NOTES
MOTOR NERVOUS SYSTEM

MOTOR CORTEX

osms.it/motor-cortex

MOTOR CORTEX BASICS

- Cerebral cortex region dedicated to voluntary movement planning, control, execution
- *Location:* posterior precentral gyrus, anterior to central sulcus

THREE INTERCONNECTED REGIONS

Premotor cortex

- Movement preparation, sensory guidance
- Emphasis on control of proximal, trunk muscles

Supplementary motor cortex

- Internally generates movement planning sequences
- Programs complex motor sequences
  - Active during mental movement rehearsal (even without physical execution)
- Coordinates two sides of body, bilateral movement

Primary motor cortex (area four)

- Topographically organized into motor homunculus
- Origin of programmed motor neuron activation patterns → movement execution
- Upper motor neurons in motor cortex become excited → transmit to brain stem, spinal cord → activate lower motor neurons → coordinated appropriate muscle contraction (voluntary movement)

MOTOR ACTIVATION PATTERN

- Supplementary motor, premotor cortices develop motor plan (specific muscles to contract, extent, sequence) → upper motor neurons in primary motor cortex → descending nerve tracts → lower motor neurons in spinal cord
- Basal ganglia, cerebellum provide additional fine tuning of motor output

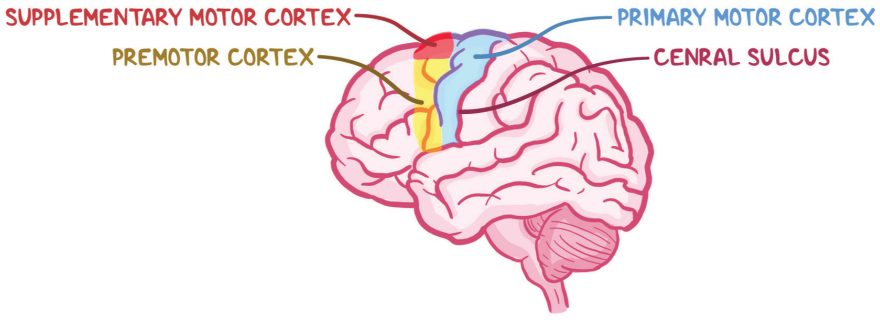

Figure 54.1 The three regions of the motor cortex.

MOTOR NEURONS & MUSCLE SPINDLES

osms.it/motor-neurons-and-muscle-spindles

MOTOR NEURONS

Motor unit

- Single motor neuron, muscle fibers it innervates
- All muscle fibers in motor unit are same fiber type (slow vs. fast twitch)
- *Fine control:* few muscle fibers per neuron (e.g. eye muscles)
- *Coarse control:* thousands of muscle fibers per neuron (e.g. postural muscles)
- *Motor neuron pool:* motor neuron collection innervating muscle fibers in same muscle

Force of contraction

- Graded action; determined by number of motor units recruited
- *Small motor neurons:* innervate few muscle fibers (generate relatively small amounts of force) → low threshold to activation → typically fire first
- *Large motor neurons:* innervate many muscle fibers (generate relatively large amounts of force) → require large action potentials to activate → typically fire last
- *Size principle:* more motor units recruited → larger motor neurons involved → greater tension developed

MUSCLE SPINDLE FIBERS

Extrafusal fibers

- Majority of skeletal muscle
- Innervated by α motor neurons
  - Large, myelinated multipolar (one axon, many dendrites) neurons that innervate extrafusal muscle fibers of skeletal muscles
  - Directly responsible for muscle contraction
  - Generate force

Intrafusal fibers

- Innervated by γ motor neurons
  - Small myelinated neurons that don't directly innervate muscle
  - Innervate intrafusal fibers → keep muscle spindles tight → allows for accurate detection of degree of stretch
- Too small to generate significant force
- Encapsulated in sheaths → form muscle spindles
  - Run parallel to extrafusal fibers
  - Abundant in muscles used for fine movements
  - Spindle-shaped organs composed of intrafusal muscle fibers
  - Innervated by sensory, motor nerve fibers
  - Attached to connective tissue

Intrafusal muscle spindle types

- Both subtypes present in every spindle
- Nuclear bag fibers
  - Larger, nuclei accumulate in central "bag" region
- Nuclear chain fibers
  - Smaller, nuclei arranged in rows (chains), more common

MUSCLE SPINDLES INNERVATION

Sensory (afferent) nerves

- Group Ia afferent nerves
  - Innervates central region of both intrafusal muscle spindle subtypes
  - Relatively large nerves → fast conduction velocity
  - Form primary endings in spiral-shaped terminal around central region of muscle spindle fibers

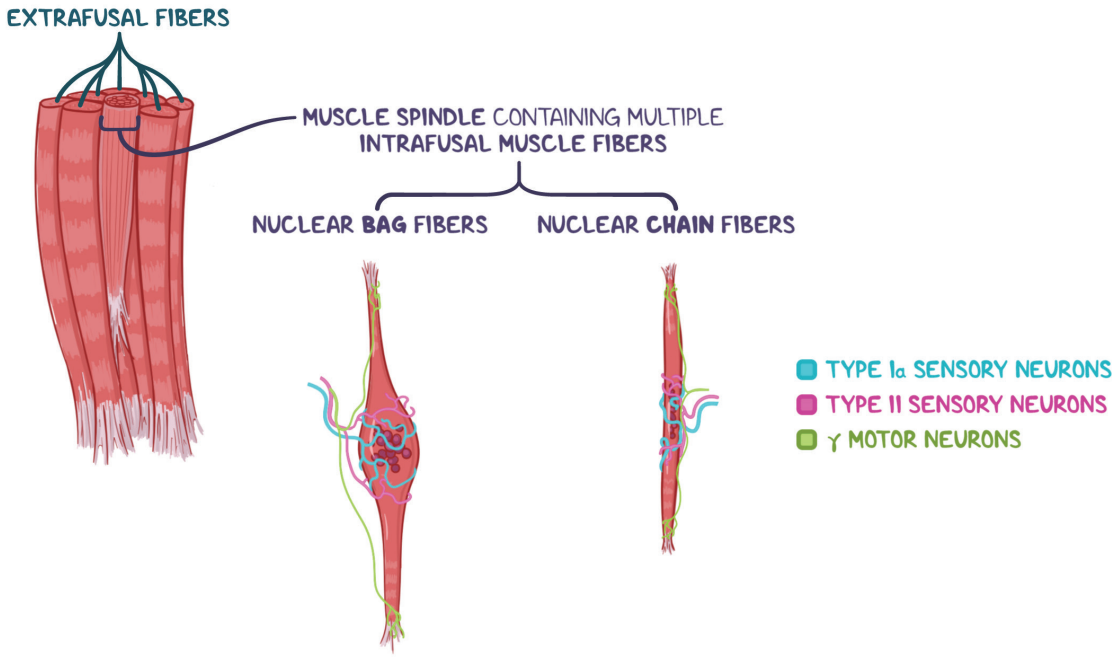

Figure 54.2 Muscles are composed of muscle fibers bundles with extrafusal muscle fibers on the outside and intrafusal fibers on the inside. There are two intrafusal fiber subtypes: nuclear bag fibers and nuclear chain fibers, determined by the nuclei arrangement within.

