. Г. Іванченко, О. В. Тихоновський. - Запоріжжя: ЗДМУ, 2018. – 77 с.

© Tykhonovska M. A., Sukhomlinova I. Ye.,
Ivanchenko D.G., Tykhonovskyi O. V., 2018
©Zaporizhzhia State Medical University, 2018

PREFACE

The current stage of professional health education as a part of a credit-modular system is characterized by a significant increase in the volume, complexity and rate of assimilation of educational material.

Instructor's Manual is constructed to meet modern teaching approaches to the educational process, training materials methodological support and professional algorithms. Under the program in physiology, the manual briefly summarizes the most significant in the present context of theoretical material, questions for discussion at the seminar, are included experimental part of the class is described, thematic tests and case studies are suggested.

The manual has a practical orientation, it summarizes many years of experience of the faculty of the Department of normal physiology of Zaporizhzhia State Medical University.

This guide can be used to prepare for practical classes in normal physiology according to the topic.

CONTENS

1. Lection: Motor End Integrative Neurophysiology.....	5
1.1The skeleton as the framework for movement.....	6
1.2 Muscle function and body movement.....	8
1.3 Peripheral nervous system components for the control of movement....	9
1.4The spinal cord in the control of movement.....	21
1.5 Supraspinal influences on motor control.....	32
1.6The role of the cerebral cortex in motor control.....	40
1.7 The basal ganglia and motor control.....	48
1.8 The cerebellum in the control of movement.....	55
2. Control questions and practical work	61
3. Control questions for independent work.....	63
4. Quizzes.....	64
5. Correct answers.....	76
6. Recommended literature	77

ABBREVIATIONS and SYMBOLS

CNS - Central nervous system

GTO – Golgi tendon organs

MI- primary motor cortex motor area

MII- The supplementary motor cortex

GPe - globus pallidus external segment

GPi - globus pallidus internal segment

SNr - substantia nigra pars reticulata

SNc - substantia nigra pars compacta region

SC - superior colliculus

MRI - Magnetic resonance imaging

MBEA - midbrain extrapyramidal area

1. Lession: Motor End Integrative Neurophysiology

Skeletal muscles - the main organ of the body's interaction with the environment and the impact on it. In the process of evolution fittest were those individuals who have motor system is the most perfect. It provides search for food, protection from enemies, procreation and other vital processes. There are two types of motor functions - the actual movement (locomotion - movement in space, ie, walking, running, jumping, swimming) and the support body positions (postures) in the space. Therefore, this system is called the locomotive. This includes muscles, tendons, joints, bones and nervous and humoral regulation mechanisms. To reduce the muscles receive signals from the motor centers located in various parts of the central nervous system. Conventionally, these centers can be divided into 5 sections: the spinal cord, hindbrain, midbrain, diencephalon and cerebral cortex.

The finger movements of a neurosurgeon manipulating microsurgical instruments while repairing a cerebral aneurysm, and the eye-hand-body control of a professional basketball player making a rimless three-point shot, are two examples of the motor control functions of the nervous system operating at high skill levels. The coordinated contraction of the hip flexors and ankle extensors to clear a slight pavement irregularity encountered during walking is a familiar example of the motor control system working at a seemingly automatic level. The stiff-legged stride of a patient who experienced a stroke and the swaying walk plus slurred speech of an intoxicated person are examples of perturbed motor control. Although our understanding of the anatomy and physiology of the motor system is still far from complete, a significant fund of knowledge exists.

1.1 THE SKELETON AS THE FRAMEWORK FOR MOVEMENT

Bones are the body's framework and system of levers. They are the elements that move. The way adjacent bones articulate determines the motion and range of movement at a joint.

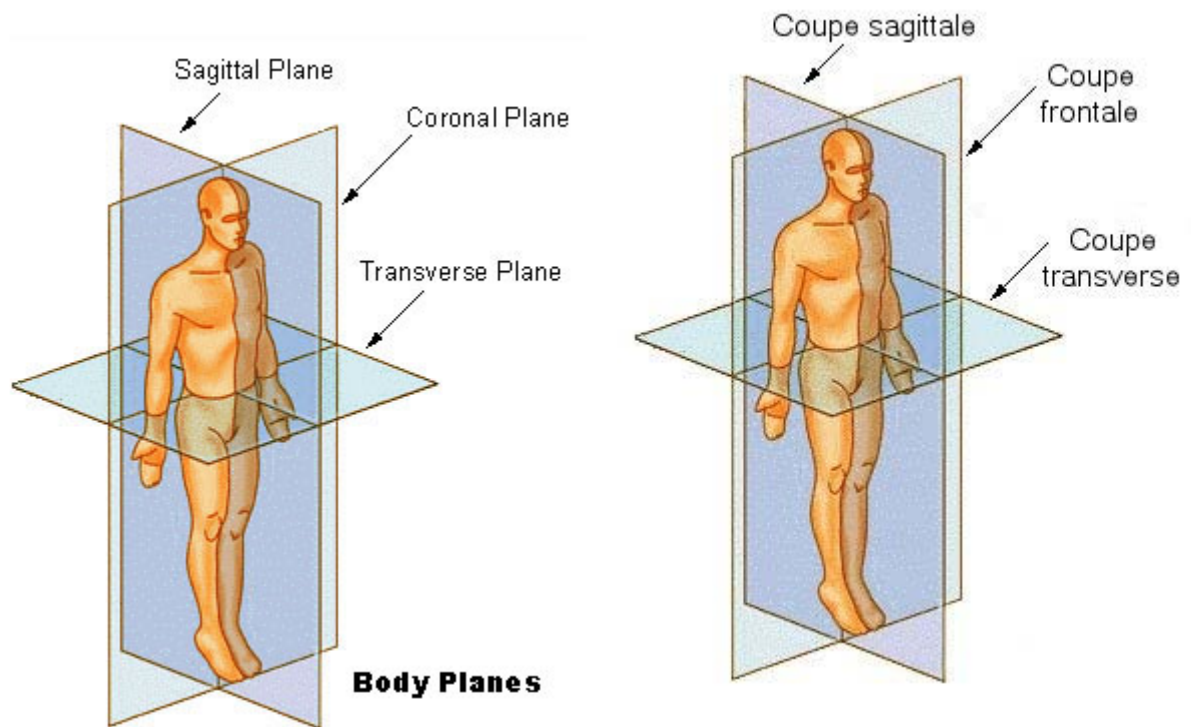


Fig. 1. **Anatomic reference planes.** The figure is shown in the standard anatomic position with the associated primary reference planes.

Ligaments hold the bones together across the joint. Movements are described based on the anatomic planes through which the skeleton moves and the physical structure of the joint. Most joints move in only one plane, but some permit movement in multiple anatomic reference planes (Fig. 1).

Hinge joints, such as the elbow, are uniaxial, permitting movements in the sagittal plane. The wrist is an example of a biaxial joint. The shoulder is a multiaxial joint; movement can occur in oblique planes as well as the three major planes of that joint. Flexion and extension describe movements in the sagittal plane. **Flexion** movements decrease the angle between the moving body segments. **Extension** describes movement in the opposite direction. **Abduction** moves the body part away from the midline, while **adduction** moves the body part toward midline.

1.2 MUSCLE FUNCTION AND BODY MOVEMENT

Muscles span joints and are attached at two or more points to the bony levers of the skeleton. The muscles provide the power that moves the body's levers. Muscles are described

in terms of their origin and insertion attachment sites. The **origin** tends to be the more fixed, less mobile location, while the **insertion** refers to the skeletal site that is more mobile. Movement occurs when a muscle generates force on its attachment sites and undergoes shortening. This type of action is termed an **isotonic** or **concentric contraction**.

Another form of muscular action is a controlled lengthening while still generating force. This is an **eccentric contraction**. A muscle may also generate force but hold its attachment sites static, as in **isometric contraction**. Because muscle contraction can produce movement in only one direction, at least two muscles opposing each other at a joint are needed to achieve motion in more than one direction. When a muscle produces movement by shortening, it is an **agonist**. The **prime mover** is the muscle that contributes most to the movement. Muscles that oppose the action of the prime mover are **antagonists**. The quadriceps and hamstring muscles are examples of agonist-antagonist pairs in knee extension and flexion. During both simple and light load skilled movements, the antagonist is relaxed. Contraction of the agonist with concomitant relaxation of the antagonist occurs by the nervous system function of **reciprocal inhibition**. Co-contraction of agonist and antagonist occurs during movements that require precise control. A muscle functions as a **synergist** if it contracts at the same time as the agonist while cooperating in producing the movement. Synergistic action can aid in producing a movement (e.g., the activity of both flexor carpi ulnaris and extensor carpi ulnaris are used in producing ulnar deviation of the wrist); eliminating unwanted movements (e.g., the activity of wrist extensors prevents flexion of the wrist when finger flexors contract in closing the hand); or stabilizing proximal joints (e.g., isometric contractions of muscles of the forearm, upper arm, shoulder, and trunk accompany a forceful grip of the hand).

1.3 PERIPHERAL NERVOUS SYSTEM COMPONENTS FOR THE CONTROL OF MOVEMENT

We can identify the components of the nervous system that are predominantly involved in the control of motor function and discuss the probable roles for each of them. It is important to appreciate that even the simplest reflex or voluntary movement requires the interaction of multiple levels of the nervous system (Fig. 2)

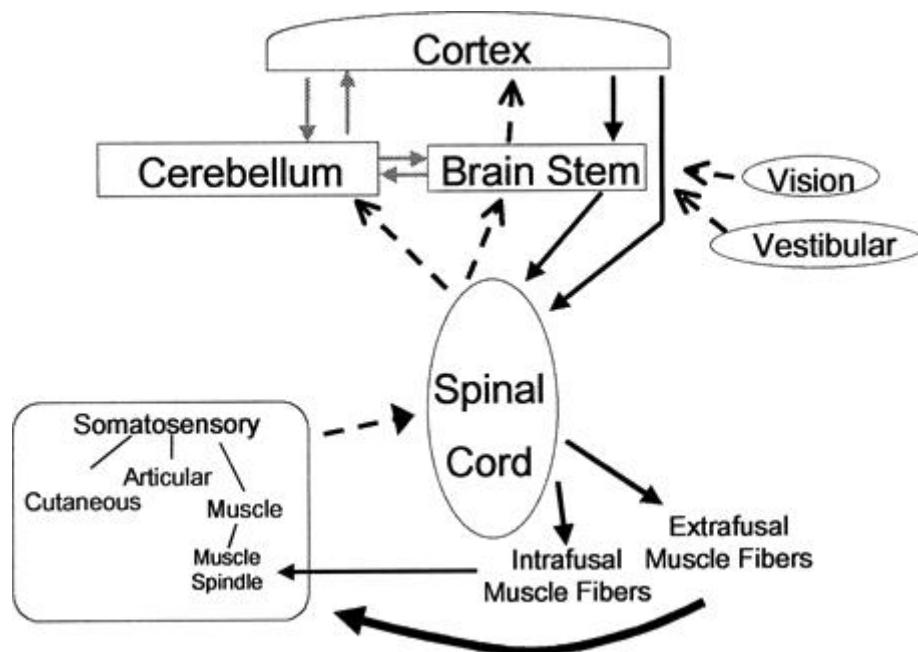


Fig. 2. **Motor control system.** Alpha motor neurons are the final common path for motor control. Peripheral sensory input and spinal cord tract signals that descend from the brainstem and cerebral cortex influence the motor neurons. The cerebellum and basal ganglia contribute to motor control by modifying brainstem and cortical activity

The motor neurons in the spinal cord and cranial nerve nuclei, plus their axons and muscle fibers, constitute the **final common path**, the route by which all central nervous activity influences the skeletal muscles. The motor neurons located in the ventral horns of the spinal gray matter and brainstem nuclei are influenced by both local reflex circuitry and by pathways that descend from the brainstem and cerebral cortex. The brainstem-derived pathways include the rubrospinal, vestibulospinal, and reticulospinal tracts; the cortical pathways are the corticospinal and corticobulbar tracts. Although some of the cortically derived axons terminate

directly on motor neurons, most of the axons of the cortical and the brainstem-derived tracts terminate on interneurons, which then influence motor neuron function. The outputs of the basal ganglia of the brain and cerebellum provide fine-tuning of cortical and brainstem influences on motor neuron functions.

Alpha Motor Neurons Are the Final Common Path for Motor Control

Motor neurons segregate into two major categories, alpha and gamma. **Alpha motor neurons** innervate the **extrafusal muscle fibers**, which are responsible for force generation.

Gamma motor neurons innervate the **intrafusal muscle fibers**), which are components of the muscle spindle. An alpha motor neuron controls several muscle fibers, 10 to 1,000, depending on the muscle. The term **motor unit** describes a motor neuron, its axon, the branches of the axon, the neuromuscular junction synapses at the distal end of each axon branch, and all of the extrafusal muscle fibers innervated by that motor neuron. When a motor neuron generates an action potential, all of its muscle fibers are activated.

Alpha motor neurons can be separated into two populations according to their cell body size and axon diameter. The larger cells have a high threshold to synaptic stimulation, have fast action potential conduction velocities, and are active in high-effort force generation. They innervate fast-twitch, high-force but fatigable muscle fibers. The smaller alpha motor neurons have lower thresholds to synaptic stimulation, conduct action potentials at a somewhat slower velocity, and innervate slow-twitch, low-force, fatigue-resistant muscle fibers. The muscle fibers of each motor unit are homogeneous, either fast twitch or slow-twitch. This property is ultimately determined by the motor neuron. Muscle fibers that are denervated secondary to disease of the axon or nerve cell body may change twitch type if reinnervated by an axon sprouted from a different twitch-type motor neuron.

The organization into different motor unit types has important functional consequences for the production of smooth, coordinated contractions. The smallest neurons have the lowest threshold and are, therefore, activated first when synaptic activity is low. These produce sustainable, relatively low-force tonic contractions

in slow twitch, fatigue-resistant muscle fibers. If additional force is required, synaptic drive from higher centers increases the action potential firing rate of the initially activated motor neurons and then activates additional motor units of the same type. If yet higher force levels are needed, the larger motor neurons are recruited, but their contribution is less sustained as a result of fatigability. This orderly process of motor unit **recruitment** obeys what is called the **size principle**—the smaller motor neurons are activated first. A logical corollary of this arrangement is that muscles concerned with endurance, such as antigravity muscles, contain predominantly slow-twitch muscle fibers in accordance with their function of continuous postural support. Muscles that contain predominantly fast-twitch fibers, including many physiological flexors, are capable of producing high-force contractions.

Afferent Muscle Innervation Provides Feedback for Motor Control

The muscles, joints, and ligaments are innervated with sensory receptors that inform the central nervous system about body position and muscle activity. Skeletal muscles contain muscle spindles, Golgi tendon organs, free nerve endings, and some Pacinian corpuscles. Joints contain Ruffini endings and Pacinian corpuscles; joint capsules contain nerve endings; ligaments contain Golgi tendon-like organs. Together, these are the **proprioceptors**, providing sensation from the deep somatic structures. These sensations, which may not reach a conscious level, include the position of the limbs and the force and speed of muscle contraction. They provide the feedback that is necessary for the control of movements.

Muscle spindles provide information about the muscle length and the velocity at which the muscle is being stretched. Golgi tendon organs provide information about the force being generated. Spindles are located in the mass of the muscle, in parallel with the extrafusal muscle fibers. Golgi tendon organs are located at the junction of the muscle and its tendons, in series with the muscle fibers (Fig. 3).

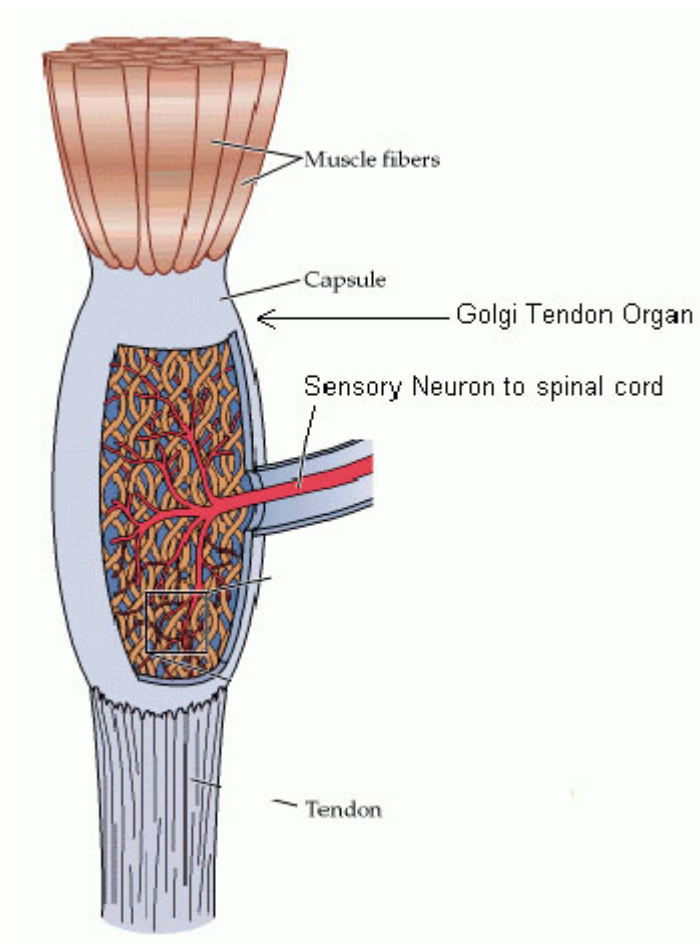


Fig. 3. An enlarged Golgi tendon organ. The sensory receptor endings interdigitate with the collagen fibers of the tendon. The axon is type Ib.

Muscle Spindles. Muscle spindles are sensory organs found in almost all of the skeletal muscles. They occur in greatest density in small muscles serving fine movements, such as those of the hand, and in the deep muscles of the neck. The muscle spindle, named for its long fusiform shape, is attached at both ends to extrafusal muscle fibers.

Within the spindle's expanded middle portion is a fluid filled capsule containing 2 to 12 specialized striated muscle fibers entwined by sensory nerve terminals. These intrafusal muscle fibers, about 300 μm long, have contractile filaments at both ends. The noncontractile midportion contains the cell nuclei (Fig. 4).

