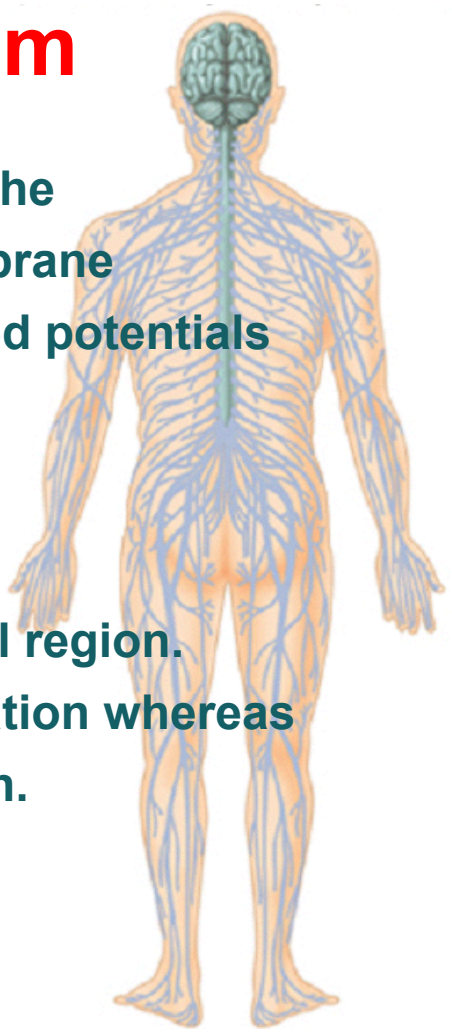
BENG 260
Supplementary neurophysiology slides

Fall 2013

Slides are taken from Vander's Human Physiology, 11th edition, McGraw Hill (ISBN 0077216091)

These slides cover:
Chapter 6, Neuronal Signaling and the Structure of the Nervous System
Chapter 8, Consciousness, Brain, and Behavior
Chapter 10, Control of Body Movement

Chapter 6
Neuronal Signaling and the Structure of the Nervous System

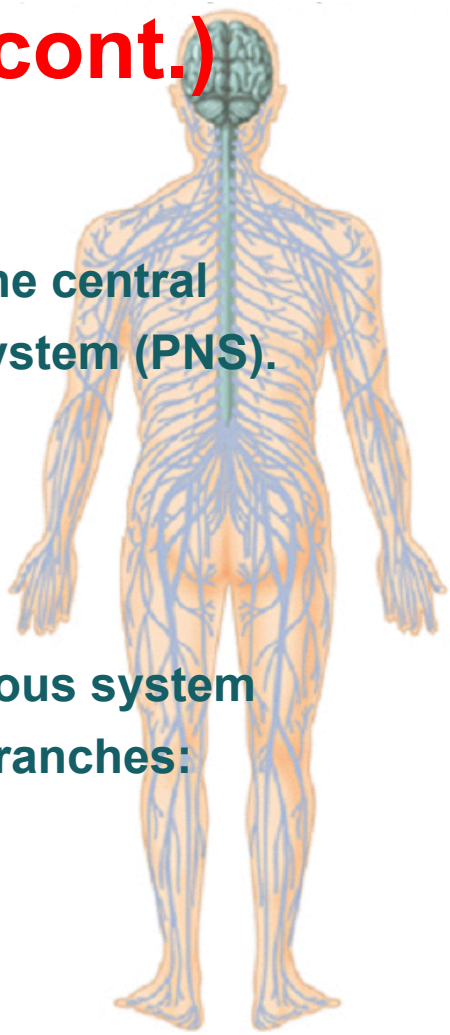


Communication by neurons is based on changes in the membrane's permeability to ions. Two types of membrane potentials are of major functional significance: graded potentials and action potentials.

A typical neuron has a dendritic region and an axonal region. The dendritic region is specialized to receive information whereas the axonal region is specialized to deliver information.

Chapter 6
Neuronal Signaling and the Structure of the Nervous System (cont.)

The two major divisions in the nervous system are the central nervous system (CNS) and the peripheral nervous system (PNS).



Within the PNS, major divisions are the somatic nervous system and the autonomic nervous system, which has two branches: the parasympathetic and the sympathetic branches.

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Dendrites: receive information, typically neurotransmitters, then undergo graded potentials.

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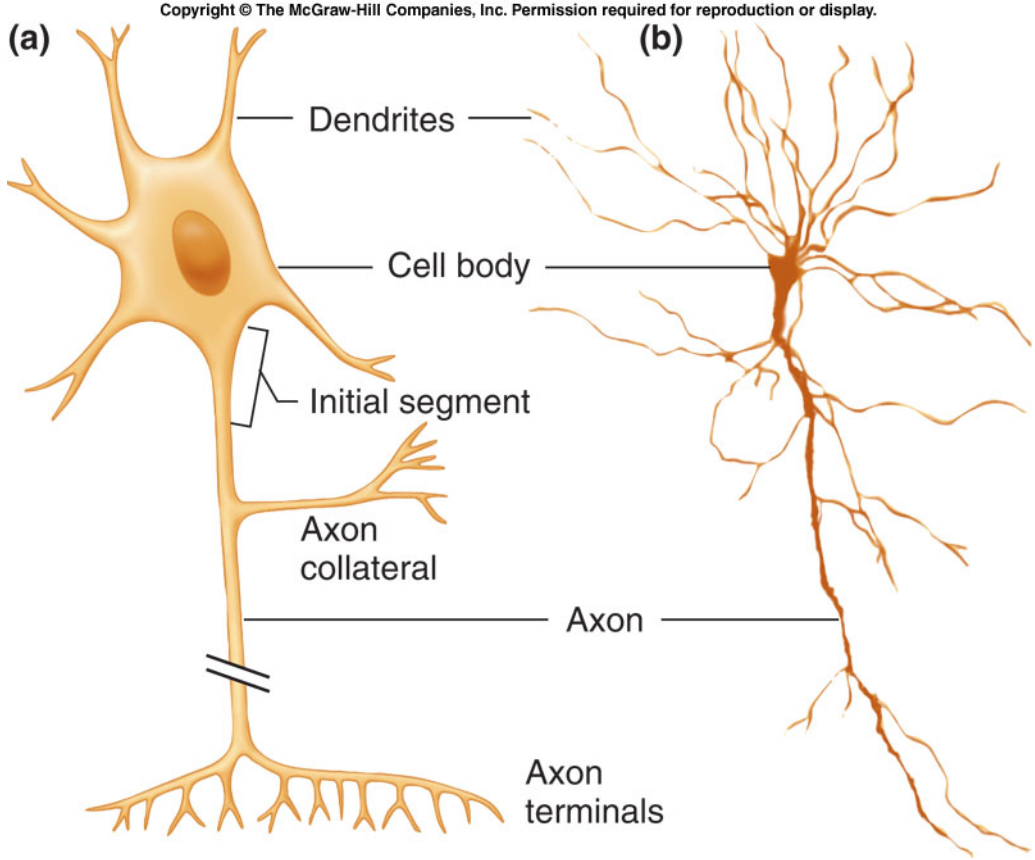

Figure 6-1

Axons: undergo action potentials to deliver information, typically neurotransmitters, from the axon terminals.

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Schwann cells form myelin on peripheral neuronal axons.

Oligodendrocytes form myelin on central neuronal axons.

Figure 6-2

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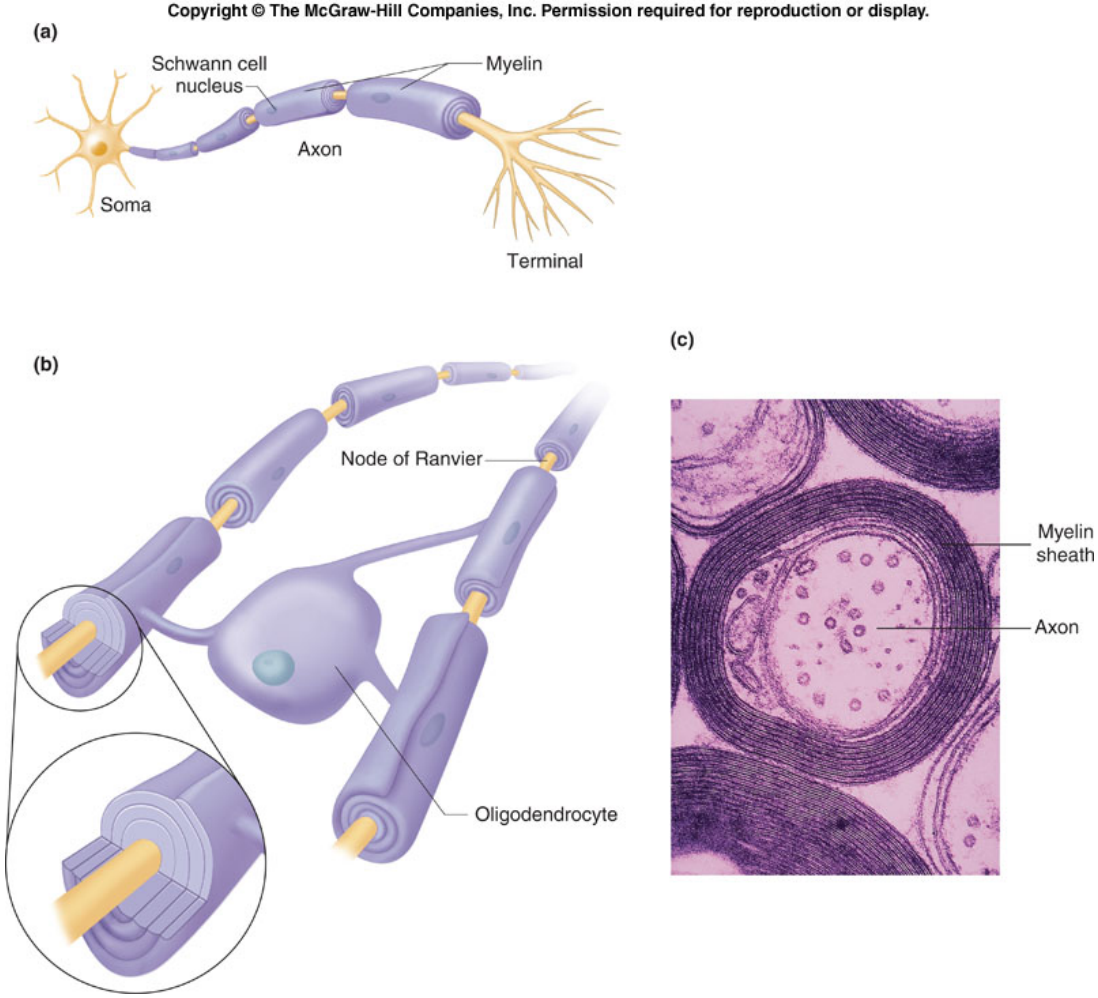

Among all types of neurons, myelinated neurons conduct action potentials most rapidly.

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Figure 6-4

CNS **PNS**

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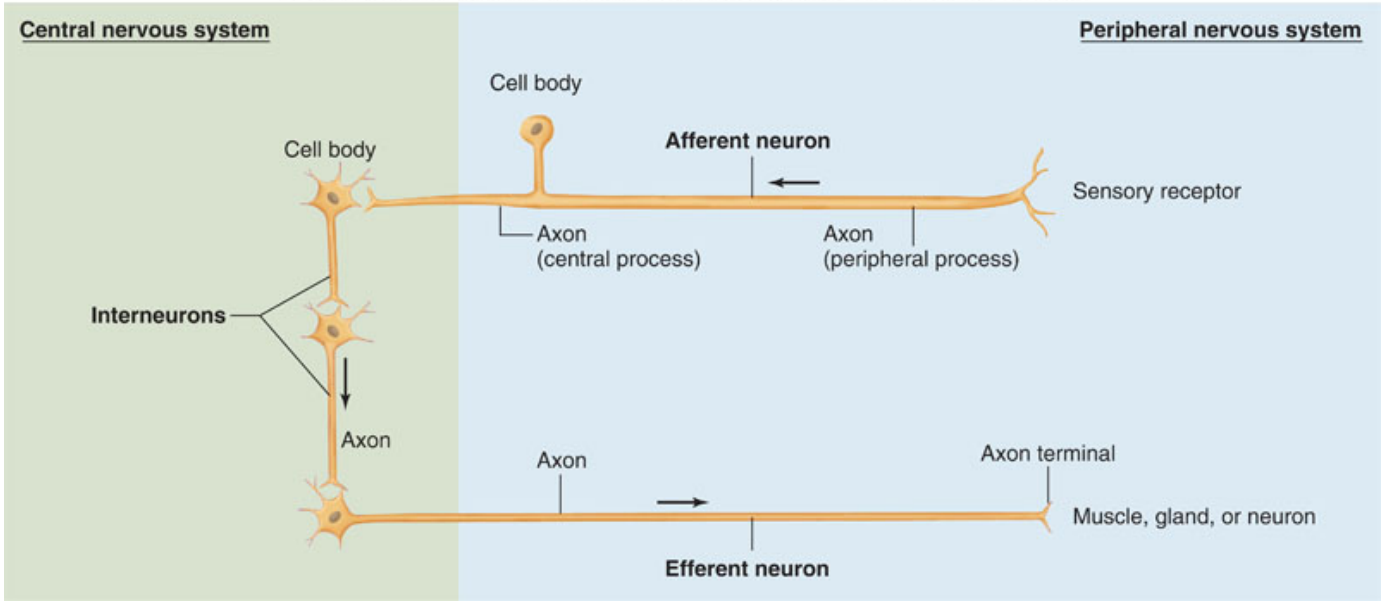

CNS = brain + spinal cord; all parts of interneurons are in the CNS.

PNS = afferent neurons (their activity "affects" what will happen next) into the CNS + efferent neurons ("effecting" change: movement, secretion, etc.) projecting out of the CNS.

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TABLE 6–1 Characteristics of Three Classes of Neurons

I. Afferent neurons

A. Transmit information into the central nervous system from receptors at their peripheral endings

B. Cell body and the long peripheral process of the axon are in the peripheral nervous system; only the short central process of the axon enters the central nervous system

C. Have no dendrites (do not receive inputs from other neurons)

II. Efferent neurons

A. Transmit information out of the central nervous system to effector cells, particularly muscles, glands, or other neurons

B. Cell body, dendrites, and a small segment of the axon are in the central nervous system; most of the axon is in the peripheral nervous system

III. Interneurons

A. Function as integrators and signal changers

B. Integrate groups of afferent and efferent neurons into reflex circuits

C. Lie entirely within the central nervous system

D. Account for 99 percent of all neurons

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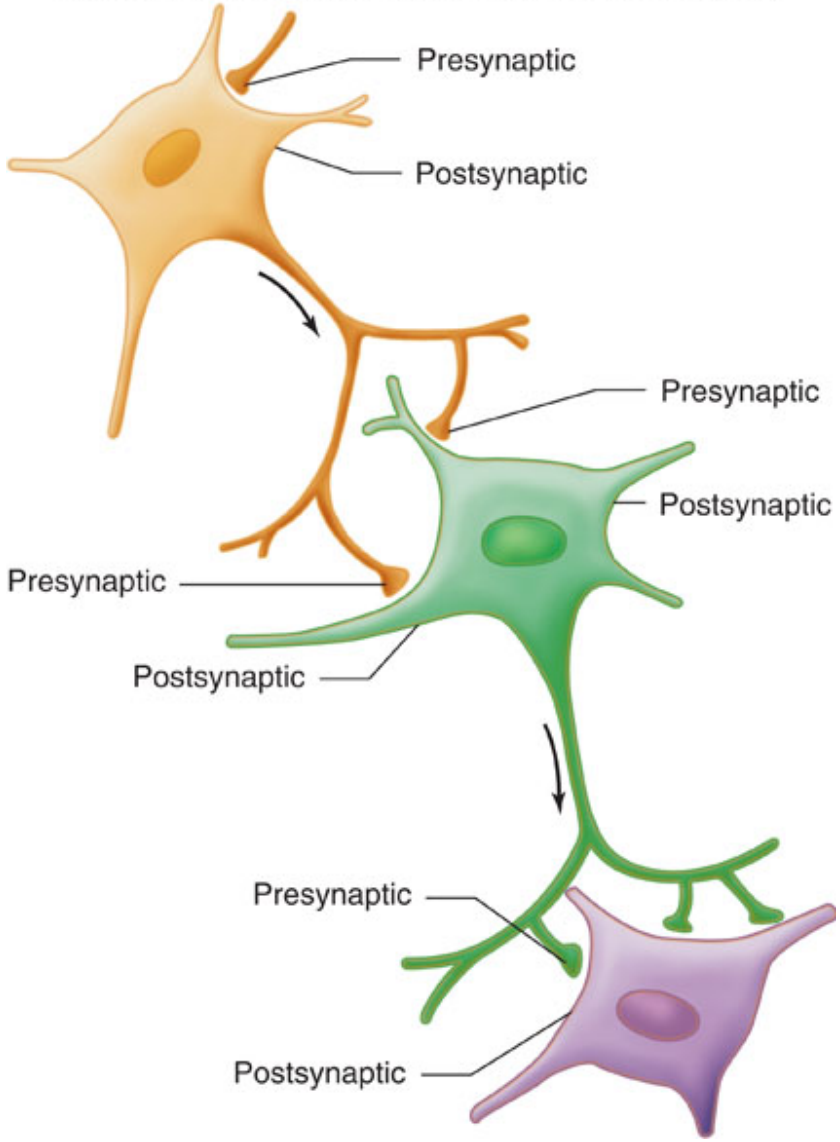

COMMUNICATION:

A single neuron postsynaptic to one cell can be presynaptic to another cell.

Figure 6-5

Figure 6-7

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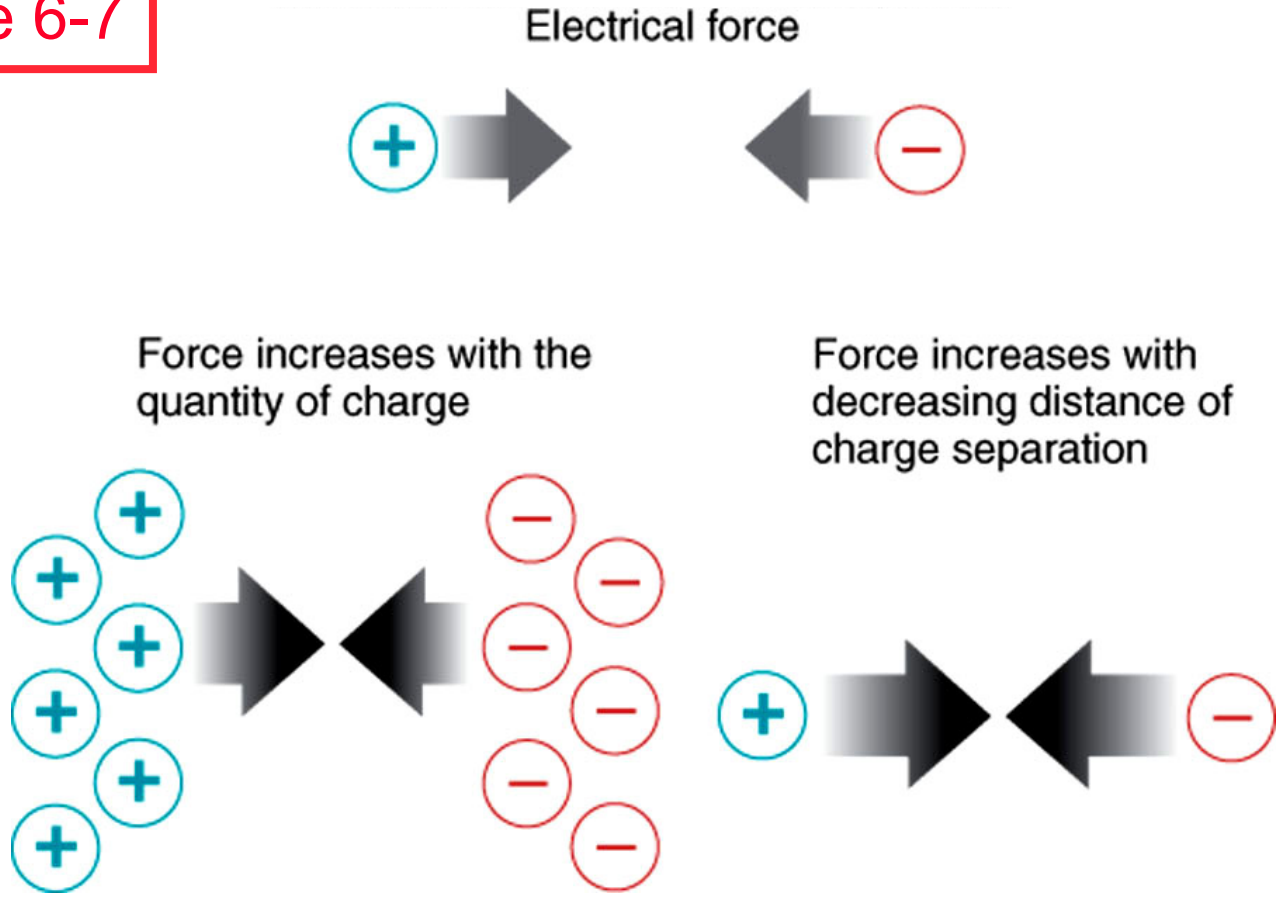

Opposite charges attract each other and will move toward each other if not separated by some barrier.

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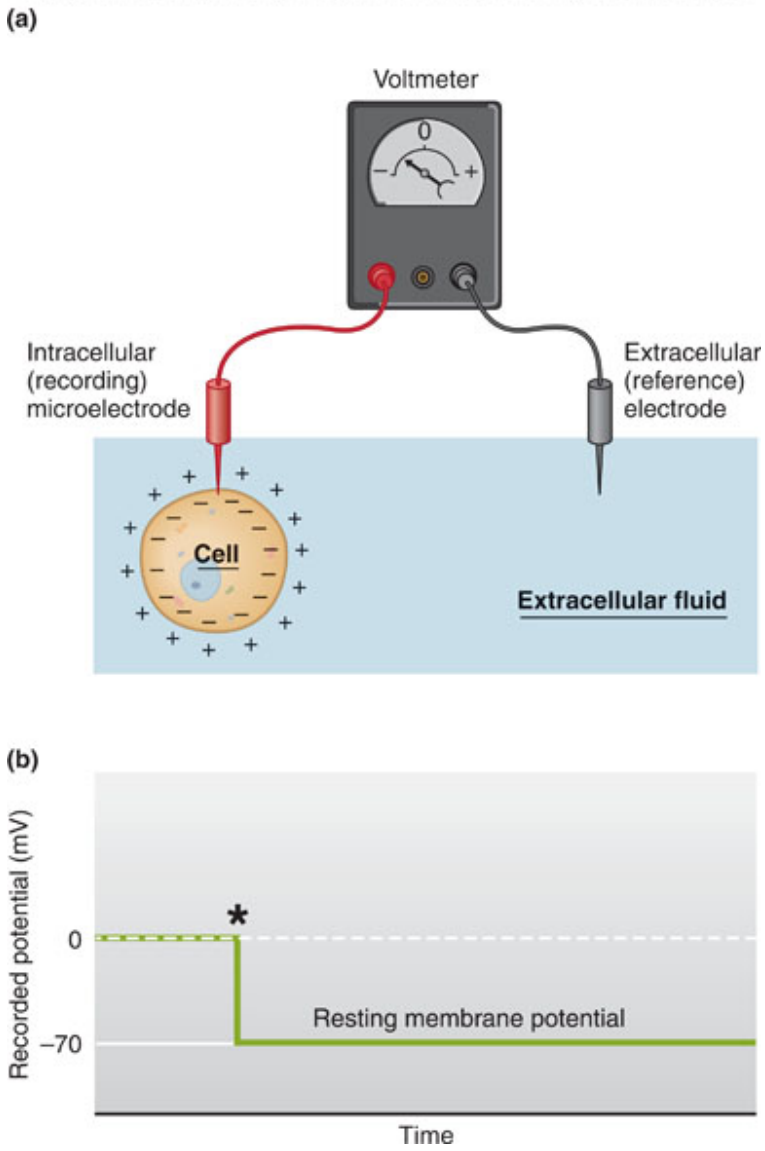

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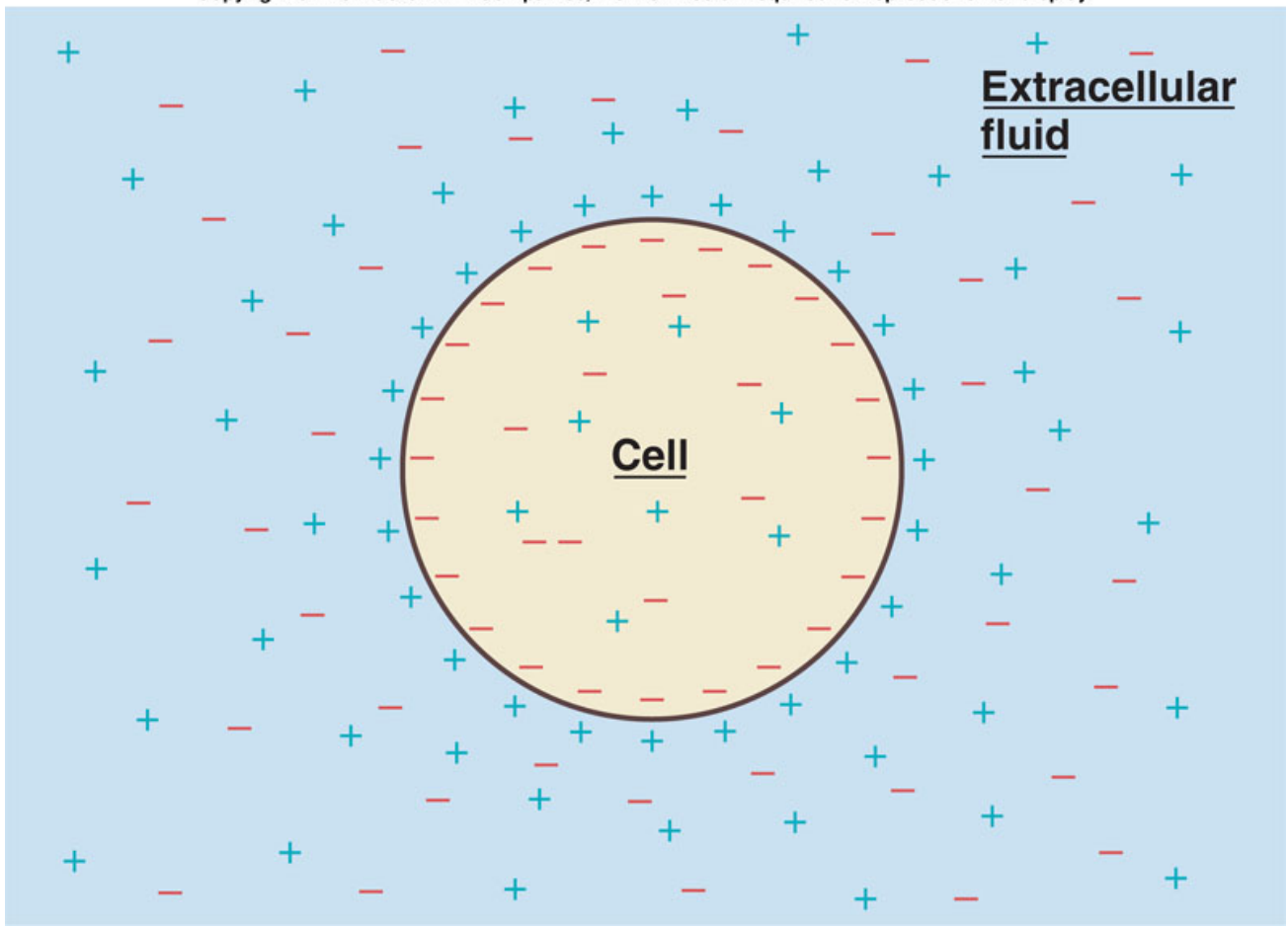

Only a very thin shell of charge difference is needed to establish a membrane potential.