- Group II afferent nerves
  - Primarily innervate nuclear chain fibers
  - Intermediate diameter → intermediate conduction velocity
  - Form secondary endings on nuclear chain fibers (primarily)

Motor (efferent) nerves

- Two types
  - Dynamic γ motor neurons → synapse on nuclear bag fibers → "plate endings"
  - Static γ motor neurons → synapse on nuclear chain fibers → form "trail endings" → spread out over longer distances

MUSCLE SPINDLES FUNCTION

- Stretch receptors
- Extrafusal muscle fibers contract/stretch → muscle spindles correct for changes in muscle length → return muscle to resting length after shortening/lengthening
  - Muscle stretch → extrafusal muscle fibers lengthen, parallel intrafusal fibers stretch
  - Increased length of intrafusal fibers detected by sensory afferent fibers innervating them; increase in length of intrafusal fibers activates group Ia, group II sensory afferent fibers (group Ia afferent fibers detect velocity of length change; group II afferent fibers detect length of muscle fibers)
  - Activation of group Ia afferent fibers stimulates α motor neurons in spinal cord → innervation of extrafusal fibers in same muscle → muscle contraction → original stretch is opposed when reflex causes muscle to contract
  - γ motor neurons coactivated with α motor neurons → muscle spindle remains sensitive to muscle length changes (even during contraction)

MOTOR NEURONS CLASSIFICATION

- α motor neurons
  - Innervate extrafusal skeletal muscle fibers → contraction
- V motor neurons
  - Smaller, slower
  - Regulate sensitivity of intrafusal muscle fibers
  - Innervate specialized intrafusal muscle fibers (part of muscle spindles that sense muscle length) → adjust sensitivity of muscle spindles → ensures appropriate response as extrafusal fibers contract
- α, γ motor neurons are co-activated → muscle spindles remain sensitive to muscle length changes as muscle contracts

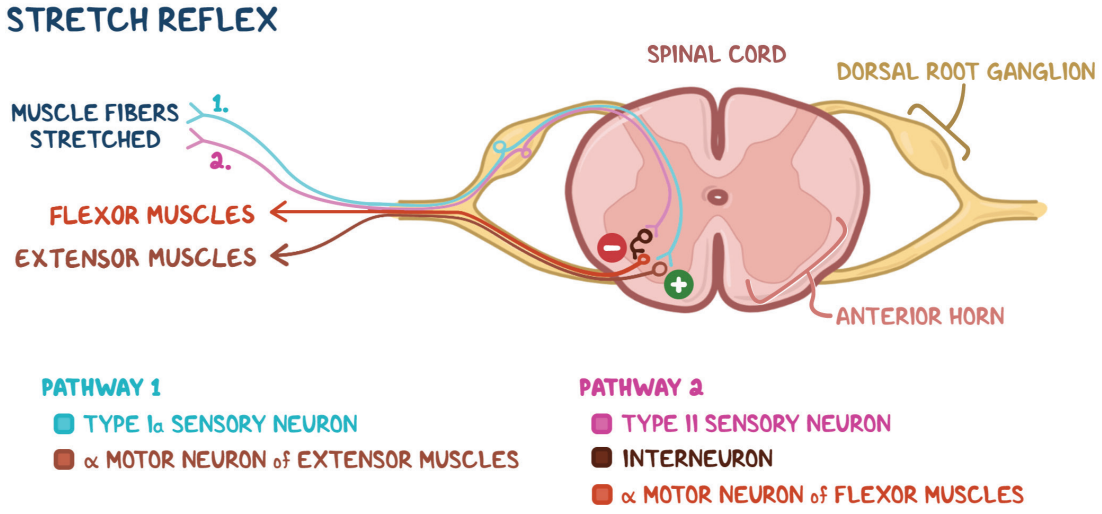

Figure 54.3 Stretch reflex when extensor muscles are stretched. Type Ia sensory neurons synapse with α motor neurons of extensor muscles, causing extensor muscle contraction. Type II sensory neurons synapse with an interneuron, which inhibits the α motor neurons to the flexor muscles → flexor muscles relax. These actions together oppose the original stretch.

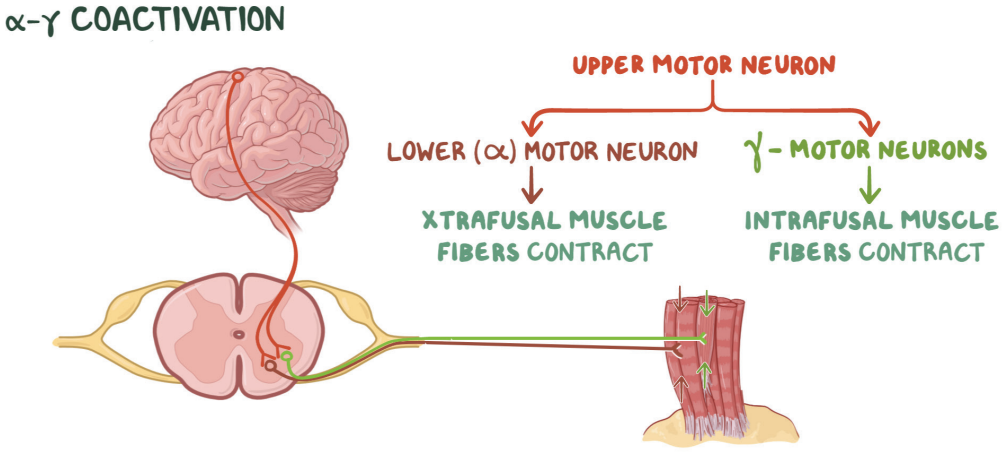

Figure 54.4 Coactivation of lower motor neurons and gamma motor neurons by upper motor neurons ensures that muscle spindle remains sensitive to muscle length changes (even during contraction).

PYRAMIDAL & EXTRAPYRAMIDAL TRACTS

osms.it/pyramidal-and-extrapyramidal-tracts

- Motor neurons descend from cerebral cortex (cortical motor areas, associated modulatory areas), brainstem via pyramidal, extrapyramidal tracts

PYRAMIDAL TRACTS

- Pass through medullary pyramids → descend onto lower motor neurons in spinal cord

Corticospinal tract

- Forms efferent nerve fibers of upper motor neurons → conduct impulses from brain to spinal cord
- Cortical motor areas (primary motor cortex, premotor cortex, supplementary motor areas), modulating sensory areas (somatosensory cortex, parietal lobe, cingulate gyrus) → posterior limb of internal capsule → cerebral peduncle (base of midbrain) → pons → medulla → spinal cord → synapse directly onto alpha motor neurons → control voluntary movement
- Forms two tracts based on where fibers cross over (decussate) to opposite side of body in medulla oblongata (decussation → muscles controlled by contralateral side of brain)
  - Lateral corticospinal tract, anterior corticospinal tract
- *Lateral corticospinal tract:* responsible for fine-motor movement of upper, lower limbs
  - Forms at level of medullary pyramids → 90% of corticospinal tract fibers decussate → lateral corticospinal tract
- *Anterior corticospinal tract:* responsible for gross, postural movement of trunk, proximal musculature
  - Forms at level of medullary pyramids → 10% of corticospinal tract fibers do not decussate → forms anterior tract; eventually decussate at spinal level they innervate
- Damage → upper motor neuron syndrome