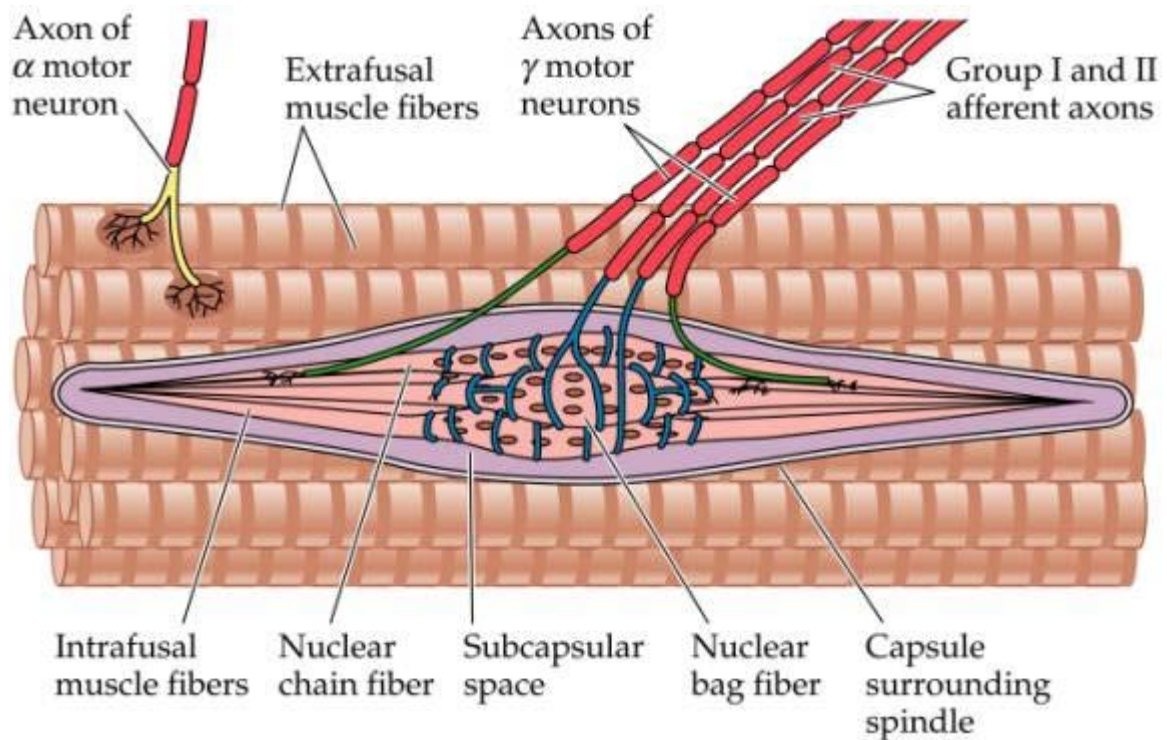


Fig. 4. Muscle spindles-consist of specialized intrafusal muscle fibers distributed among ordinary (extrafusal) muscle fibers; detect changes in muscle length.

Gamma motor neurons innervate the contractile elements. There are two types of intrafusal fibers: **nuclear bag fibers**, named for the large number of nuclei packed into the midportion, and **nuclear chain fibers**, in which the nuclei are arranged in a longitudinal row. There are about twice as many nuclear chain fibers as nuclear bag fibers per spindle. The nuclear bag type fibers are further classified as bag1 and bag2, based on whether they respond best in the dynamic or static phase of muscle stretch, respectively. Sensory axons surround both the noncontractile midportion and paracentral region of the contractile ends of the intrafusal fiber. The sensory axons are categorized as **primary (type Ia)** and **secondary (type II)**. The axons of both types are myelinated. Type Ia axons are larger in diameter (12 to 20 μm) than type II axons (6 to 12 μm) and have faster conduction velocities. Type Ia axons have spiral shaped endings that wrap around the middle of the intrafusal muscle fiber (see Fig. 5).

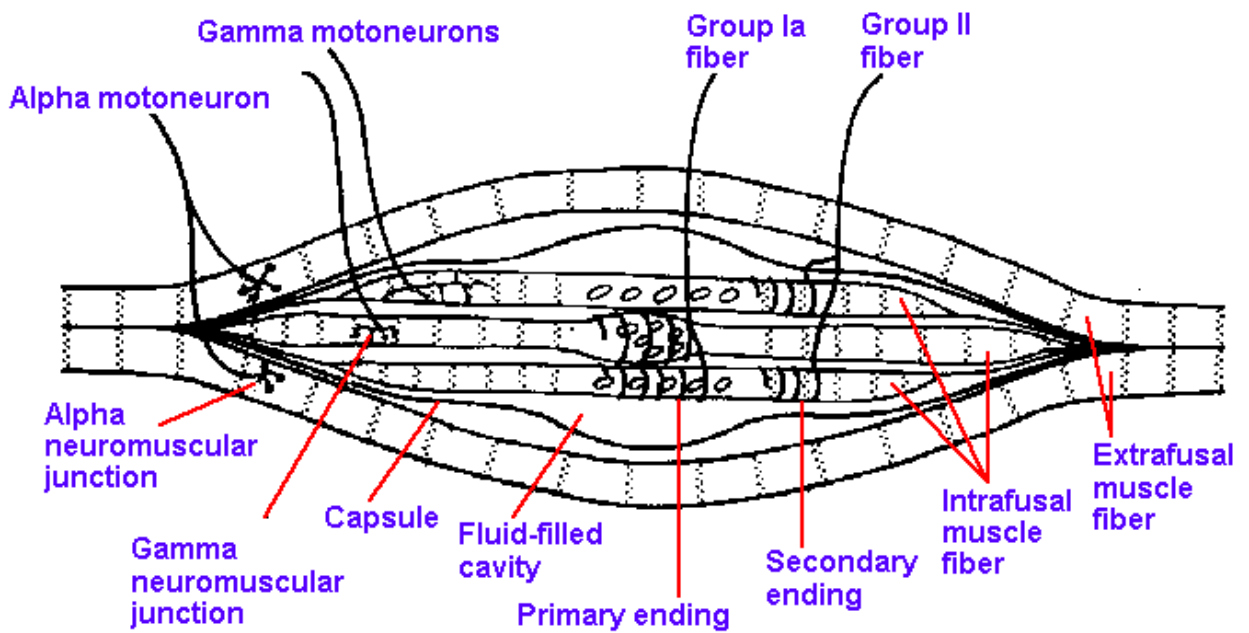


Fig. 5. This enlarged spindle shows nuclear bag and nuclear chain types of intrafusal fibers; afferent innervation by Ia axons, which provide primary endings to both types of fibers; type II axons, which have secondary endings mainly on chain fibers; and motor innervation by the two types of gamma motor axons, static and dynamic

Both nuclear bag and nuclear chain fibers are innervated by type Ia axons. Type II axons innervate mainly nuclear chain fibers and have nerve endings that are located along the contractile components on either side of the type Ia spiral ending. The nerve endings of both primary and secondary sensory axons of the muscle spindles respond to stretch by generating action potentials that convey information to the central nervous system about changes in muscle length and the velocity of length change (Fig 7). The primary endings temporarily cease generating action potentials during the release of a muscle stretch (Fig. 8).

Golgi Tendon Organs. **Golgi tendon organs** (GTOs) are 1 mm long, slender receptors encapsulated within the tendons of the skeletal muscles (see Fig. 6 A and B). The distal pole of a GTO is anchored in collagen fibers of the tendon.

The proximal pole is attached to the ends of the extrafusal muscle fibers. This arrangement places the GTO in series with the extrafusal muscle fibers such that contractions of the muscle stretch the GTO

A large-diameter, myelinated type Ib afferent axon arises from each GTO. These axons are slightly smaller in diameter than the type Ia variety, which innervate the muscle spindle. Muscle contraction stretches the GTO and generates action potentials in type Ib axons. The GTO output provides information to the central nervous system about the force of the muscle contraction.

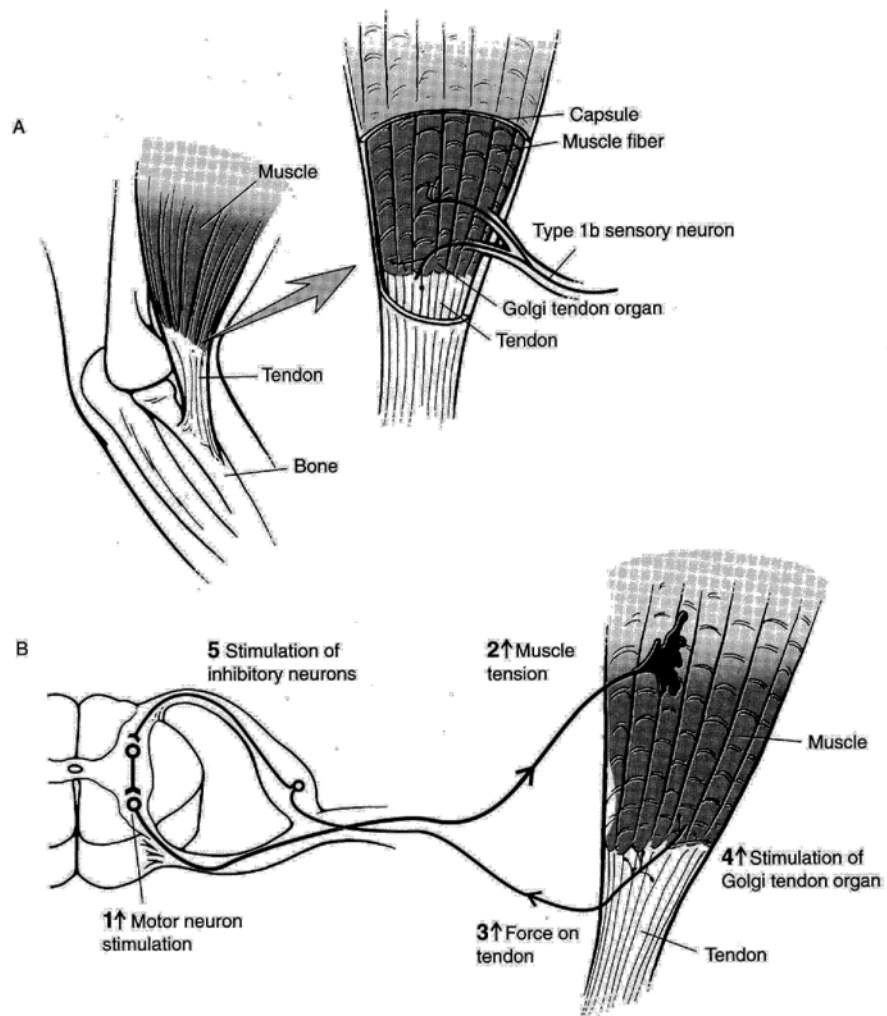


Fig 6 (A) The **Golgi tendon organ** (GTO) is located at the muscle –tendon junction. (B) When there is tension developed at this site, the GTO sends information into the spinal cord via type Ib afferent neurons. The sensory input from the GTO facilitates a relaxation of the muscle via stimulation of inhibitory interneurons. This response is known as the inverse stretch reflex or autogenic inhibition.

Information entering the spinal cord via type Ia and Ib axons is directed to many targets, including the spinal interneurons that give rise to the **spinocerebellar tracts**. These tracts convey information to the cerebellum about the status of muscle length and tension.

Gamma Motor Neurons. Alpha motor neurons innervate the extrafusal muscle fibers, and gamma motor neurons innervate the intrafusal fibers. Cells bodies of both alpha and gamma motor neurons reside in the ventral horns of the spinal cord and in nuclei of the cranial motor nerves.

Nearly one third of all motor nerve axons are destined for intrafusal muscle fibers. This high number reflects the complex role of the spindles in motor system control. Intrafusal muscle fibers likewise constitute a significant portion of the total number of muscle cells, yet they contribute little or nothing to the total force generated when the muscle contracts. Rather, the contractions of intrafusal fibers play a modulating role in sensation, as they alter the length and, thereby, the sensitivity of the muscle spindles. Even when the muscle is at rest, the muscle spindles are slightly stretched, and type Ia afferent nerves exhibit a slow discharge of action potentials.

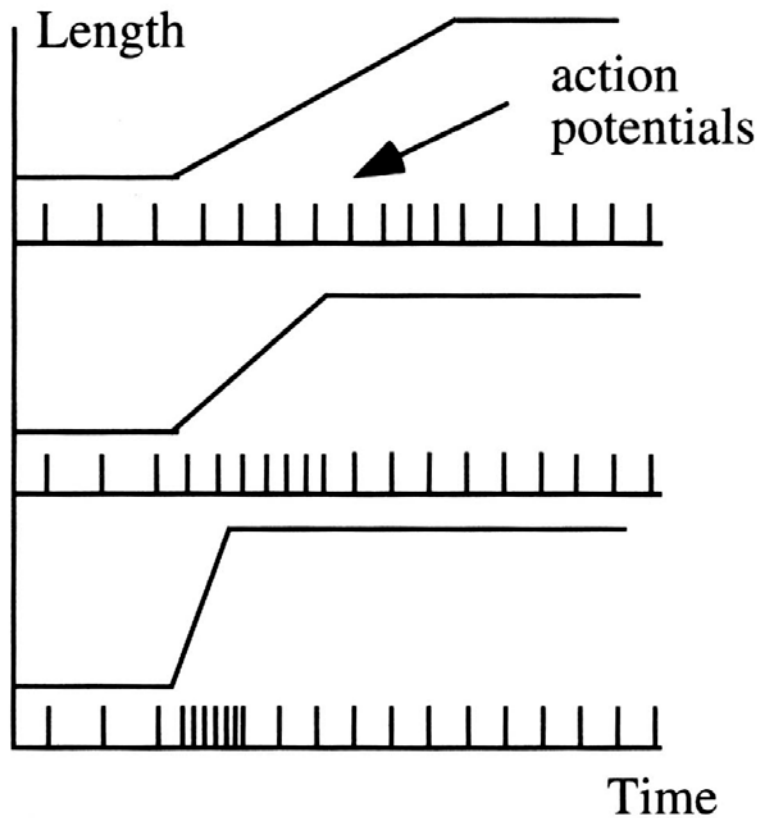


Fig. 7 Typical responses of a primary spindle ending to an externally imposed muscle stretch at different velocities. Note that the response increases with muscle length and with velocity of stretch.

Contraction of the muscle increases the firing rate in type Ib axons from Golgi tendon organs, whereas type Ia axons temporarily cease or reduce firing because the shortening of the surrounding extrafusal fibers unloads the intrafusal muscle fibers. If a load on the-spindle were reinstated, the Ia nerve endings would resume their sensitivity to stretch. The role of the gamma motor neurons is to “reload” the spindle during muscle contraction by activating the contractile elements of the intrafusal fibers. This is accomplished by coordinated activation of the alpha and gamma motor neurons during muscle contraction (see Fig. 7).

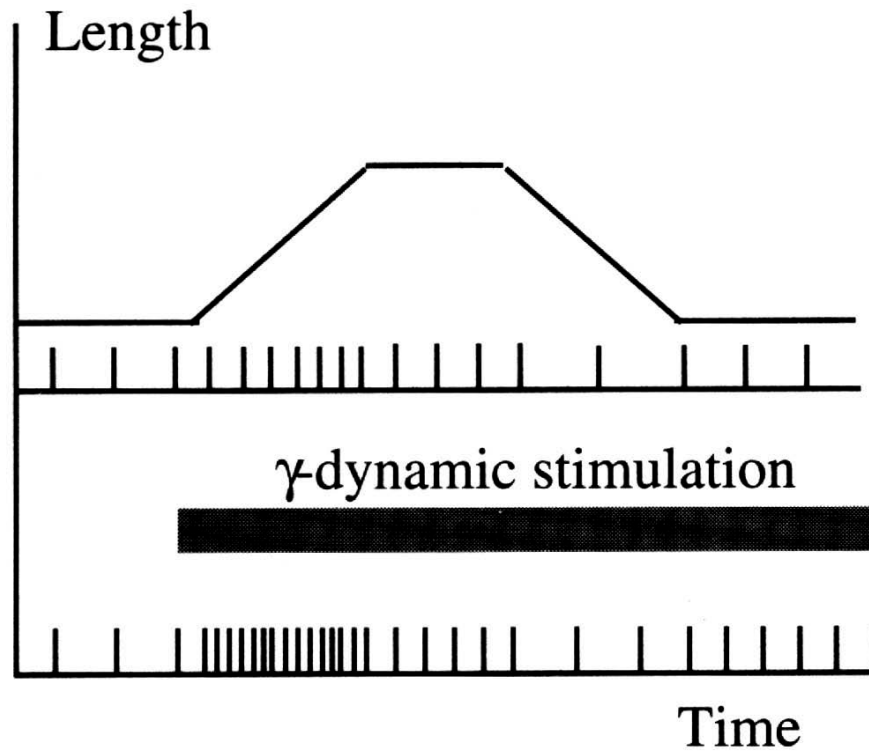


Fig. 8. The effects of activation of dynamic Gamma motor neurons on response of a primary spindle ending to muscle stretch and shortening. In lower graph, a **Gamma** - dynamic stimulation was applied during the same changes in muscle length.

The gamma motor neurons and the intrafusal fibers they innervate are traditionally referred to as the **fusimotor system**. Axons of the gamma neurons terminate in one of two types of endings, each located distal to the sensory endings on the striated poles of the spindle's muscle fibers (see Fig. 9).

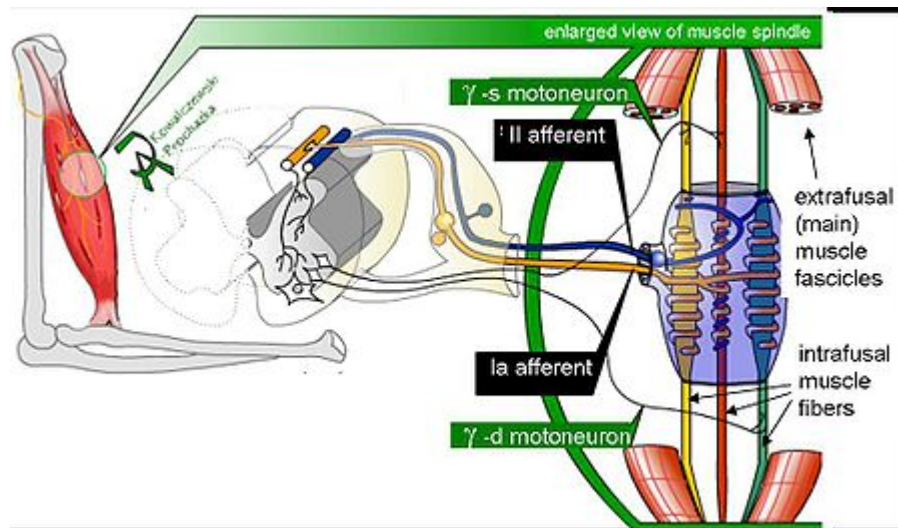


Fig. 9. The spindle is a stretch receptor with its own motor supply comprised of several intrafusal muscle fibres. The sensory endings of a primary (group Ia) afferent and a secondary (group II) afferent coil around the non-contractile central portions of the intrafusal fibres. Gamma motoneurons activate the intrafusal muscle fibres, changing the resting firing rate and stretch-sensitivity of the afferents.

The nerve terminals are either plate endings or trail endings; each intrafusal fiber has only one of these two types of endings. **Plate endings** occur predominantly on bag1 fibers (dynamic), whereas **trail endings**, primarily on chain fibers, are also seen on bag2 (static) fibers. This arrangement allows for largely independent control of the nuclear bag and nuclear chain fibers in the spindle.

Gamma motor neurons with plate endings are designated **dynamic** and those with trail endings are designated **static**.