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Table 6–2 Distribution of Major Mobile Ions Across the Plasma Membrane of a Typical Nerve Cell

	Concentration, mmol/L	
Ion	**Extracellular**	**Intracellular**
Na^+	145	15
Cl^-	100	7*
K^+	5	150

A more accurate measure of electrical driving force can be obtained using mEq/L, which factors in ion valence. Since all the ions in this table have a valence of 1, the mEq/L is the same as the mmol/L concentration.

*Intracellular chloride concentration varies significantly between neurons due to differences in expression of membrane transporters and channels.

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Begin:

K^+ in Compartment 2,
Na^+ in Compartment 1;
BUT only K^+ can move.

Ion movement:
K^+ crosses into Compartment 1;
Na^+ stays in Compartment 1.

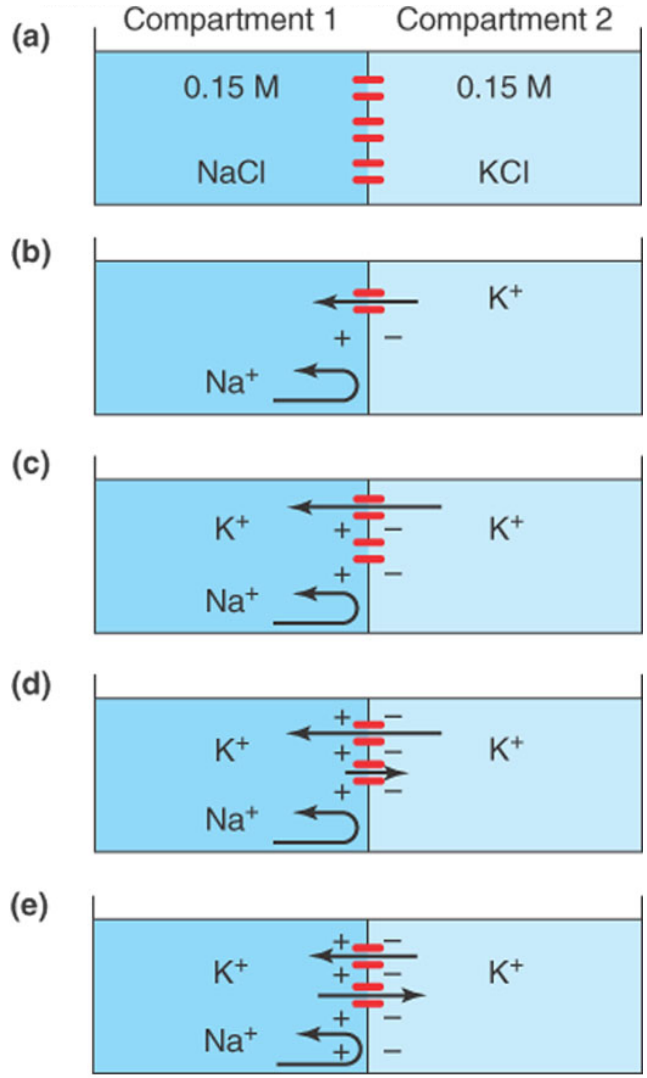

Figure 6-10

At the potassium equilibrium potential:
buildup of positive charge in Compartment 1 produces an electrical potential that exactly offsets the K^+ chemical concentration gradient.

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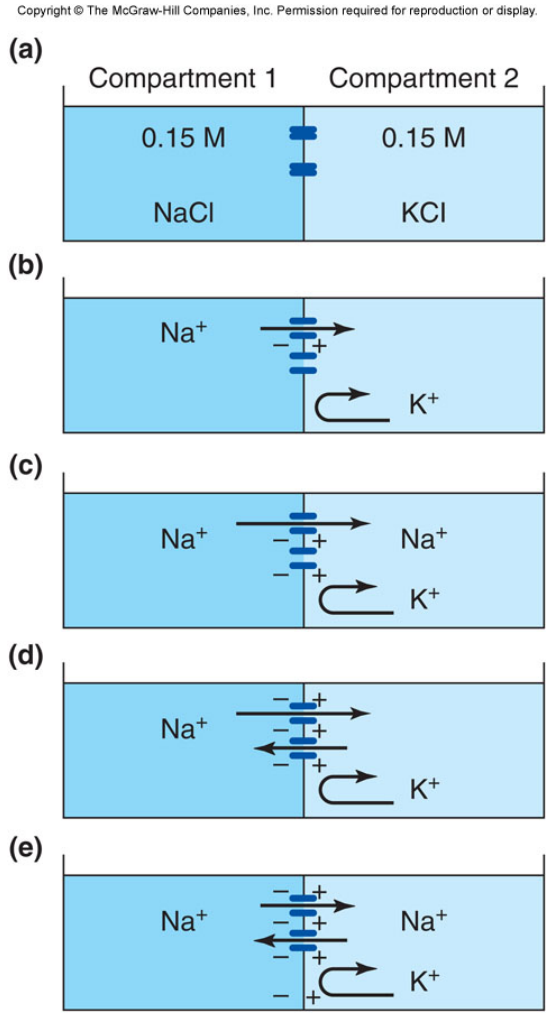
Begin:

K^+ in Compartment 2,
Na^+ in Compartment 1;
BUT only Na^+ can move.

Ion movement:
Na^+ crosses into Compartment 2;
but K^+ stays in Compartment 2.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At the sodium equilibrium potential:

buildup of positive charge in Compartment 2 produces an electrical potential that exactly offsets the Na^+ chemical concentration gradient.

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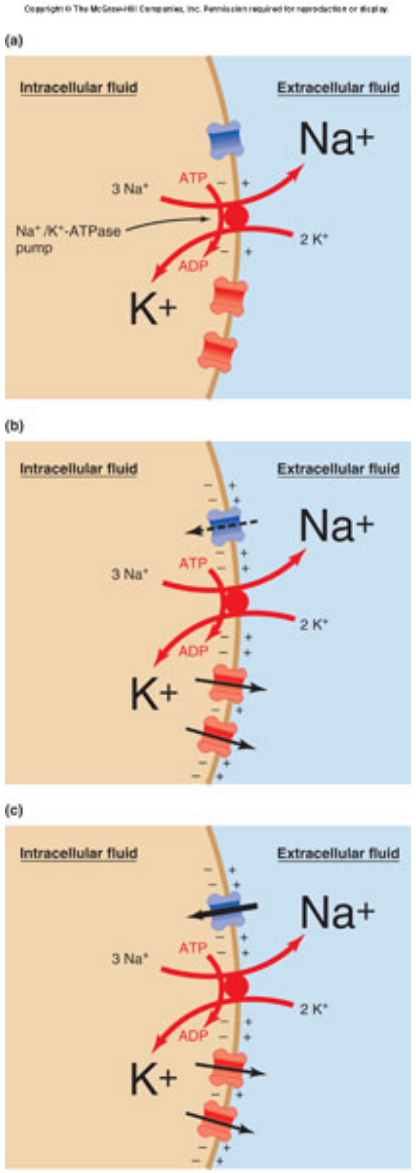
Figure 6-13



Establishment of resting membrane potential:

Na+/K+ pump establishes concentration gradient generating a small negative potential; pump uses up to 40% of the ATP produced by that cell!

Figure 6-14

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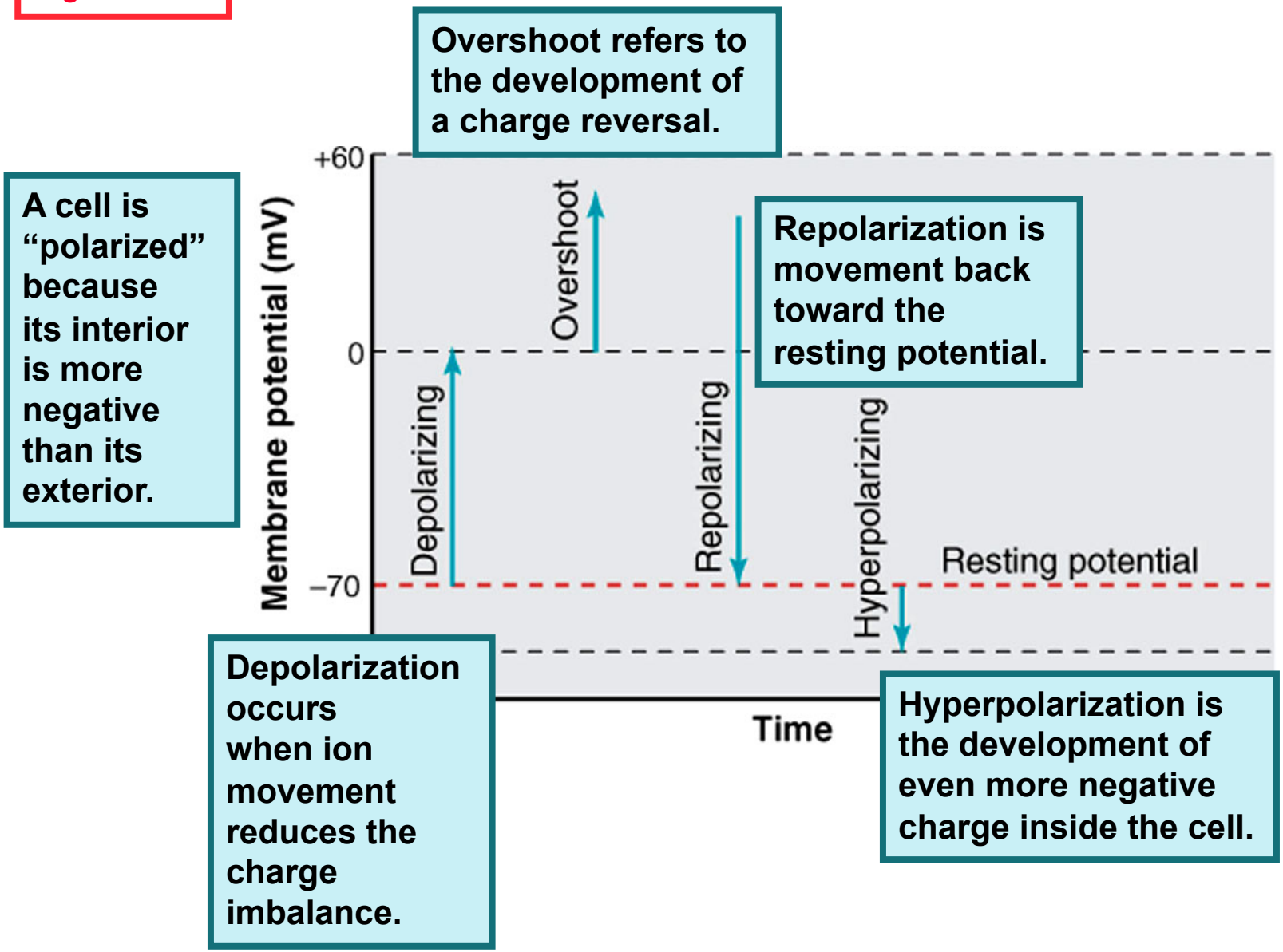

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TABLE 6–3 A Miniglossary of Terms Describing the Membrane Potential

Potential = potential difference	The voltage difference between two points.
Membrane potential = transmembrane potential	The voltage difference between the inside and outside of a cell.
Equilibrium potential	The voltage difference across a membrane that produces a flux of a given ion species that is equal but opposite to the flux due to the concentration gradient of that same ion species.
Resting membrane potential = resting potential	The steady transmembrane potential of a cell that is not producing an electric signal.
Graded potential	A potential change of variable amplitude and duration that is conducted decrementally; it has no threshold or refractory period.
Action potential	A brief all-or-none depolarization of the membrane, reversing polarity in neurons; it has a threshold and refractory period and is conducted without decrement.
Synaptic potential	A graded potential change produced in the postsynaptic neuron in response to the release of a neurotransmitter by a presynaptic terminal; it may be depolarizing (an excitatory postsynaptic potential or EPSP) or hyperpolarizing (an inhibitory postsynaptic potential or IPSP).
Receptor potential	A graded potential produced at the peripheral endings of afferent neurons (or in separate receptor cells) in response to a stimulus.
Pacemaker potential	A spontaneously occurring graded potential change that occurs in certain specialized cells.
Threshold potential	The membrane potential at which an action potential is initiated.

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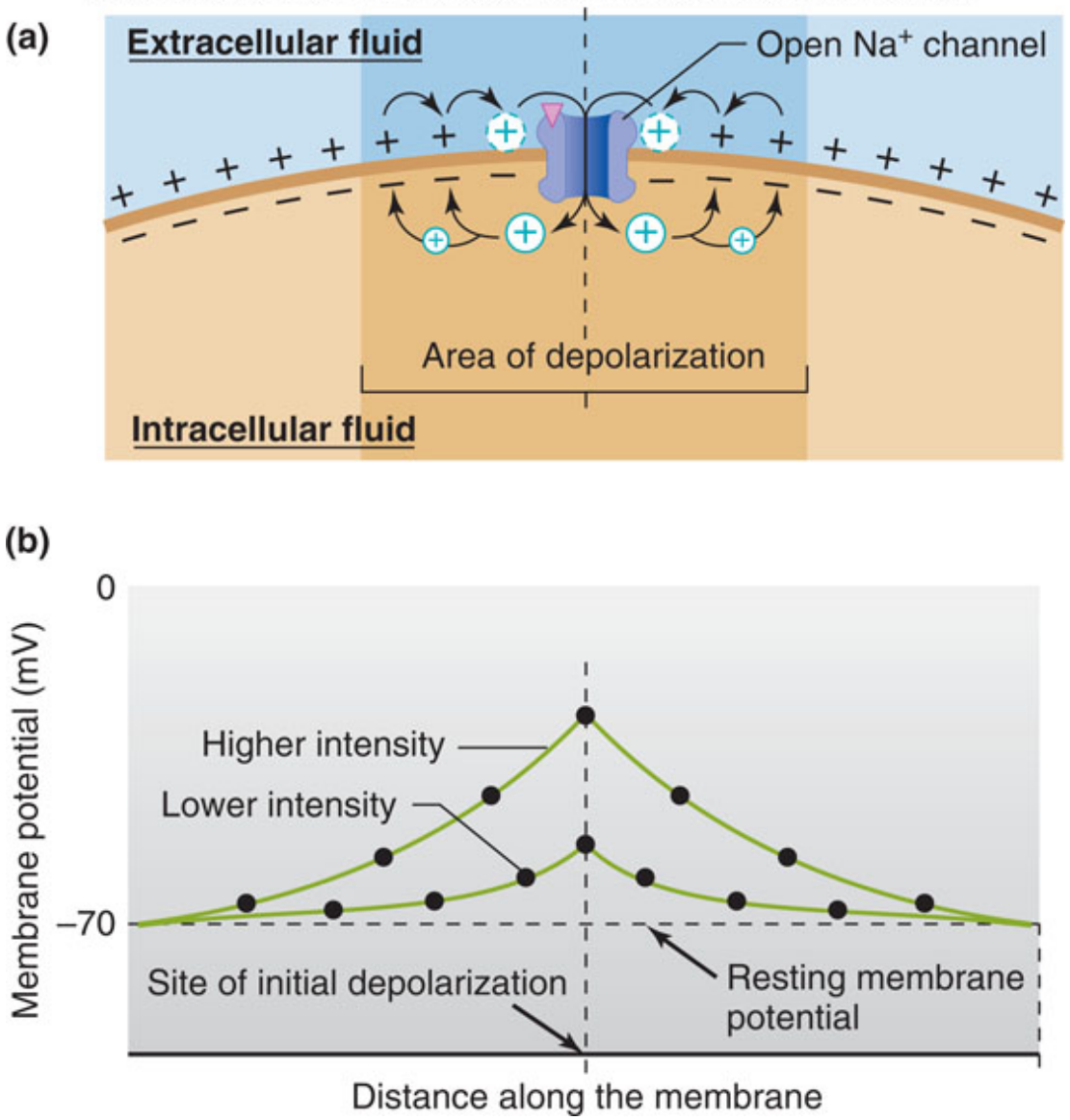

The size of a graded potential (here, graded depolarizations) is proportionate to the intensity of the stimulus.

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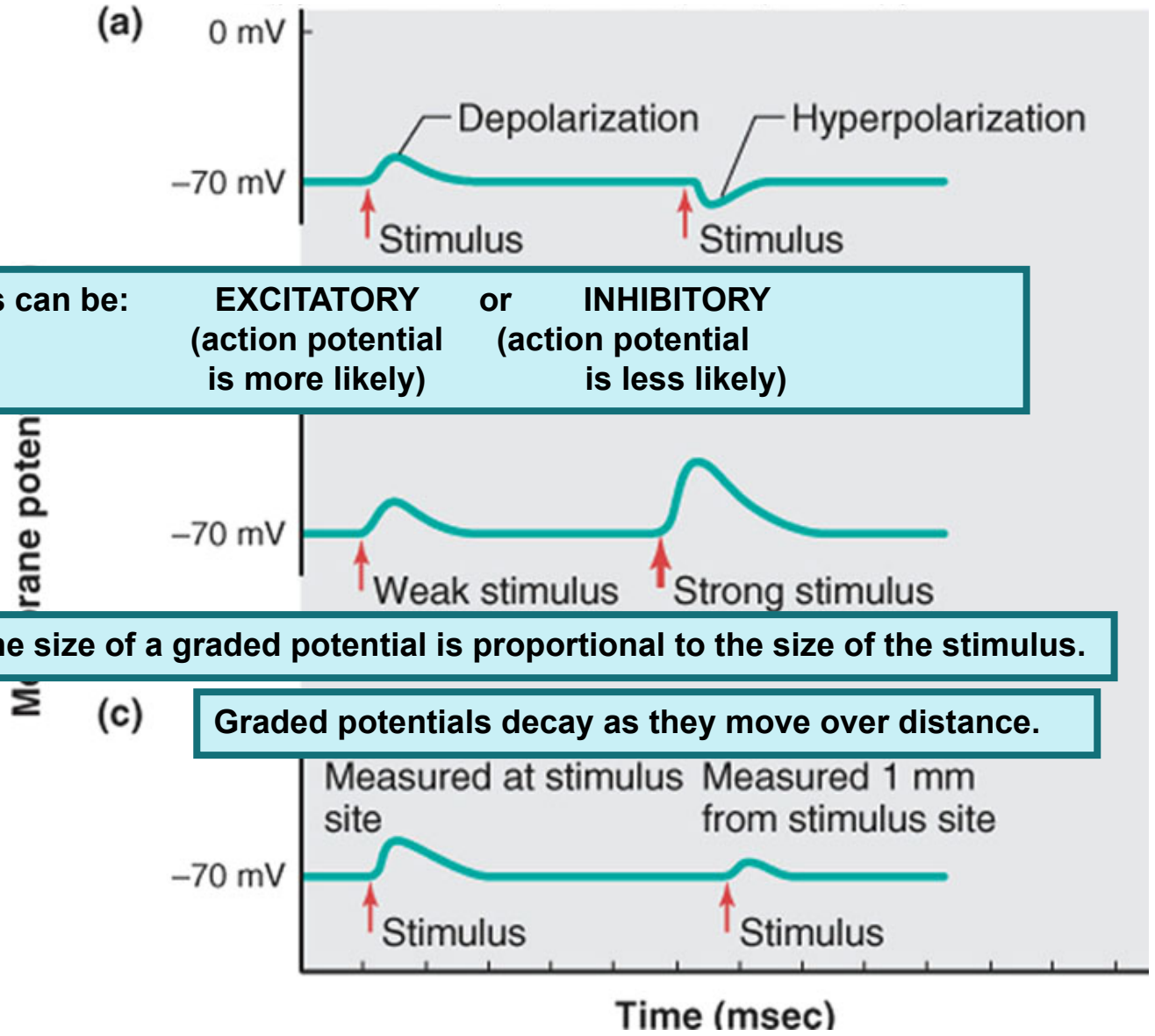

Graded potentials can be: **EXCITATORY (action potential is more likely)** **or** **INHIBITORY (action potential is less likely)**

The size of a graded potential is proportional to the size of the stimulus.

Graded potentials decay as they move over distance.

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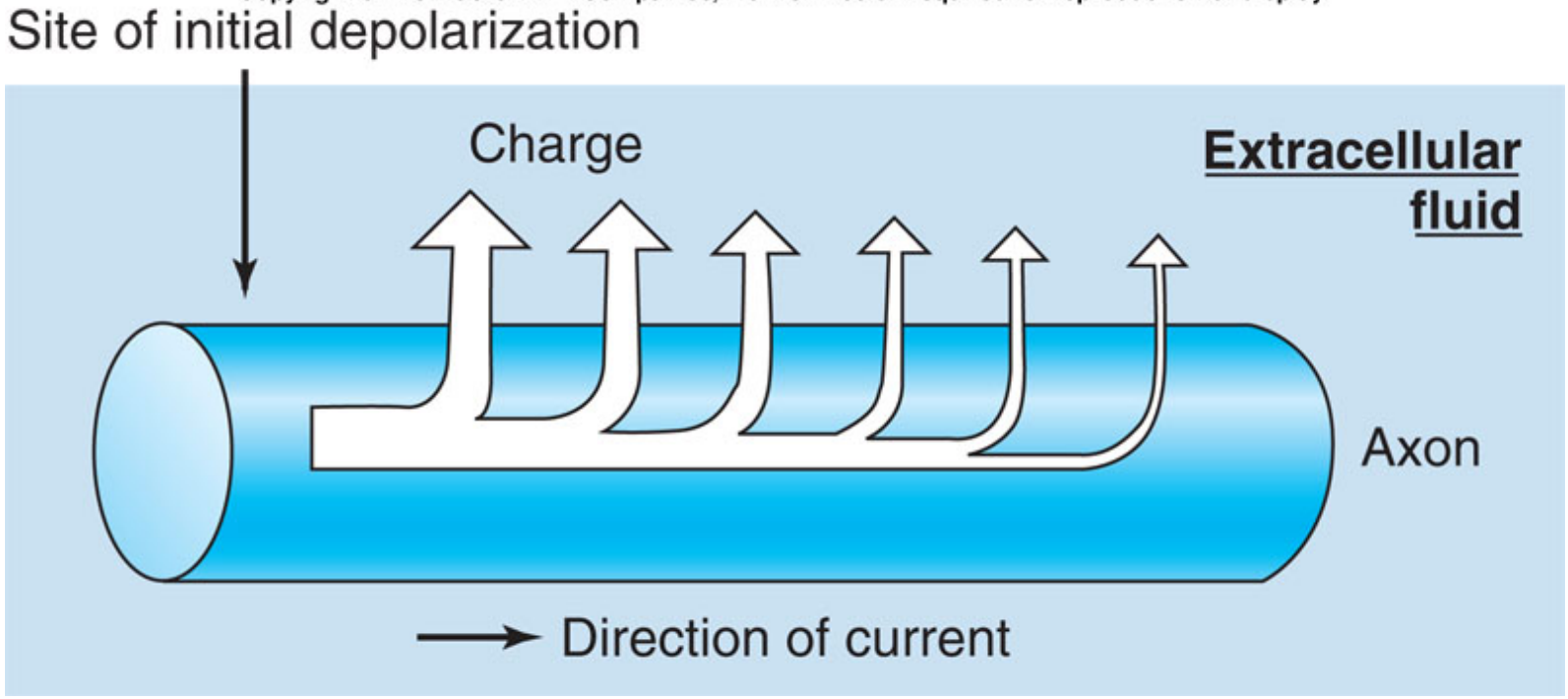

Graded potentials decay as they move over distance.