Corticobulbar tract

- Conducts impulses from brain → cranial nerves
- *Primary motor cortex:* projects through corona radiata, genu of internal capsule/ some fibers through posterior limb of internal capsule → midbrain
- *Midbrain:* internal capsule becomes cerebral peduncles, ventral white matter of cerebral peduncles form crus cerebri → middle third of crus cerebri forms corticobulbar (and corticospinal fibres) → corticobulbar fibers exit brainstem at appropriate level to synapse on lower motor neurons of cranial nerves
- Controls facial, neck muscles (expression, mastication, swallowing)
- Only nerves controlling muscles of lower face decussate
- *Damage:* unilateral → only involves lower face; bilateral → pseudobulbar palsy (inability to control facial muscles)
  - *Pseudobulbar palsy signs, symptoms:* slow, indistinct speech; dysphagia; small/stiff/spastic tongue; brisk jaw jerk, labile affect with/without evidence of upper motor lesion also affecting limbs

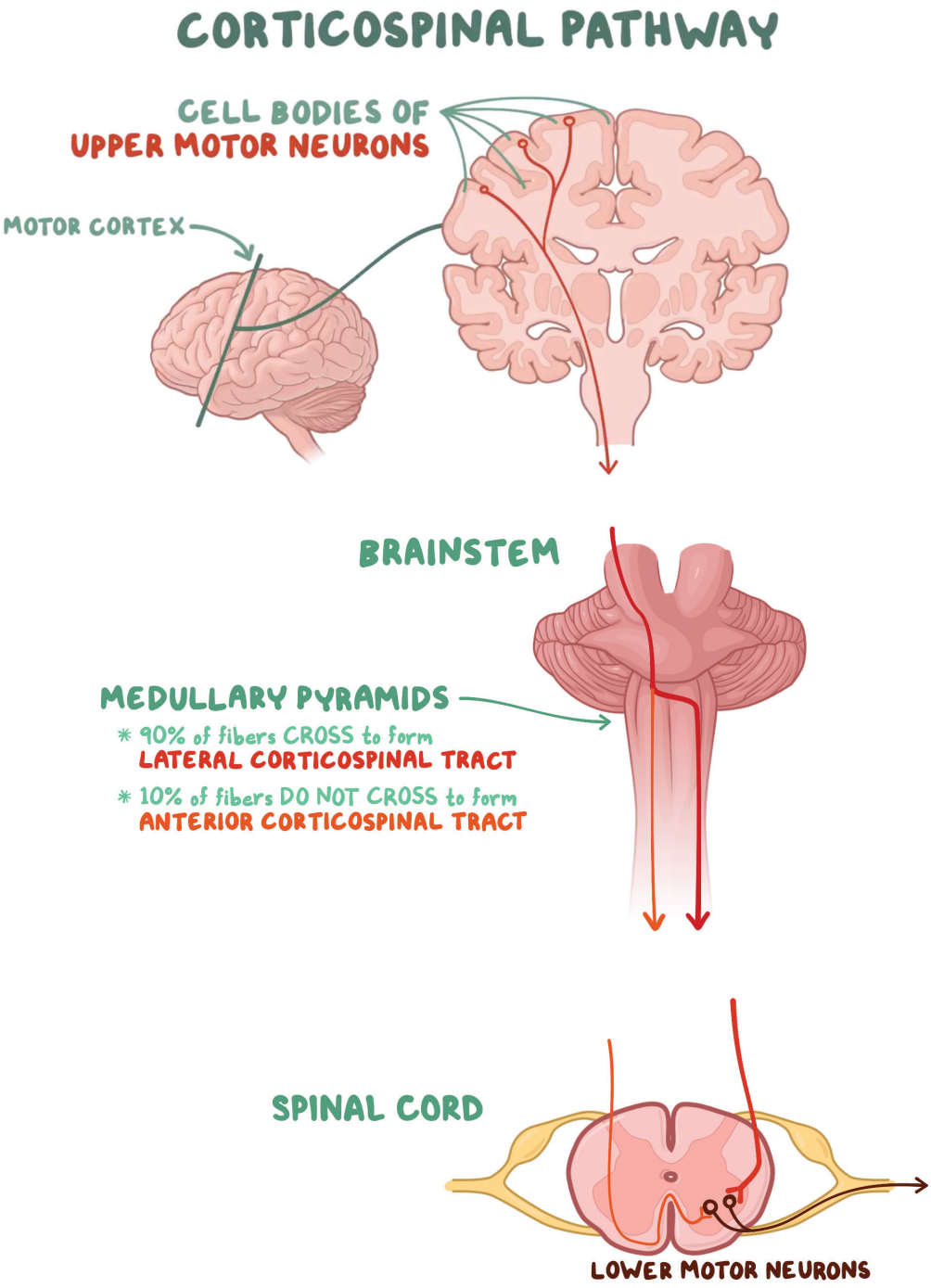

Figure 54.5 Upper motor neuron pathway in corticospinal tract. Lateral corticospinal tract fibers decussate in medulla, while anterior corticospinal tract fibers decussate at the level of the lower motor neuron (which they synapse with).