This functional distinction is based on experimental findings showing that stimulation of gamma neurons with plate endings enhanced the response of type Ia sensory axons to stretch, but only during the dynamic (muscle length changing) phase of a muscle stretch. During the static phase of the stretch (muscle length increase maintained) stimulation of the gamma neurons with trail endings enhanced the response of type II sensory axons. Static gamma neurons can affect the responses of both types Ia and II sensory axons; dynamic gamma neurons affect the response of only type Ia axons. These differences suggest that the motor

system has the ability to monitor muscle length more precisely in some muscles and the speed of contraction in others.

1.4 THE SPINAL CORD IN THE CONTROL OF MOVEMENT

Muscles interact extensively in the maintenance of posture and the production of coordinated movement. The circuitry of the spinal cord automatically controls much of this interaction. Sensory feedback from muscles reaches motor neurons of related muscles and, to a lesser degree, of more distant muscles. In addition to activating local circuits, muscles and joints transmit sensory information up the spinal cord to higher centers. This information is processed and can be relayed back to influence spinal cord circuits.

The Structural Arrangement of Spinal Motor Systems Correlates With Function

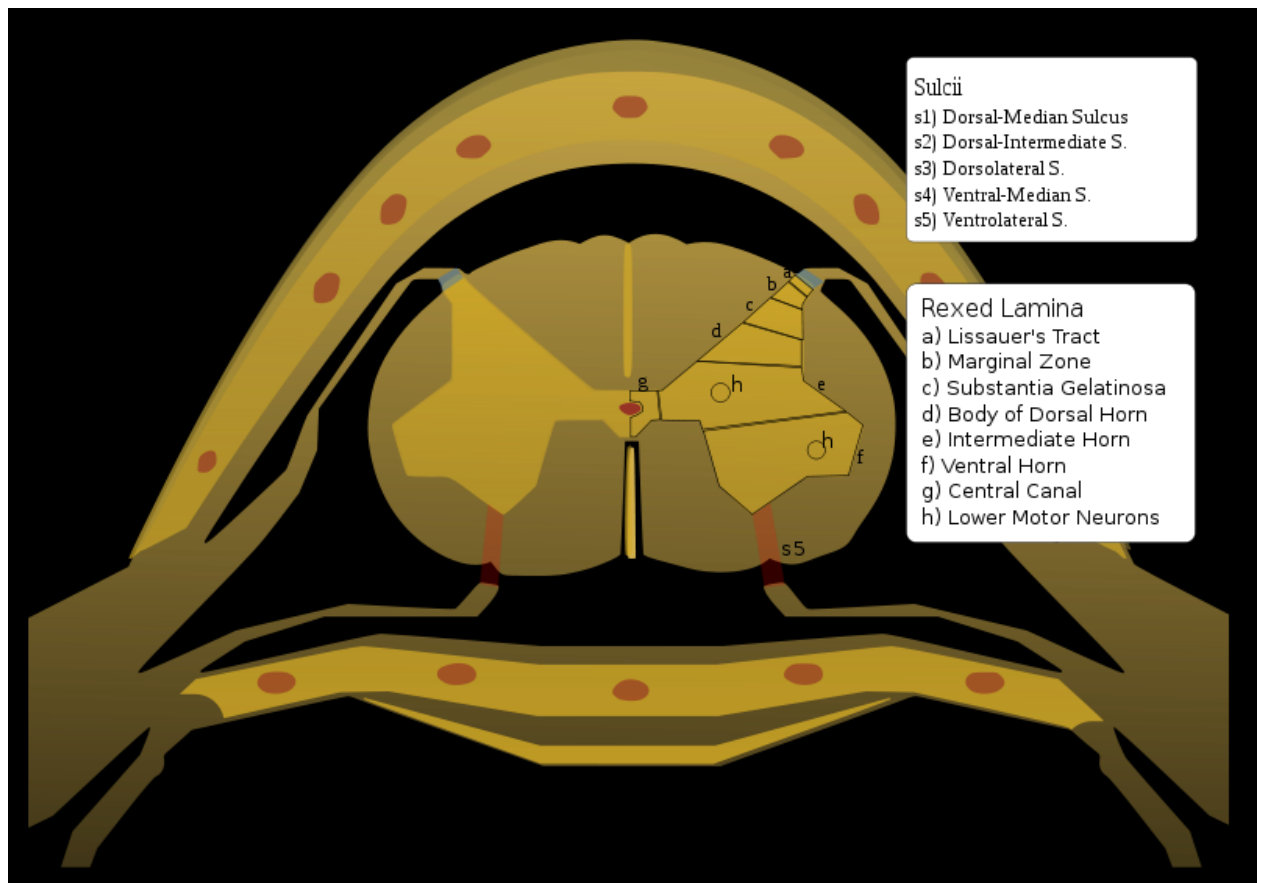


Fig. 10. **Spinal cord motor neuron pools.** Motor neurons controlling axial, girdle, and limb muscles are grouped in pools oriented in a medial-to-lateral fashion. Limb flexor and extensor motor neurons also segregate into pools.

The cell bodies of the spinal cord motor neurons are grouped into pools in the ventral horns. A **pool** consists of the motor neurons that serve a particular muscle.

The number of motor neurons that control a muscle varies in direct proportion to the delicacy of control required. The motor neurons are organized so that those innervating the axial muscles are grouped medially and those innervating the limbs are located laterally (Fig. 10).

The lateral limb motor neuron areas are further organized so that proximal actions, such as girdle movements, are controlled from relatively medial locations, while distal actions, such as finger movements, are located the most laterally. Neurons innervating flexors and extensors are also segregated. A motor neuron pool may extend over several spinal segments in the form of a column of motor neurons. This is mirrored by the innervation serving a single muscle emerging from the spinal cord in two or even three adjacent spinal nerve root levels.

A physiological advantage to such an arrangement is that injury to a single nerve root, as could be produced by herniation of an intervertebral disk, will not completely paralyze a muscle.

A zone between the medial and lateral pools contains interneurons that project to limb motor neuron pools ipsilaterally and axial pools bilaterally. Between the spinal cord's dorsal and ventral horns lies the intermediate zone, which contains an extensive network of interneurons that interconnect motor neuron pools (see Fig. 10). Some interneurons make connections in their own cord segment; others have longer axon projections that travel in the white matter to terminate in other segments of the spinal cord. These longer axon interneurons, termed **propriospinal cells**, carry information that aids coordinated movement. The importance of spinal cord interneurons is reflected in the fact that they comprise the majority of neurons in the spinal cord and provide the majority of the motor neuron synapses.

The Spinal Cord Mediates Reflex Activity

The spinal cord contains neural circuitry to generate **reflexes**, stereotypical actions produced in response to a peripherally applied stimulus. One function of a reflex is to generate a rapid response. A familiar example is the rapid, involuntary

withdrawal of a hand after touching a danger. This type of reflex protects the organism before higher CNS levels identify the problem. Some reflexes are simple, others much more complex. Even the simplest requires coordinated action in which the agonist contracts while the antagonist relaxes. The functional unit of a reflex consists of a sensor, an afferent pathway, an integrating center, an efferent pathway, and an effector. The sensory receptors for spinal reflexes are the proprioceptors and cutaneous receptors. Impulses initiated in these receptors travel along afferent nerves to the spinal cord, where interneurons and motor neurons constitute the integrating center. The final common path, or motor neurons, make up the efferent pathway to the effector organs, the skeletal muscles. The responsiveness of such a functional unit can be modulated by higher motor centers acting through descending pathways to facilitate or inhibit its activation.

Study of the three types of spinal reflexes—the myotatic, the inverse myotatic, and the flexor withdrawal— provides a basis for understanding the general mechanism of reflexes.

The Myotatic (Muscle Stretch) Reflex. Stretching or elongating a muscle—such as when the patellar tendon is tapped with a reflex hammer or when a quick change in posture is made—causes it to contract within a short time period.

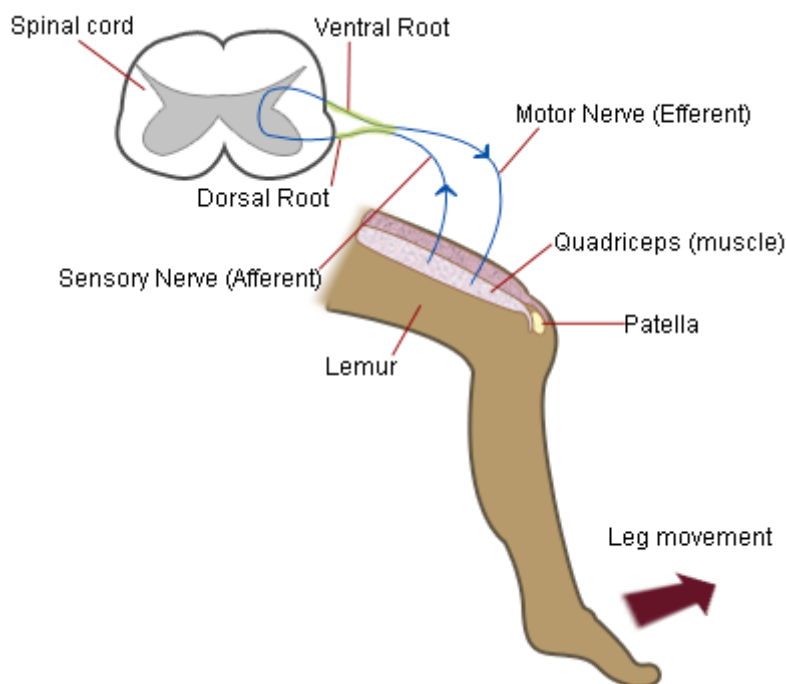


Fig 11 Myotatic reflex circuitry.

The period between the onset of a stimulus and the response, the **latency period**, is on the order of 30 msec for a knee-jerk reflex in a human. This response, called the **myotatic** or **muscle stretch reflex**, is due to monosynaptic circuitry, where an afferent sensory neuron synapses directly on the efferent motor neuron (Fig 11). The stretch activates muscle spindles. Type Ia sensory axons from the spindle carry action potentials to the spinal cord, where they synapse directly on motor neurons of the same (homonymous) muscle that was stretched and on motor neurons of synergistic (heteronymous) muscles. These synapses are excitatory and utilize glutamate as the neurotransmitter.

Monosynaptic type Ia synapses occur predominantly on alpha motor neurons; gamma motor neurons seemingly lack such connections. Collateral branches of type Ia axons also synapse on interneurons, whose action then inhibits motor neurons of antagonist muscles (see Fig 12 A, B).

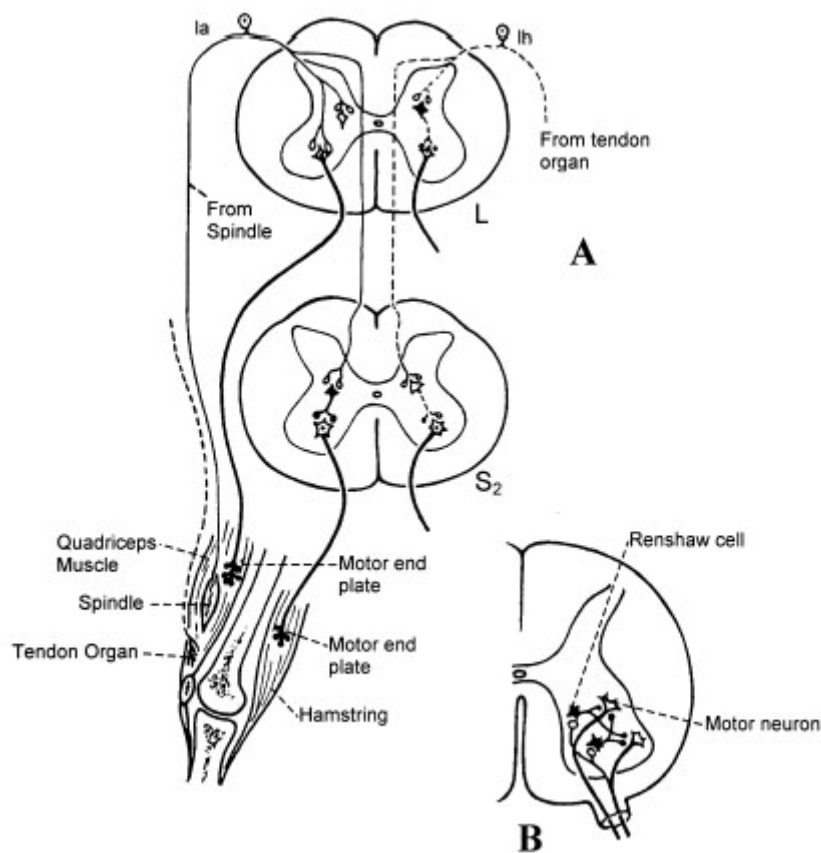


Fig 12 Myotatic or stretch reflex. A. Solid lines and open structures are facilitatory; broken lines and solid structures are inhibitory. After stretch of the quadriceps, the

muscle spindle (Ia fibers) stimulates motor cells in the spinal cord to contract the muscle, while GTO (Ib fibers) inhibit contraction in the quadriceps. B. Proposed mechanism for inhibition.

This synaptic pattern, called **reciprocal inhibition** (Fig 13), serves to coordinate muscles of opposing function around a joint. Secondary (type II) spindle afferent fibers also synapse with homonymous motor neurons, providing excitatory input through both monosynaptic and polysynaptic pathways.

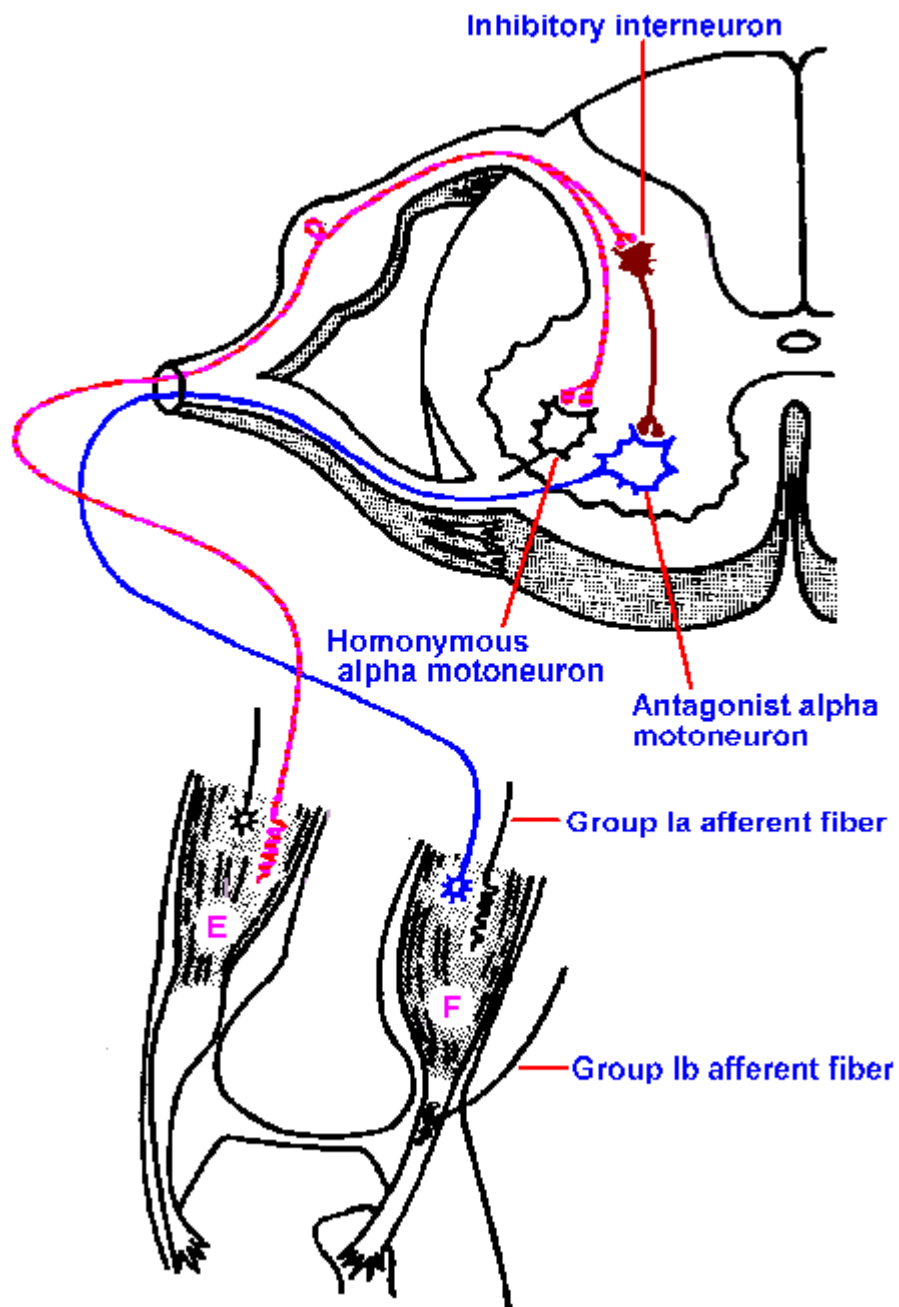


Fig 13. Reciprocal innervation. Ia afferent axons from the muscle spindle make excitatory monosynaptic contact with homonymous motor neurons and with

inhibitory interneurons that synapse on motor neurons of antagonist muscles. F and E indicate flexor and extensor muscles.

Golgi tendon organ input via type Ib axons has an inhibitory influence on homonymous motor neurons.