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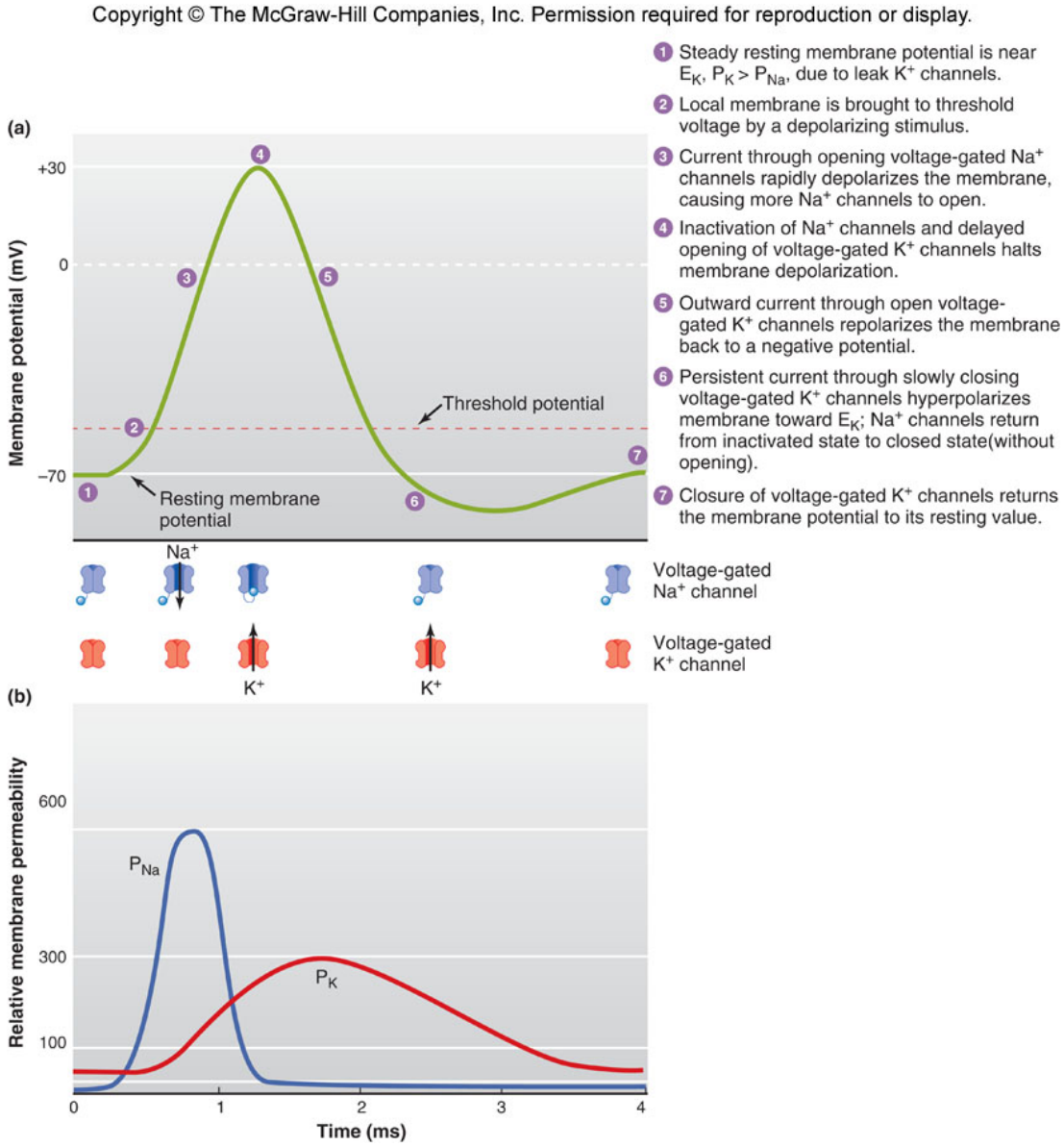
An action potential is an "all-or-none" sequence of changes in membrane potential resulting from an all-or-none sequence of changes in ion permeability due to the operation of voltage-gated Na+ and K + channels.



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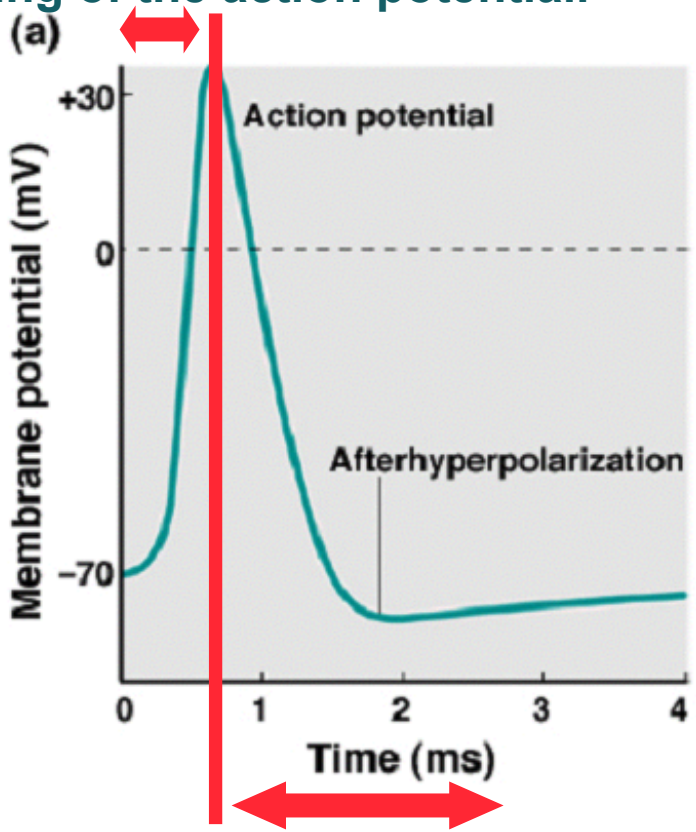
The rapid opening of voltage-gated Na^+ channels explains the rapid-depolarization phase at the beginning of the action potential.



The slower opening of voltage-gated K^+ channels explains the repolarization and after hyperpolarization phases that complete the action potential.

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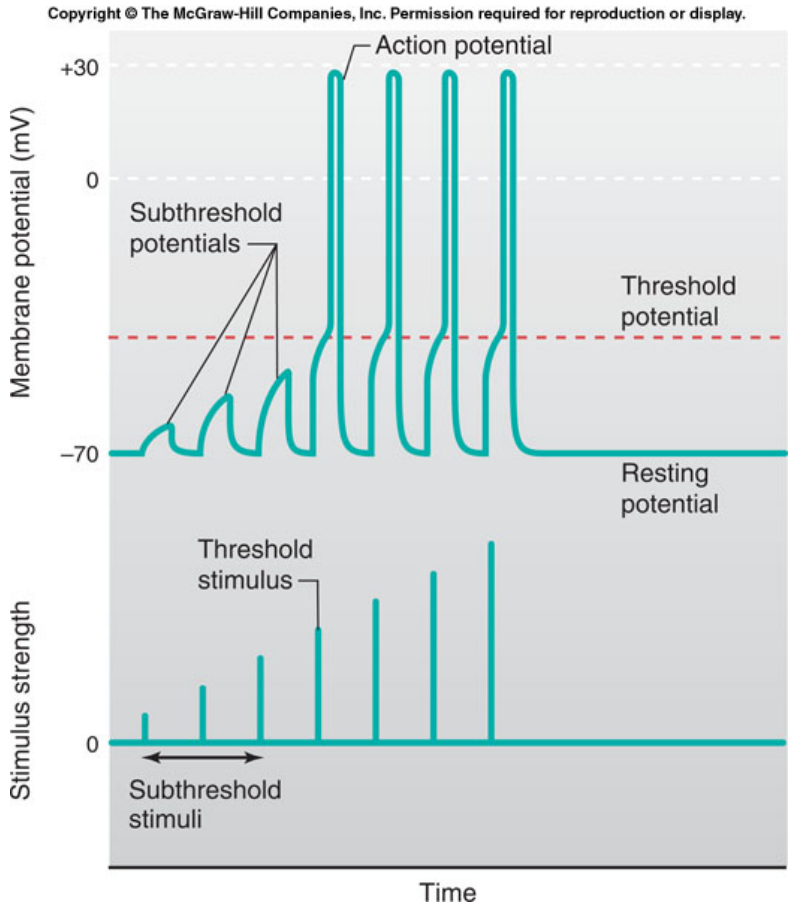

Four action potentials, each the result of a stimulus strong enough to cause deloplarization,are shown in the right half of the figure.

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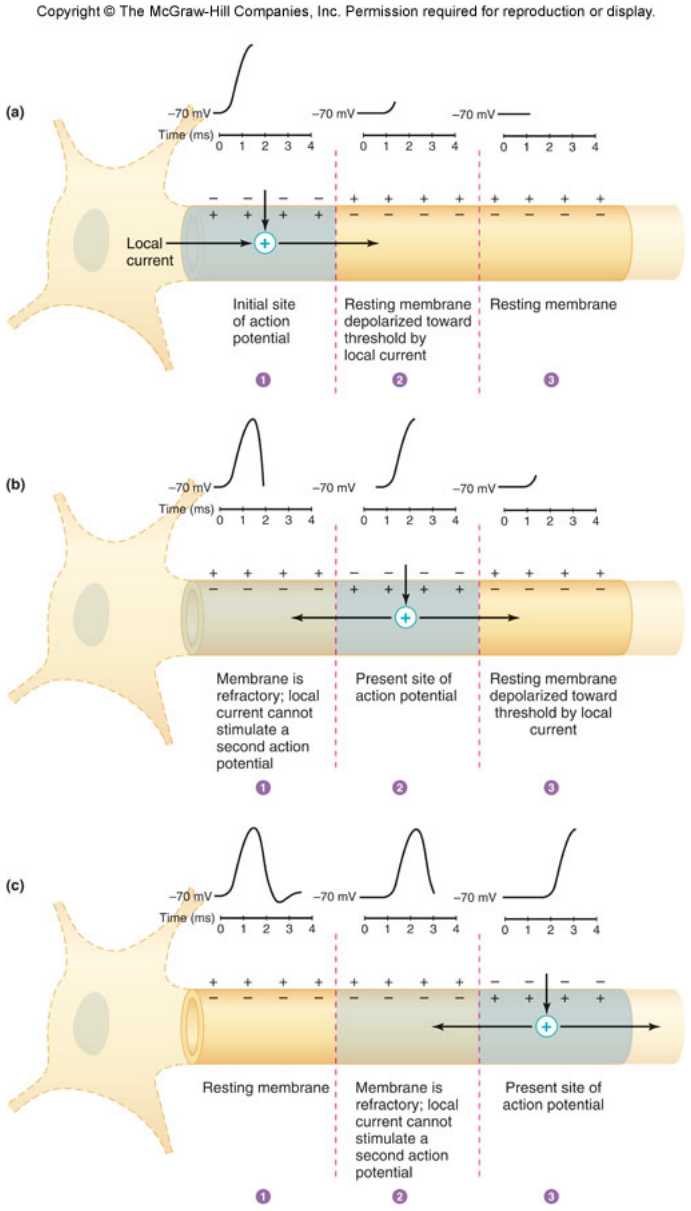
The propagation of the action potential from the dendritic to the axon-terminal end is typically one-way because the absolute refractory period follows along in the "wake" of the moving action potential.



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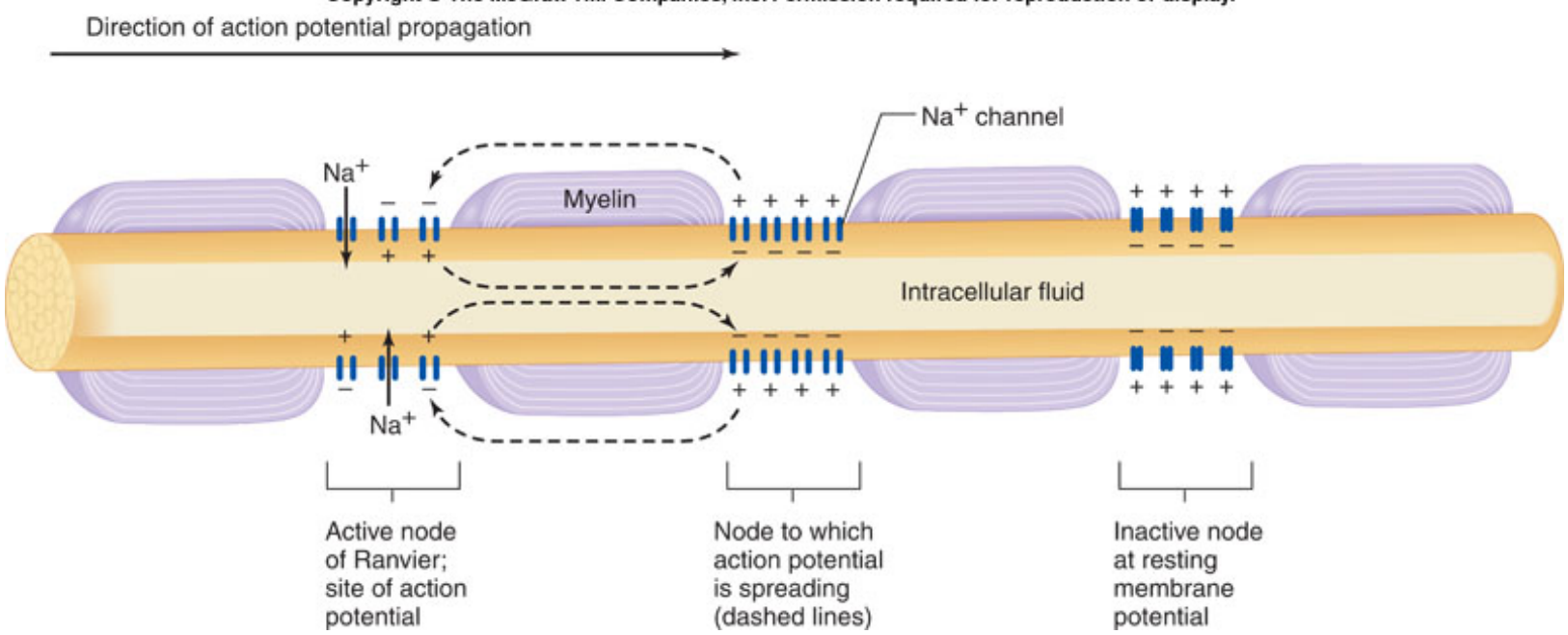

Saltatorial Conduction: Action potentials jump from one node to the next as they propagate along a myelinated axon.

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Table 6–4 Differences between Graded Potentials and Action Potentials

Graded Potential	Action Potential
Amplitude varies with size of the initiating event.	All-or-none. Once membrane is depolarized to threshold, amplitude is independent of the size of the initiating event.
Can be summed.	Cannot be summed.
Has no threshold.	Has a threshold that is usually about 15 mV depolarized relative to the resting potential.
Has no refractory period.	Has a refractory period.
Is conducted decrementally; that is, amplitude decreases with distance.	Is conducted without decrement; the depolarization is amplified to a constant value at each point along the membrane.
Duration varies with initiating conditions.	Duration is constant for a given cell type under constant conditions.
Can be a depolarization or a hyperpolarization.	Is only a depolarization.
Initiated by environmental stimulus (receptor), by neurotransmitter (synapse), or spontaneously.	Initiated by a graded potential.
Mechanism depends on ligand-gated channels or other chemical or physical changes.	Mechanism depends on voltage-gated channels.

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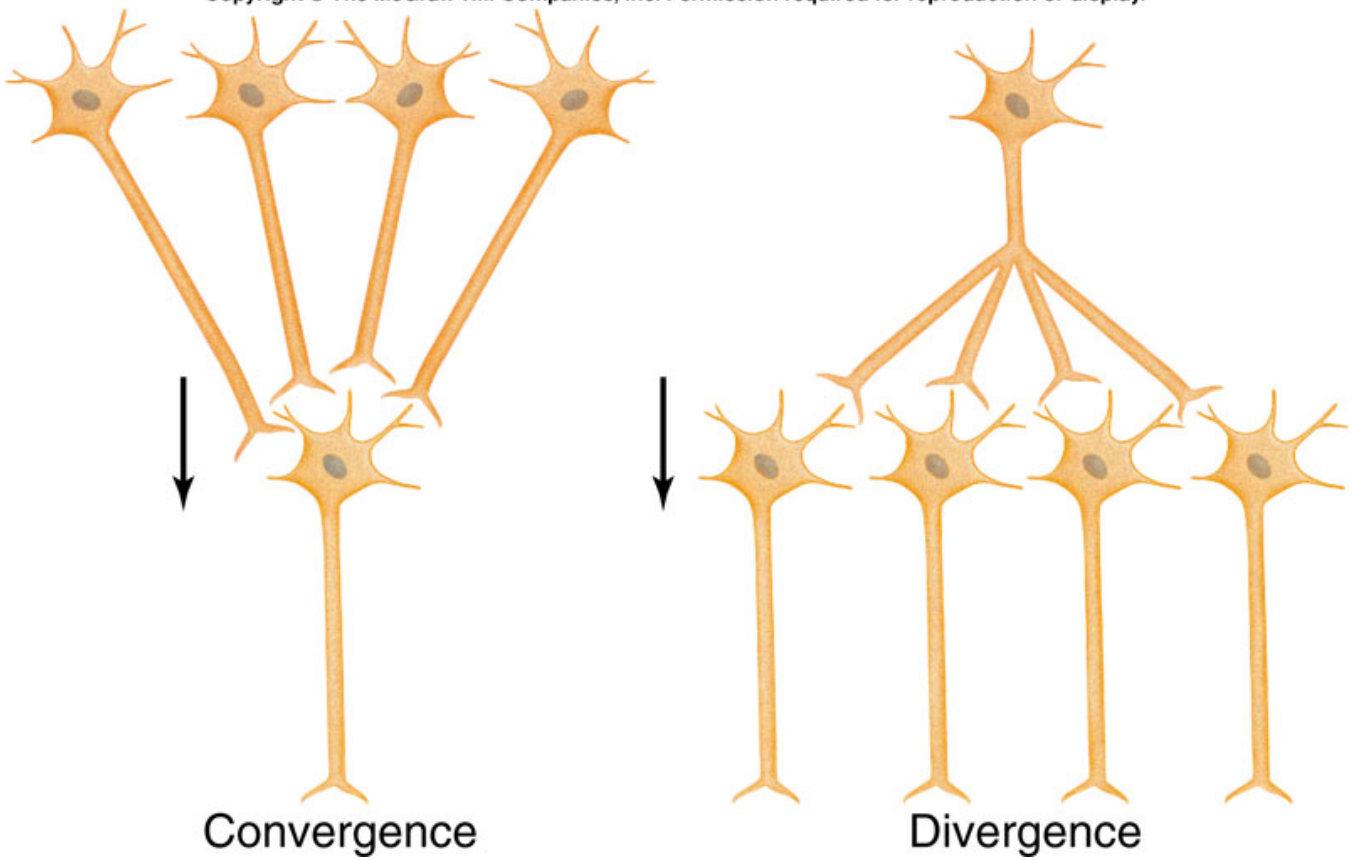

Four primary neurons communicate to one secondary neuron.

One primary neuron communicates to four secondary neurons.

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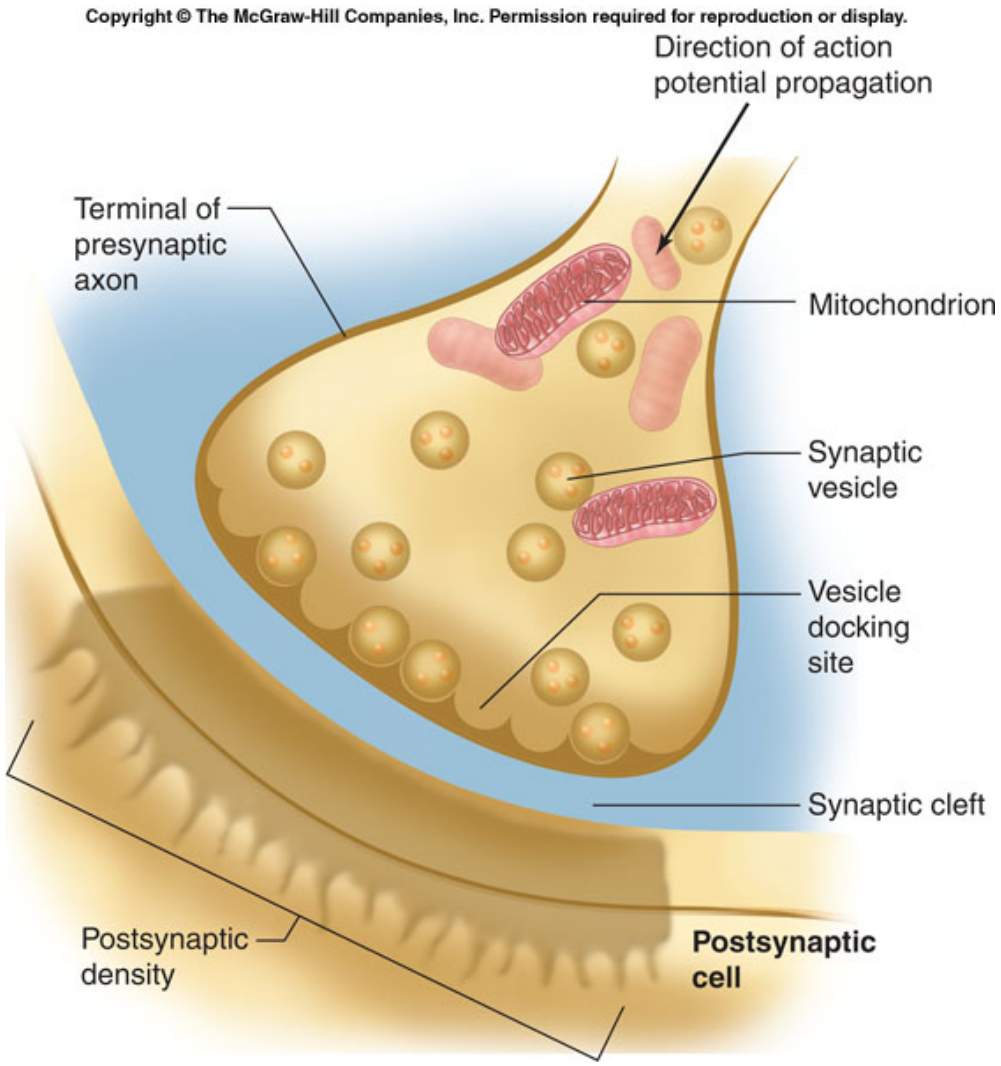
The synapse is the point of communication between two neurons that operate sequentially.



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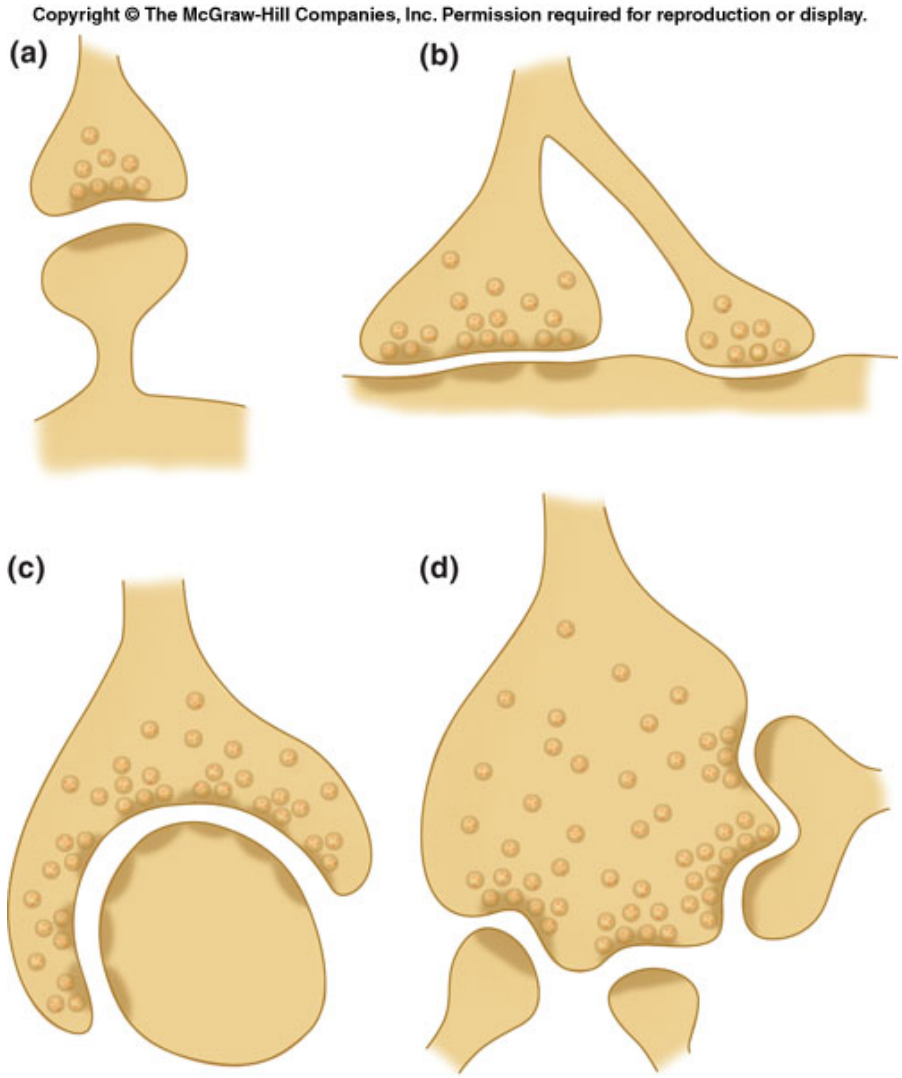
Diversity in synaptic form allows the nervous system to achieve diversity and flexibility.