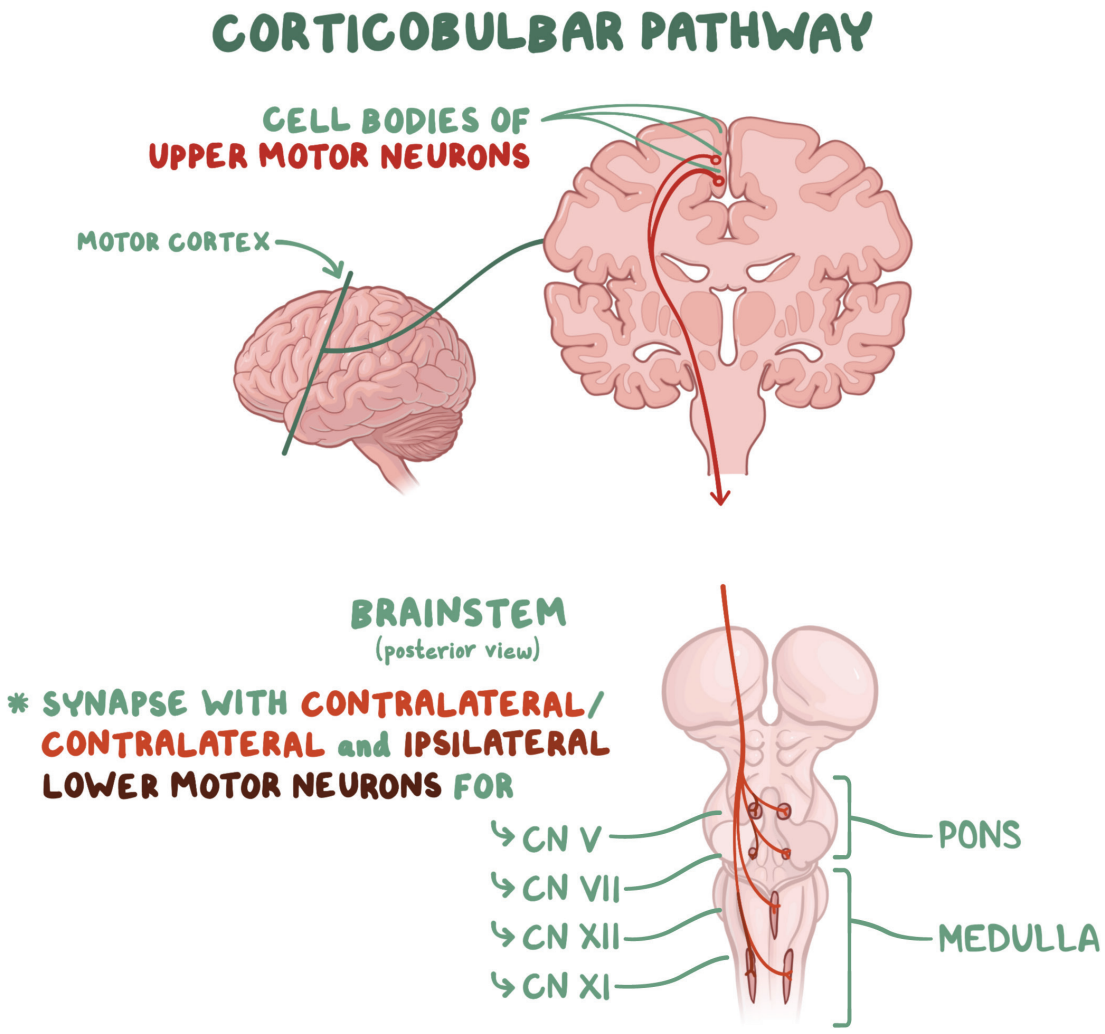

Figure 54.6 Pathway of upper motor neurons in corticobulbar tract. The fibers that decussate do so at the cranial nerve level (which they synapse with). Cranial nerve lower motor neurons that receive upper motor neuron branches from both ipsilateral, contralateral sides include: CN V, XI, and portion of VII (that innervates muscles of the face's upper half). Cranial nerves that only receive upper motor neuron signals from the contralateral side include: CN XII and the part of VII that controls muscles of the face'slower half.

EXTRAPYRAMIDAL TRACTS

- Motor neurons from motor cortex that don't pass through pyramids of medulla; tracts run through pons, medulla, target lower motor neurons in spinal cord
- Control reflexes, locomotion, complex movements, posture
- Modulated by nigrostriatal pathway, basal ganglia, cerebellum, vestibular nuclei, sensory areas of cerebral cortex
- *Extrapyramidal tract damage:* various types of dyskinesias (involuntary movement disorders)

Rubrospinal tract

- Originates in red nucleus of midbrain, projects to motor neurons in lateral spinal cord (runs adjacent to lateral corticospinal tract), terminates in cervical spinal cord
- Mediates voluntary movement (primarily upper limbs)
- Activates flexor muscles
- Inhibits extensor muscles
- Can assume function of corticospinal tract if corticospinal tract is injured
- *Damage:* temporary slowness of movement

Lateral vestibulospinal tract

- Originates in lateral vestibular nucleus (Deiters nucleus), projects to ipsilateral motor neurons in spinal cord
- Activates extensors; inhibits flexors
- Maintains upright balance, posture through action on muscles of trunk, legs
- Receives input from cerebellum

Reticulospinal tract

- Coordinates automatic locomotion, posture movements
- Facilitates, inhibits voluntary movement
- Mediates autonomic function
- Modulates pain
- Damage at/just below level of red nucleus
  - *Decerebration:* unopposed extension of head, limbs
- *Pontine (medial) reticulospinal tract:* originates in nuclei of pons, projects to ventromedial spinal cord; activates anti-gravity extensor muscles
- *Medullary (lateral) reticulospinal tract:* originates in medullary reticular formation, projects to spinal cord; inhibits excitatory axial extensors

Tectospinal tract (colliculospinal tract)

- Originates in superior colliculus, projects to cervical spinal cord
- *Controls neck muscles:* mediates reflex, postural movements of head in response to visual, auditory stimuli

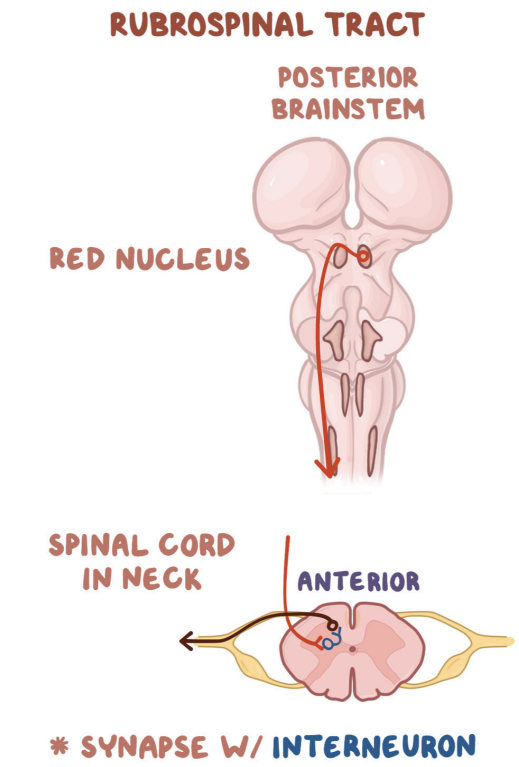

Figure 54.7 Rubrospinal tract.

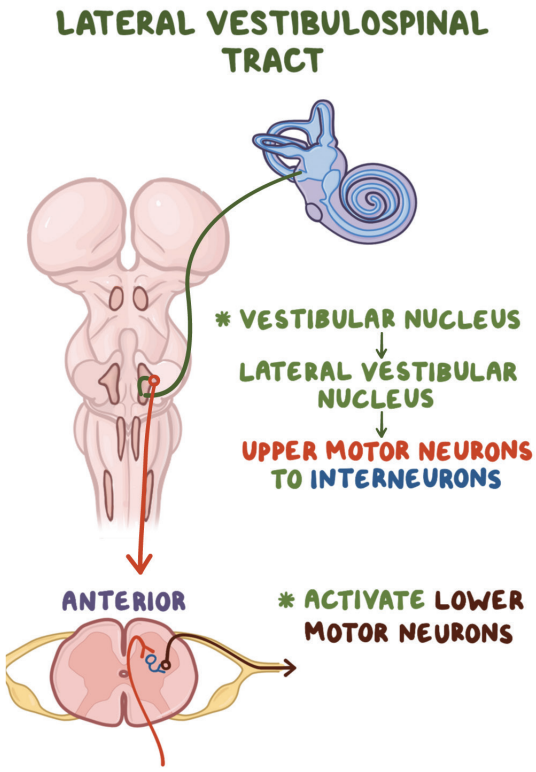

Figure 54.8 Lateral vestibulospinal tract.