The myotatic reflex has two components: a phasic part, exemplified by tendon jerks, and a tonic part, thought to be important for maintaining posture. The phasic component is more familiar. These components blend together, but either one may predominate, depending on whether other synaptic activity, such as from cutaneous afferent neurons or pathways descending from higher centers, influences the motor response. Primary spindle afferent fibers probably mediate the tendon jerk, with secondary afferent fibers contributing mainly to the tonic phase of the reflex. The myotatic reflex performs many functions. At the most general level, it produces rapid corrections of motor output in the moment-to-moment control of movement. It also forms the basis for postural reflexes, which maintain body position despite a varying range of loads and/or external forces on the body.(Fig 14)

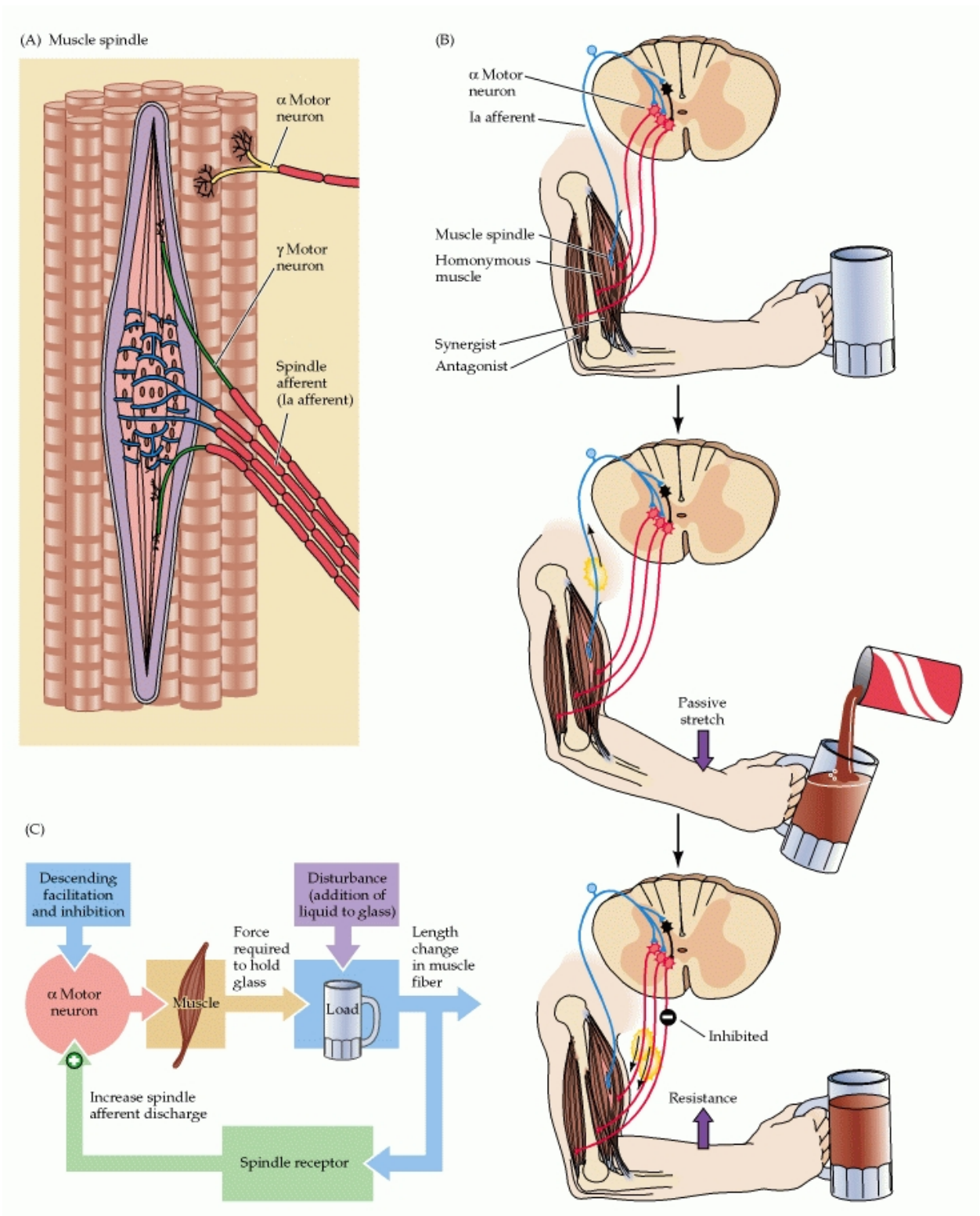


Fig 14 Stretch reflex circuitry. (A) Diagram of muscle spindle, the sensory receptor that initiates the stretch reflex. (B) Stretching a muscle spindle leads to increased activity in Ia afferents and an increase in the activity of α motor neurons that innervate the same muscle. Ia afferents also excite the motor neurons that innervate synergistic muscles, and inhibit the motor neurons that innervate antagonists. (C) The stretch reflex operates as a negative feedback loop to regulate muscle length.

The Inverse Myotatic Reflex. The active contraction of a muscle also causes reflex inhibition of the contraction. This response is called the **inverse myotatic reflex** because it produces an effect that is opposite to that of the myotatic reflex. Active muscle contraction stimulates Golgi tendon organs, producing action potentials in the type Ib afferent axons. Those axons synapse on inhibitory interneurons that influence homonymous and heteronymous motor neurons and on excitatory interneurons that influence motor neurons of antagonists (Fig 6 B).

The function of the inverse myotatic reflex appears to be a tension feedback system that can adjust the strength of contraction during sustained activity. The inverse myotatic reflex does not have the same function as reciprocal inhibition. Reciprocal inhibition acts primarily on the antagonist, while the inverse myotatic reflex acts on the agonist.

The inverse myotatic reflex, like the myotatic reflex, has a more potent influence on the physiological extensor muscles than on the flexor muscles, suggesting that the two reflexes act together to maintain optimal responses in the antigravity muscles during postural adjustments. Another hypothesis about the conjoint function is that both of these muscles by regulating muscle stiffness.

The Flexor Withdrawal Reflex. Cutaneous stimulation— such as touch, pressure, heat, cold, or tissue damage—can elicit a **flexor withdrawal reflex**. This reflex consists of a contraction of flexors and a relaxation of extensors in the stimulated limb. The action may be accompanied by a contraction of the extensors on the contralateral side. The axons of cutaneous sensory receptors synapse on interneurons in the dorsal horn. Those interneurons act ipsilaterally to excite the motor neurons of flexor muscles and inhibit those of extensor muscles. Collaterals of interneurons cross the midline to excite contralateral extensor motor neurons and inhibit flexors (Fig. 15.).

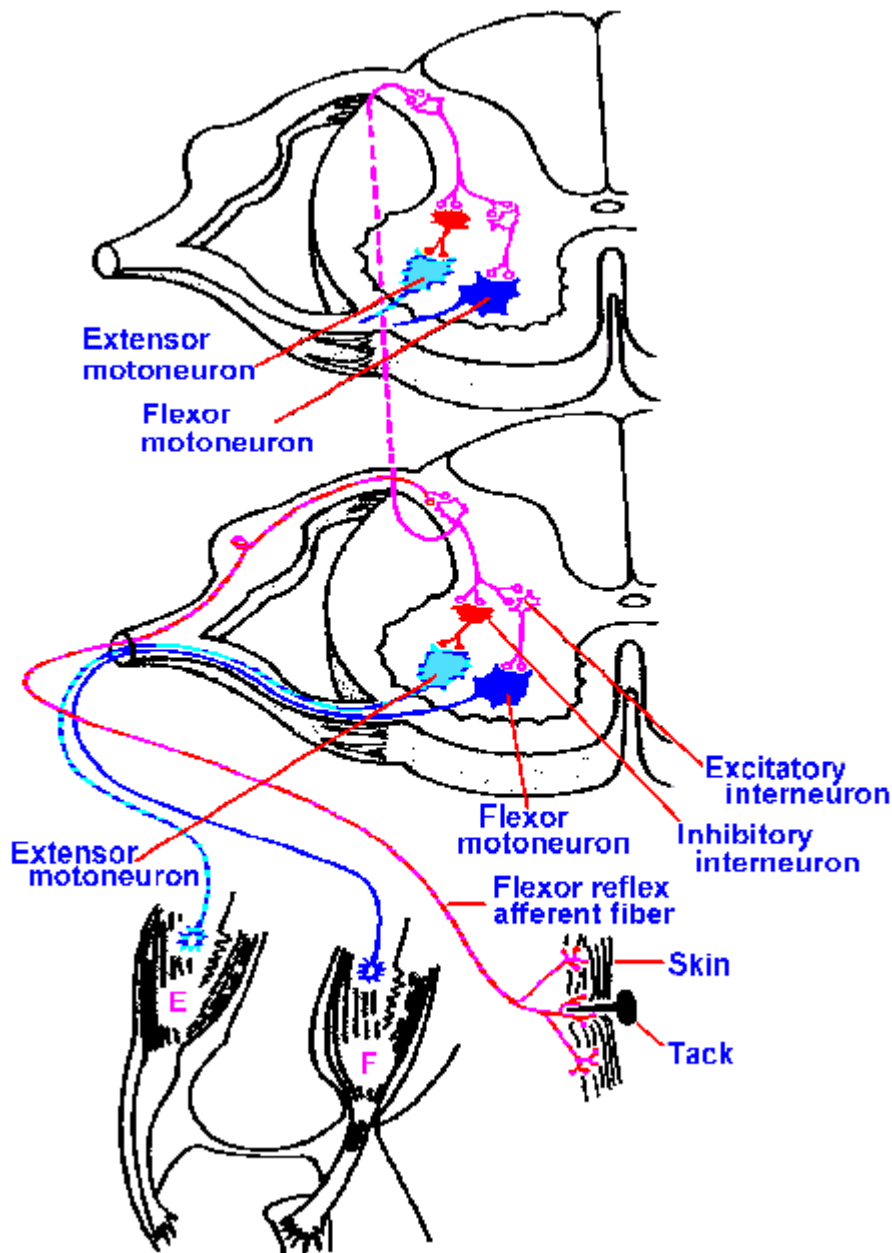


Fig 15. Flexion reflex. A circuit diagram of the flexion reflex showing afferent fibers from skin, interneurons, and flexor alpha-motoneurons in two spinal cord segments. Note that some interneurons are intersegmental. F and E indicate flexor and extensor muscles.

There are two types of flexor withdrawal reflexes: those that result from innocuous stimuli and those that result from potentially injurious stimulation. The first type produces a localized flexor response accompanied by slight or no limb withdrawal; the second type produces widespread flexor contraction throughout the limb and abrupt withdrawal.

The function of the first type of reflex is less obvious, but may be a general mechanism for adjusting the movement of a body part when an obstacle is detected by cutaneous sensory input. The function of the second type is protection of the individual. The endangered body part is rapidly removed, and postural support of the opposite side is strengthened if needed (e.g., if the foot is being withdrawn). Collectively, these reflexes provide for stability and postural support (the myotatic and inverse myotatic) and mobility (flexor withdrawal). The reflexes provide a foundation of automatic responses on which more complicated voluntary movements are built

The Spinal Cord Can Produce Basic Locomotor Actions

For locomotion, muscle action must occur in the limbs, but the posture of the trunk must also be controlled to provide a foundation from which the limb muscles can act. For example, when a human takes a step forward, not only must the advancing leg flex at the hip and knee, the opposite leg and bilateral truncal muscles must also be properly activated to prevent collapse of the body as weight is shifted from one leg to the other. Responsibility for the different functions that come together in successful locomotion is divided between several levels of the central nervous system. Studies in experimental animals, mostly cats, have demonstrated that the spinal cord contains the capability for generating basic locomotor movements. This neural circuitry, called a **central pattern generator**, can produce the alternating contraction of limb flexors and extensors that is needed for walking. It has been shown experimentally that application of an excitatory amino acid like glutamate to the spinal cord produces rhythmic action potentials in motor neurons. Each limb has its own pattern generator, and the actions of different limbs are then coordinated. The normal strategy for generating basic locomotion engages central pattern generators and uses both sensory feedback-and efferent impulses from higher motor control centers for the refinement of control.

Spinal Cord Injury Alters Voluntary and Reflex Motor Activity

When the spinal cord of a human or other mammal is severely injured, voluntary and reflex movements are immediately lost caudal to the level of injury. This acute impairment of function is called **spinal shock**. The loss of voluntary motor control is termed **plegia**, and the loss of reflexes is termed **areflexia**. Spinal shock may last from days to months, depending on the severity of cord injury. Reflexes tend to return, as may some degree of voluntary control. As recovery proceeds, myotatic reflexes become hyperactive, as demonstrated by an excessively vigorous response to tapping the muscle tendon with a reflex hammer. Tendon tapping, or even limb repositioning that produces a change in the muscle length, may also provoke **clonus**, a condition characterized by repetitive contraction and relaxation of a muscle in an oscillating fashion every second or so, in response to a single stimulus. Flexor withdrawal reflexes may also reappear and be provoked by lesser stimuli than would be normally required. The acute loss and eventual over activity of all of these reflexes results from the lack of influence of the neural tracts that descend from higher motor control centers to the motor neurons and associated interneuron pools.

1.5 SUPRASPINAL INFLUENCES ON MOTOR CONTROL

Descending signals from the cervical spinal cord, brainstem, and cortex can influence the rate of motor neuron firing and the recruitment of additional motor neurons to increase the speed and force of muscle contraction. The influence of higher motor control centers is illustrated by a walking dog whose right and left limbs show alternating contractions and then change to a running pattern in which both sides contract in synchrony.

The brainstem contains the neural circuitry for initiating locomotion and for controlling posture. The maintenance of posture requires coordinated activity of both axial and limb muscles in response to input from proprioceptors and spatial position sensors, such as the inner ear. Cerebral cortex input through the corticospinal system is necessary for the control of fine individual movements of the distal limbs and digits. Each higher level of the nervous system acts on lower levels to produce appropriate, more refined movements.

The Brainstem Is the Origin of Three Descending Tracts That Influence Movement

Three brainstem nuclear groups give rise to descending motor tracts that influence motor neurons and their associated interneurons. These consist of the **red nucleus**, the **vestibular nuclear complex**, and the **reticular formation**. The other major descending influence on the motor neurons is the corticospinal tract, the only volitional control pathway in the motor system. In most cases, the descending pathways act through synaptic connections on interneurons. The connection is less commonly made directly with motor neurons.

The Rubrospinal Tract. The red nucleus of the mesencephalon receives major input from both the cerebellum and the cerebral cortical motor areas. Output via the **rubrospinal tract** is directed predominantly to contralateral spinal motor neurons that are involved with movements of the distal limbs (Fig.16).

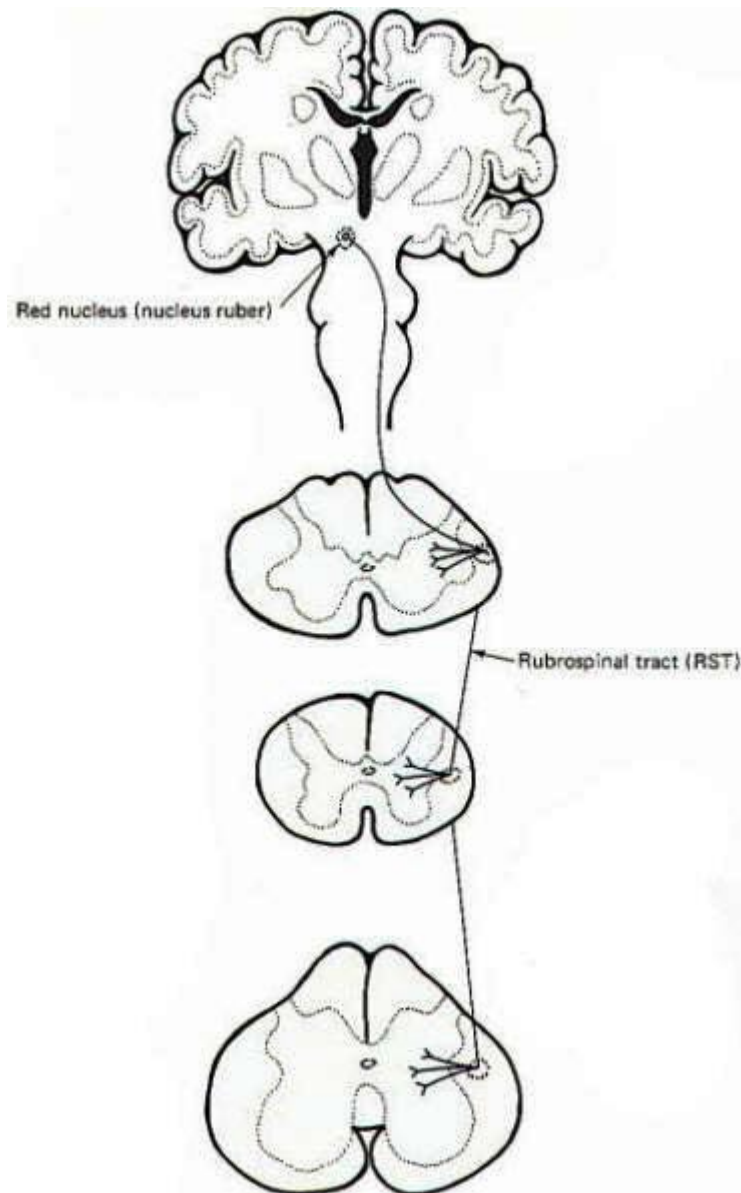


Fig. 16. The rubrospinal tract influences motor neurons controlling distal limb muscles.

The axons of the rubrospinal tract are located in the lateral spinal white matter, just anterior to the corticospinal tract. Rubrospinal action enhances the function of motor neurons innervating limb flexor muscles while inhibiting extensors. This tract may also influence gamma motor neuron function. Electrophysiological studies reveal that many rubrospinal neurons are active during locomotion, with more than half showing increased activity during the swing phase of stepping, when the flexors are most active. This system appears to be important for the production of movement, especially in the distal limbs. Experimental lesions that interrupt rubrospinal axons produce deficits in distal limb flexion, with little

change in more proximal muscles. In higher animals, the corticospinal tract supersedes some of the function of the rubrospinal tract.

The Vestibulospinal Tract. The vestibular system regulates muscular function for the maintenance of posture in response to changes in the position of the head in space and accelerations of the body. There are four major nuclei in the vestibular complex: the **superior, lateral, medial, and inferior vestibular nuclei**. These nuclei, located in the pons and medulla, receive afferent action potentials from the vestibular portion of the ear, which includes the semicircular canals, the utricle, and the saccule. Information about rotatory and linear motions of the head and body are conveyed by this system. The vestibular nuclei are reciprocally connected with the superior colliculus on the dorsal surface of the mesencephalon. Input from the retina is received there and is utilized in adjusting eye position during movement of the head. Reciprocal connections to the vestibular nuclei are also made with the cerebellum and reticular formation.

The chief output to the spinal cord is the **vestibulospinal tract** (Fig 17), which originates predominantly from the lateral vestibular nucleus. The tract's axons are located in the anterior-lateral white matter and carry excitatory action potentials to ipsilateral extensor motor neuron pools, both alpha and gamma. The extensor motor neurons and their musculature are important in the maintenance of posture. Lesions in the brainstem secondary to stroke or trauma may abnormally enhance the influence of the vestibulospinal tract and produce dramatic clinical manifestations.