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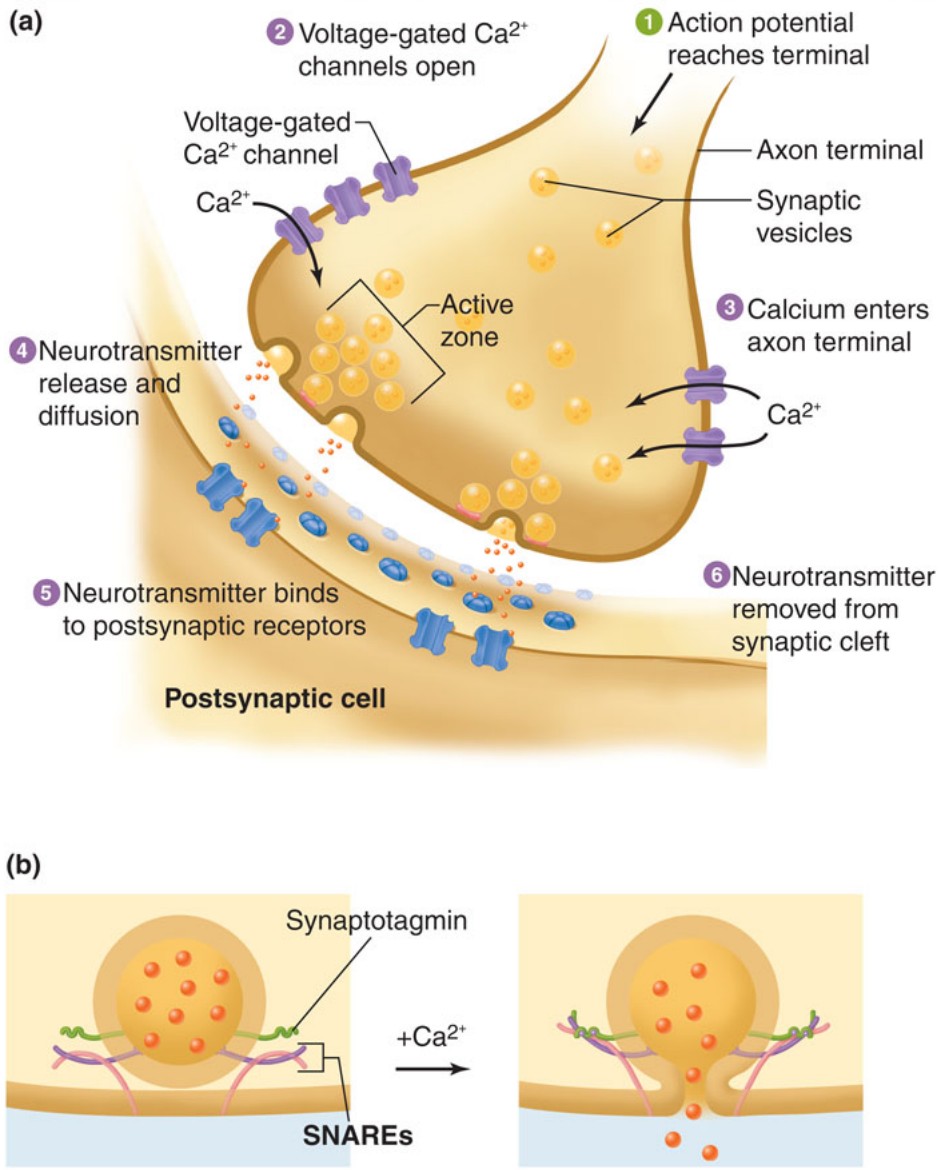

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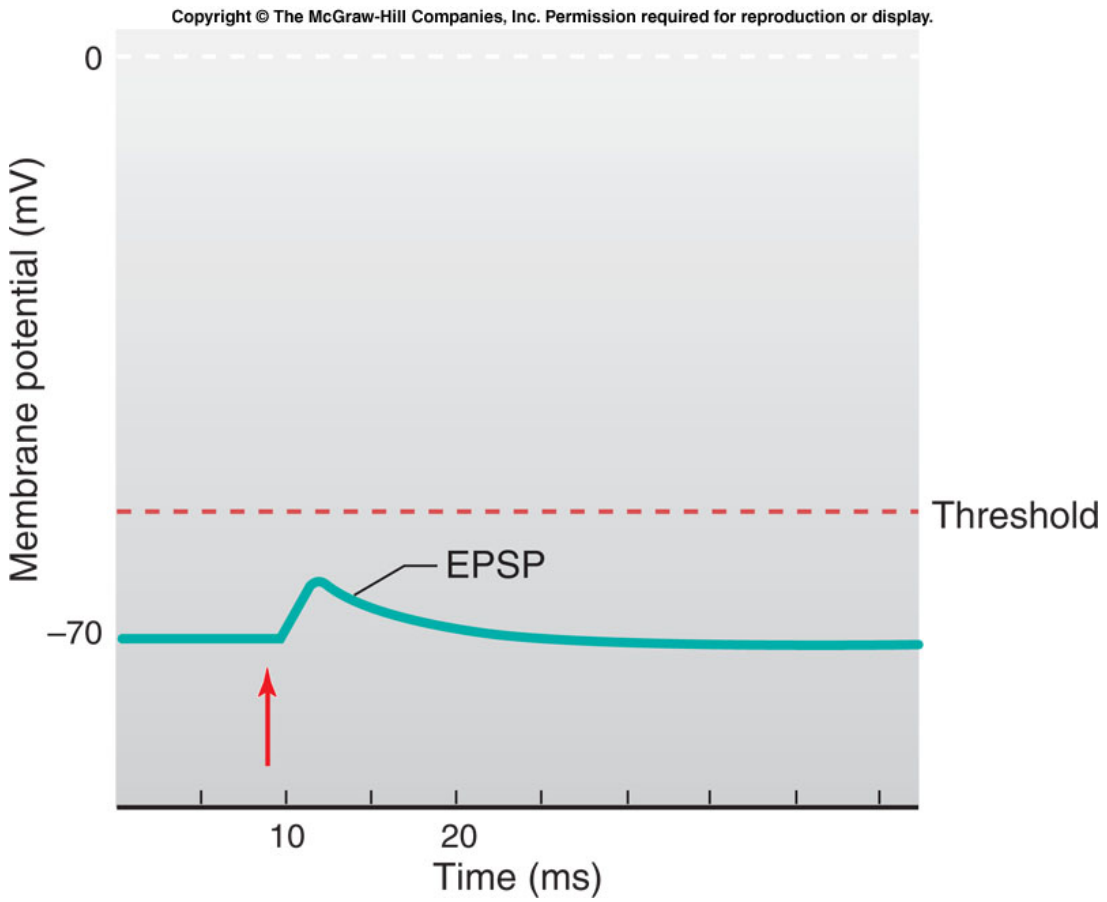
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An excitatory postsynaptic potential (EPSP) is a graded depolarization that moves the membrane potential closer to the threshold for firing an action potential (excitement).

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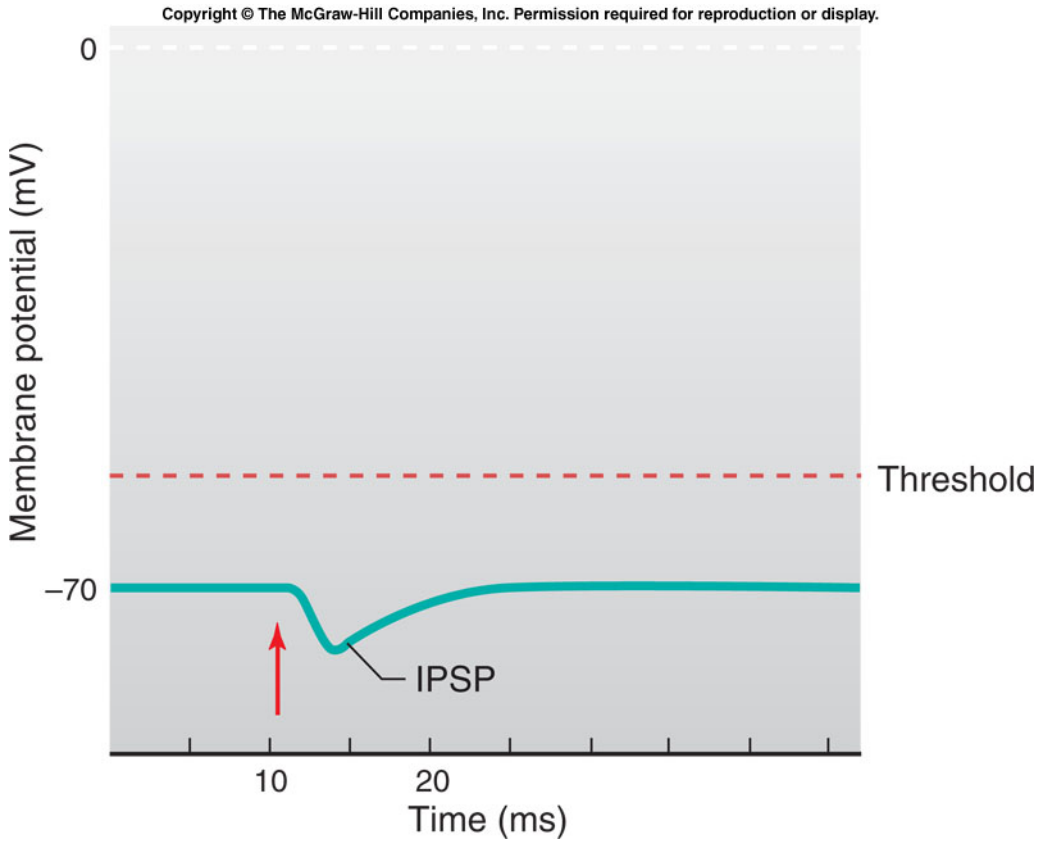

An inhibitory postsynaptic potential (IPSP) is a graded hyperpolarization that moves the membrane potential further from the threshold for firing an action potential (inhibition).

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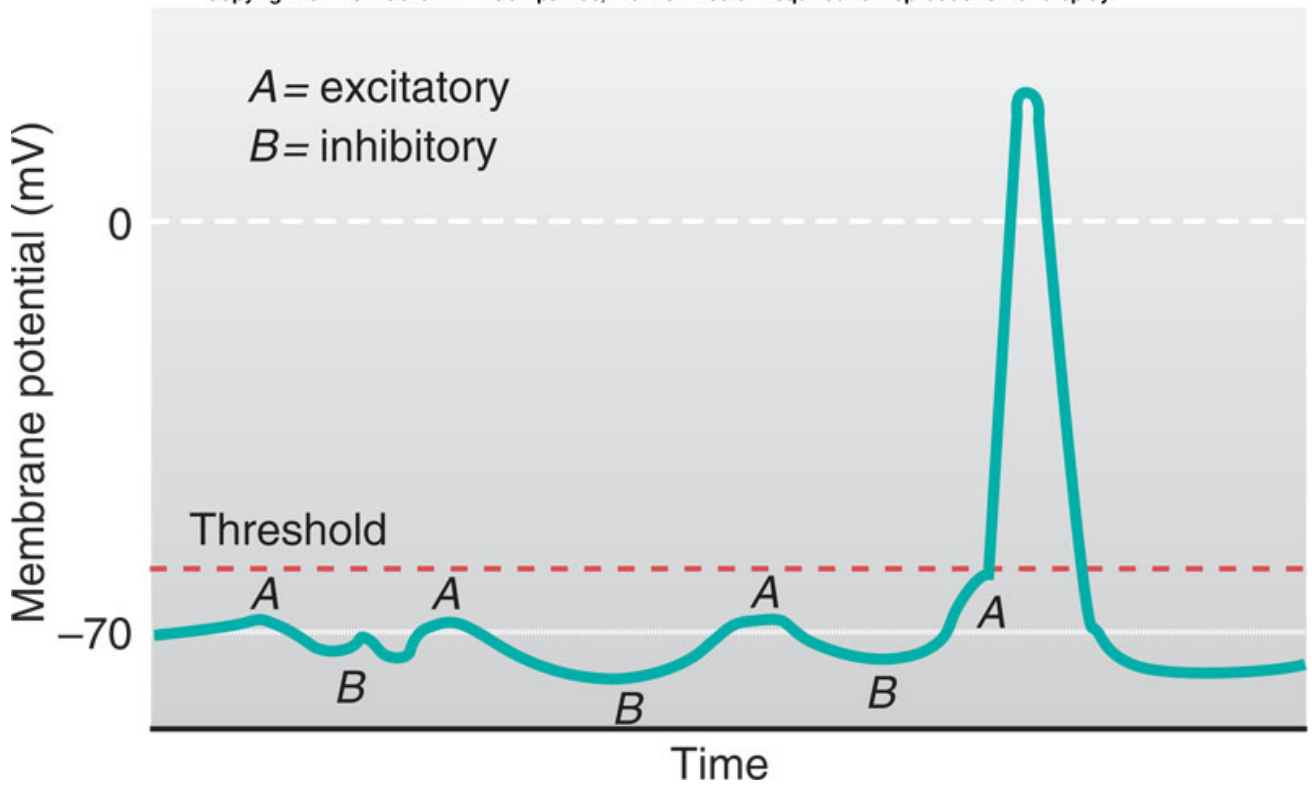

The membrane potential of a real neuron typically undergoes many EPSPs (A) and IPSPs (B), since it constantly receives excitatory and inhibitory input from the axons terminals that reach it.

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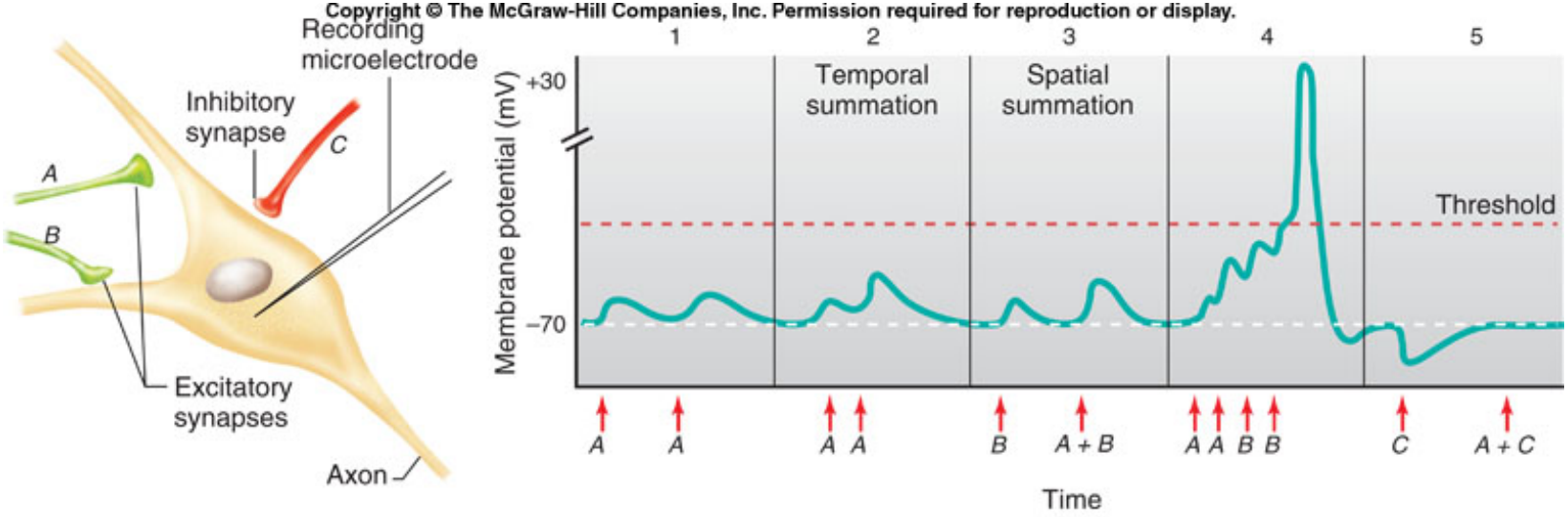

Panel 1: Two distinct, non-overlapping, graded depolarizations.
Panel 2: Two overlapping graded depolarizations demonstrate temporal summation.
Panel 3: Distinct actions of stimulating neurons A and B demonstrate spatial summation.
Panel 4: A and B are stimulated enough to cause a suprathreshold graded depolarization, so an action potential results.
Panel 5: Neuron C causes a graded hyperpolarization; A and C effects add, cancel each other out.

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Real neurons receive as many as 200,000 terminals.

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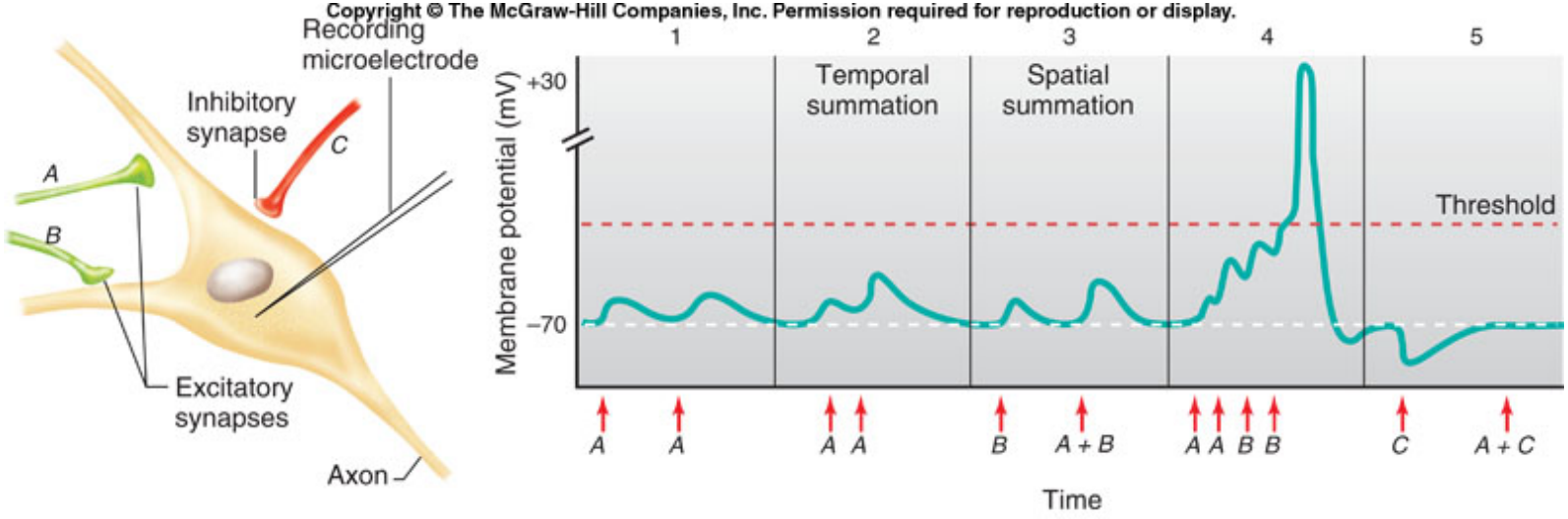

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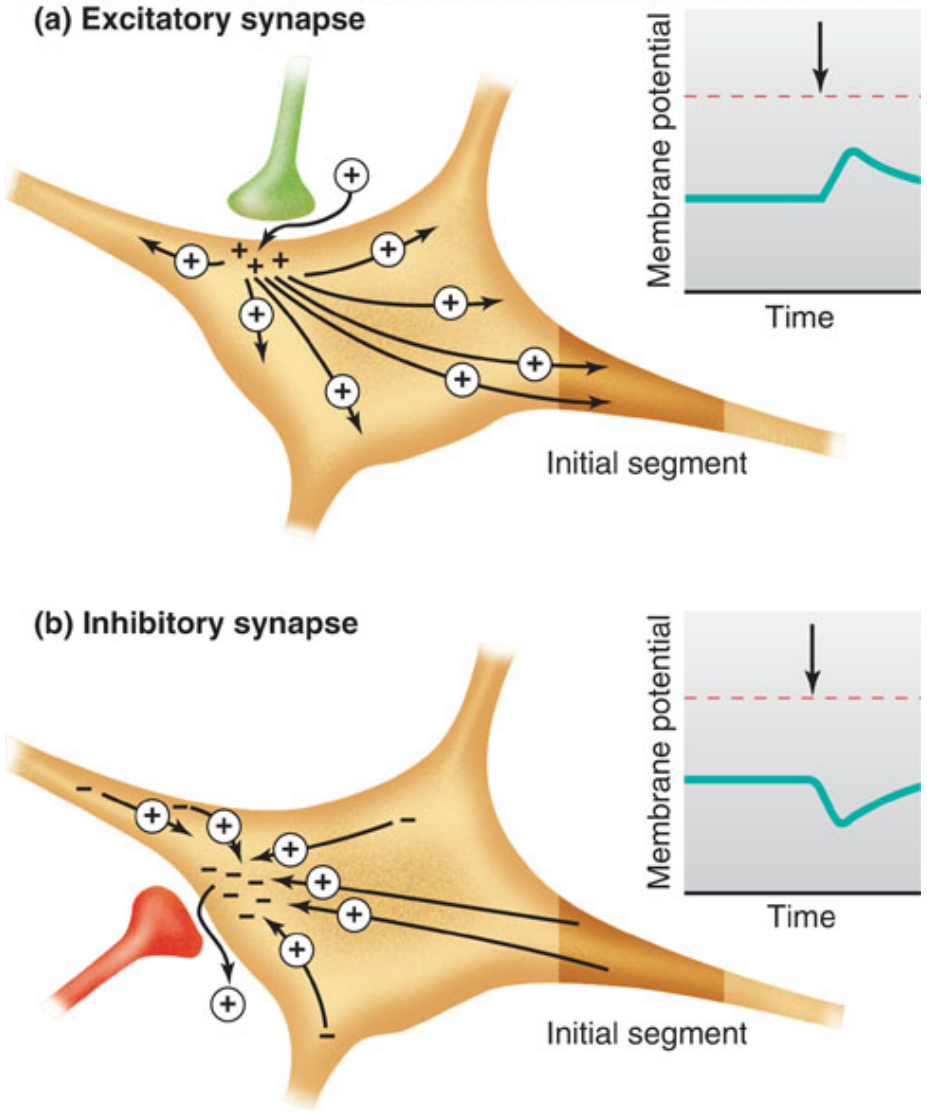

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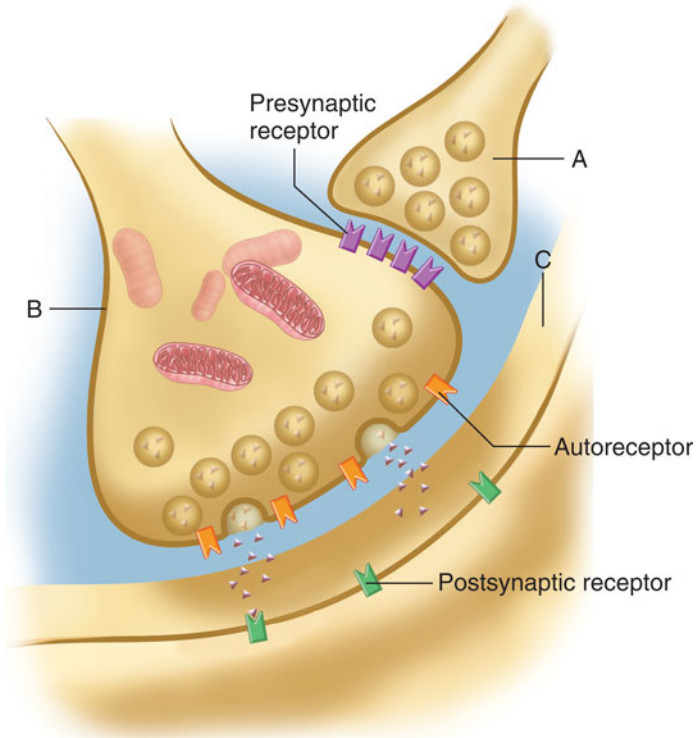

Axo-axonal communication (here, between A & B) can modify classical synaptic communication (here, between B & C); this can result in presynaptic inhibition or presynaptic facilitation.

Figure 6-33

Note: the Terminal B must have receptors for the signal released from A.