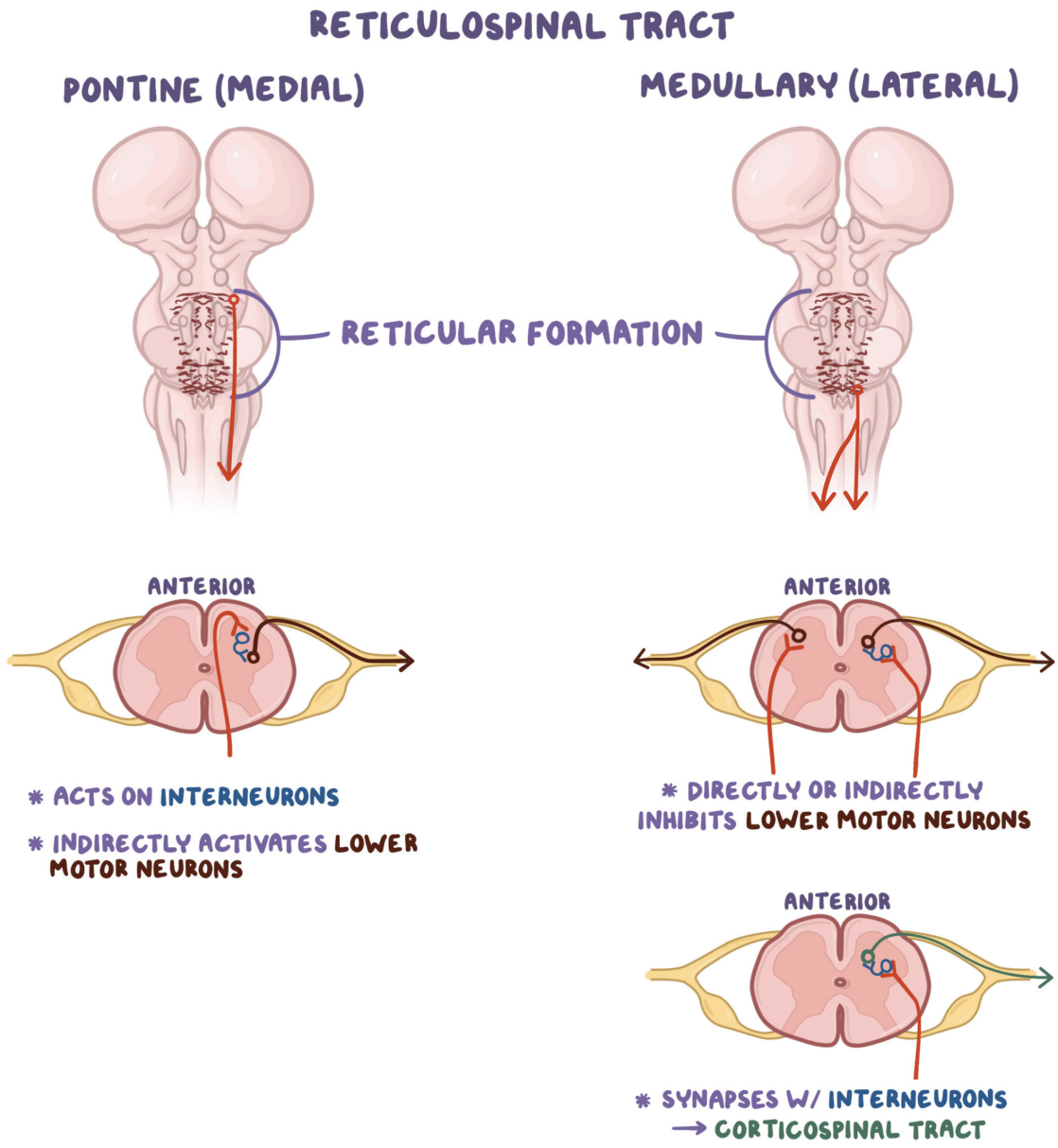

Figure 54.9 Pontine and medullary reticulospinal tracts.

SIGNS OF UPPER VS. LOWER MOTOR NEURON LESIONS

	UPPER MOTOR NEURON LESION	LOWER MOTOR NEURON LESION
LESION	Lesion anywhere in motor tract above anterior horn cell of spinal cord	Lesion affects nerve fibers travelling from anterior horn of spinal cord to relevant muscle
TONE	↑ Spasticity: (↑, involuntary, velocity-dependent muscle tone, causes resistance to movement) Clonus: (involuntary, rhythmic, muscular contractions, relaxation) Clasp-knife response: initial higher resistance to movement followed by lesser resistance	↓
POWER	Upper limb → primarily extensor weakness Lower limb → primarily flexor weakness	Distal > proximal distribution Flexors, extensors equally affected
DEEP TENDON REFLEXES	↑	↓/absent
BABINSKI REFLEX*	Babinski sign present	Absent
MUSCLE WASTING	Not notably wasted	Present
MUSCLE FASCICULATION	Absent	Present

*Babinski reflex: sole of the foot is stimulated with a pointed instrument → hallux flexion (normal) or extension (sign of pathology)

CEREBELLUM

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CEREBELLUM

- *Location:* posterior fossa below occipital lobe
- Connected to brain stem by three cerebellar peduncles containing afferent, efferent fibers
- *Regulates movement, posture:* controls movement synergy (rate, range, force, direction)

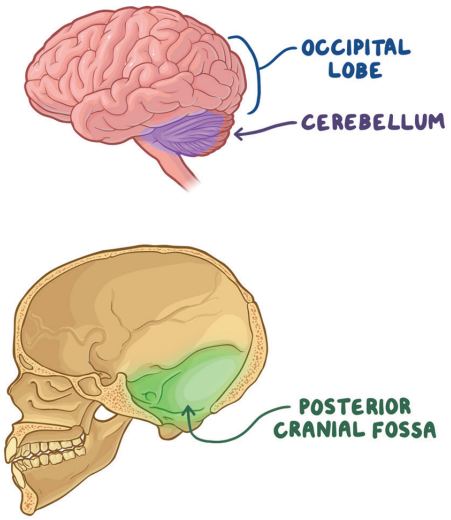

Figure 54.10 Cerebellum location relative to brain and skull.

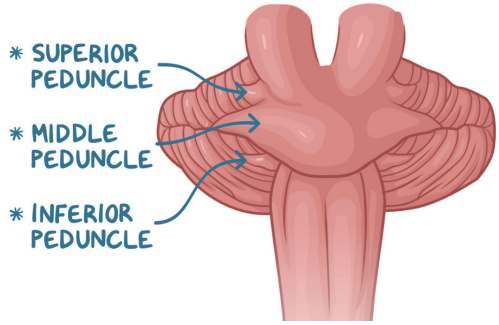

Figure 54.11 Superior, middle, inferior peduncles attach cerebellum to brain stem.