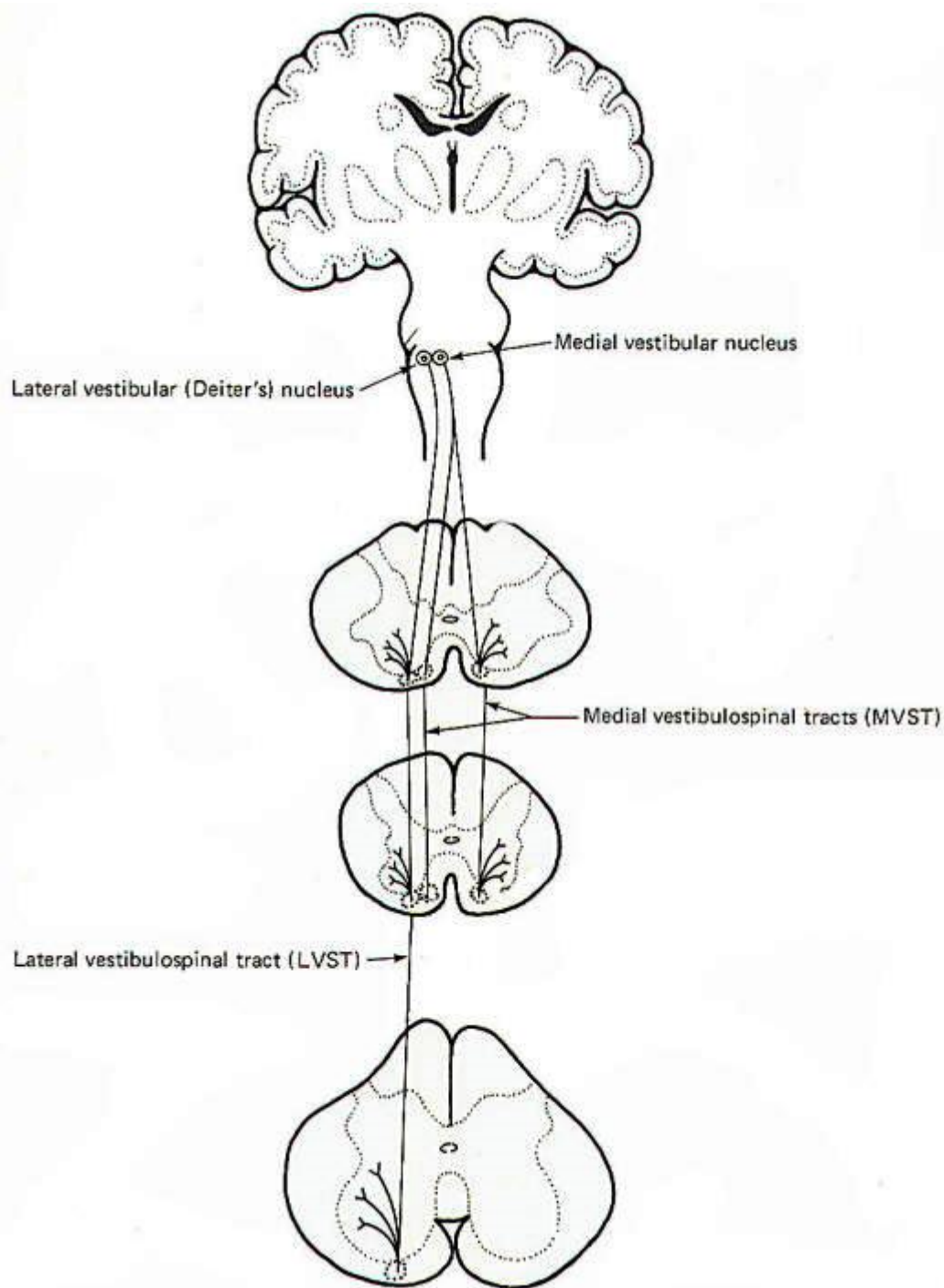


Fig. 17. The vestibulospinal tracts influence motor neurons that control axial and proximal limb muscles.

The Reticulospinal Tract. The reticular formation in the central gray matter core of the brainstem contains many axon bundles interwoven with cells of various shapes and sizes. A prominent characteristic of reticular formation neurons is that their axons project widely in ascending and descending pathways, making multiple synaptic connections throughout the neuraxis. The medial region of the reticular formation contains large neurons that project upward to the thalamus, as well as

downward to the spinal cord. Afferent input to the reticular formation comes from the spinal cord, vestibular nuclei, cerebellum, lateral hypothalamus, globus pallidus, tectum, and sensorimotor cortex. Two areas of the reticular formation are important in the control of motor neurons. The descending tracts arise from the **nucleus reticularis pontis oralis** and **nucleus reticularis pontis caudalis** in the pons, and from the **nucleus reticularis gigantocellularis** in the medulla. The pontine reticular area gives rise to the ipsilateral **pontine reticulospinal tract**, whose axons descend in the medial spinal cord white matter. These axons carry excitatory action potentials to interneurons that influence alpha and gamma motor neuron pools of axial muscles. The medullary area gives rise to the **medullary reticulospinal tract** (Fig 18), whose axons descend mostly ipsilateral in the anterior spinal white matter. These axons have inhibitory influences on interneurons that modulate extensor motor neurons.

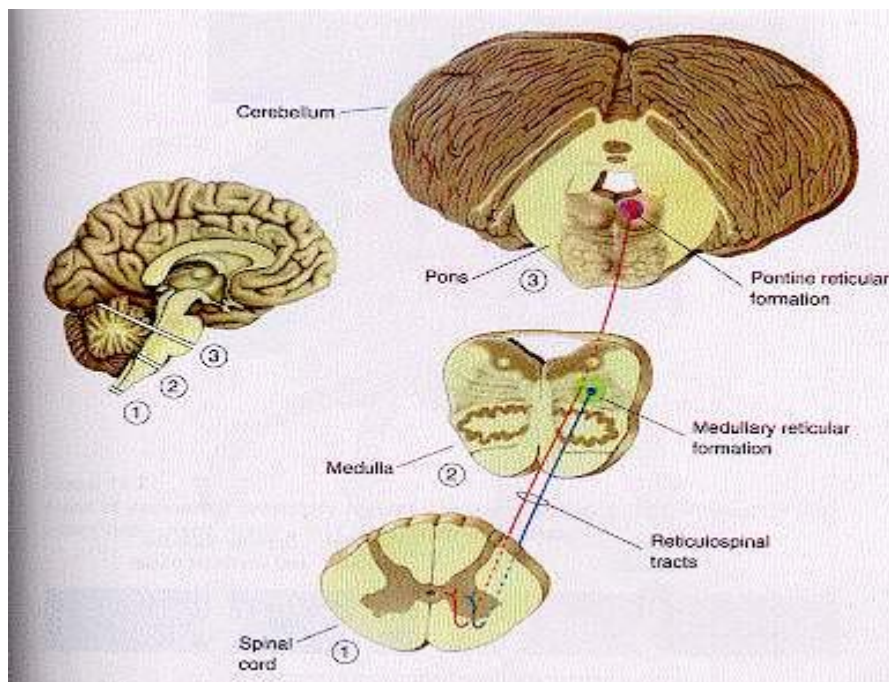


Fig 18. The pontine (medial) and medullary (lateral) reticulospinal tracts. These components of the ventromedial pathway control posture of the trunk and the antigravity muscles of the limbs

The Terminations of the Brainstem Motor Tracts Correlate With Their Functions

The vestibulospinal and reticulospinal tracts descend medially in the spinal cord and terminate in the ventromedial part of the intermediate zone, an area in the gray matter containing propriospinal interneurons. There are also some direct connections with motor neurons of the neck and back muscles and the proximal limb muscles. These tracts are the main CNS pathways for maintaining posture and head position during movement.

The rubrospinal tract descends laterally in the spinal cord and terminates mostly on interneurons in the lateral spinal intermediate zone, but it also has some monosynaptic connections directly on motor neurons to muscles of the distal extremities. This tract supplements the medial descending pathways in postural control and the corticospinal tract for independent movements of the extremities.

In accordance with their medial or lateral distributions to spinal motor neurons, the reticulospinal and vestibulospinal tracts are thought to be most important for the control of axial and proximal limb muscles, whereas the rubrospinal (and corticospinal) tracts are most important for the control of distal limb muscles, particularly the flexors.

CLINICAL Decerebrate Rigidity

A patient with a history of poorly controlled hypertension, a result of noncompliance with his medication, is brought to the emergency department because of sudden collapse and subsequent unresponsiveness. A neurological examination performed about 30 minutes after onset of the collapse shows no response to verbal stimuli. No spontaneous movements of the limbs are observable. A mildly painful stimulus, compression of the soft tissue of the supraorbital ridge, causes immediate extension of the neck and both arms and legs. This posture relaxes within a few seconds after the stimulation is stopped. After the patient is stabilized medically, he undergoes a magnetic resonance imaging (MRI) study of the brain. The study demonstrates a large area of hemorrhage bilaterally in the

upper pons and lower mesencephalon. The posture this patient demonstrated in response to a noxious stimulus is termed **decerebrate rigidity** (Fig. 19). Its presence is associated with lesions of the mesencephalon that isolate the portions of the brainstem below that level from the influence of higher centers. The abnormal posture is a result of extreme activation of the antigravity extensor muscles by the unopposed action of the lateral vestibular nucleus and the vestibulospinal tract. A model of this condition can be produced in experimental animals by a surgical lesion located between the mesencephalon and pons. It can also be shown in experimental animals that a destructive lesion of the lateral vestibular nucleus relieves the rigidity on that side.



Fig. 19. The posture this patient demonstrated in response to a noxious stimulus is termed decerebrate rigidity

Sensory and Motor Systems Work Together to Control Posture

The maintenance of an upright posture in humans requires active muscular resistance against gravity. For movement to occur, the initial posture must be altered by flexing some body parts against gravity. Balance must be maintained during movement, which is achieved by postural reflexes initiated by several key

sensory systems. Vision, the vestibular system, and the somatosensory system are important for postural reflexes.

Somatosensory input provides information about the position and movement of one part of the body with respect to others. The vestibular system provides information about the position and movement of the head and neck with respect to the external world. Vision provides both types of information, as well as information about objects in the external world. Visual and vestibular reflexes interact to produce coordinated head and eye movements associated with a shift in gaze. Vestibular reflexes and somatosensory neck reflexes interact to produce reflex changes in limb muscle activity. The quickest of these compensations occurs at about twice the latency of the monosynaptic myotatic reflex. These response types are termed **long loop reflexes**. The extra time reflects the action of other neurons at different anatomic levels of the nervous system.

1.6 THE ROLE OF THE CEREBRAL CORTEX IN MOTOR CONTROL

The cerebral cortical areas concerned with motor function exert the highest level of motor control. It is difficult to formulate an unequivocal definition of a **cortical motor area**, but three criteria may be used. An area is said to have a motor function if

- Stimulation using very low current strengths elicits movements.
- Destruction of the area results in a loss of motor function.
- The area has output connections going directly or relatively directly (i.e., with a minimal number of intermediate connections) to the motor neurons.

Some cortical areas fulfill all of these criteria and have exclusively motor functions. Other areas fulfill only some of the criteria yet are involved in movement, particularly volitional movement.

Distinct Cortical Areas Participate in Voluntary Movement

The **primary motor cortex** (MI), Brodmann's area 4, fulfills all three criteria for a motor area (Fig. 20). The **supplementary motor cortex** (MII), which also fulfills all three criteria, is rostral and medial to MI in Brodmann's area 6. Other areas that fulfill some of the criteria include the rest of Brodmann's area 6; areas 1, 2, and 3 of the postcentral gyrus; and areas 5 and 7 of the parietal lobe. All of these areas contribute fibers to the **corticospinal tract**, the efferent motor pathway from the cortex.

The Primary Motor Cortex (MI). This cortical area corresponds to Brodmann's area 4 in the precentral gyrus. Area 4 is structured in six well-defined layers (I to VI), with layer I being closest to the pial surface. Afferent fibers terminate in layers I to V. Thalamic afferent fibers terminate in two layers; those that carry somatosensory information end in layer IV, and those from nonspecific nuclei end in layer I.

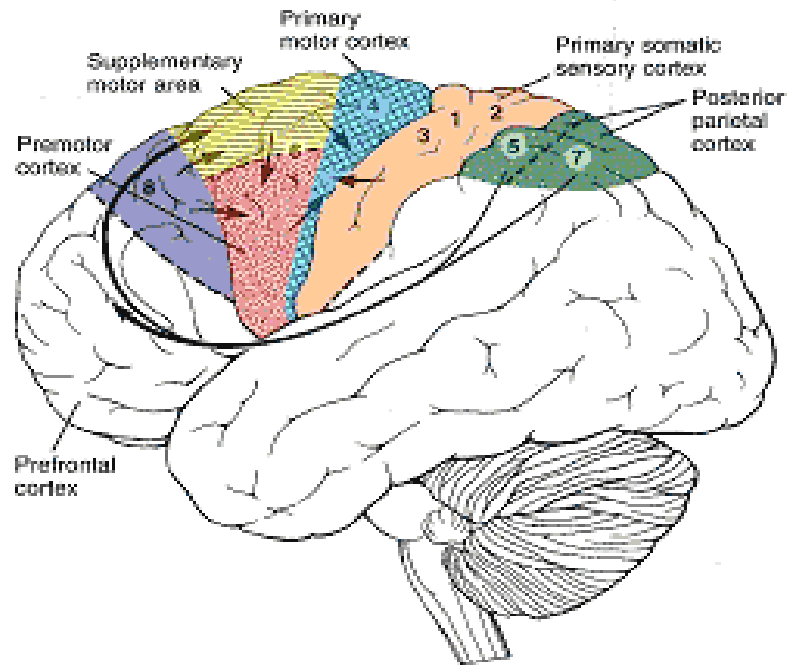


Fig. 20. **Brodman's cytoarchitectural map of the human cerebral cortex.** Area 4 is the primary motor cortex (MI); area 6 is the premotor cortex and includes the supplementary motor area (MII) on the medial aspect of the hemisphere; area 8 influences voluntary eye movements; areas 1, 2, 3, 5, and 7 have sensory functions but also contribute axons to the corticospinal tract.

Cerebellar afferents terminate in layer IV. Efferent axons arise in layers V and VI to descend as the corticospinal tract. Body areas are represented in an orderly manner, as **somatotopic maps**, in the motor and sensory cortical areas (Fig 21). Those parts of the body that perform fine movements, such as the digits and the facial muscles, are controlled by a greater number of neurons that occupy more cortical territory than the neurons for the body parts only capable of gross movements. Low-level electrical stimulation of MI produces twitch like contraction of a few muscles or, less commonly, a single muscle. Slightly stronger stimuli also produce responses in adjacent muscles. Movements elicited from area 4 have the lowest stimulation thresholds and are the most discrete of any movements elicited by stimulation. Stimulation of MI limb areas produces contralateral movement, while cranial cortical areas may produce bilateral motor responses.

Destruction of any part of the primary motor cortex leads to immediate paralysis of the muscles controlled by that area. In humans, some function may return weeks to months later, but the movements lack the fine degree muscle control of the normal state. For example, after a lesion in the arm area of MI, the use of the hand recovers, but the capacity for discrete finger movements does not.

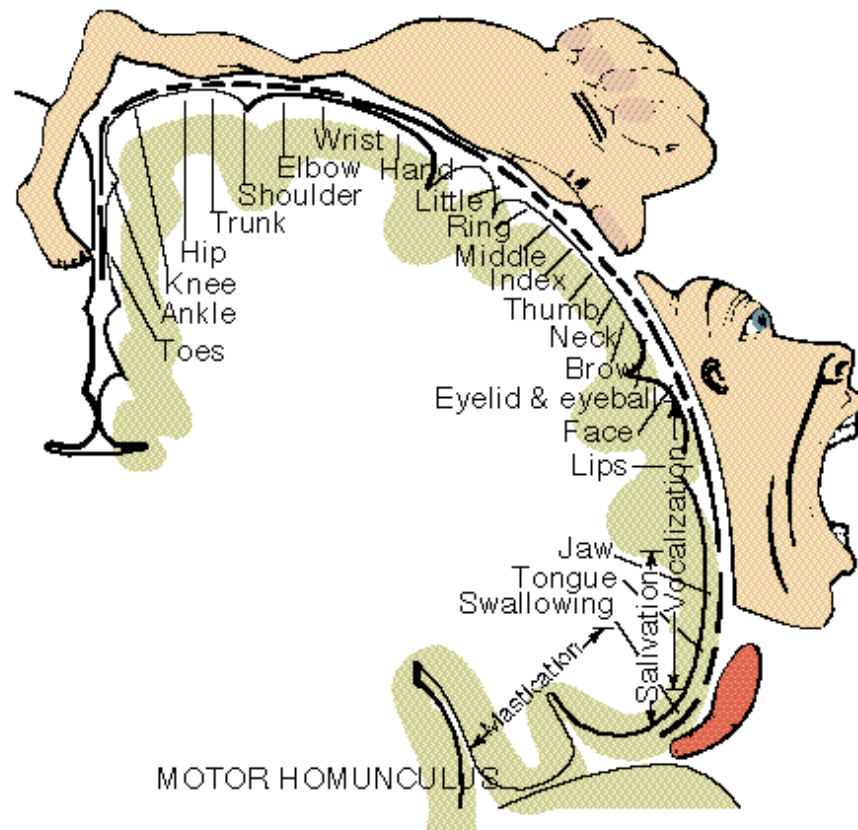


Fig. 21. **A cortical map of motor functions.** Primary motor cortex (MI).

Neurons in MI encode the capability to control muscle force, muscle length, joint movement, and position. The area receives somatosensory input, both cutaneous and proprioceptive, via the ventrobasal thalamus. The cerebellum projects to MI via the red nucleus and ventrolateral thalamus.

Other afferent projections come from the nonspecific nuclei of the thalamus, the contralateral motor cortex, and many other ipsilateral cortical areas. There are many axons between the precentral (motor) and postcentral (somatosensory) gyri and many connections to the visual cortical areas. Because of their connections

with the somatosensory cortex, the cortical motor neurons can also respond to sensory stimulation. For example, cells innervating a particular muscle may respond to cutaneous stimuli originating in the area of skin that moves when that muscle is active, and they may respond to proprioceptive stimulation from the muscle to which they are related.

Many efferent fibers from the primary motor cortex terminate in brain areas that contribute to ascending somatic sensory pathways. Through these connections, the motor cortex can control the flow of somatosensory information to motor control centers.

The close coupling of sensory and motor functions may play a role in two cortically controlled reflexes that were originally described in experimental animals as being important for maintaining normal body support during locomotion—the placing and hopping reactions. The **placing reaction** can be demonstrated in a cat by holding it so that its limbs hang freely. Contact of any part of the animal's foot with the edge of a table provokes immediate placement of the foot on the table surface. The **hopping reaction** is demonstrated by holding an animal so that it stands on one leg. If the body is moved forward, backward, or to the side, the leg hops in the direction of the movement so that the foot is kept directly under the shoulder or hip, stabilizing the body position. Lesions of the contralateral precentral or postcentral gyrus abolish placing. Hopping is abolished by a contralateral lesion of the precentral gyrus.

The Supplementary Motor Cortex (MII).

The MII cortical area is located on the medial surface of the hemispheres, above the cingulate sulcus, and rostral to the leg area of the primary motor cortex (see Fig. 20). This cortical region within Brodmann's area 6 has no clear cytoarchitectural boundaries; that is, the shapes and sizes of cells and their processes are not obviously compartmentalized, as in the layers of MI.

Electrical stimulation of MII produces movements, but a greater strength of stimulating current is required than for MI. The movements produced by

stimulation are also qualitatively different from MI; they last longer, the postures elicited may remain after the stimulation is over, and the movements are less discrete. Bilateral responses are common. MII is reciprocally connected with MI, and receives input from other motor cortical areas. Experimental lesions in MI eliminate the ability of MII stimulation to produce movements.

Current knowledge is insufficient to adequately describe the unique role of MII in higher motor functions. MII is thought to be active in bimanual tasks, in learning and preparing for the execution of skilled movements, and in the control of muscle tone. The mechanisms that underlie the more complex aspects of movement, such as thinking about and performing skilled movements and using complex sensory information to guide movement, remain incompletely understood.

The Primary Somatosensory Cortex and Superior Parietal Lobe.

The **primary somatosensory cortex** (Brodmann's areas 1, 2, and 3) lies on the postcentral gyrus (see Fig. 20.) and has a role in movement. Electrical stimulation here can produce movement, but thresholds are 2 to 3 times higher than in MI. The somatosensory cortex is reciprocally interconnected with MI in a somatotopic pattern— for example, arm areas of sensory cortex project to arm areas of motor cortex. Efferent fibers from areas 1, 2, and 3 travel in the corticospinal tract and terminate in the dorsal horn areas of the spinal cord.