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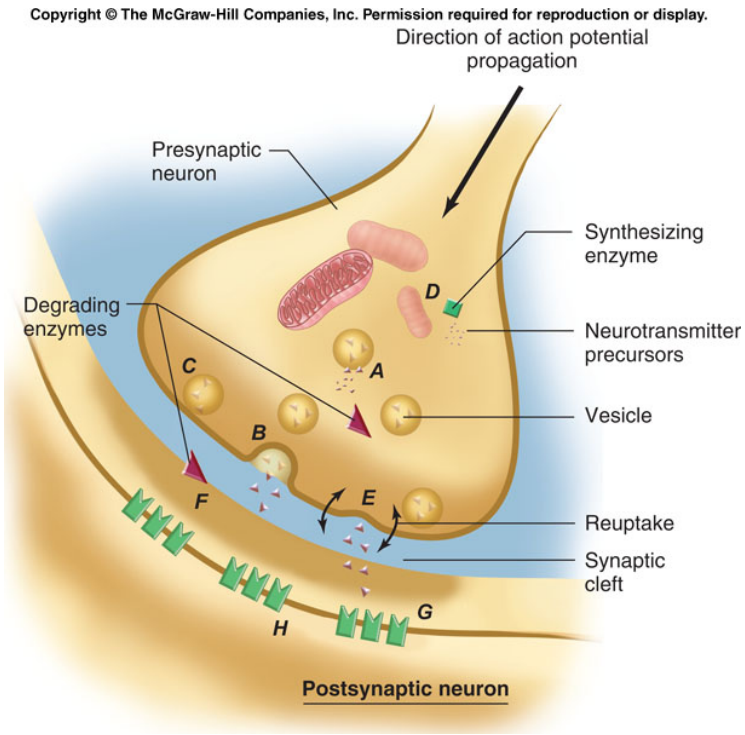
Possible drug effects on synaptic effectiveness:



A. release and degradation of the neurotransmitter *inside* the axon terminal.

B. increased neurotransmitter release into the synapse.

C. prevention of neurotransmitter release into the synapse.

D. inhibition of synthesis of the neurotransmitter.

E. reduced reuptake of the neurotransmitter from the synapse.

F. reduced degradation of the neurotransmitter in the synapse.

G. agonists (evoke same response as neurotransmitter) or antagonists (block response to neurotransmitter) can occupy the receptors.

H. reduced biochemical response inside the dendrite.

Figure 6-34

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Table 6–5 Factors that Determine Synaptic Strength

I. PRESYNAPTIC FACTORS
- A. *Availability of neurotransmitter*
  1. Availability of precursor molecules
  2. Amount (or activity) of the rate-limiting enzyme in the pathway for neurotransmitter synthesis
- B. *Axon terminal membrane potential*
- C. *Axon terminal calcium*
- D. *Activation of membrane receptors on presynaptic terminal*
  1. Axo-axonic synapses
  2. Autoreceptors
  3. Other receptors
- E. *Certain drugs and diseases, which act via the above mechanisms A–D*

II. POSTSYNAPTIC FACTORS
- A. *Immediate past history of electrical state of postsynaptic membrane (e.g., excitation or inhibition from temporal or spatial summation)*
- B. *Effects of other neurotransmitters or neuromodulators acting on postsynaptic neuron*
- C. *Up- or down-regulation and desensitization of receptors*
- D. *Certain drugs and diseases*

III. GENERAL FACTORS
- A. *Area of synaptic contact*
- B. *Enzymatic destruction of neurotransmitter*
- C. *Geometry of diffusion path*
- D. *Neurotransmitter reuptake*

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Table 6–6 Classes of Some of the Chemicals Known or Presumed to Be Neurotransmitters or Neuromodulators

1. Acetylcholine (ACh)

2. Biogenic amines
   - Catecholamines
     - Dopamine (DA)
     - Norepinephrine (NE)
     - Epinephrine (Epi)
   - Serotonin (5-hydroxytryptamine, 5-HT)
   - Histamine

3. Amino acids
   - Excitatory amino acids; for example, glutamate
   - Inhibitory amino acids; for example, gamma-aminobutyric acid (GABA) and glycine

4. Neuropeptides
   - For example, endogenous opioids, oxytocin, tachykinins

5. Miscellaneous
   - Gases; for example, nitric oxide
   - Purines; for example, adenosine and ATP

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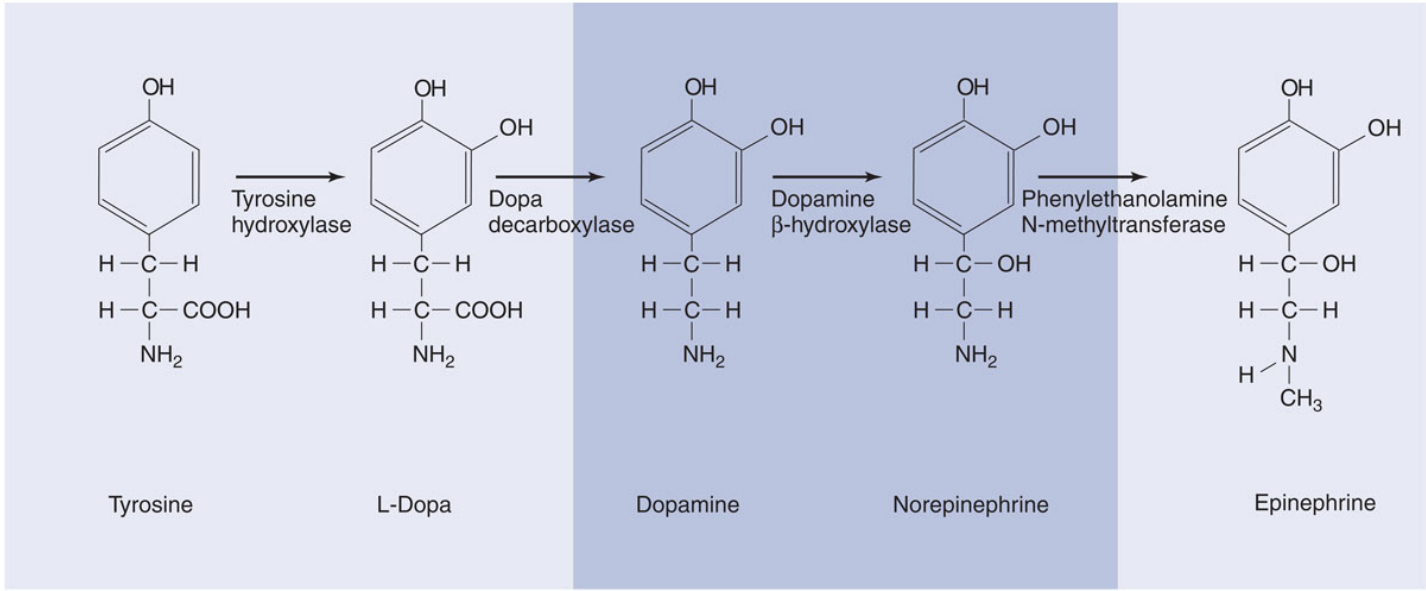

The catecholamines are formed from the amino acid tyrosine and share the same two initial steps in their biosynthetic pathway.

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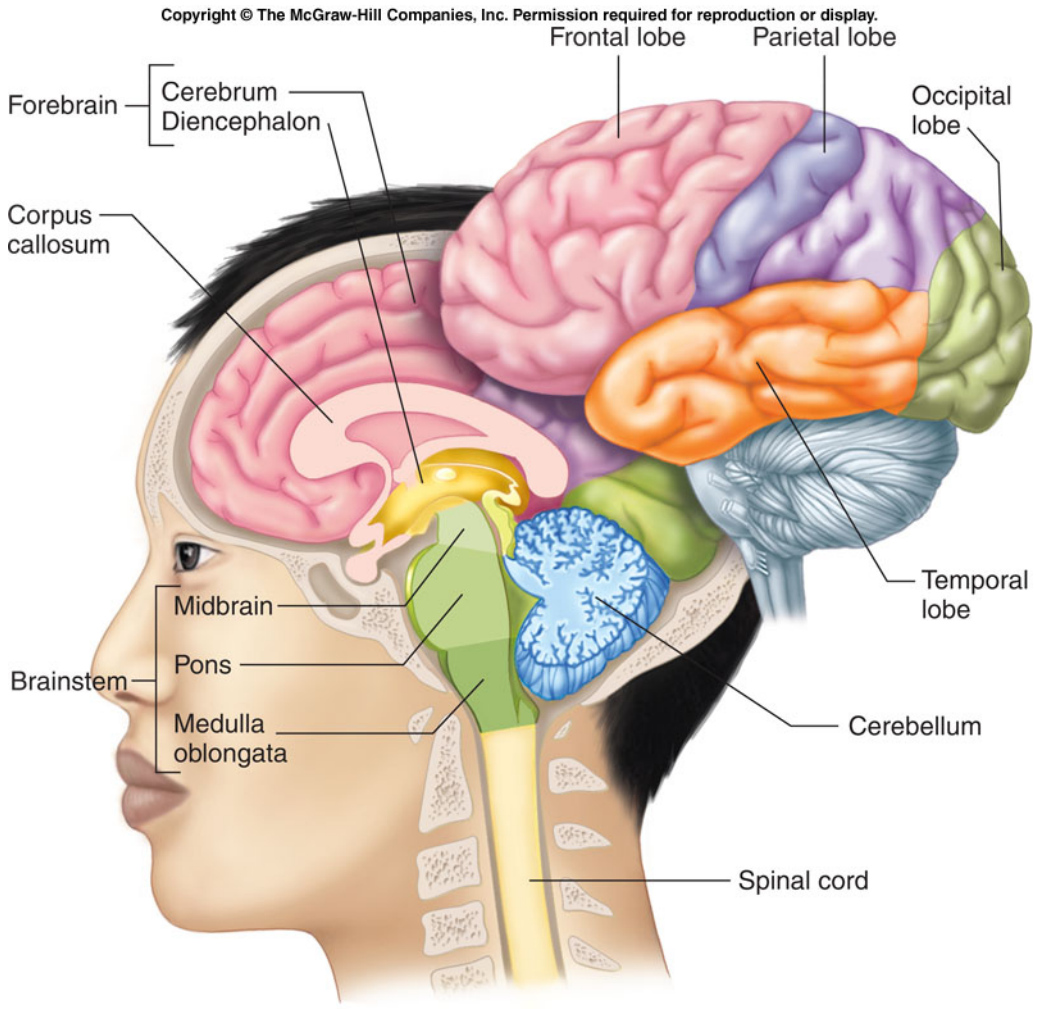

Figure 6-38 **Major landmarks of the Central Nervous System**

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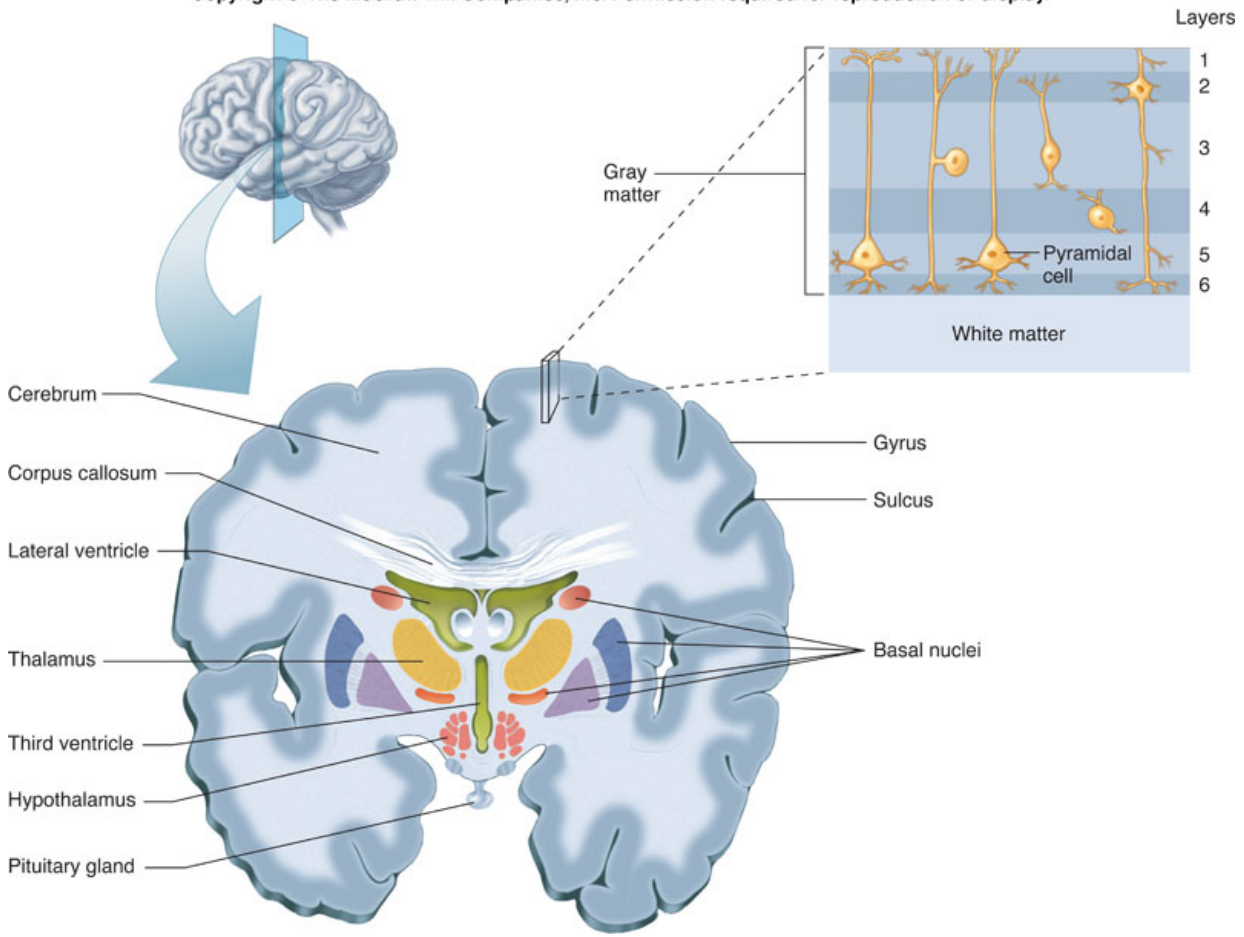

Organization of neurons in the cerebral cortex reveals six layers.

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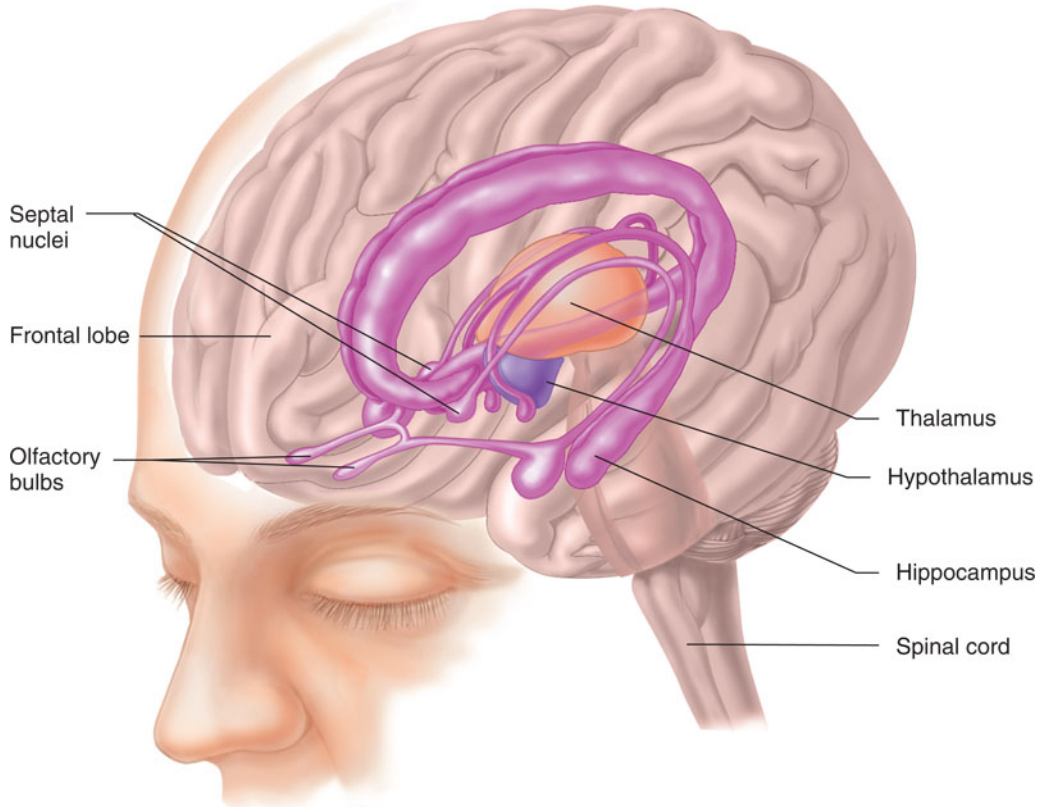

Figure 6-40

Functions of the limbic system:

- learning
- emotion
- appetite (visceral function)
- sex
- endocrine integration

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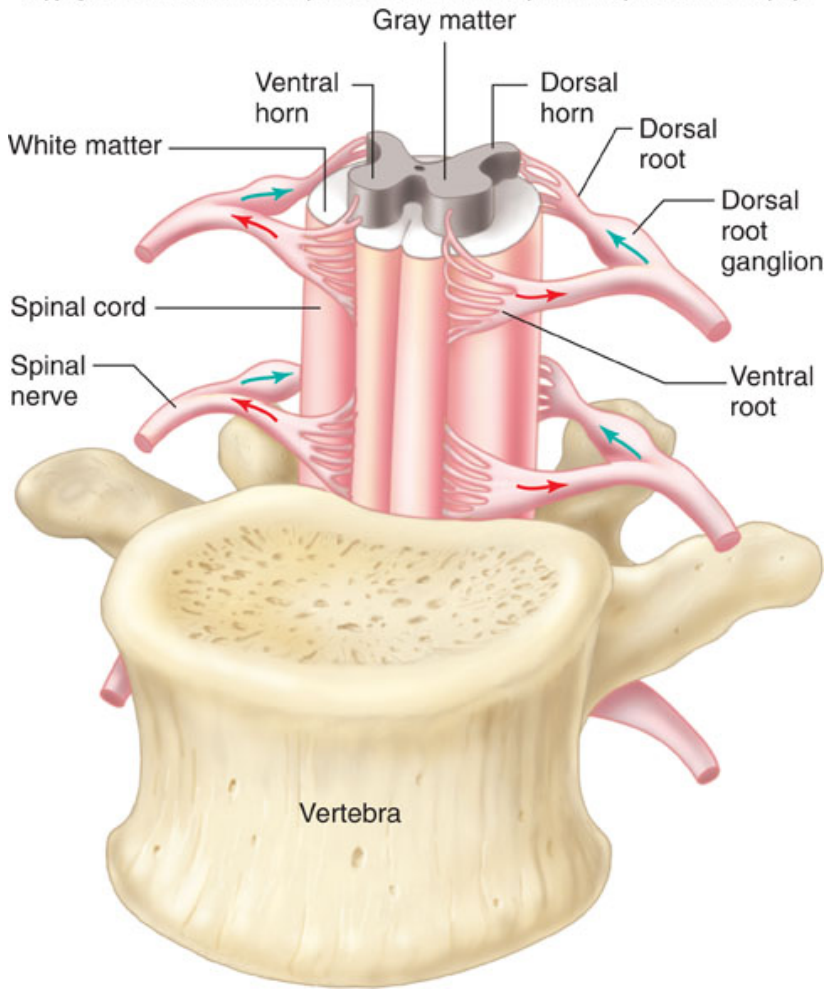

Figure 6-41

Anterior view of one vertebra and the nearby section of the spinal cord.

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Table 6–8 The Cranial Nerves

Name	Fibers	Comments
I. Olfactory	Afferent	Carries input from receptors in olfactory (smell) neuroepithelium. Not a true nerve.
II. Optic	Afferent	Carries input from receptors in eye. Not a true nerve.
III. Oculomotor	Efferent	Innervates skeletal muscles that move eyeball up, down, and medially and raise upper eyelid; innervates smooth muscles that constrict pupil and alter lens shape for near and far vision.
	Afferent	Transmits information from receptors in muscles.
IV. Trochlear	Efferent	Innervates skeletal muscles that move eyeball downward and laterally.
	Afferent	Transmits information from receptors in muscles.
V. Trigeminal	Efferent	Innervates skeletal chewing muscles.
	Afferent	Transmits information from receptors in skin; skeletal muscles of face, nose, and mouth; and teeth sockets.
VI. Abducens	Efferent	Innervates skeletal muscles that move eyeball laterally.
	Afferent	Transmits information from receptors in muscles.
VII. Facial	Efferent	Innervates skeletal muscles of facial expression and swallowing; innervates nose, palate, and lacrimal and salivary glands.
	Afferent	Transmits information from taste buds in front of tongue and mouth.
VIII. Vestibulocochlear	Afferent	Transmits information from receptors in ear.
IX. Glossopharyngeal	Efferent	Innervates skeletal muscles involved in swallowing and parotid salivary gland.
	Afferent	Transmits information from taste buds at back of tongue and receptors in auditory-tube skin.
X. Vagus	Efferent	Innervates skeletal muscles of pharynx and larynx and smooth muscle and glands of thorax and abdomen.
	Afferent	Transmits information from receptors in thorax and abdomen.
XI. Accessory	Efferent	Innervates neck skeletal muscles.
XII. Hypoglossal	Efferent	Innervates skeletal muscles of tongue.

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Table 6–9 Peripheral Nervous System: Somatic and Autonomic Divisions

Somatic
1. Consists of a single neuron between central nervous system and skeletal muscle cells
2. Innervates skeletal muscle
3. Can lead only to muscle excitation
Autonomic
1. Has two-neuron chain (connected by a synapse) between central nervous system and effector organ
2. Innervates smooth and cardiac muscle, glands, and GI neurons
3. Can be either excitatory or inhibitory

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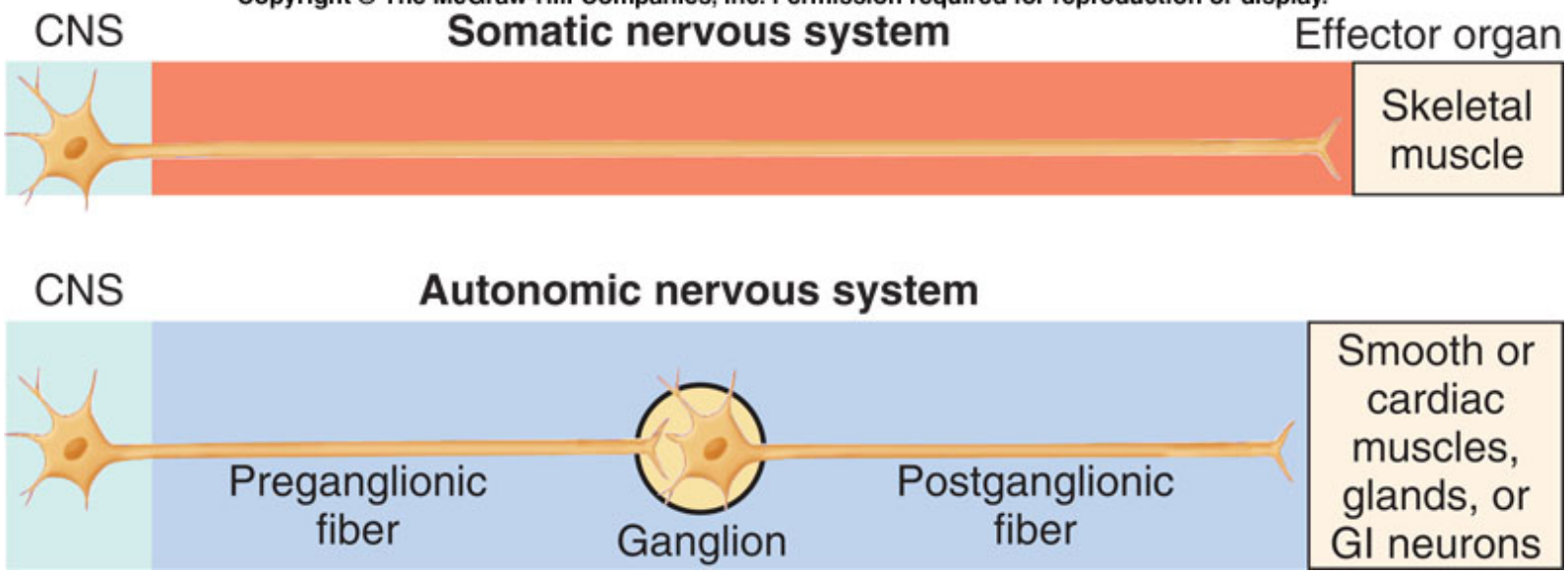

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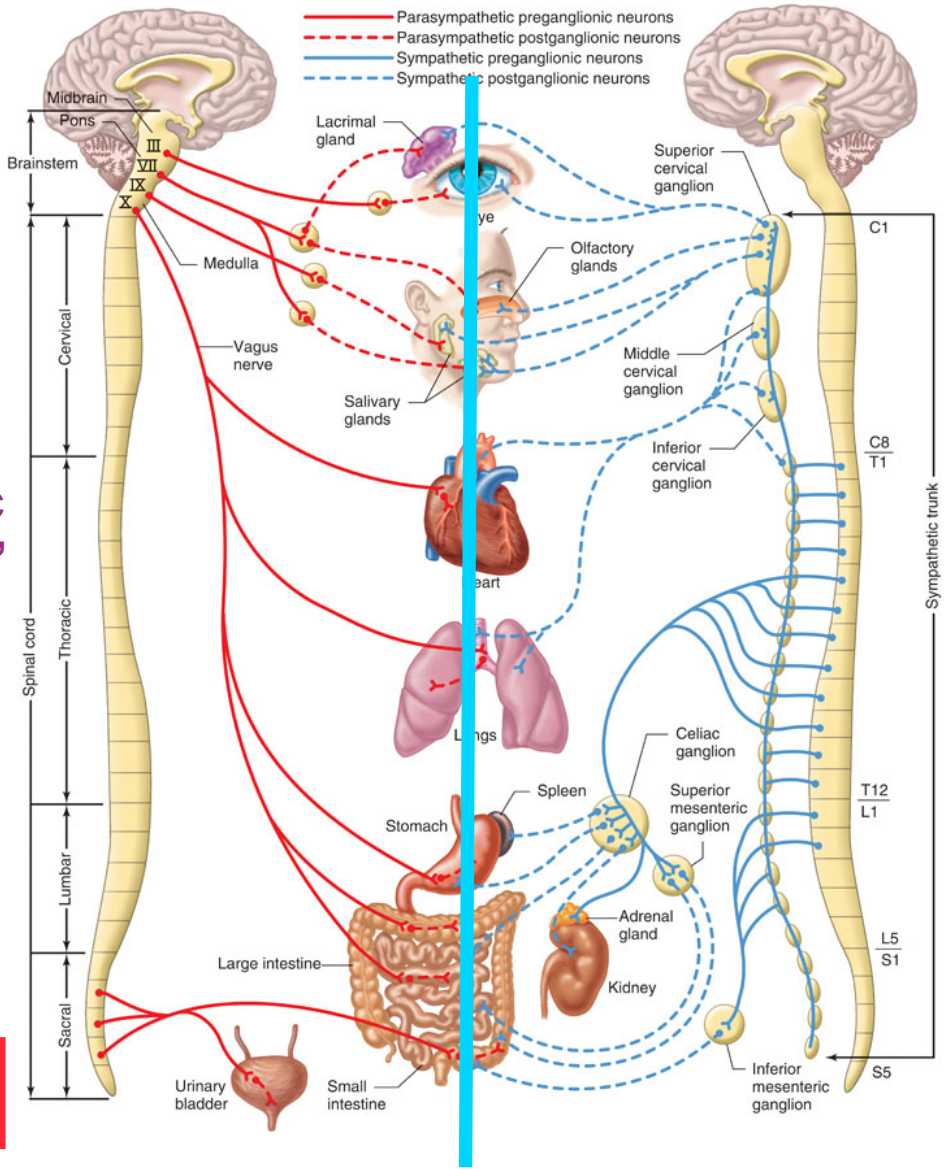
Parasympathetic
"rest and digest'



Sympathetic:
"emergency responses"

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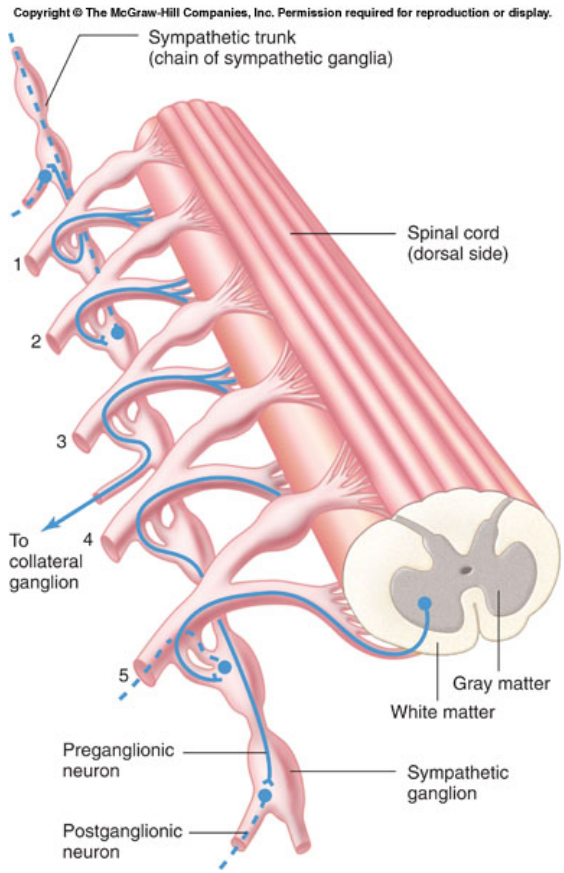

The sympathetic trunks are chains of sympathetic ganglia that are parallel to either side of the spinal cord; the trunk interacts closely with the associated spinal nerves.