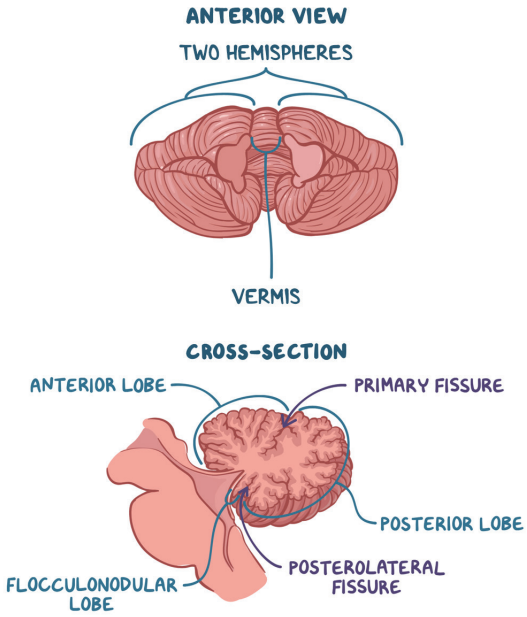

Figure 54.12 Cerebellum divisions from anterior, lateral views. The vermis is the narrow ridge separating the two hemispheres, fissures separate the lobes.

FUNCTIONAL DIVISIONS

Vestibulocerebellum

- *Anatomical components:* flocculonodular lobe (plus immediately adjacent vermis)
- *Vestibular input:* balance, eye movement

Spinocerebellum

- *Anatomical components:* vermis, intermediate parts of hemispheres
- *Spinal cord input (proprioception):* regulation of movement synergy

Pontocerebellum

- *Anatomical components:* lateral part of cerebellar hemispheres
- *Cerebral input (via pontine nuclei):* controls planning, movement initiation

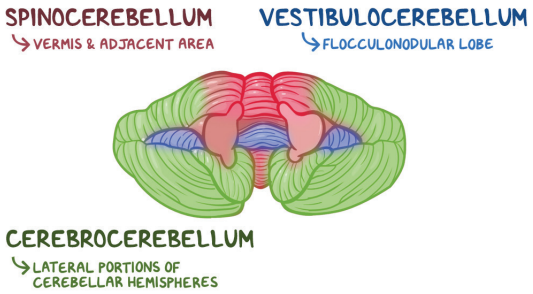

Figure 54.13 Functional cerebellum divisions (anterior view).

CEREBELLAR CORTEX

Three layers

- Molecular layer
  - *Outermost layer:* contains outer stellate cells, basket cells, dendrites of Purkinje, Golgi II cells, axons of granule cells
  - *Two inhibitory interneuron types:* stellate cells, basket cells (inhibit Purkinje cells, basket cells, outer stellate cells, Golgi type II cells)
- Purkinje cell layer
  - *Middle layer:* contains Purkinje cells
  - Primary integrative neurons of cerebellar cortex
  - Provides sole output of cerebellar cortex
  - Exclusively inhibitory output onto deep cerebellar neurons, vestibular nuclei of brainstem
- Granular layer
  - *Innermost layer:* contains granule cells, Golgi II cells, glomeruli
  - Excitatory mossy fibers from pontine nuclei enter granular layer, deep cerebellar nuclei; in glomeruli axons of mossy fibers from spinocerebellar, pontocerebellar tracts synapse on dendrites of granules, Golgi type II cells

Cerebellar cortex output

- Purkinje cell axons → always inhibitory (GABAergic)
- Purkinje cells axons project topographically to deep cerebellar nuclei, lateral vestibular nuclei
- Regulates movement synergy

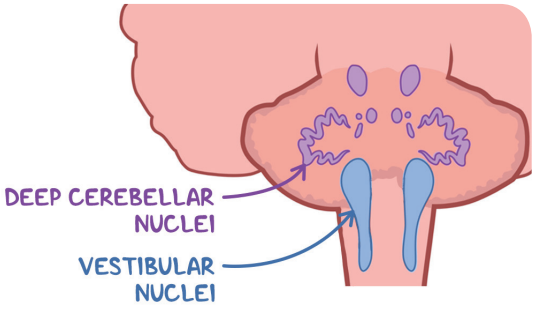

Figure 54.15 Deep cerebellar and vestibular nuclei, to which Purkinje cells project. The lateral vestibular nuclei are in the medulla.

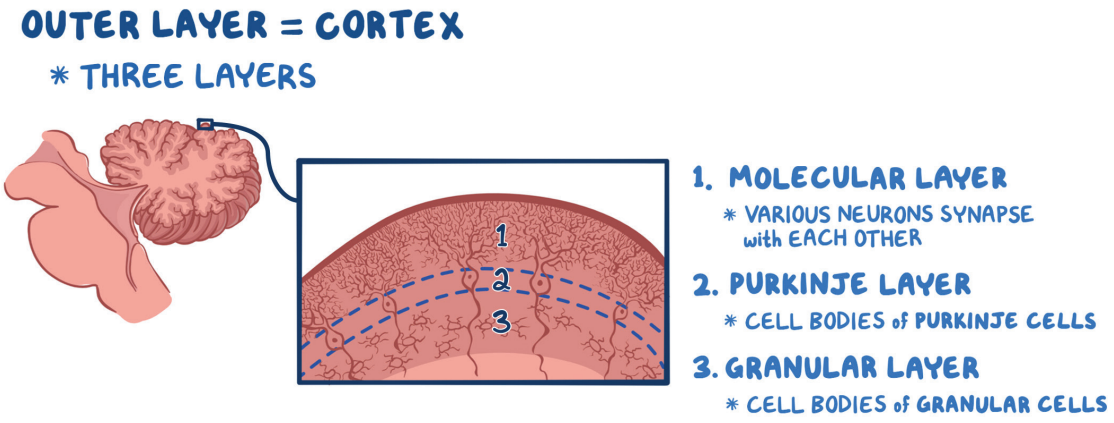

Figure 54.14 The three layers of the cerebellar cortex, from superficial to deep.