The **superior parietal lobe** (Brodmann's areas 5 and 7) also has important motor functions. In addition to contributing fibers to the corticospinal tract, it is well connected to the motor areas in the frontal lobe. Lesion studies in animals and humans suggest this area is important for the utilization of complex sensory information in the production of movement.

The Corticospinal Tract Is the Primary Efferent Path From the Cortex

Traditionally, the descending motor tract originating in the cerebral cortex has been called the **pyramidal tract** because it traverses the medullary pyramids on its way to the spinal cord (Fig. 22.). This path is the **corticospinal tract**. All other

descending motor tracts emanating from the brainstem were generally grouped together as the extrapyramidal system. Cells in Brodmann's area 4 (MI) contribute 30% of the corticospinal fibers; area 6 (MII) is the origin of 30% of the fibers; and the parietal lobe, especially Brodmann's areas 1, 2, and 3, supplies 40%. In primates, 10 to 20% of corticospinal fibers ends directly on motor neurons; the others end on interneurons associated with motor neurons.

From the cerebral cortex, the corticospinal tract axons descend through the brain along a path located between the basal ganglia and the thalamus, known as the **internal capsule**. They then continue along the ventral brainstem as the **cerebral peduncles** and on through the pyramids of the medulla. Most of the corticospinal axons cross the midline in the medullary pyramids; thus, the motor cortex in each hemisphere controls the muscles on the contralateral side of the body. After crossing in the medulla, the corticospinal axons descend in the dorsal lateral columns of the spinal cord and terminate in lateral motor pools that control distal muscles of the limbs. A smaller group of axons do not cross in the medulla and descend in the ventral spinal columns. These axons terminate in the motor pools and adjacent intermediate zones that control the axial and proximal musculature.

The corticospinal tract is estimated to contain about 1 million axons at the level of the medullary pyramid. The largest-diameter, heavily myelinated axons are between 9 and 20 μm in diameter, but that population accounts for only a small fraction of the total. Most corticospinal axons are small, 1 to 4 μm in diameter, and half are unmyelinated.

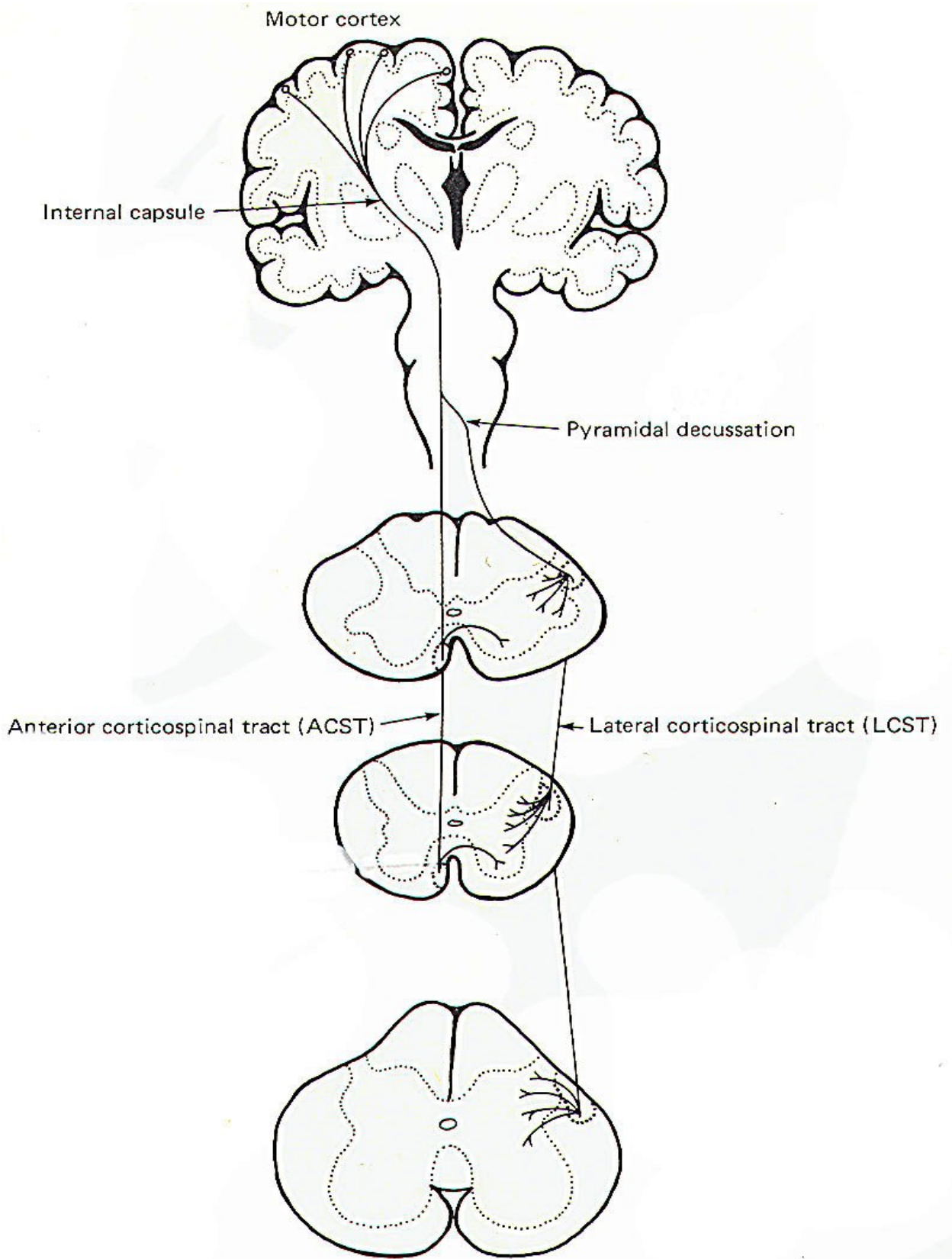


Fig. 22. **The corticospinal tract.** Axons arising from cortical neurons, including the primary motor area, descend through the internal capsule, decussate in the medulla, travel in the lateral area of the spinal cord as the lateral corticospinal tract,

and terminate on motor neurons and interneurons in the ventral horn areas of the spinal cord. Note the upper and lower motor neuron designations.

In addition to the direct corticospinal tract, there are other indirect pathways by which cortical fibers influence motor function. Some cortical efferent fibers project to the reticular formation, then to the spinal cord via the reticulospinal tract; others project to the red nucleus, then to the spinal cord via the rubrospinal tract. Despite the fact that these pathways involve intermediate neurons on the way to the cord, volleys relayed through the reticular formation can reach the spinal cord motor circuitry at the same time as, or earlier than, some volleys along the corticospinal tract.

1.7 THE BASAL GANGLIA AND MOTOR CONTROL

The **basal ganglia** are a group of subcortical nuclei located primarily in the base of the forebrain, with some in the diencephalon and upper brainstem. The striatum, globus pallidus, subthalamic nucleus, and substantia nigra comprise the basal ganglia. Input is derived from the cerebral cortex and output is directed to the cortical and brainstem areas concerned with movement. Basal ganglia action influences the entire motor system and plays a role in the preparation and execution of coordinated movements.

The forebrain (telencephalic) components of the basal ganglia consist of the **striatum**, which is made up of the **caudate nucleus** and the **putamen**, and the **globus pallidus**. The caudate nucleus and putamen are histologically identical but are separated anatomically by fibers of the anterior limb of the internal capsule. The globus pallidus has two subdivisions: the **external segment (GPe)**, adjacent to the medial aspect of the putamen, and the **internal segment (GPi)**, medial to the GPe. The other main nuclei of the basal ganglia are the **subthalamic nucleus** in the diencephalon and the **substantia nigra** in the mesencephalon.

The Basal Ganglia Are Extensively Interconnected

Although the circuitry of the basal ganglia appears complex at first glance, it can be simplified into input, output, and internal pathways (Fig. 23.). Input is derived from the cerebral cortex and is directed to the striatum and the subthalamic nucleus. The predominant nerve cell type in the striatum is termed the **medium spiny neuron**, based on its cell body size and dendritic structure. This type of neuron receives input from all of the cerebral cortex except for the primary visual and auditory areas. The input is roughly somatotopic and is via neurons that use glutamate as the neurotransmitter. The putamen receives the majority of the cortical input from sensorimotor areas. Input to the subthalamus is from the cortical areas concerned with motor function, including eye movement, and is also via glutamate-releasing neurons.

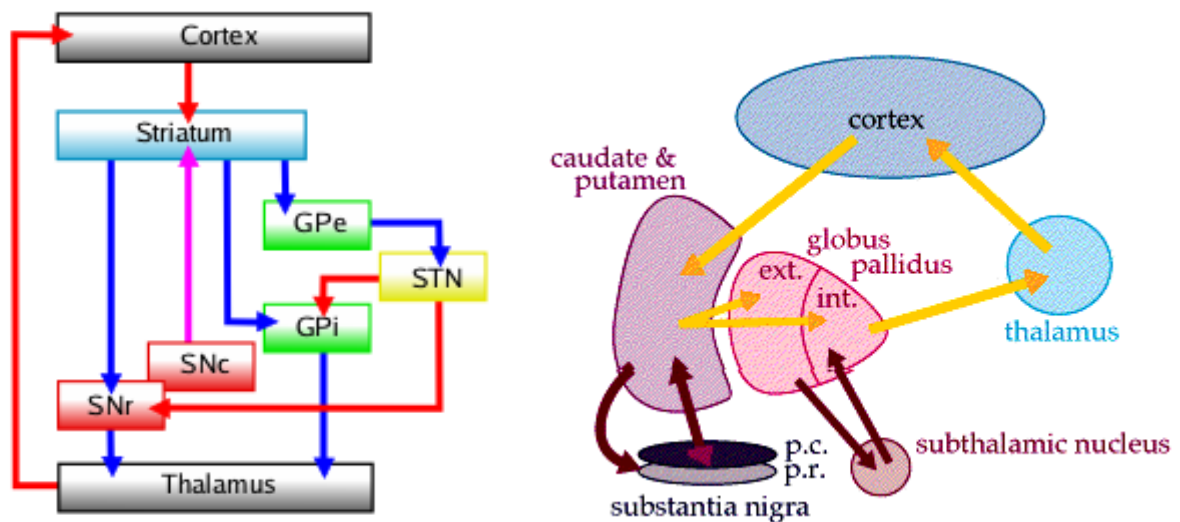


Fig. 23. **Basal ganglia nuclei and circuitry.** The circuit of cerebral cortex to striatum to GPi to thalamus and back to the cortex is the main pathway for basal ganglia influence on motor control. Note the direct and indirect pathways involving the striatum, GPi, GPe, and subthalamic nucleus. GPi output is also directed to the midbrain extrapyramidal area (MBEA). The SNr to SC pathway is important in eye movements. Excitatory pathways are shown in red, inhibitory pathways are in black. GPe and GPi, globus pallidus externa and interna; SUB, subthalamic nucleus; SNc and SNr, substantia nigra pars compacta and pars reticulata; SC, superior colliculus.

Basal ganglia output is from the internal segment of the globus pallidus (GPi) and one segment of the substantia nigra. The GPi output is directed to ventrolateral and ventral anterior nuclei of the thalamus, which feed back to the cortical motor areas. The output of the GPi is also directed to a region in the upper brainstem termed the **midbrain extrapyramidal area**. This latter area then projects to the neurons of the reticulospinal tract. The substantia nigra output arises from the **pars reticulata** (SNr), which is histologically similar to the GPi. The output is directed to the superior colliculus of the mesencephalon, which is involved in eye movement control. The GPi and SNr output is inhibitory via neurons that use GABA as the neurotransmitter.

The internal pathway circuits link the various nuclei of the basal ganglia. The globus pallidus externa (GPe), the subthalamic nucleus, and the pars compacta region of the substantia nigra (SNc) are the nuclei in these pathways. The GPe receives inhibitory input from the striatum via GABA-releasing neurons. The output of the GPe is also inhibitory via GABA release and is directed to the GPi and the subthalamic nucleus. The subthalamic nucleus output is excitatory and is directed to the GPi and the SNr. This striatum- GPe-subthalamic nucleus-GPi circuit has been termed the **indirect pathway** in contrast to the **direct pathway** of striatum to GPi (see Fig. 23.). The SNc receives inhibitory input from the striatum and produces output back to the striatum via dopamine-releasing neurons. The output can be either excitatory or inhibitory depending on the receptor type of the target neurons in the striatum. The action of the SNc may modulate cortical input to the striatum.

The Functions of the Basal Ganglia Are Partially Revealed by Disease

Basal ganglia diseases produce profound motor dysfunction in humans and experimental animals. The disorders can result in reduced motor activity, **hypokinesia**, or abnormally enhanced activity, **hyperkinesia**. Two well-known neuro- logical conditions that show histological abnormality in basal ganglia structures, Parkinson's disease and Huntington's disease, illustrate the effects of basal ganglia dysfunction.

Patients with **Parkinson's disease** show a general slowness of initiation of movement and paucity of movement when in motion. The latter takes the form of reduced arm swing and lack of truncal swagger when walking. These patients also have a resting tremor of the hands, described as "pill rolling." The tremor stops when the hand goes into active motion. At autopsy, patients with Parkinson's

disease show a severe loss of dopamine-containing neurons in the SNc region.

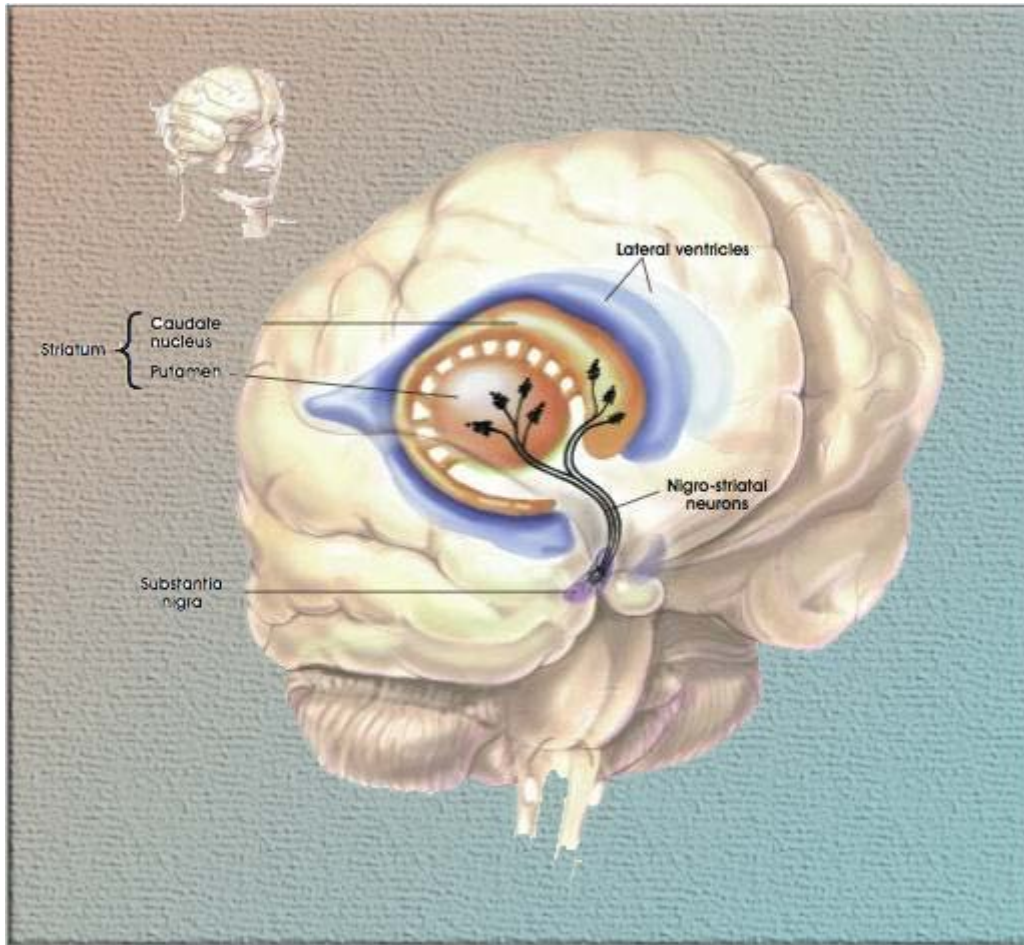


Fig. 24. Area of the brain damaged by Parkinson's disease.

Patients with **Huntington's disease** have uncontrollable, quick, brief movements of individual limbs. These movements are similar to what a normal individual might show when flicking a fly off a hand or when quickly reaching up to scratch an itchy nose. At autopsy, a severe loss of striatal neurons is found (Fig. 25).



Fig. 25. The human brain, showing the impact of **Huntington's disease** (HD) on brain structure in the basal ganglia region of a person with HD (top) and a normal brain (bottom).

The function of the basal ganglia in normal individuals remains unclear. One theory is that the primary action is to inhibit undesirable movements, thereby, allowing desired motions to proceed. Neuronal activity is increased in the appropriate areas of the basal ganglia prior to the actual execution of movement. The basal ganglia act as a brake on undesirable motion by the inhibitory output of the GPi back to the cortex through the thalamus. Enhanced output from the GPi increases this braking effect. The loss of dopamine-releasing neurons in Parkinson's disease is thought to produce this type of result by reducing inhibitory influence on the striatum and, thereby, increasing the excitatory action of the subthalamic nucleus on the GPi through the indirect basal ganglia pathway (see Fig. 24). Hyperkinetic disorders like Huntington's disease are thought to result from decreased GPi output secondary to the loss of inhibitory influence of the striatum through the direct pathway.

Stereotactic Neurosurgery for Parkinson's disease

Parkinson's disease is a CNS disorder producing a generalized slowness of movement and resting tremor of the hands. Loss of dopamine-producing neurons in the substantia nigra pars compacta is the cause of the condition. Treatment with medications that stimulate an increased production of dopamine by the surviving substantia nigra neurons has revolutionized the management of Parkinson's disease. Unfortunately, the benefit of the medications tends to lessen after 5 to 10 years of treatment. Increasing difficulty in initiating movement and worsening slowness of movement are features of a declining responsiveness to medication. Improved knowledge of basal ganglia circuitry has enabled neurosurgeons to develop surgical procedures to ameliorate some of the effects of the advancing disease.