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Table 6–10 Locations of Receptors for Acetylcholine, Norepinephrine, and Epinephrine

I. Receptors for acetylcholine
   a. Nicotinic receptors
      On postganglionic neurons in the autonomic ganglia
      At neuromuscular junctions of skeletal muscle
      On some central nervous system neurons
   b. Muscarinic receptors
      On smooth muscle
      On cardiac muscle
      On gland cells
      On some central nervous system neurons
      On some neurons of autonomic ganglia (although the great majority of receptors at this site are nicotinic)

II. Receptors for norepinephrine and epinephrine
   On smooth muscle
   On cardiac muscle
   On gland cells
   On some central nervous system neurons

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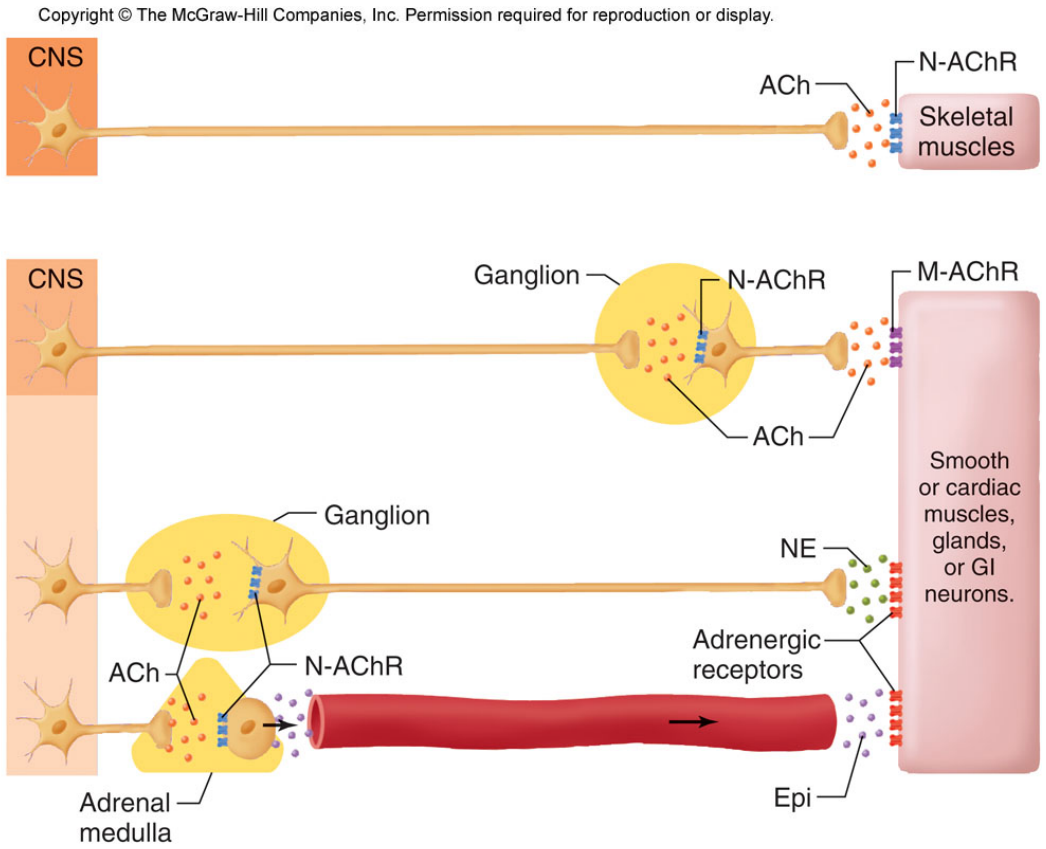
Voluntary command: Move!



Skeletal muscle contraction

Involuntary command: Rest & digest.

Involuntary command: Emergency!

Heart, smooth muscle, glands, many "involuntary" targets.

Figure 6-46

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Table 6–11 Some Effects of Autonomic Nervous System Activity

Effector Organ	Receptor Type*	Sympathetic Nervous System Effect	Parasympathetic Nervous System Effect†
Eyes			
Iris muscle	α_1	Contracts radial muscle (widens pupil)	Contracts sphincter muscle (makes pupil smaller)
Ciliary muscle	β_2	Relaxes (flattens lens for far vision)	Contracts (allows lens to become more convex for near vision)
Heart			
SA node	β_1	Increases heart rate	Decreases heart rate
Atria	β_1, β_2	Increases contractility	Decreases contractility
AV node	β_1, β_2	Increases conduction velocity	Decreases conduction velocity
Ventricles	β_1, β_2	Increases contractility	Decreases contractility slightly
Arterioles			
Coronary	α_1, α_2	Constricts	—‡
	β_2	Dilates	
Skin	α_1, α_2	Constricts	—
Skeletal muscle	α_1	Constricts	—
	β_2	Dilates	
Abdominal viscera	α_1	Constricts	—
Kidneys	α_1	Constricts	—
Salivary glands	α_1, α_2	Constricts	Dilates
Veins	α_1, α_2	Constricts	—
	β_2	Dilates	
Lungs			
Bronchial muscle	β_2	Relaxes	Contracts
Salivary glands	α_1	Stimulates watery secretion	Stimulates watery secretion
	β	Stimulates enzyme secretion	
Stomach			
Motility, tone	α_1, α_2, β_2	Decreases	Increases
Sphincters	α_1	Contracts	Relaxes
Secretion	(?)	Inhibits (?)	Stimulates
Intestine			
Motility	α_1, α_2, β_1, β_2	Decreases	Increases
Sphincters	α_1	Contracts (usually)	Relaxes (usually)
Secretion	α_2	Inhibits	Stimulates
Gallbladder	β_2	Relaxes	Contracts
Liver	α_1, β_2	Glycogenolysis and gluconeogenesis	—
Pancreas			
Exocrine glands	α	Inhibits secretion	Stimulates secretion
Endocrine glands	α_2	Inhibits secretion	—
	β_2	Stimulates secretion	
Fat cells	α_2, β_3	Increases fat breakdown	—
Kidneys	β_1	Increases renin secretion	—
Urinary bladder			
Bladder wall	β_2	Relaxes	Contracts
Sphincter	α_1	Contracts	Relaxes
Uterus	α_1	Contracts in pregnancy	Variable
	β_2	Relaxes	
Reproductive tract (male)	α_1	Ejaculation	Erection
Skin			
Muscles causing hair erection	α_1	Contracts	—
Sweat glands	α_1	Secretion from hands, feet, and armpits	—
	AChR	Generalized abundant, dilute secretion	—

Table adapted from "Goodman and Gilman's The Pharmacological Basis of Therapeutics," Laurence L. Brunton, John S. Lazo, and Keither L. Parker, eds., 11th ed., McGraw-Hill, New York, 2006.

*Note that many effector organs contain both alpha-adrenergic and beta-adrenergic receptors. Activation of these receptors may produce either the same or opposing effects. For simplicity, except for the arterioles and a few other cases, only the dominant sympathetic effect is given when the two receptors oppose each other.

†These effects are all mediated by muscarinic receptors.

‡A dash means these cells are not innervated by this branch of the autonomic nervous system or that these nerves do not play a significant physiological role.

Chapter 8
Consciousness, Brain, and Behavior

Electroencephalography: a window on the brain

- **States of wakefulness and sleep**
- **Limbic system: motivation and reward**
- **Neurochemistry of drug abuse**
- **Learning and memory**

Figure 8-1

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VOLTAGE
(typically 20-100 microvolts)

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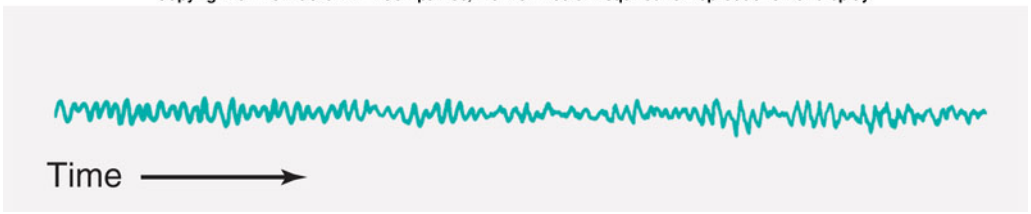

The electroencephalograph (EEG) is the printout of an electronic device that uses scalp electrodes to monitor the internal neural activity in the brain; this is a record from the parietal or occipital lobes of an awake person.

Figure 8-2

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VOLTAGE
(20 to 100 microvolts)

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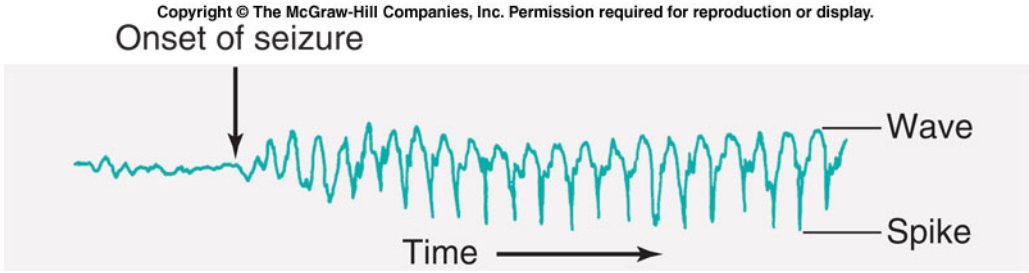

EEGs provide diagnostic information about the location of abnormal activity in the brain, such as shown in this record typical of a patient undergoing an epileptic seizure.

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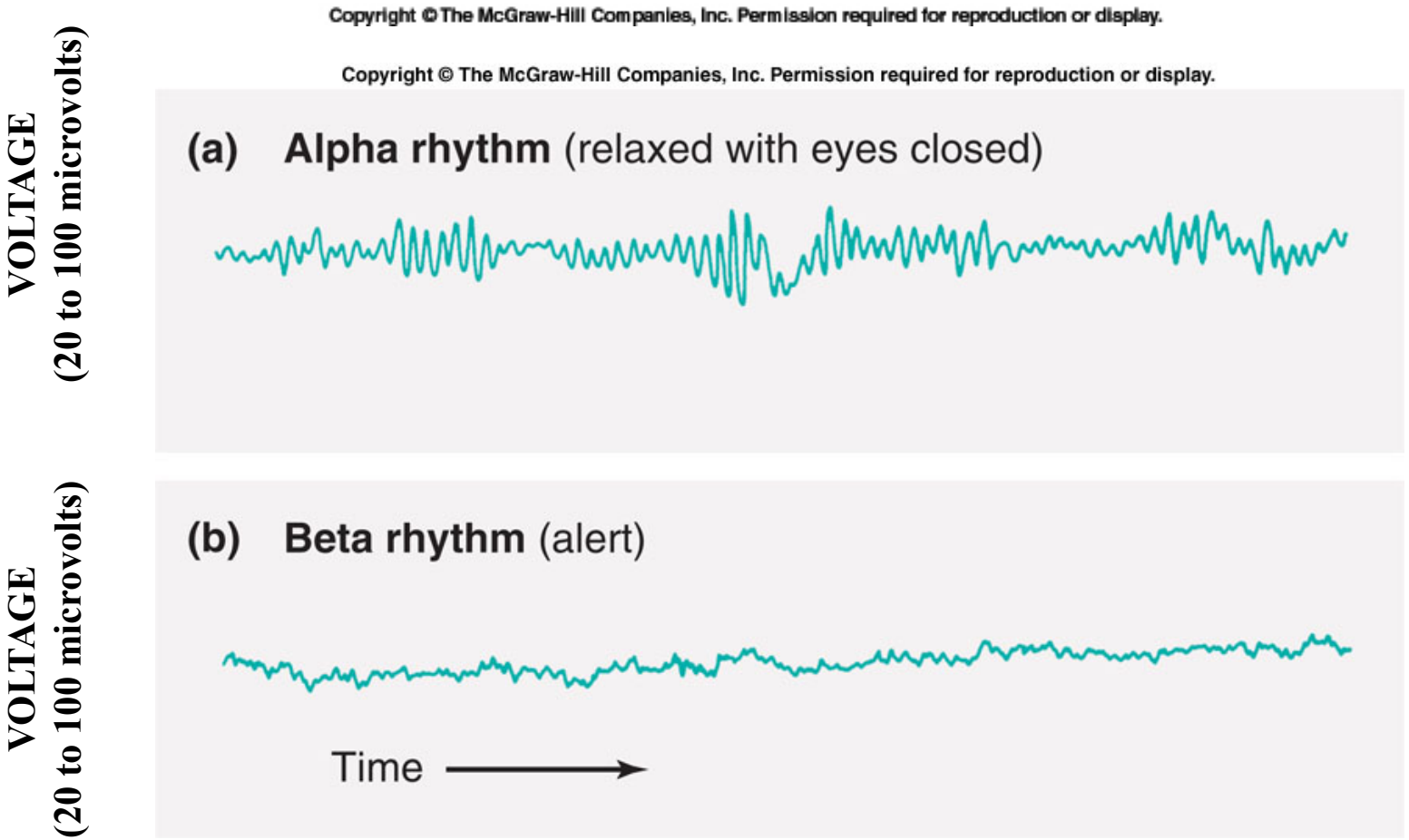

Figure 8-3

EEGs reflect mental state: contrasted here are mental relaxation (a) versus concentration (b).

Figure 8-4

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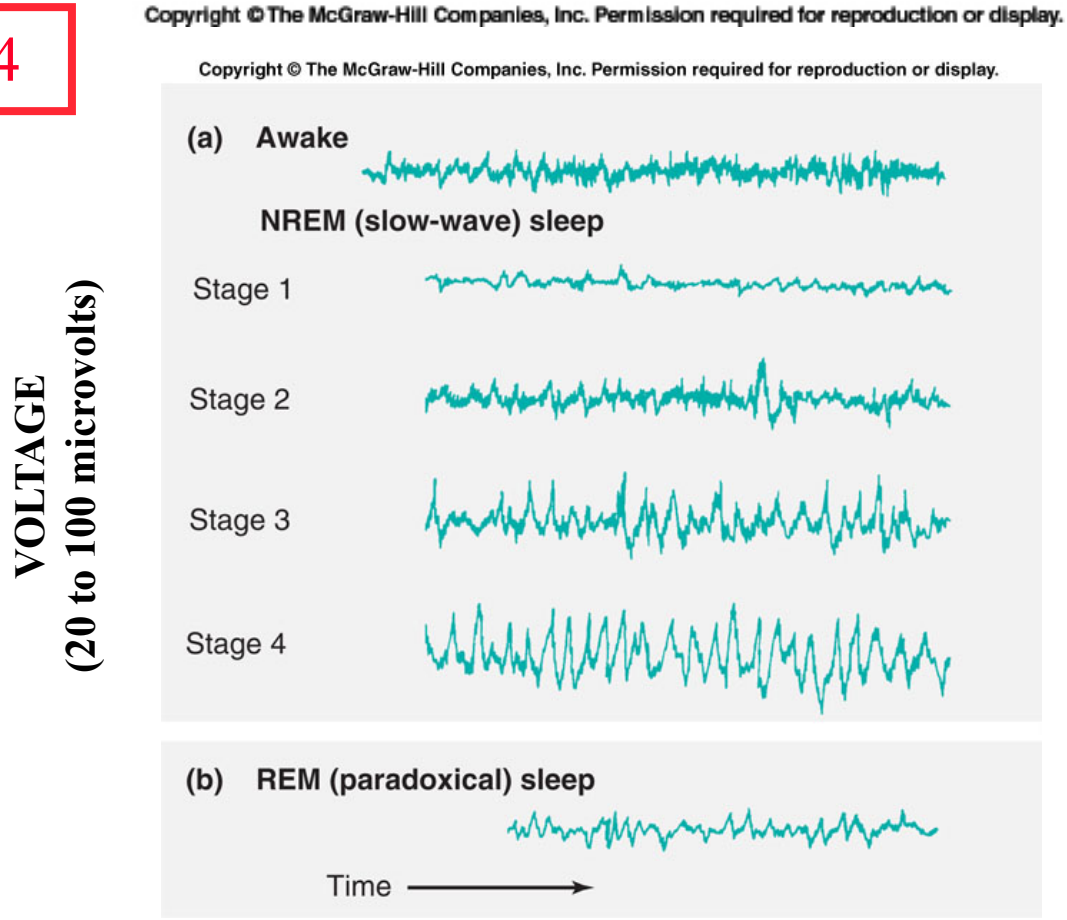

EEG patterns undergo characteristic shifts in a sleeping person, reflecting the four stages of sleep; the duration of the series is typically ~90 minutes, and the entire pattern cycles 4 to 8 times per night.

Figure 8-5

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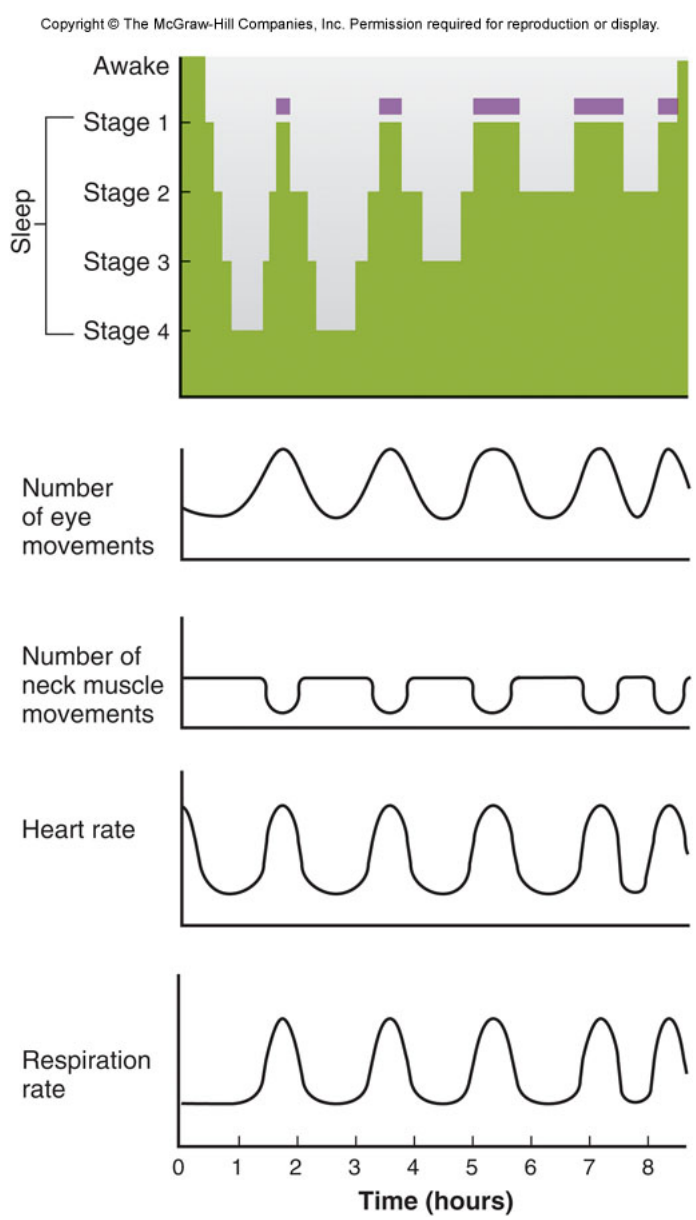

The EEG pattern was analyzed to produce this graph of a full night's sequence of sleep stages; also shown are cyclic patterns in the periphery.

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TABLE 8–1 Sleep–Wakefulness Stages

STAGE	BEHAVIOR	EEG (see Figures 8–3 and 8–4)
Alert wakefulness	Awake, alert with eyes open.	Beta rhythm (faster than 13 Hz).
Relaxed wakefulness	Awake, relaxed with eyes closed.	Mainly alpha rhythm (8–13 Hz) over the parietal and occipital lobes. Changes to beta rhythm in response to internal or external stimuli.
Relaxed drowsiness	Fatigued, tired, or bored; eyelids may narrow and close; head may start to droop; momentary lapses of attention and alertness. Sleepy but not asleep.	Decrease in alpha-wave amplitude and frequency.
NREM (slow-wave) sleep		
Stage 1	Light sleep; easily aroused by moderate stimuli or even by neck muscle jerks triggered by muscle stretch receptors as head nods; continuous lack of awareness.	Alpha waves reduced in frequency, amplitude, and percentage of time present; gaps in alpha rhythm filled with theta (4–8 Hz) and delta (slower than 4 Hz) activity.
Stage 2	Further lack of sensitivity to activation and arousal.	Alpha waves replaced by random waves of greater amplitude.
Stages 3 and 4	Deep sleep; in stage 4, activation and arousal occur only with vigorous stimulation.	Much theta and delta activity, predominant delta in stage 4.
REM (paradoxical) sleep	Deepest sleep; greatest relaxation and difficulty of arousal; begins 50–90 min after sleep onset, episodes are repeated every 60–90 min, each episode lasting about 10 min; dreaming occurs, rapid eye movements behind closed eyelids; marked increase in brain O_2 consumption.	EEG resembles that of alert awake state.