Excitatory input to cerebellar cortex

- *Arises from two systems:* climbing fibers, mossy fiber system (both project to deep cerebellar nuclei)
- *Climbing fibers:* originate in inferior olive of medulla, project directly to Purkinje cells in 1:1 ratio
  - Single action potential → multiple excitatory bursts of descending amplitude (complex spikes) in Purkinje dendrites
  - Modulate Purkinje cell response to mossy fiber input
  - May be involved in cerebellar learning
- *Mossy fiber system:* majority of cerebellar input
  - Vestibulocerebellar, spinocerebellar pontocerebellar afferents
  - Project to granule cells (excitatory interneurons) → found in synapse collections which form glomeruli → axons from granule cells ascend to molecular layer → bifurcate → form parallel fibers
  - Parallel fibers synapse with many Purkinje cell dendrites → excitation beams across Purkinje cell row
  - Each Purkinje cell's dendritic tree may receive input from up to 250, 000 parallel fibers (contrast with climbing fiber input to Purkinje dendrites → 1:1)
  - Mossy fiber input produces single action potential (AKA simple spikes)
  - Parallel fibers also synapse on cerebellar interneurons (basket, stellate, Golgi II)
- Excitatory projection from cerebellar cortex → activates secondary circuits → modulate output of cerebellar nuclei via Purkinje cells

Cerebellar interneurons

- Modulate Purkinje cell output
- All cerebellar interneurons are inhibitory (except granule cells)
  - Granule cells offer excitatory input for basket cells, stellate cells, Golgi II cells, Purkinje cells
  - Basket, stellate cells inhibit Purkinje cells (parallel fibers)
  - Golgi II cells inhibit granule cells → reduce excitatory effect on Purkinje cells

LESION DISORDERS

- Lesions → lack of voluntary coordination of muscle movements, limbs, posture, gait (ataxia)

General signs and symptoms

- Lack of coordination → errors in fine movement control
- Delayed onset of movement/poor execution of sequences
- Overshoot target, stop before reaching
- *Dysdiadochokinesia:* unable to perform rapid alternating movements
- *Intention tremor:* tremor perpendicular to direction of voluntary movement, increases near end of movement

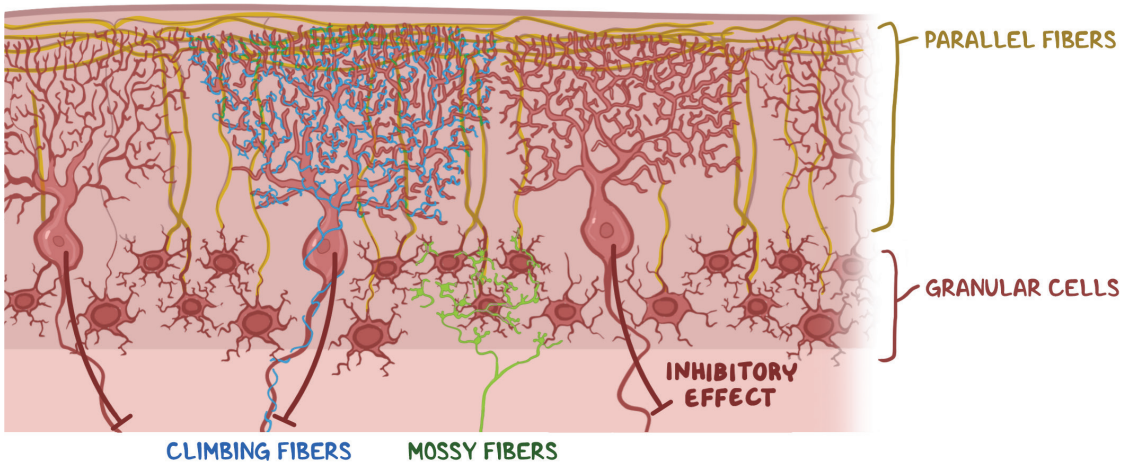

Figure 54.16 Projection destinations for climbing and mossy fibers.

- *Rebound phenomenon:* inability to stop movement

Specific signs and symptoms

- According to affected portion of cerebellum
  - *Posterior (flocculonodular lobe):* nystagmus, poor postural control, gait dysfunction
  - *Midline (vermis):* truncal, gait ataxia
  - *Lateral (hemispheric):* limb ataxia, dysmetria, dysdiadochokinesia, intention tremor, dysarthria, hypotonia

BASAL GANGLIA: DIRECT & INDIRECT PATHWAY OF MOVEMENT

osms.it/basal-ganglia-direct-indirect-pathways

BASAL GANGLIA

- Collection of subcortical nuclei
- Consists of globus pallidus, striatum (caudate nucleus, putamen, amygdala)
- *Associated nuclei:* ventral anterior, ventral lateral nuclei of thalamus; subthalamic nucleus of diencephalon; substantia nigra of midbrain
- *Function:* influence motor cortex via pathways through thalamus
  - Aid in planning, execution of smooth movements; contribute to affective, cognitive function

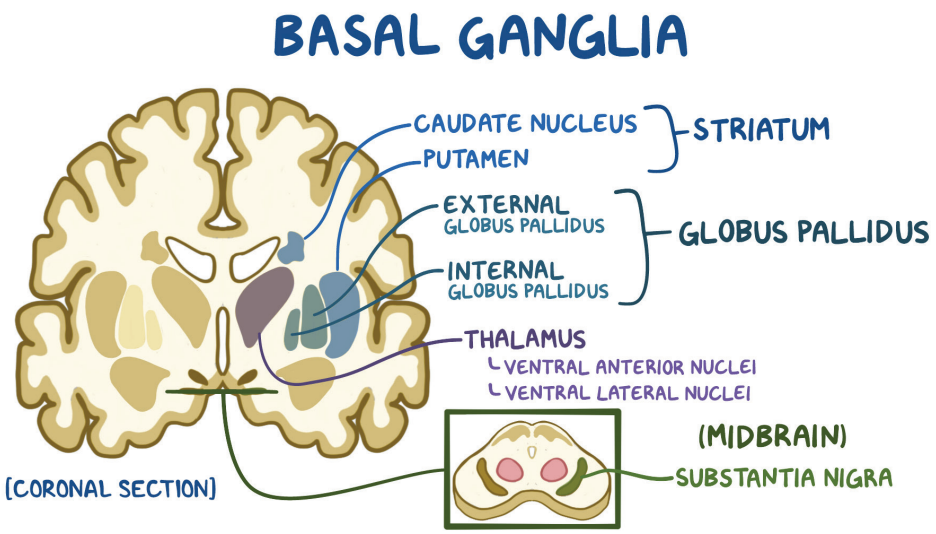

Figure 54.17 Location of basal ganglia and associated structures in coronal slice of the brain.

COMPLEX AFFERENT & EFFERENT PATHWAYS

- Excitatory pathways use glutamate as neurotransmitter
- Inhibitory pathways use GABA (γ-aminobutyric acid) as neurotransmitter
- Almost all cerebral cortex areas project topographically onto striatum, input from motor cortex → striatum → thalamus → back to the cortex via indirect/direct pathways
- Outputs of indirect, direct pathways from basal ganglia to motor cortex are opposed, balanced
  - Disturbance of output → upsets balance of motor control → activity increases/decreases
- Back-and-forth connection between striatum, pars compacta of substantia nigra are dopaminergic
  - Dopaminergic pathway is inhibitory via D2 receptors on indirect pathway; excitatory effect via D1 receptors on direct pathway

Direct pathway (excitatory)

- Striatum → inhibits → internal segment of globus pallidus, pars reticulata of substantia nigra (structures that would inhibit otherwise excitatory structures)
- Substantia nigra → inhibitory input to thalamus
- Thalamus → excitatory input to motor cortex
- Overall input is excitatory

Indirect pathway (inhibitory)