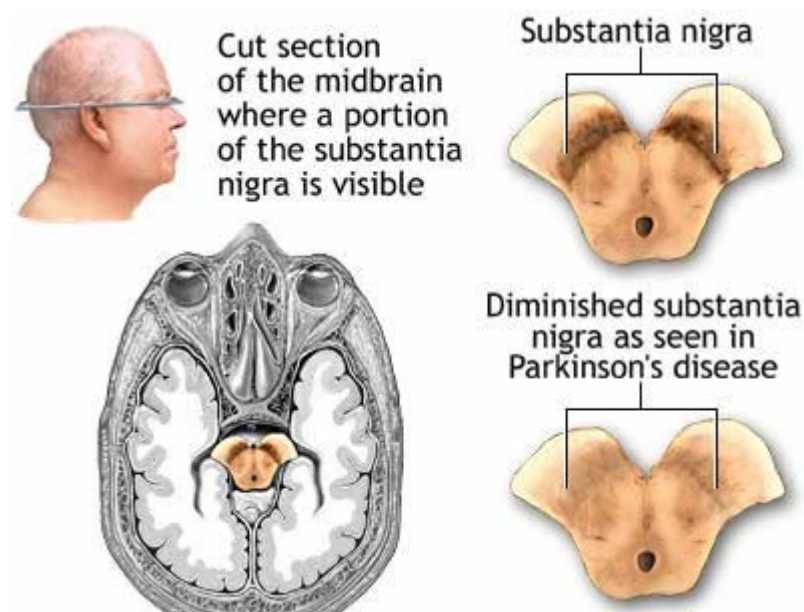


Fig. 26. Substantia nigra and Parkinson's Disease

Degeneration of the dopamine-releasing cells of the substantia nigra reduces excitatory input to the putamen (Fig. 26). Inhibitory output of the putamen to the GPe greatly increases via the indirect pathway. This results in decreased inhibitory GPe output to the subthalamic nucleus, which, in turn, acts unrestrained to

stimulate the GPi. Stimulation of the GPi enhances its inhibitory influence on the thalamus and results in decreased excitatory drive back to the cerebral cortex. Stereotactic neurosurgery is a technique in which a small probe can be precisely placed into a target within the brain (Fig. 27) . Magnetic resonance imaging (MRI) of the brain defines the three-dimensional location of the GPi. The surgical probe is introduced into the brain through a small hole made in the skull and is guided to the target by the surgeon using the MRI coordinates. The correct positioning of the probe into the GPi can be further confirmed by recording the electrical activity of the GPi neurons with an electrode located at the tip of the probe. GPi neurons have a continuous, high frequency firing pattern that, when amplified and presented on a loudspeaker, sounds like heavy rain striking a metal roof. When the target location is reached, the probe is heated to a temperature that destroys a precisely controllable amount of the GPi. The inhibitory outflow of the GPi is reduced and movement improves.



Fig. 27. Stereotactic Neurosurgery for Parkinson's disease

The use of implantable stimulators to modify activity of the basal ganglia nuclei is also being investigated to improve function in patients with Parkinson's disease and other types of movement disorders.

1.8 THE CEREBELLUM IN THE CONTROL OF MOVEMENT

The **cerebellum**, or “little brain,” lies caudal to the occipital lobe and is attached to the posterior aspect of the brainstem through three paired fiber tracts: the **inferior**, **middle**, and **superior cerebellar peduncles**. Input to the cerebellum comes from peripheral sensory receptors, the brainstem, and the cerebral cortex. The inferior, middle and, to a lesser degree, superior cerebellar peduncles carry the input. The output projections are mainly, if not totally, to other motor control areas of the central nervous system and are mostly carried in the superior cerebellar peduncle. The cerebellum contains three pairs of intrinsic nuclei: the **fastigial**, **interpositus** (interposed), and **dentate**. In some classification schemes, the interposed nucleus is further divided into the **emboliform** and **globose** nuclei.

The Structural Divisions of the Cerebellum Correlate With Function

The cerebellar surface is arranged in multiple, parallel, longitudinal folds termed **folia**. Several deep fissures divide the cerebellum into three main morphological components— the **anterior**, **posterior**, and **flocculonodular lobes**, which also correspond with the functional subdivisions of the cerebellum (Fig. 28). The functional divisions are the vestibulocerebellum, the spinocerebellum, and the cerebrocerebellum. These divisions appear in sequence during evolution. The lateral cerebellar hemispheres increase in size along with expansion of the cerebral cortex. The three divisions have similar intrinsic circuitry; thus, the function of each depends on the nature of the output nucleus to which it projects.

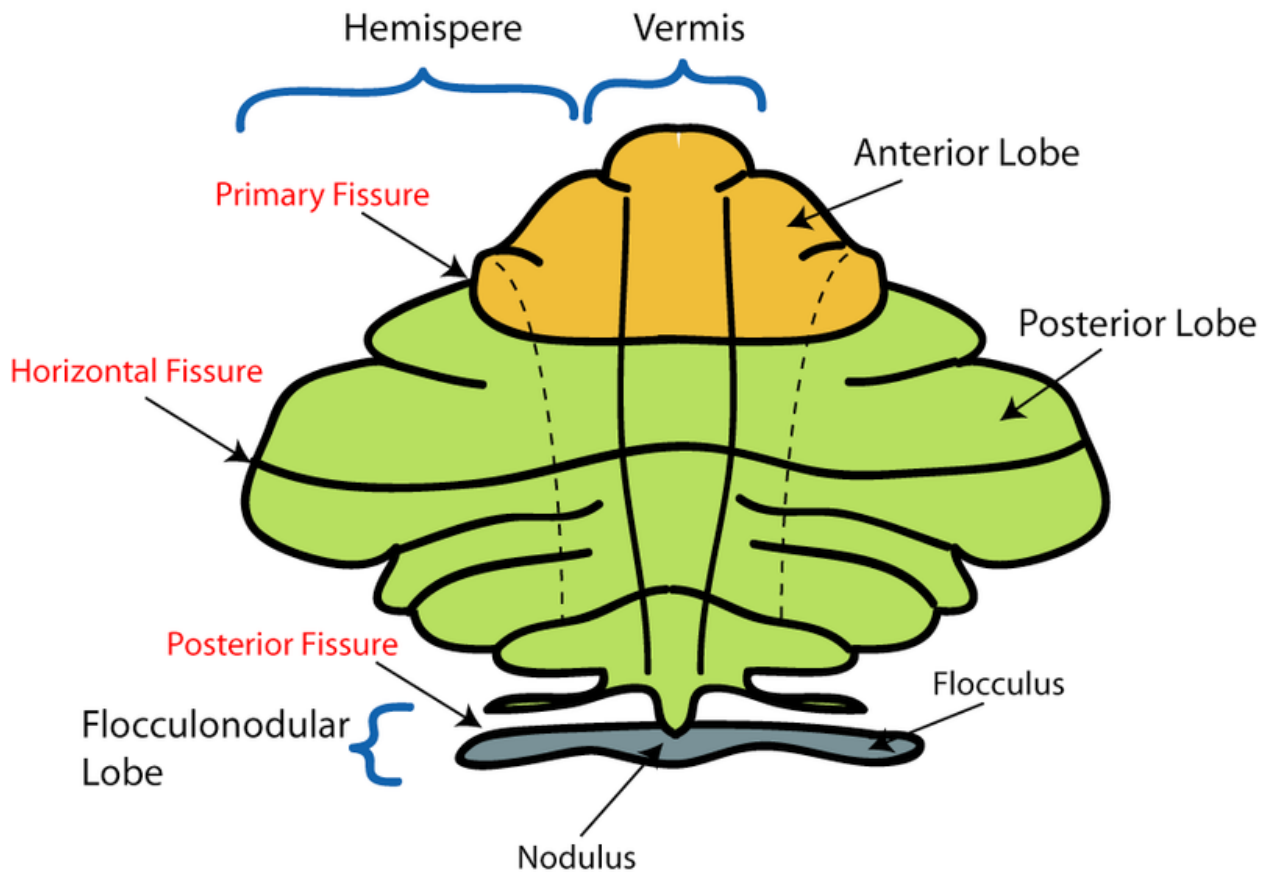


Fig. 27. **The structure of the cerebellum.** The three lobes are shown: anterior, posterior, and flocculonodular.

The **vestibulocerebellum** is composed of the flocculonodular lobe. It receives input from the vestibular system and visual areas. Output goes to the vestibular nuclei, which can, in a sense, be considered as an additional pair of intrinsic cerebellar nuclei. The vestibulocerebellum functions to control equilibrium and eye movements.

The medially placed **spinocerebellum** consists of the midline **vermis** plus the medial portion of the lateral hemispheres, called the **intermediate zones**. Spinocerebellar pathways carrying somatosensory information terminate in the vermis and intermediate zones in somatotopic arrangements. The auditory, visual, and vestibular systems and sensorimotor cortex also project to this portion of the cerebellum. Output from the vermis is directed to the fastigial nuclei (Fig. 28), which project through the inferior cerebellar peduncle to the vestibular nuclei and reticular formation of the pons and medulla. Output from the intermediate zones

goes to the interposed nuclei and from there to the red nucleus and, ultimately, to the motor cortex via the ventrolateral nucleus of the thalamus. It is believed that both the fastigial and interposed nuclei contain a complete representation of the muscles of the body. The fastigial output system controls antigravity muscles in posture and locomotion, while the interposed nuclei, perhaps, act on stretch reflexes and other somatosensory reflexes.

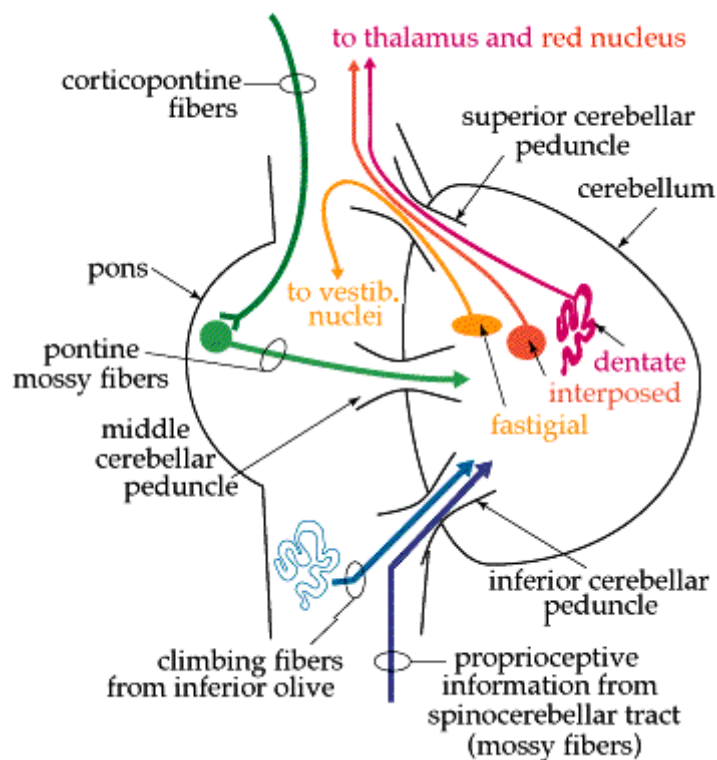


Fig. 28. The 3 deep nuclei are the **fastigial**, **interposed**, and **dentate nuclei**. The fastigial nucleus is primarily concerned with balance, and sends information mainly to vestibular and reticular nuclei. The dentate and interposed nuclei are concerned more with voluntary movement, and send axons mainly to thalamus and the red nucleus.

The **cerebrocerebellum** occupies the lateral aspects of the cerebellar hemispheres. Input comes exclusively from the cerebral cortex, relayed through the middle cerebellar peduncles of the pons. The cortical areas that are prominent in motor control are the sources for most of this input. Output is directed to the dentate

nuclei and from there via the ventrolateral thalamus back to the motor and premotor cortices.

The Intrinsic Circuitry of the Cerebellum Is Very Regular

The cerebellar cortex is composed of five types of neurons arranged into three layers (Fig. 29). The molecular layer is the outermost and consists mostly of axons and dendrites plus two types of interneurons, **stellate cells** and **basket cells**. The next layer contains the dramatic **Purkinje cells**, whose dendrites reach upward into the molecular layer in a fan-like array. The Purkinje cells are the only efferent neurons of the cerebellar cortex. Their action is inhibitory via GABA as the neurotransmitter. Deep to the Purkinje cells is the **granular layer**, containing **Golgi cells**, and small local circuit neurons, the **granule cells**. The granule cells are numerous; there are more granule cells in the cerebellum than neurons in the entire cerebral cortex!

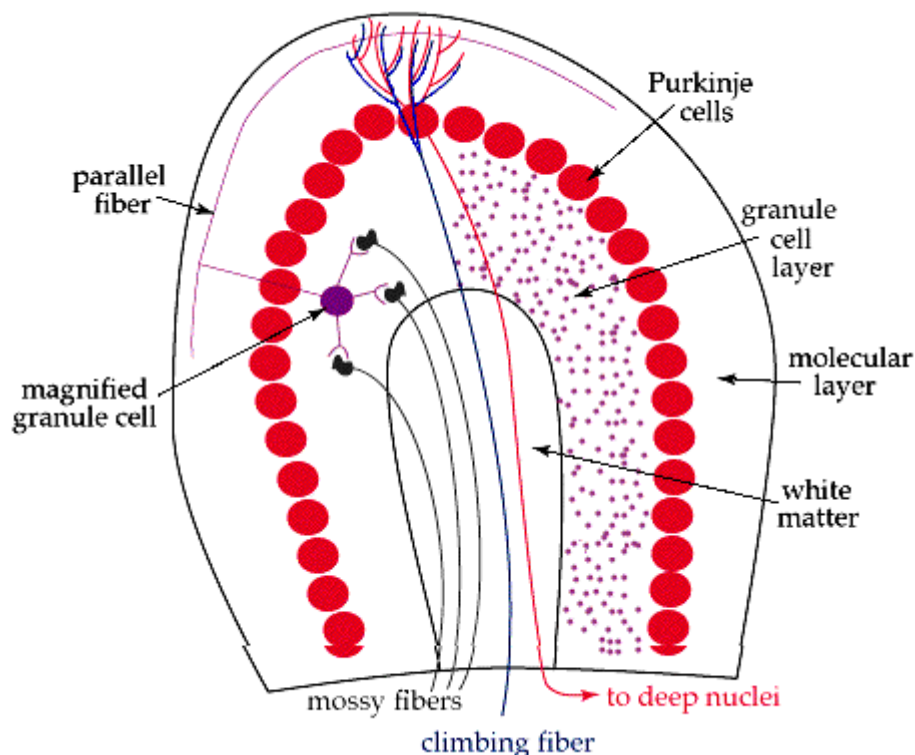


Fig. 29. **Cerebellar circuitry.** The cell types and action potential pathways are shown. Mossy fibers bring afferent input from the spinal cord and the cerebral cortex. Climbing fibers bring afferent input from the inferior olive nucleus in the medulla and synapse directly on the Purkinje cells. The Purkinje cells are the efferent pathways of the cerebellum.

Afferent axons to the cerebellar cortex are of two types: mossy fibers and climbing fibers. **Mossy fibers** arise from the spinal cord and brainstem neurons, including those of the pons that receive input from the cerebral cortex. Mossy fibers make complex multicontact synapses on granule cells. The granule cell axons then ascend to the molecular layer and bifurcate, forming the **parallel fibers**. These travel perpendicular to and synapse with the dendrites of Purkinje cells, providing excitatory input via glutamate. Mossy fibers discharge at high tonic rates, 50 to 100 Hz, which increases further during voluntary movement. When mossy fiber input is of sufficient strength to bring a Purkinje cell to threshold, a single action potential results.

Climbing fibers arise from the **inferior olive**, a nucleus in the medulla. Each climbing fiber synapses directly on the dendrites of a Purkinje cell and exerts a strong excitatory influence. One action potential in a climbing fiber produces a burst of action potentials in the Purkinje cell called a complex spike.

Climbing fibers also synapse with basket, Golgi, and stellate interneurons, which then make inhibitory contact with adjacent Purkinje cells. This circuitry allows a climbing fiber to produce excitation in a single Purkinje cell and inhibition in the surrounding ones.

Mossy and climbing fibers also give off excitatory collateral axons to the deep cerebellar nuclei before reaching the cerebellar cortex. The cerebellar cortical output (Purkinje cell efferents) is inhibitory to the cerebellar and vestibular nuclei, but the ultimate output of the cerebellar nuclei is mostly excitatory. A smaller population of neurons of the deep cerebellar nuclei produces inhibitory outflow directed mainly back to the inferior olive.

Lesions Reveal the Function of the Cerebellum.

Lesions of the cerebellum produce impairment in the coordinated action of agonists, antagonists, and synergists. This impairment is clinically known as **ataxia**. The control of limb, axial, and cranial muscles may be impaired depending on the site of the cerebellar lesion. Limb ataxia might manifest as the coarse

jerking motions of an arm and hand during reaching for an object instead of the expected, smooth actions. This jerking type of motion is also referred to as **action tremor**. The swaying walk of an intoxicated individual is a vivid example of truncal ataxia.

Cerebellar lesions can also produce a reduction in muscle tone, **hypotonia**. This condition is manifest as a notable decrease in the low level of resistance to passive joint movement detectable in normally relaxed individuals. Myotatic reflexes produced by tapping a tendon with a reflex hammer reverberate for several cycles (pendular reflexes) because of impaired damping from the reduced muscle tone. The hypotonia is likely a result of impaired processing of cerebellar afferent action potentials from the muscle spindles and Golgi tendon organs.

While these lesions establish a picture of the absence of cerebellar function, we are left without a firm idea of what the cerebellum does in the normal state. Cerebellar function is sometimes described as comparing the intended with the actual movement and adjusting motor system output in ongoing movements. Other putative functions include a role in learning new motor and even cognitive skills.

2. Control questions and practical work

After studying this chapter, students should be able to . . .

- Describe how skilled movements are planned and carried out.
- Describe the Organization of General Principles controlling of Posture and Movement.
- Organization of the Spinal Cord for Motor Functions: muscle sensory receptors (Muscle Spindles and Golgi Tendon organs); Spinal cord's neurons (the alpha motor neurons, the gamma motor neurons and interneurons).
- The Spinal stretch reflexes (or Deep Tendon Reflex, or Myotatic reflex): the adequate stimulus for the stretch reflex; the sensory ending and afferent fibers; Reflex action.
- What do you know about the control of sensitivity of the Spinal reflex
- Explain the Clasp Knife Phenomenon; the Flexion (Withdrawal) Reflex; the Crossed Extensor reflex.
- Name the posture-regulating parts of the CNS and discuss the role of each.
- Define spinal shock, and describe the initial and long-term changes in spinal reflexes that follow transection of the spinal cord.
- Define decerebrate and decorticate rigidity, and comment on the cause and physiologic significance of each.
- What do you know about the Corticospinal and the Corticobulbar Tracts?
- Explain the Anatomy and Function of the Cortical Motor Areas, their plasticity
- The Supplementary Motor Area, the Premotor Cortex, the Posterior Parietal Cortex and its Role in Movement and influence on Stretch reflexes
- Describe the basal ganglia, and list the path ways that interconnect them, along with the neurotransmitters in each pathway.
- Describe and explain the symptoms of Parkinson s disease and Huntington's disease.

- List the pathways to and from the cerebellum and the connections of each within the cerebellum.
- Discuss the functions of the cerebellum and the neurologic abnormalities produced by diseases of this part of the brain.

3. QUESTIONS for INDEPENDENT WORK

1. What is meant by the terms upper motor neuron and lower motor neuron?

Contrast the effects of lower motor neuron lesions with those of lesions affecting each of the types of upper motor neurons.