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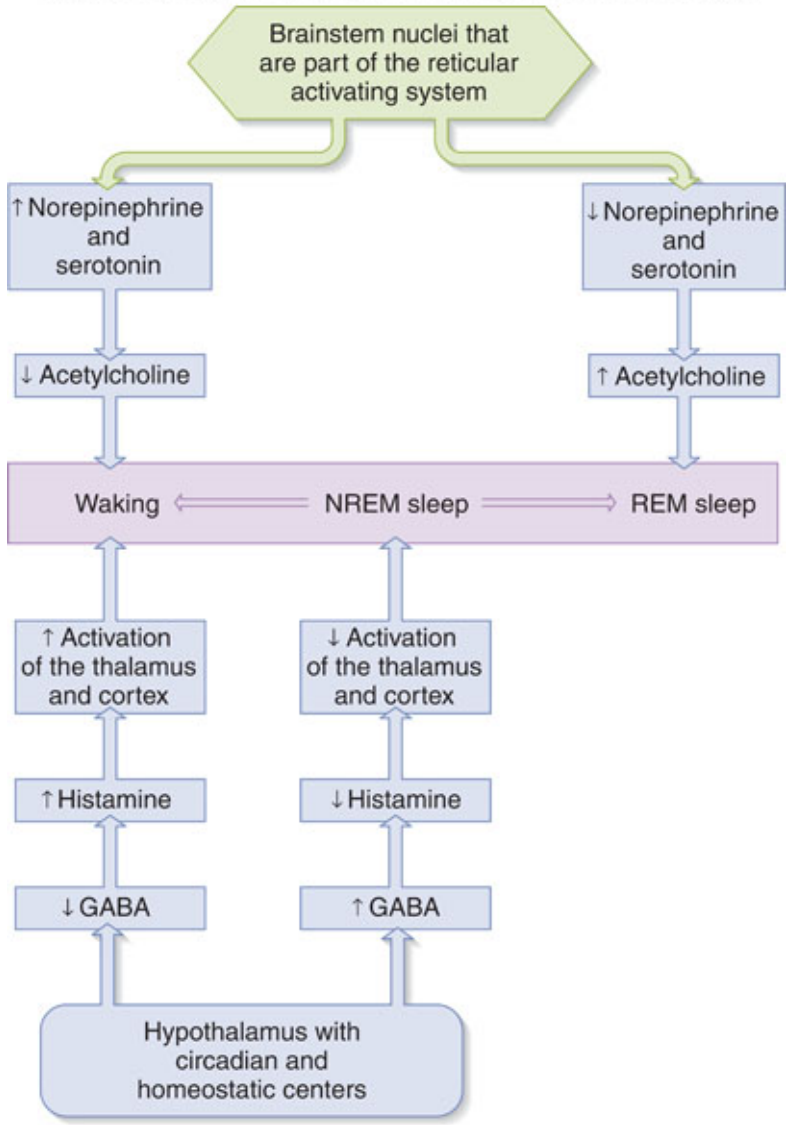

A model of some of the neurochemical changes across the sleep-wake continuum; cause-and-effect relationships are under study.

Figure 8-7

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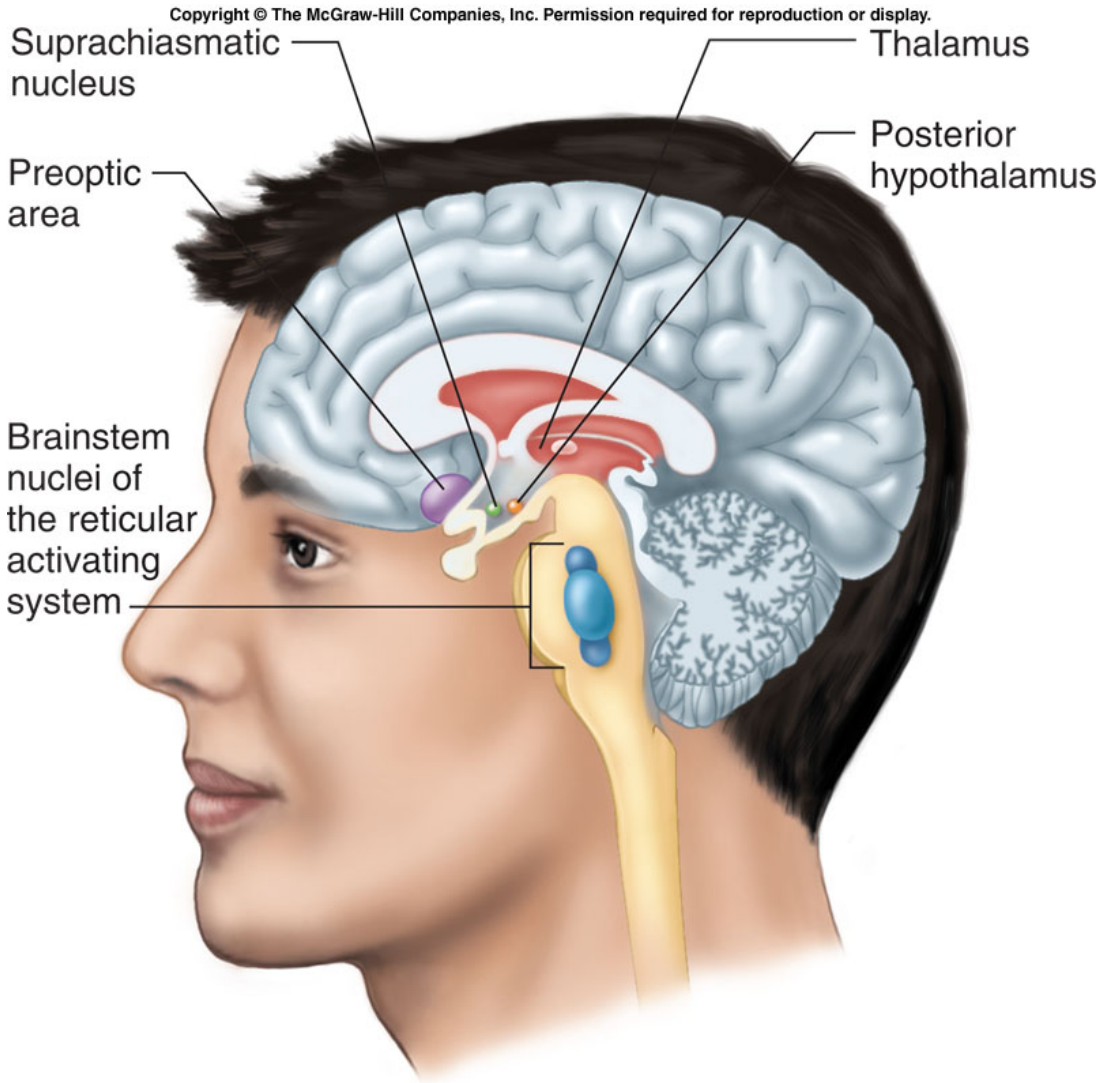

Neuronal changes in these CNS structures appear to be essential participants in sleep-wake transitions and in biological rhythms.

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TABLE 8–2 Criteria for Brain Death

I. The nature and duration of the coma must be known.
   a. Known structural damage to brain or irreversible systemic metabolic disease.
   b. No chance of drug intoxication, especially from paralyzing or sedative drugs.
   c. No sign of brain function for 6 h in cases of known structural cause and when no drug or alcohol is involved; otherwise, 12–24 h without signs of brain function plus a negative drug screen.

II. Cerebral and brainstem function are absent.
   a. No response to painful stimuli administered above the spinal cord.
   b. Pupils unresponsive to light.
   c. No eye movement in response to ice-water stimulation of the vestibular reflex.
   d. Apnea (no spontaneous breathing) for 10 min.
   e. Systemic circulation may be intact.
   f. Purely spinal reflexes may be retained.

III. Supplementary (optional) criteria.
   a. Flat EEG (wave amplitudes less than 2 μV).
   b. Responses absent in vital brainstem structures.
   c. Greatly reduced cerebral circulation.

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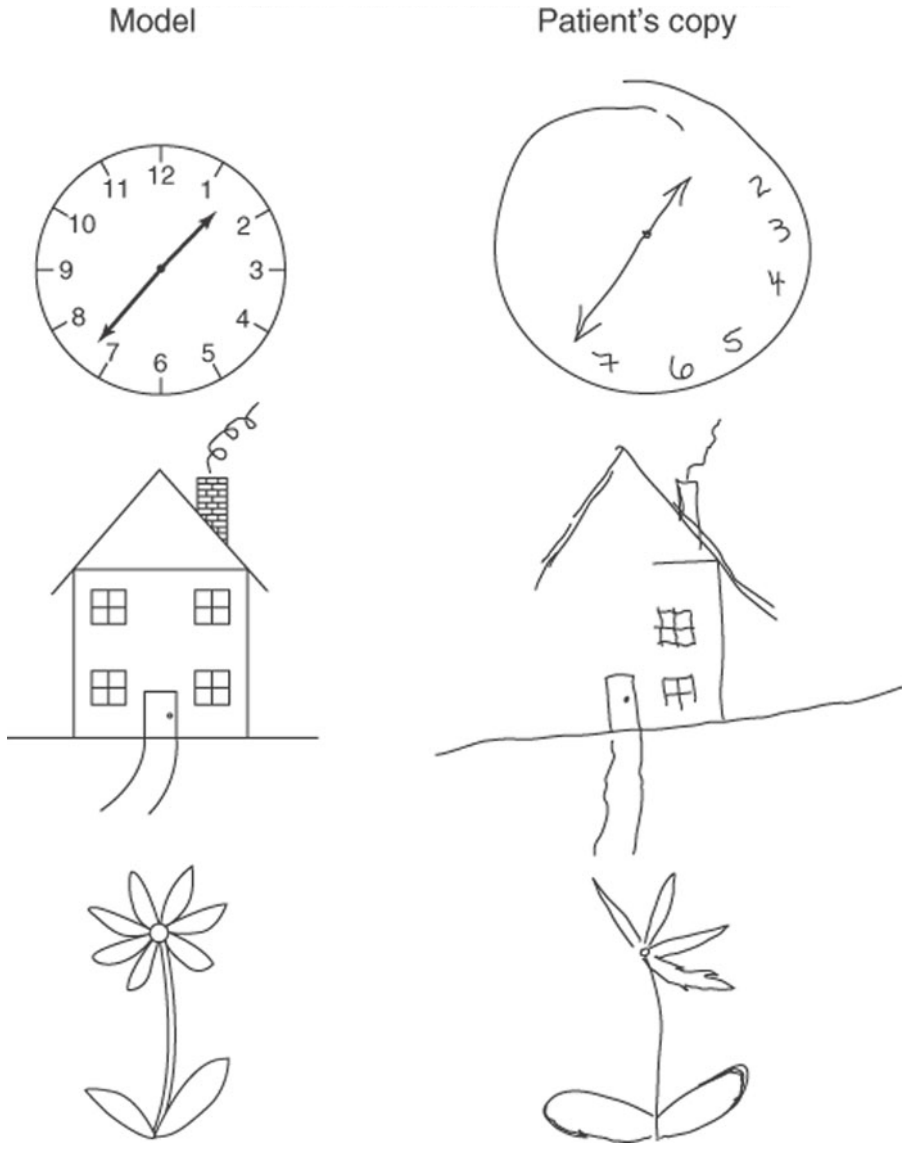
Figure 8-8



Neural damage in the right parietal lobe of this patient results in the unilateral visual neglect seen in this drawing task. Although patient is not impaired visually, does not *perceive* part of visual world.

Figure 8-9

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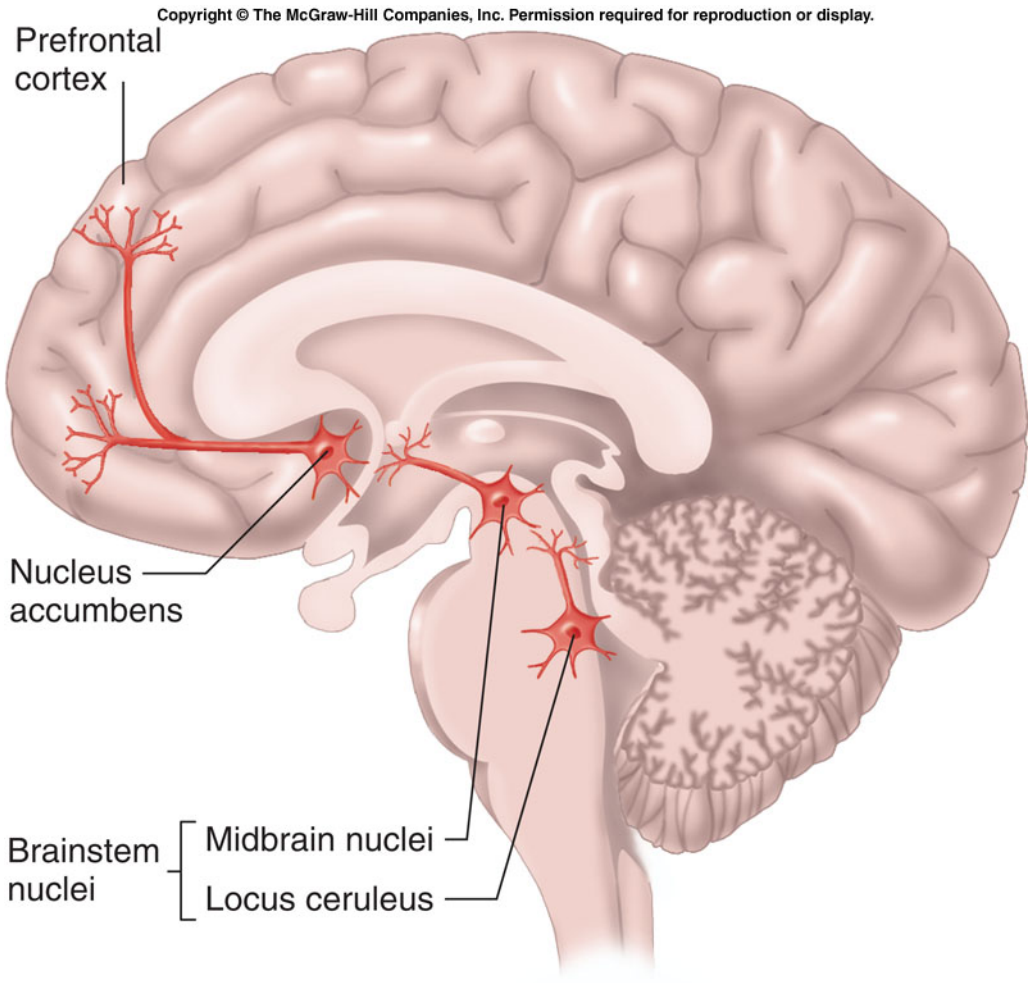

Alterations in the mesolimbic dopamine pathway (shown here) appear to be a primary mechanism by which psychoactive drugs change behavior.

Figure 8-10

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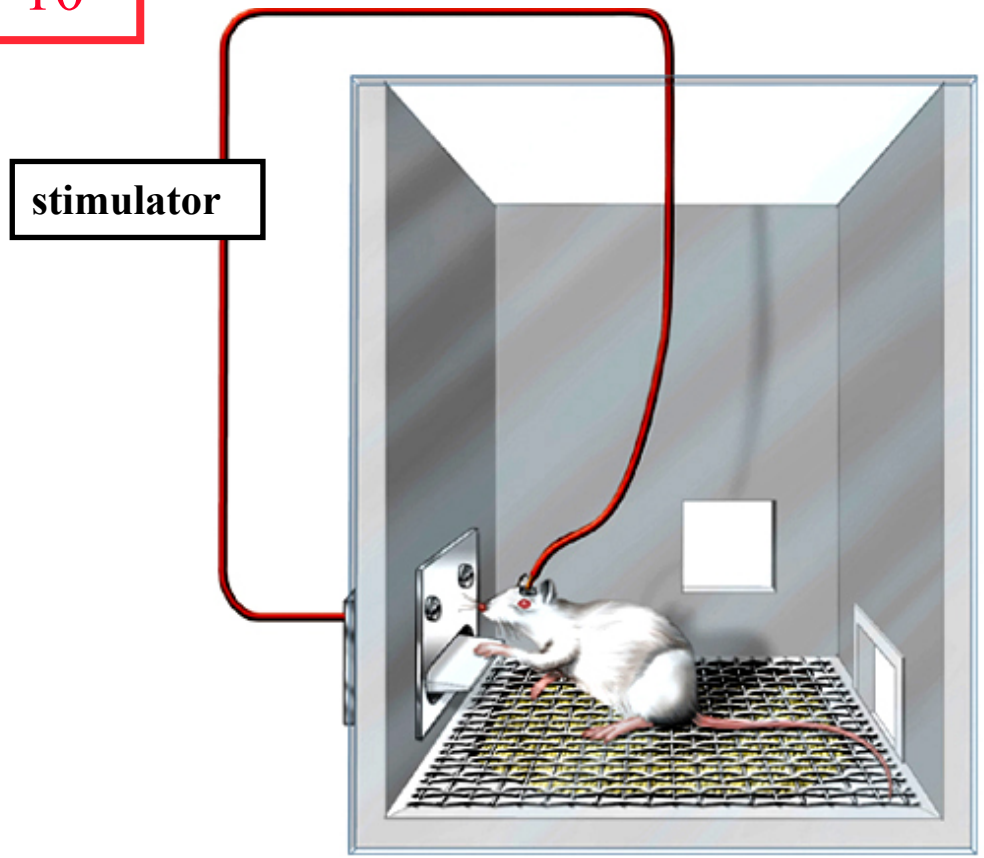

Animal models, such as this rat performing lever-presses to receive rewarding neural stimulation through electrodes implanted in its brain, have provided detailed insights into the anatomical and neurochemical organization of the brain.

Figure 8-11

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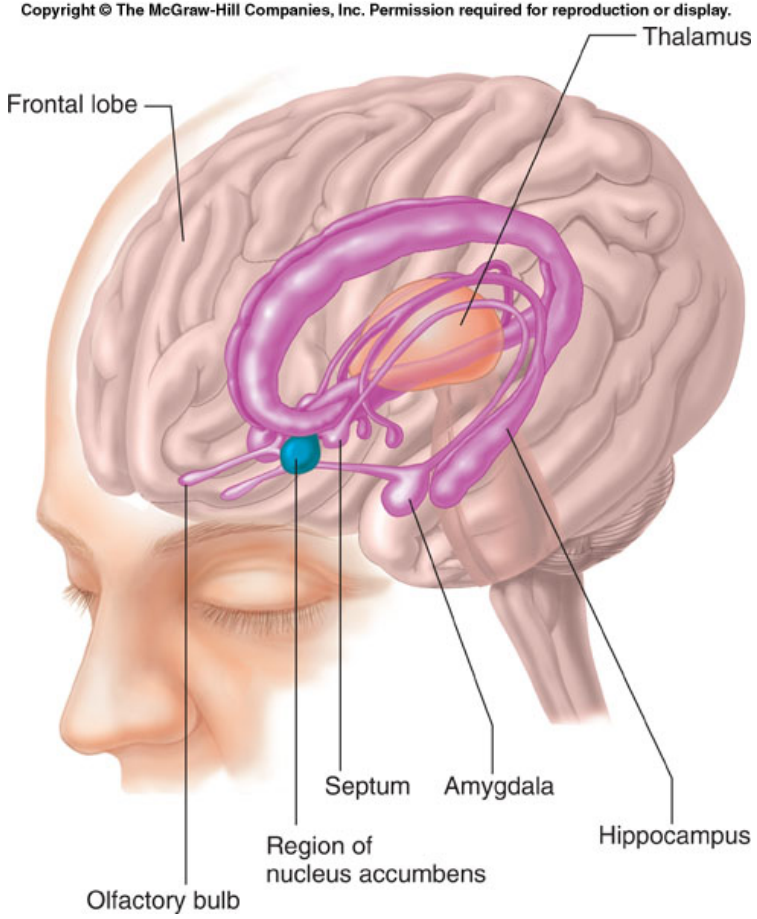

Changes in activity of the limbic system underlie some of the primary needs of the organism, including learning, motivation, appetite, and emotional response; its malfunction is associated with affective disorders.

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Psychoactive drugs that affect serotonin-receptors share structural similarities with serotonin.

Psychoactive drugs that affect dopamine-receptors share structural similarities with dopamine.

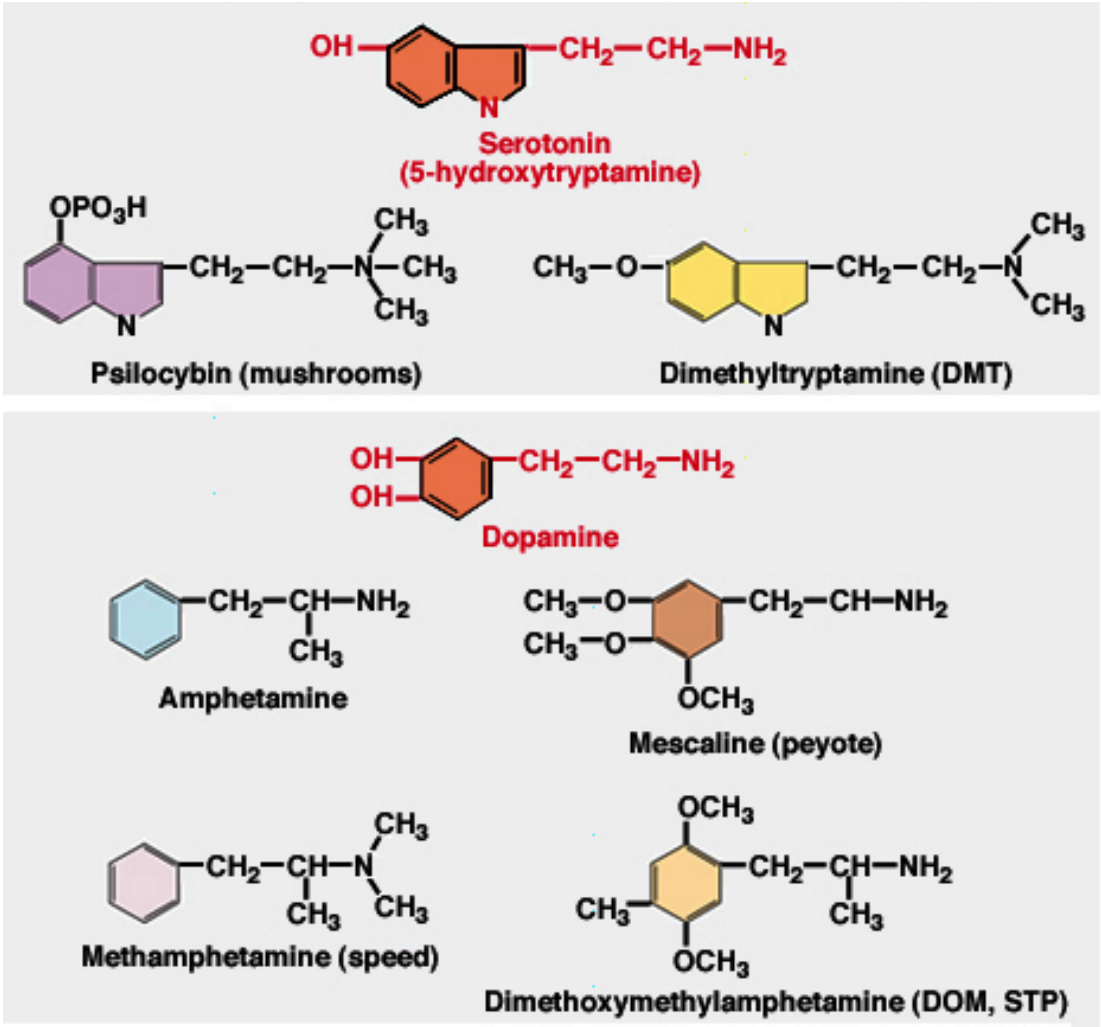

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TABLE 8–3 Diagnostic Criteria for Substance Dependence

Substance dependence is indicated when three or more of the following occur within a 12-month period.

1. Tolerance, as indicated by
   a. a need for increasing amounts of the substance to achieve the desired effect, or
   b. decreasing effects when continuing to use the same amount of the substance
2. Withdrawal, as indicated by
   a. appearance of the characteristic withdrawal symptoms upon stopping use of the substance, or
   b. use of the substance (or one closely related to it) to relieve or avoid withdrawal symptoms
3. Use of the substance in larger amounts or for longer periods of time than intended
4. Persistent desire for the substance; unsuccessful attempts to cut down or control use of the substance
5. A great deal of time is spent in activities necessary to obtain the substance, use it, or recover from its effects.
6. Occupational, social, or recreational activities are given up or reduced because of substance use.
7. Use of the substance is continued despite knowledge that one has a physical or psychological problem that is likely to be exacerbated by the substance.

Adapted from DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, 4th edn. American Psychiatric Association, Washington, D.C., 1994.

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TABLE 8–4 Potential of Various Substances to Cause Dependence

If 100 people regularly use a substance, how many will become dependent on it?	
Nicotine	33
Heroin	25
Cocaine	16
Alcohol	15
Amphetamines	11
Marijuana	9

Figure 8-14

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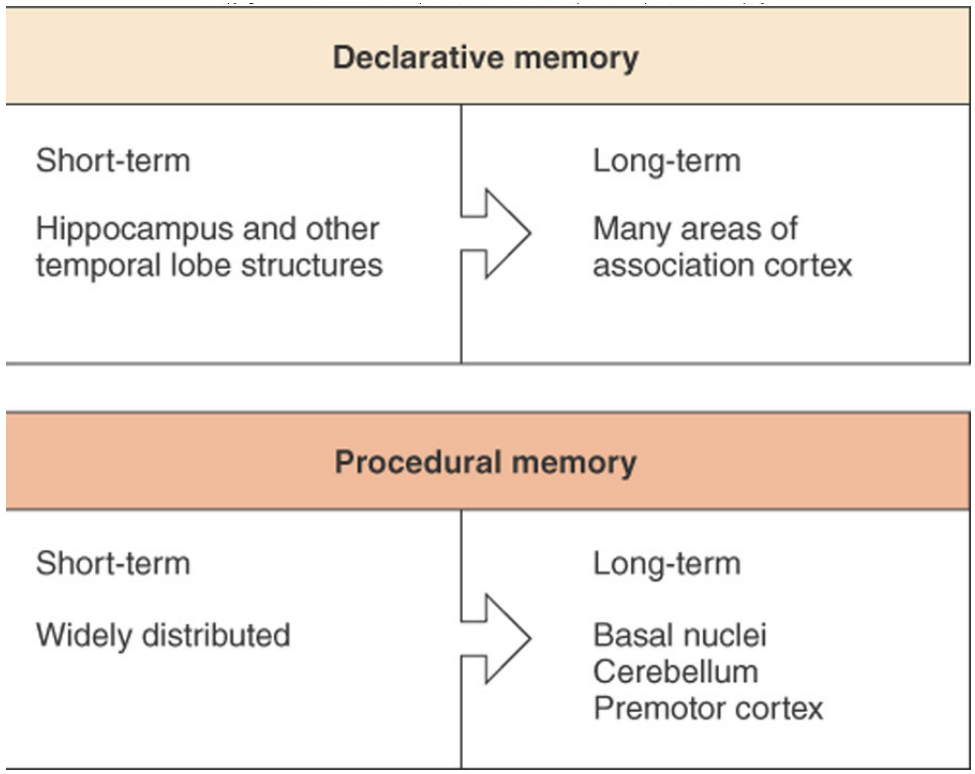
Declarative memory is associated with actual events in a person's direct experience.