- Striatum → inhibits → external segment of globus pallidus → inhibits → subthalamic nucleus
- Subthalamic nucleus projects excitatory input to internal segment of globus pallidus → internal segment of globus pallidus, pars reticulata of substantia nigra → inhibits → thalamus
- Thalamus → excitatory input to motor cortex
- Overall input of indirect pathway is inhibitory

BASAL GANGLIA DISEASES

Parkinson's disease

- Cellular damage → cells of pars compacta of substantia nigra degenerate → reduce inhibition via indirect pathway, reduce excitation via direct pathway
- Initial accumulation in olfactory bulb, medulla oblongata, pontine tegmentum; early non-motor symptoms (loss of smell, sleep disturbances, autonomic dysfunction)
- *Progression:* affects midbrain, basal forebrain, neocortex, typical Parkinson's symptoms (resting tremor; movement slowness, delay; shuffling gait)
- *Treatment:* aim to ↑ dopamine level in brain/mimic its action with dopaminergic drugs
  - L-DOPA (dopamine precursor) → remaining dopamine neurons produce, secrete more dopamine
  - Dopamine agonists (e.g. bromocriptine) → bind to postsynaptic dopaminergic receptors
  - MAO-B inhibitors → impede dopamine breakdown

Huntington's disease

- Hereditary disorder caused by destruction of striatal, cortical cholinergic neurons, inhibitory GABAergic neurons
- Presents with chorea (writhing movements), dementia
- No known cure

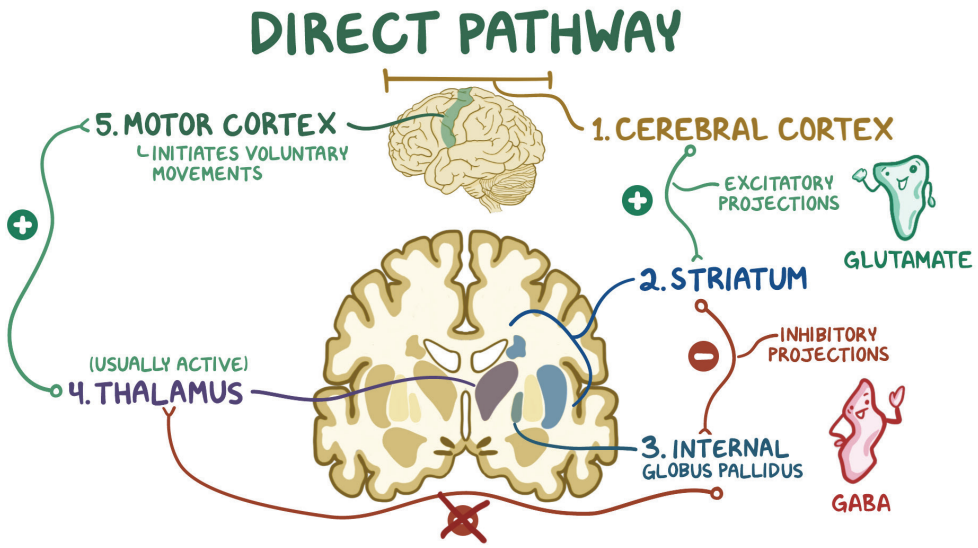

Figure 54.18 Direct pathway. Cerebral cortex sends excitatory projections to striatum → sends inhibitory projections to internal globus pallidus → sends inhibitory projections to thalamus. When striatum inhibits internal globus pallidus, internal globus pallidus can't inhibit thalamus → thalamus is free to send excitatory signals to motor cortex.

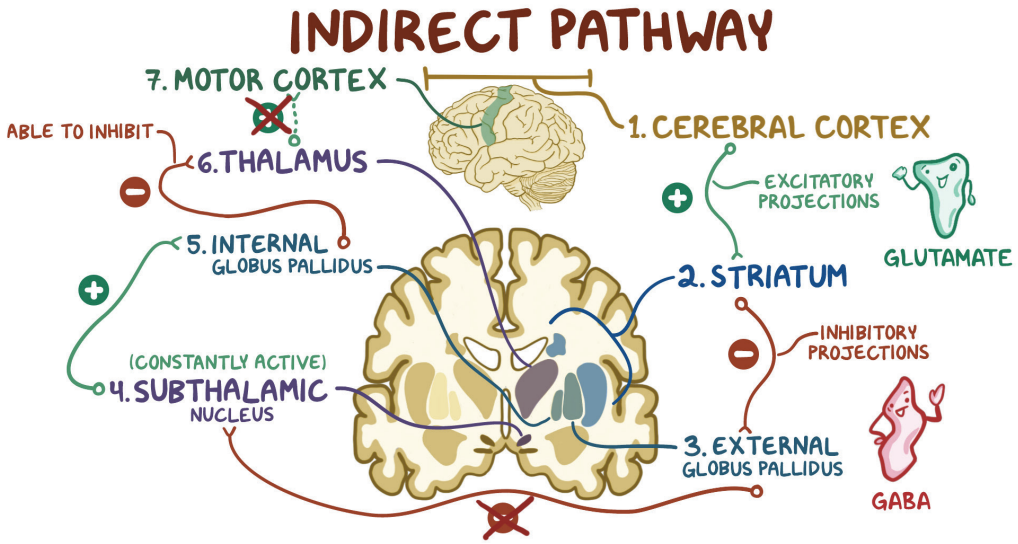

Figure 54.19 Indirect pathway. Cerebral cortex sends excitatory projections to striatum → sends inhibitory projections to external globus pallidus → sends inhibitory projections to subthalamic nucleus. When striatum inhibits external globus pallidus, external globus pallidus can't inhibit subthalamic nucleus → subthalamic nucleus is free to send excitatory signals to internal globus pallidus. Internal globus pallidus inhibits thalamus, preventing it from sending excitatory signals to the motor cortex.

SPINAL CORD REFLEXES

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Intrinsic reflex

- Involuntary, unlearned, rapid, predictable response to stimulus
  - Prevents need for conscious thought about all actions (e.g. staying upright, withdrawing from pain, controlling visceral reactions)
  - Subject to modification if necessary

Acquired reflex

- Acquired after sufficient repetition (e.g. complex sequence of reactions that occur while driving a car)
  - Process is automatic, but had to be learned initially

REFLEX ARC COMPONENTS

- *Receptor:* detects stimulus
- *Sensory neuron:* transmits afferent impulse to central nervous system (CNS)
- *Integration center:* processes information, dictates response
  - *Simple reflex arcs:* single synapse between sensory neuron, motor neurons (monosynaptic reflex)
  - *Complex reflex arcs:* multiple synapses with chains of interneurons (polysynaptic reflex)
- *Motor neuron:* conducts efferent impulse from integration center to effector
- *Effector:* muscle fiber/gland that responds to efferent impulse (contracts/secretes)

CLASSIFICATION

Somatic

- Activates skeletal muscle
- Voluntary; occasionally non-voluntary (reflexes)

Autonomic (visceral)

- Activates visceral organ effectors
  - *Smooth muscle:* involuntary; forms walls of hollow organs, glands, blood vessels, tracts of respiratory, urinary, reproductive systems
  - *Cardiac muscle:* involuntary; forms heart walls