2. What is the Babinski sign? What is its physiologic and pathologic significance?

3. What is the mass reflex? Why does it occur after transection of the spinal cord?

4. Define athetosis, ballism, and chorea, and describe the disease processes that produce each of them.

5. List three drugs and two surgical procedures used in the treatment of Parkinson's disease, and explain why each is of value.

6. List five types of neurons found in the cerebellar cortex, and describe the morphology and function of each.

7. What is an intention tremor? Why does it occur in cerebellar disease?

8. Describe the Clinical applications of the Stretch reflex (Biceps tendon reflex, Triceps tendon reflex, Knee Jerk Reflex, and Achilles tendon reflex).

9. Explain the role of the Medullary Components in movement and origin of Tonic Labyrinthine Reflexes and the Tonic Neck Reflexes.

10. Explain the role of the Midbrain Components in movement and origin of Righting Reflexes, the Grasp Reflex and other Midbrain Responses

11. Explain the role of the Thalamus and the Hypothalamus in movement.

4. Quizzes

TESTS:

1. What symptoms can a person have after lesions of the C5 -C6 of Spinal cord?
 - A. the inability to learned sequences of movements such as eating with a knife
 - B. the difficulty in speech
 - C. the difficulty in bimanual coordination
 - D. the difficulty in walking
 - E all answers are correct

- 2.The spinal cord is contiguous superiorally with the
 - A. cerebellum.
 - B. medulla oblongata
 - C. midbrain
 - D. pons
 - E.cortex

3. The lateral horns of the gray matter in the thoracic region of the spinal cord contain the cell bodies of
 - A. somatic motor neurons
 - B. parasympathetic motor neurons
 - C. sympathetic motor neurons
 - D. sensory neurons
 - E. all answers are correct

4. The knee-jerk reflex involves which of the following?
 - A. sensory and motor neurons
 - B. sensory, association and motor neurons
 - C. sensory, motor and interneurons
 - D. sensory and association neurons
 - E. all answers are incorrect

5. Which of the following reflexes inhibits skeletal muscle contraction?
 - A. crossed extensor reflex
 - B. Golgi tendon reflex
 - C. stretch reflex
 - D. withdrawal reflex
 - E. all answers are incorrect

6. Inhibitory interneurons are involved in which of the following spinal reflexes?
 - A. Golgi tendon reflex
 - B. knee jerk reflex
 - C. stretch reflex

- D. withdrawal reflex
- E. all answers are incorrect

7. Excitatory interneurons are involved in which of the following spinal reflexes?

- A. Golgi tendon reflex
- B. knee jerk reflex
- C. stretch reflex
- D. withdrawal reflex
- E. all of the above

8. After severely breaking his left humerus in an accident, a man lost sensation on the posterior aspect of the limb and was unable to extend his forearm, wrist, or fingers. What nerve was damaged?

- A. axillary
- B. musculocutaneous
- C. radial
- D. ulnar
- E. sciatic

9. Compression of what nerve arising from the brachial plexus results in numbness, tingling, and pain in the fingers, a condition called carpal tunnel syndrome?

- A. axillary nerve
- B. radial nerve
- C. median nerve
- D. musculocutaneous nerve
- E. sciatic nerve

10. The ventral branches or rami of spinal nerves do NOT join to form nerve plexuses in which of the following spinal cord areas?

- A. cervical
- B. lumbar
- C. sacra
- D. thoracic
- E. coccygeal

11. Given these components of a reflex:

- 1. association neuron
- 2. skeletal muscle
- 3. afferent neuron
- 4. efferent neuron
- 5. sensory receptor

Choose the sequence below that best represents the order followed in a reflex, from stimulus to response.

- A. 5, 4, 3, 2, 1
- B. 5, 3, 2, 4, 1

- C. 5, 4, 3, 1, 2
- D. 5, 3, 1, 4, 2
- E. 5, 4, 1, 3, 2

12. Stretch reflexes

- A. cause muscles to contract in response to a stretching force being applied to them
- B. involves a sensory receptor (muscle spindle).
- C. involve sensory neurons that directly synapse with motor neurons in the spinal cord
- D. help maintain posture
- E. all of the above

13. The Golgi tendon reflex

- A. involves the synapse of sensory neurons from the Golgi tendon organs with stimulating interneurons at the spinal cord
- B. prevents contracting muscles from applying excessive tension to tendons
- C. involves the stimulation of alpha neurons leading back to the muscles that are stretching tendons
- D. results in increased tension at tendons
- E. all of the above

14. The withdrawal reflex

- A. includes the Golgi tendon organs
- B. includes the synapse of sensory neurons directly with alpha motor neurons
- C. helps to protect the body from painful stimuli
- D. is a response to increased tension at a tendon
- E. all of the above

15. Which of these events occur when a person steps on a tack with their right foot?

- A. The right foot is pulled away from the tack because of the Golgi tendon reflex
- B. The left leg is extended to support the body because of the stretch reflex
- C. The flexor muscles of the thigh contract, and the extensor muscles relax because of reciprocal innervation
- D. The extensor muscles of both thighs contract because of the crossed extensor reflex
- E. all of these

16. A knee-jerk reflex is an example of the

- A. crossed extensor reflex
- B. withdrawal reflex
- C. Golgi tendon reflex

- D. stretch reflex
- E. all of the above

17. Reflexes

- A. are never homeostatic
- B. are automatic responses to a stimulus
- C. cannot be suppressed by higher brain functions
- D. are always simple pathways containing three neurons
- E. all of these

18. Which of the following is NOT true about the knee jerk reflex?

- A. It is a spinal reflex
- B. It is a somatic reflex
- C. It helps to maintain an upright posture
- D. B and C
- E. It involves conduction of nerve impulses from sensory neuron to interneuron to motor neuron.

19. John injured the quadriceps muscles in his legs due to increased tension and tearing while horsing around on the football field. What reflex failed to protect the muscles?

- A. Golgi tendon reflex
- B. flexor reflex
- C. knee jerk reflex
- D. stretch reflex
- E. all of the above

20. You are walking barefoot and step on a tack with your right foot. All of the following will occur. EXCEPT

- A. flexor muscles in your right thigh and leg contract to remove your foot
- B. reciprocal innervation inhibits extensor muscles in the same limb
- C. collaterals of sensory neurons stimulate alpha motor neurons that cause extension in the opposite limb
- D. collaterals of interneurons stimulate a crossed extensor reflex
- E. all answers are incorrect

21. The reflex arc contains a

- A. sensory reception
- B. sensory neuron
- C. motor neuron
- D. A, B and C
- E. all answers are incorrect

22. A man was in an accident and severed his spinal cord between C6 and C7. Which of the following would NOT occur?

- A. loss of sensation in the trunk below the shoulders, the lower limbs and portions of the arms
 - B. damage to the phrenic nerves, which would therefore affect breathing
 - C. loss of movement in the lower limbs
 - D. damage to the intercostal nerves, which would affect breathing because the intercostal muscles would be paralyzed
 - E. all answers are incorrect
23. After severely injuring her hip in an accident, a woman was unable to extend her right leg. What nerve was damaged?
- A. femoral
 - B. obturator
 - C. tibial
 - D. pudental
 - E. sciatic
24. The ventral rami of adjacent spinal nerves intermingle with each other to form a...
- A. ganglion
 - B. fascicle
 - C. plexus
 - D. tract
 - E. way
25. The nerves that supply the lower limbs enter or exit from this area of the spinal cord.
- A. brachial plexuses
 - B. cervical enlargement
 - C. cervical plexuses
 - D. lumbar enlargement
 - E. sacral plexuses
26. What symptoms can a person have after lesions of the L2-L4 of Spinal cord?
- A. the inability to learn sequences of movements such as eating with a knife
 - B. the difficulty in speech
 - C. the difficulty in bimanual coordination
 - D. the difficulty in walking
 - E. all answers are correct
27. Damage to the dorsal root of a spinal nerve results in
- A. loss of motor control
 - B. loss of parasympathetic function
 - C. loss of sensory input

- D. A and B
- E. all of these

28. In spinal cord injury

- A. there may be loss of sensation and motor functions
- B. classification is done according to the vertebral level at which the injury occurred, the extent of the cord damage, and the mechanism of the injury.
- C. there is primary and secondary damage
- D. treatment may include the use of steroids to decrease total damage
- E. all of the above

29. A man suffers a shoulder injury, and as a result has very little strength when he tries to flex his forearm. The nerve most likely damaged is the

- A. axillary nerve
- B. median nerve
- C. musculocutaneous nerve
- D. radial nerve
- E. ulnar nerve

30. The Spinal Center of Achilles tendon reflex in

- A. C5- C6
- B. C6-C8
- C. L2 to L4
- D. L4 to S1
- E. L5 to S2

31. Where do the feet represent in the brain?

- A. Midbrain
- B. top of the Cortex precentral gyrus
- C. below of the Cortex precentral gyrus
- D. Cerebellum
- E. Basal Ganglia

32. What part of the Body can represent bilaterally at the Cortex Brain?

- A. a foot
- B. an arm
- C. a face
- D. a leg
- E. a hand

33. Which of the following is NOT one of the three large nerve tracts connecting the cerebellum to the rest of the central nervous system?

- A superior cerebellar peduncles

- B. middle cerebellar peduncles
- C inferior cerebellar peduncles
- D anterior cerebellar peduncles
- E. A and B

34. What symptoms a person can have after lesions of the Posterior Parietal Cortex?

- A. the inability to learned sequences of movements such as eating with a knife
- B. the difficulty in speech
- C. the difficulty in bimanual coordination
- D. the difficulty in walking
- E. the awkwardness in complex activities performing

35. What are the enlargements on the medulla oblongata that are involved in conscious skeletal muscle control?

- A cardiac center
- B. olives
- C. pyramids
- D. decussate
- E. A and B

36. Which of the following brain regions does NOT belong with the others?

- A medulla oblongata
- B. midbrain
- C. pons
- D. thalamus
- E. A and B

37. Choose the symptoms of Chorea

- A. .the movements are excessive and abnormal
- B. the continuous slow writhing movements
- C. A and B
- D the involuntary flailing intense and violent movements
- E. the difficulty in initiating movement and decreased spontaneous movement

38. What symptoms can a person have after lesions of the Supplementary Motor Area?

- A. he hasn't any symptoms
- B. the difficulty in speech
- C. the difficulty in walking
- D. the difficulty in bimanual coordination
- E. all answers are correct

39. Which lobes of the cerebrum serve as the main center for receiving and processing of sensory information EXCEPT for smell, hearing and vision?
- A. frontal
 - B. occipital
 - C. parietal
 - D. temporal
 - E. frontal and occipital
40. Which of the following serves as a motor center that is involved in maintaining muscle tone and coordinating movements?
- A. inferior olivary nucleus
 - B. red nucleus
 - C. suprachiasmatic nucleus
 - D. inferior olivary nucleus and red nucleus
 - E. substantia nigra
41. Because of injuries received in an automobile accident, a young man remains hospitalized in a coma. It is likely the injuries affected his
- A. amygdala
 - B. hippocampus
 - C. limbic system
 - D. reticular formation
 - E. A and C
42. Which of the following is or are located within the white matter of the cerebrum?
- A. basal nuclei
 - B. red nucleus
 - C. substantia nigra
 - D. suprachiasmatic nucleus
 - E. A and B
43. The anesthetic a dentist injects before drilling to clean and repair a cavity is done to block sensory impulses from a branch of what cranial nerve?
- A. abducens
 - B. facial
 - C. trigeminal
 - D. trochlear
 - E. C and D
44. Which cranial nerve controls the muscles involved in chewing?
- A. facial
 - B. glossopharyngeal
 - C. trigeminal

- D. vagus
- E. A and D

45. Select the nerves that are somatic motor/proprioceptive only.

- A. 1,2,3,4,5
- B. 1,2,4,5
- C. 1,3,4,5
- D. 2,3,4
- E. 3,4,5

46. Where does the face represent in the brain?

- A. Midbrain
- B. top of the Cortex precentral gyrus
- C. below of the Cortex precentral gyrus
- D. Cerebellum
- E. Basal Ganglia

47. Which of the following cranial nerves does NOT contain only sensory fibers?

- A. olfactory
- B. optic
- C. trigeminal
- D. vestibulocochlear
- E. A and B

48. What symptoms can a person have after lesions of the Premotor Cortex?

- A. he hasn't any symptoms
- B. the difficulty in speech
- C. the difficulty in walking
- D. the difficulty in bimanual coordination
- E. awkwardness with setting posture in the start of planned movement

49. What type of nuclei in the cerebrum are involved in control of motor functions?.

- A. ependymal cells
- B. caudate nucleus
- C. basal nucleus
- D. lentiform nucleus
- E. A and B

50. Choose the symptoms of Akinesia

- A. the movements are excessive and abnormal
- B. the involuntary dancing movements
- C. the continuous slow writhing movements

- D. the involuntary flailing intense and violent movements
- E. the difficulty in initiating movement and decreased spontaneous movement

51. What symptoms can a person have after lesions of the Posterior Parietal Cortex?

- A. difficulty in breathing
- B. awkwardness with hand –eye coordination
- C. difficulty in bimanual coordination
- D. difficulty in walking
- E. awkwardness in performing complex activities

52. Which lobes receive and interpret sensory input for smell and / or hearing?

- A. frontal and temporal
- B. frontal and occipital
- C. parietal and occipital
- D. temporal and occipital
- E. frontal, occipital and temporal

53. What cranial nerve causes movements of the tongue involved in speaking, manipulating food and swallowing?

- A. facial
- B. trigeminal
- C. hypoglossal
- D. vagus
- E. A and B

54. The two main kinds of stretch receptors or proprioceptors that transmit information to the spinal cord and brain about muscle tension and length are

- A. Meissner's corpuscles and pacinian corpuscles
- B. Meissner's corpuscles and Golgi tendon organs
- C. muscle spindles and Golgi tendon organs
- D. pacinian corpuscles and Golgi tendon organs
- E. pacinian corpuscles

55. What part of the Body can represent unilaterally in the Cortex Brain?

- A. a foot
- B. an arm
- C. a face
- D. an leg
- E. a hand, a foot, a leg, an arm

56. The neuron of the spinothalamic system that ascends within the spinal cord and carries sensory information from a dorsal horn to the thalamus is designated a

- A. primary neuron
- B. tertiary neuron
- C. quaternary neuron
- D. A and B
- E. secondary neuron

57. The primary somatic sensory cortex is located in the

- A. angular gyrus
- B. cingulated gyrus
- C. precentral gyrus
- D. postcentral gyrus
- E. A and B

58. An injury to the lateral portion of the postcentral gyrus in the right hemisphere would result in some loss of sensation in the

- A. left lower limb
- B. right lower limb
- C. left facial area
- D. right facial area
- E. A and B

59. The medial lemniscus

- A. is a tract of nerve fibers that conveys sensory signals to the thalamus
- B. receives information from the brain and spinal cord and relays it to the cerebellum
- C. provides an avenue of communication between the two cerebral hemispheres
- D. links the primary motor area to the primary sensory area
- E. B and C

60. Which of the following tracts carry motor impulses to trunk and upper and lower limb muscles to maintain posture?

- A. corticospinal tracts
- B. reticulospinal tracts
- C. spinocerebellular tracts
- D. spinothalamic tracts
- E. A and D

5. CORRECT ANSWERS

1. Correct Answer: C
2. Correct Answer: B
3. Correct Answer: C
4. Correct Answer: A
5. Correct Answer: B
6. Correct Answer: A
7. Correct Answer: D
8. Correct Answer: C
9. Correct Answer: C
10. Correct Answer: D
11. Correct Answer: D
12. Correct Answer: E
13. Correct Answer: B
14. Correct Answer: C
15. Correct Answer: C
16. Correct Answer: D
17. Correct Answer: B
18. Correct Answer: E
19. Correct Answer: A
20. Correct Answer: C
21. Correct Answer: D
22. Correct Answer: B
23. Correct Answer: A
24. Correct Answer: C
25. Correct Answer: D
26. Correct Answer: D
27. Correct Answer: C
28. Correct Answer: E
29. Correct Answer: C
30. Correct Answer: E
31. Correct Answer: B
32. Correct Answer: C
33. Correct Answer: D
34. Correct Answer: A
35. Correct Answer: C
36. Correct Answer: D
37. Correct Answer: A
38. Correct Answer: D
39. Correct Answer: C
40. Correct Answer: E
41. Correct Answer: D
42. Correct Answer: A
43. Correct Answer: C
44. Correct Answer: C
45. Correct Answer: B
46. Correct Answer: C
47. Correct Answer: C
48. Correct Answer: E
49. Correct Answer: A
50. Correct Answer: C
51. Correct Answer: D
52. Correct Answer: A
53. Correct Answer: C
54. Correct Answer: C
55. Correct Answer: E
56. Correct Answer: E
57. Correct Answer: D
58. Correct Answer: C
59. Correct Answer: A
60. Correct Answer: B

6. RECOMMENDED LITERATURE

1. Rizzolatti G, Luppino G .The Cortical Motor System. Neuron 31: 889-901, 2001
2. Rodney A. Rhoades, David R. Bell Medical Principles for Clinical Medicine - 2012, 821p.
3. Latash, Mark L; Scholz, John P; Schönner, Gregor. "Toward a new theory of motor synergies". 2007, 276–308.
4. Wise, Stephen P.; Shadmehr, Reza (July 10, 2002). "Motor Control". Encyclopedia of the Human Brain. Academic Press. pp. 137–157.
5. Guyton A. C. Textbook of Medical Physiology: Philadelphia, 2011, 1116p.
6. Meier, J.D., Aflalo, T.N., Kastner, S. (2008). "Complex organization of human primary motor cortex: A high-resolution fMRI study". J. Neurophysiol (4): 1800–1812.
7. Hanong V.F. Human physiology: Lvov: BaK, 2009. – 784p.
8. Hentze M.W, Muckenthaler MU, Andrews NC: Balancing acts: molecular control of mammalian iron metabolism. Cell 117:285, 2004.
9. Rivara C.B, Sherwood C.C, Bouras C, and Hof PR (2003). "Stereologic characterization and spatial distribution patterns of Betz cells in the human primary motor cortex". The anatomical record. Part A, Discoveries in molecular, cellular, and evolutionary biology 270(2): 137–151
10. Cisik, P and Kalaska, J.F (2005). "Neural correlates of reaching decisions in dorsal premotor cortex: specification of multiple direction choices and final selection of action".Neuron 45 (5): 801–814.
11. Churchland M.M., Yu B.M., Ryu, S.I., Santhanam G. and Shenoy K.V (2006). "Neural variability in premotor cortex provides a signature of motor preparation". J. Neurosc 26(14): 3697–3712.
12. Picard N. and Strick P.L (2003). "Activation of the supplementary motor area (SMA) during performance of visually guided movements". Cereb. Cortex 13 (9): 977–986.
13. Sembulingam K. Essentials of Medical Physiology. Jaypee, 2013, 895p.

14. Cisik P and Kalaska J.F (2005). "Neural correlates of reaching decisions in dorsal premotor cortex: specification of multiple direction choices and final selection of action".Neuron 45 (5): 801–814.
15. Sujit K. Chaudhuri. Concise Medical Physiology.-New Central Book Agency, 2013.-752p.