Procedural memory is associated knowledge of the sequence of events and relationships between events.

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TABLE 8–5 General Principles about Learning and Memory

1. There are multiple memory systems in the brain.
2. Working memory requires changes in existing neural circuits, whereas long-term memory requires new protein synthesis and growth.
3. These changes may involve multiple cellular mechanisms within single neurons.
4. Second-messenger systems appear to play a role in mediating cellular changes.
5. Changes in the properties of membrane channels are often correlated with learning and memory.

Adapted from John M. Beggs et al. "Learning and Memory: Basic Mechanisms," in Michael J. Zigmond, Floyd E. Bloom, Story C. Landis, James L. Roberts, and Larry R. Squire, eds., Fundamental Neuroscience, Academic Press, San Diego, CA, 1999.

Figure 8-17

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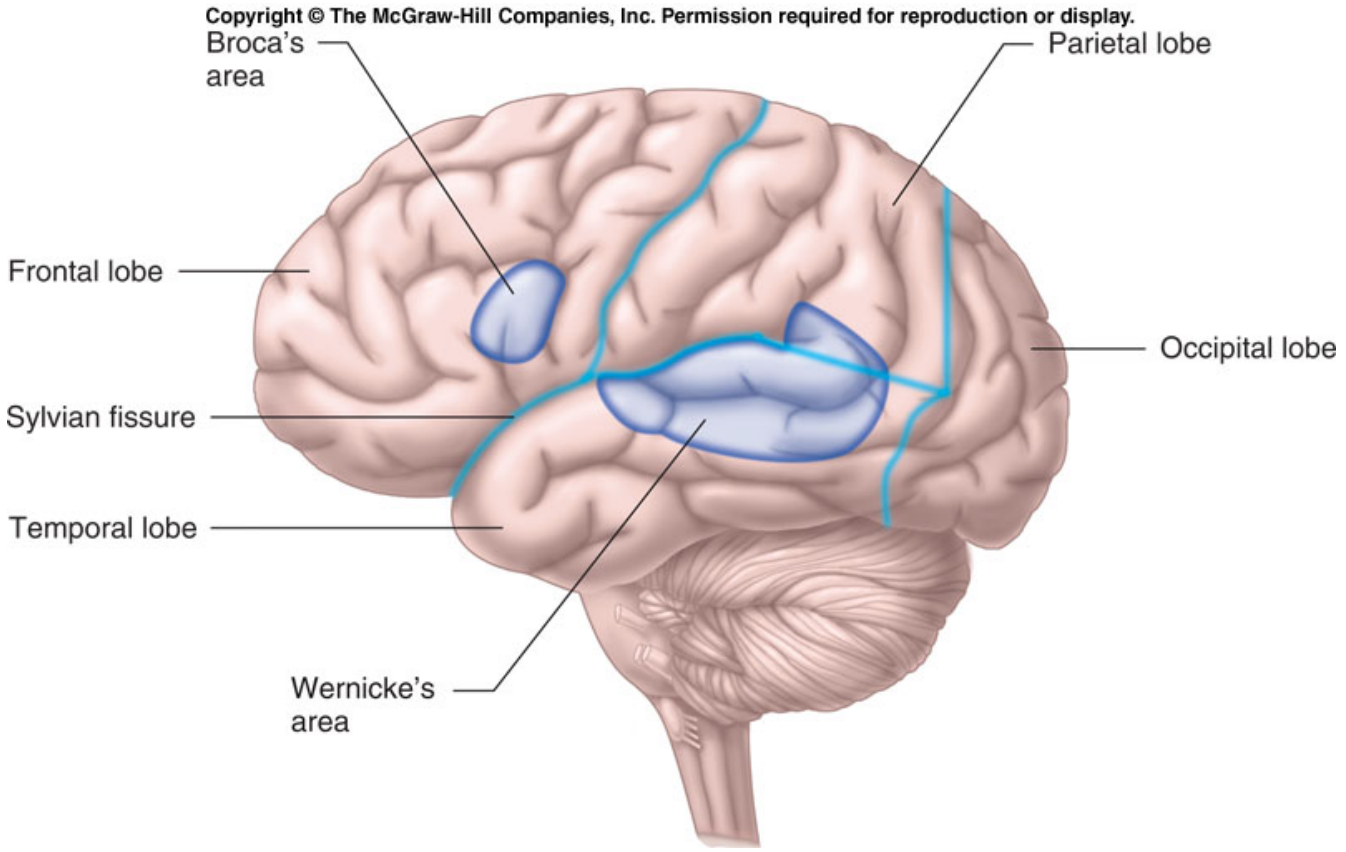
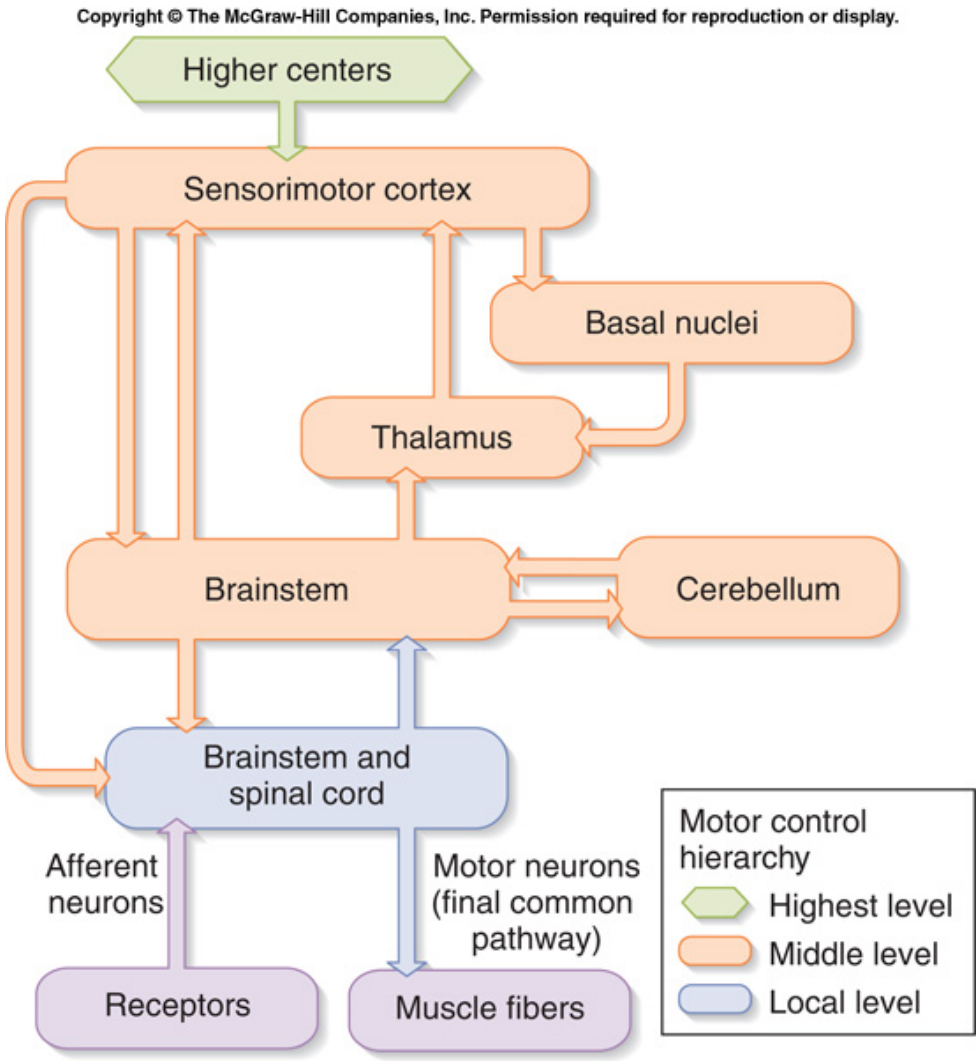


The primary loci underlying the comprehension of speech are in Wernicke's area, whereas the primary loci for the production of speech are located in Broca's area.

Motor commands from the brain have been modified by a variety of excitatory and inhibitory control systems, including essential feedback from sensory afferent neurons, along with vision and balance cues (not shown).



Side and cross-sectional views of some of the neural components regulating motor commands. Altered processing abilities in these components can cause motor problems such as Parkinsonism.

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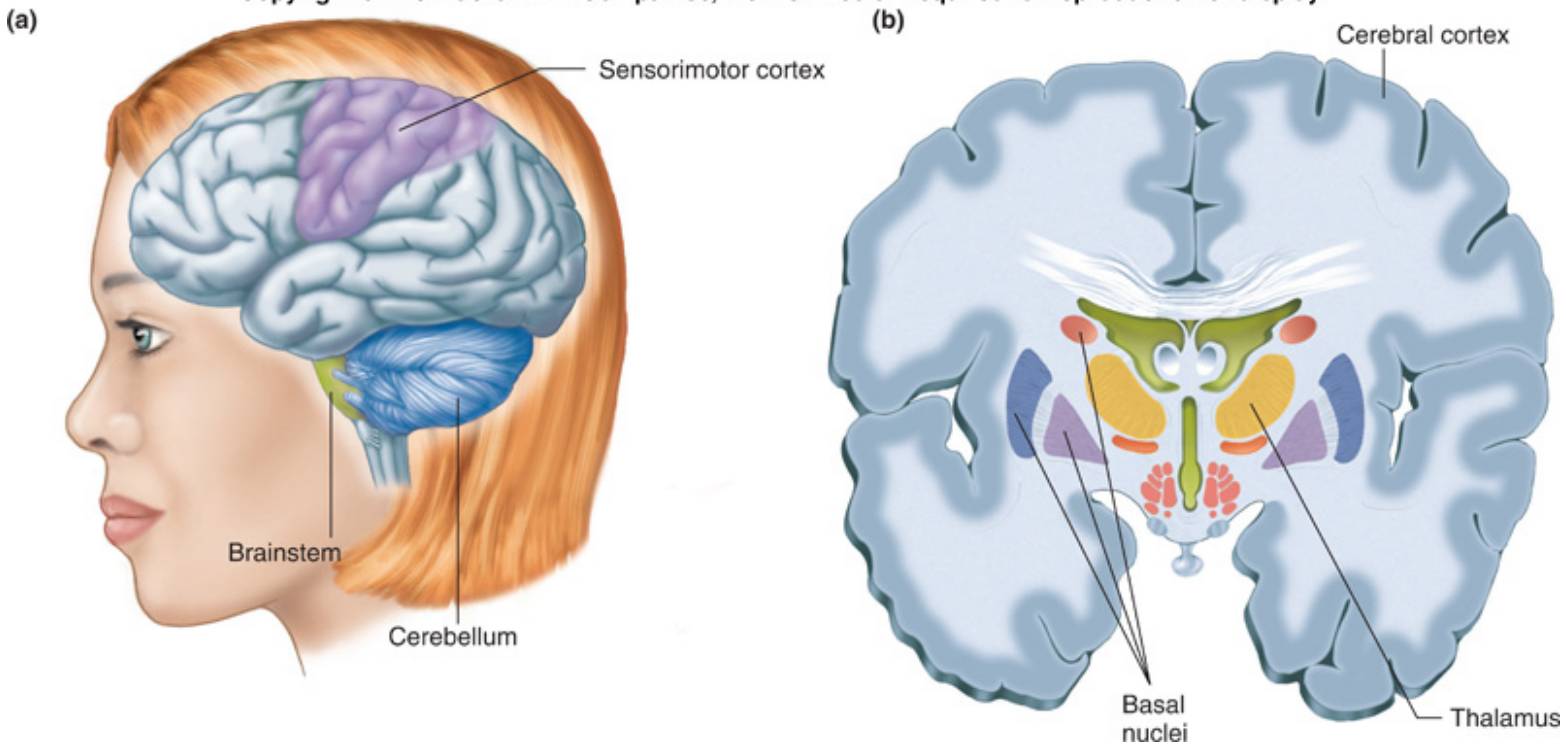

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TABLE 10–1 Conceptual Motor Control Hierarchy for Voluntary Movements

I. Higher centers

a. Function: forms complex plans according to individual's intention and communicates with the middle level via "command neurons."

b. Structures: areas involved with memory and emotions, supplementary motor area, and association cortex. All these structures receive and correlate input from many other brain structures.

II. The middle level

a. Function: converts plans received from the highest level to a number of smaller motor programs, which determine the pattern of neural activation required to perform the movement. These programs are broken down into subprograms that determine the movements of individual joints. The programs and subprograms are transmitted through descending pathways to the lowest control level.

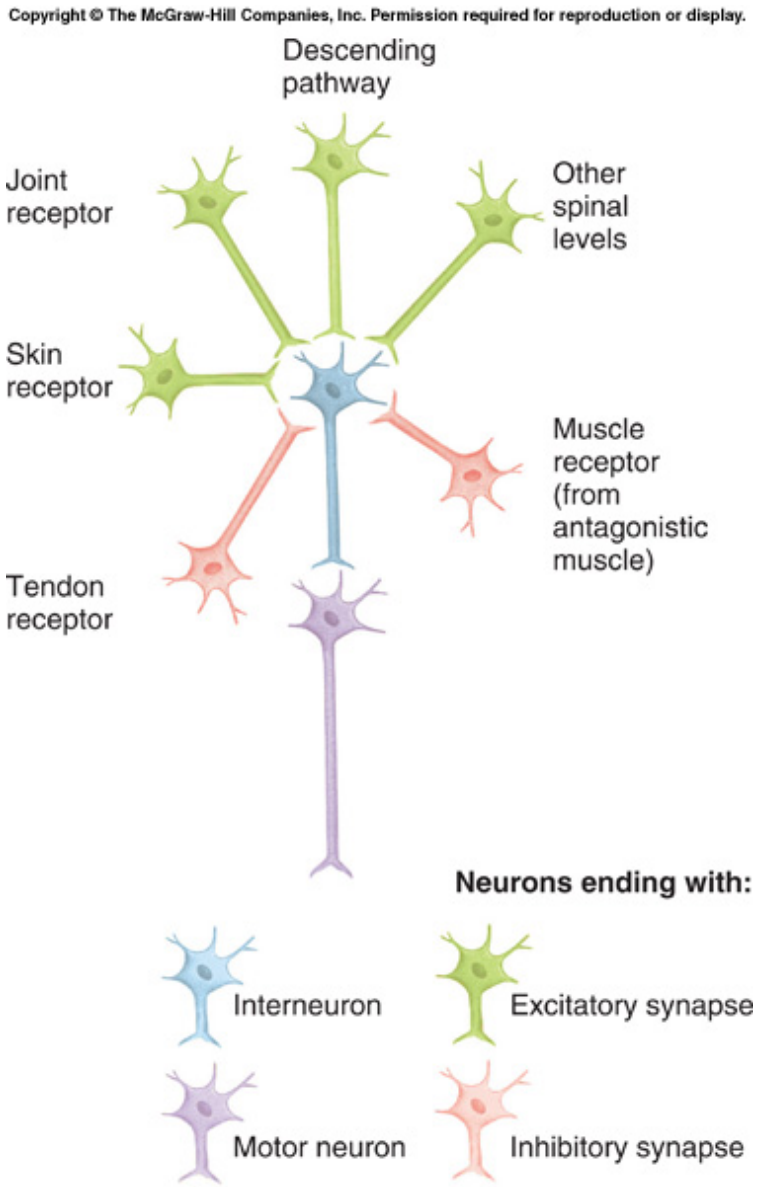
b. Structures: sensorimotor cortex, cerebellum, parts of basal nuclei, some brainstem nuclei.

III. The lowest level (the local level)

a. Function: specifies tension of particular muscles and angle of specific joints at specific times necessary to carry out the programs and subprograms transmitted from the middle control levels.

b. Structures: levels of brainstem or spinal cord from which motor neurons exit.

Examples of the categories of information and their underlying neuronal substrates modifying the production of motor commands from the brain.



Acting on local reflex circuits and by relaying impulses to the brain, muscle spindles and Golgi tendon organs provide information about muscle position and stretch in order to finely regulate the speed and intensity of muscle contraction.

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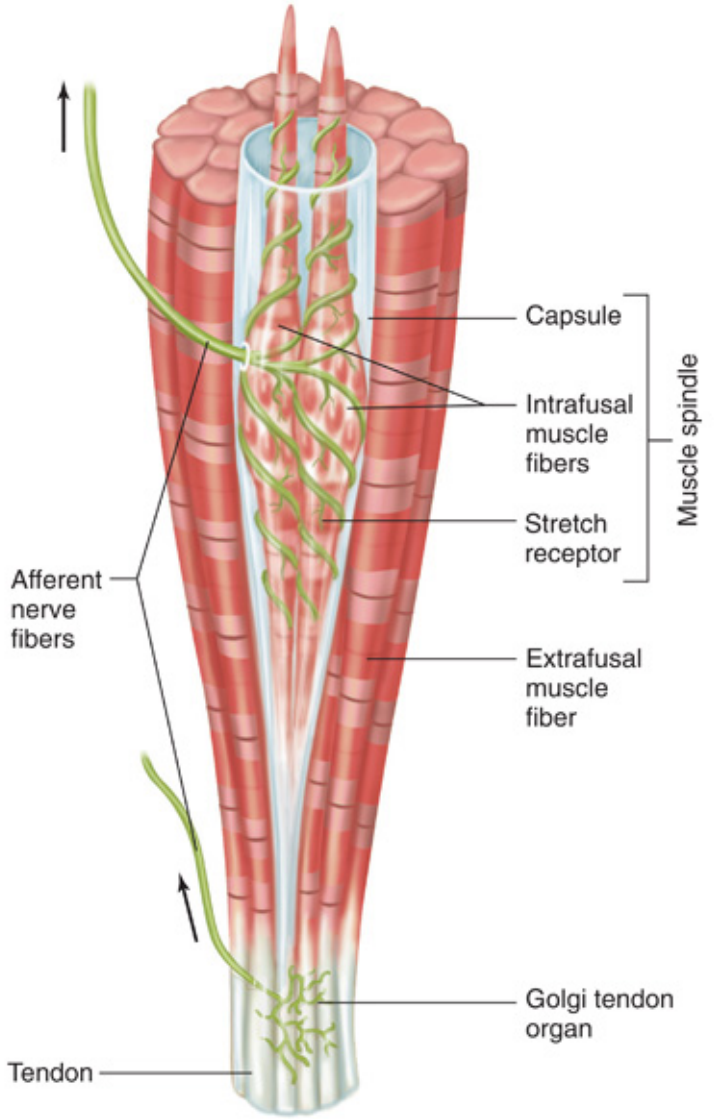
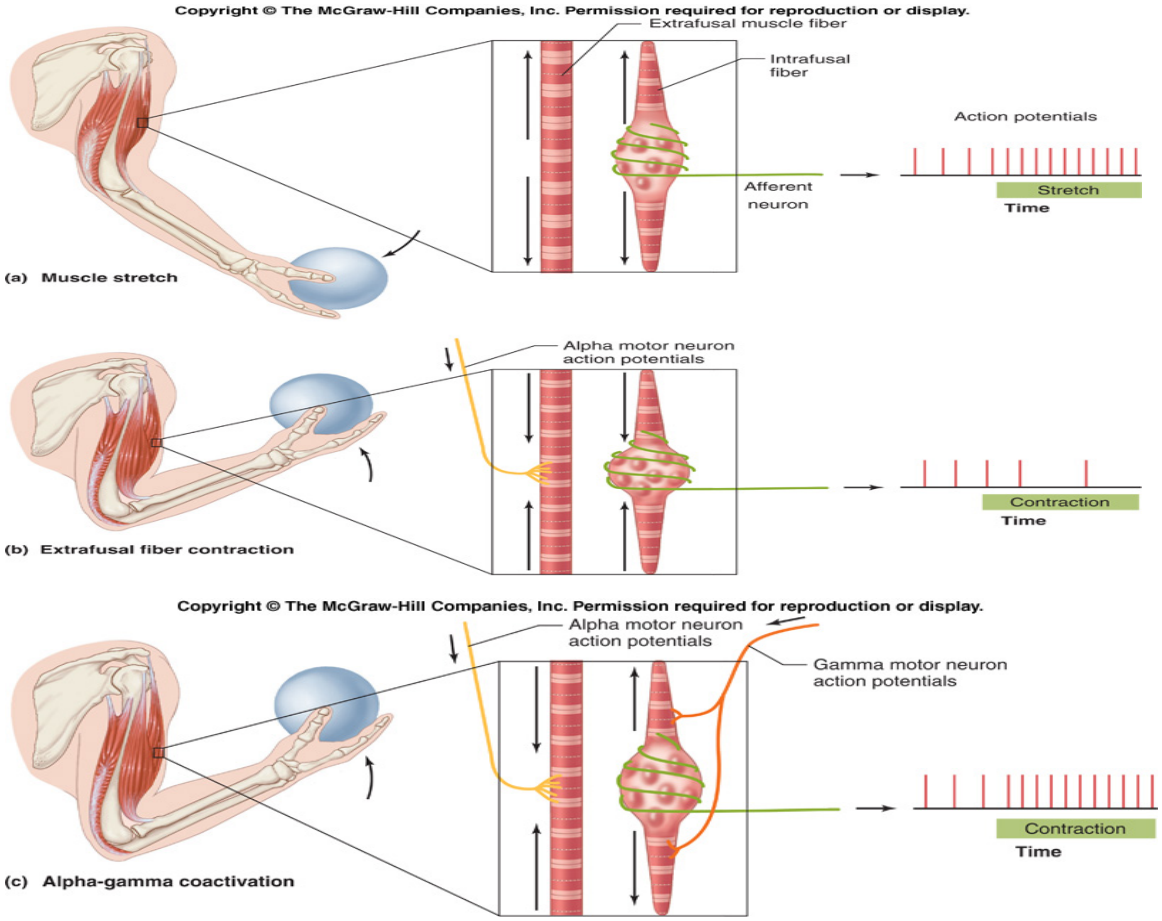


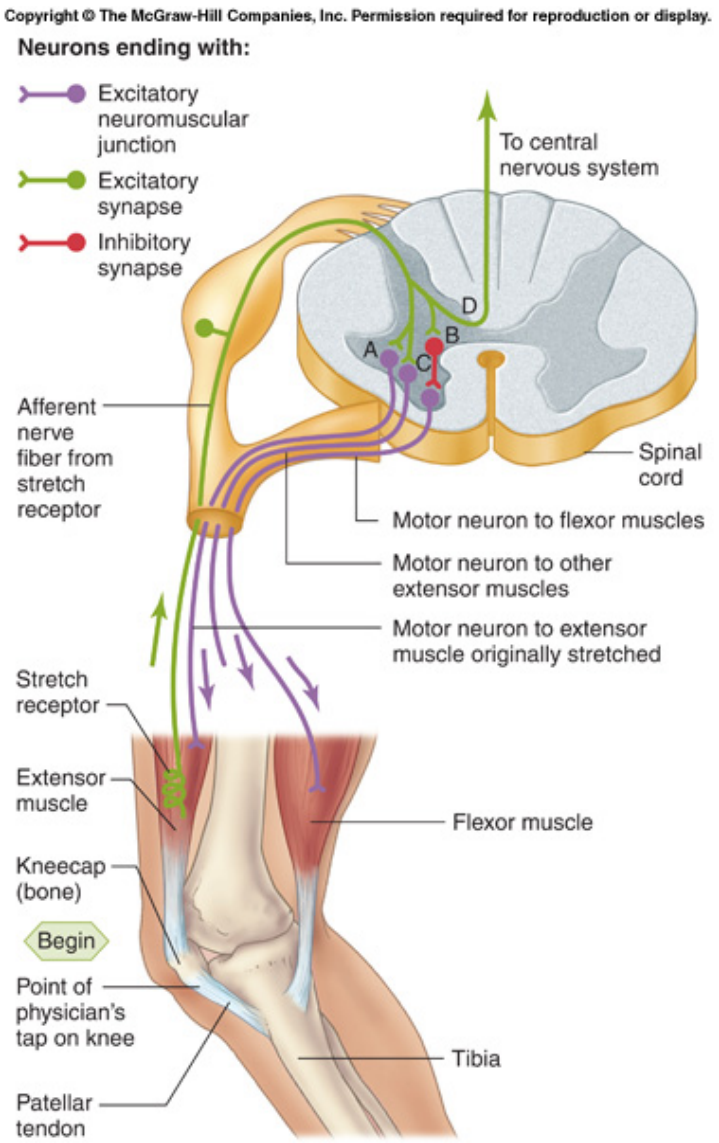
Regardless of the reason for a change in length, the stretched spindle in scenario (a) generates a burst of action potentials as the muscle is lengthened; in scenario (b), the shortened spindle produces fewer action potentials from the spindle.



Tapping the patellar tendon lengthens the stretch receptor in the associated extensor muscle in the thigh; responses include:

compensatory contraction in that muscle (A and C), relaxation in the opposing flexor (B), and sensory afferent delivery to the brain.

Note: NMJ = neuromuscular junction



Activation of Golgi tendon organs. Compared to when a muscle is contracting, passive stretch of the relaxed muscle produces less stretch of the tendon and fewer action potentials from the Golgi tendon organ.

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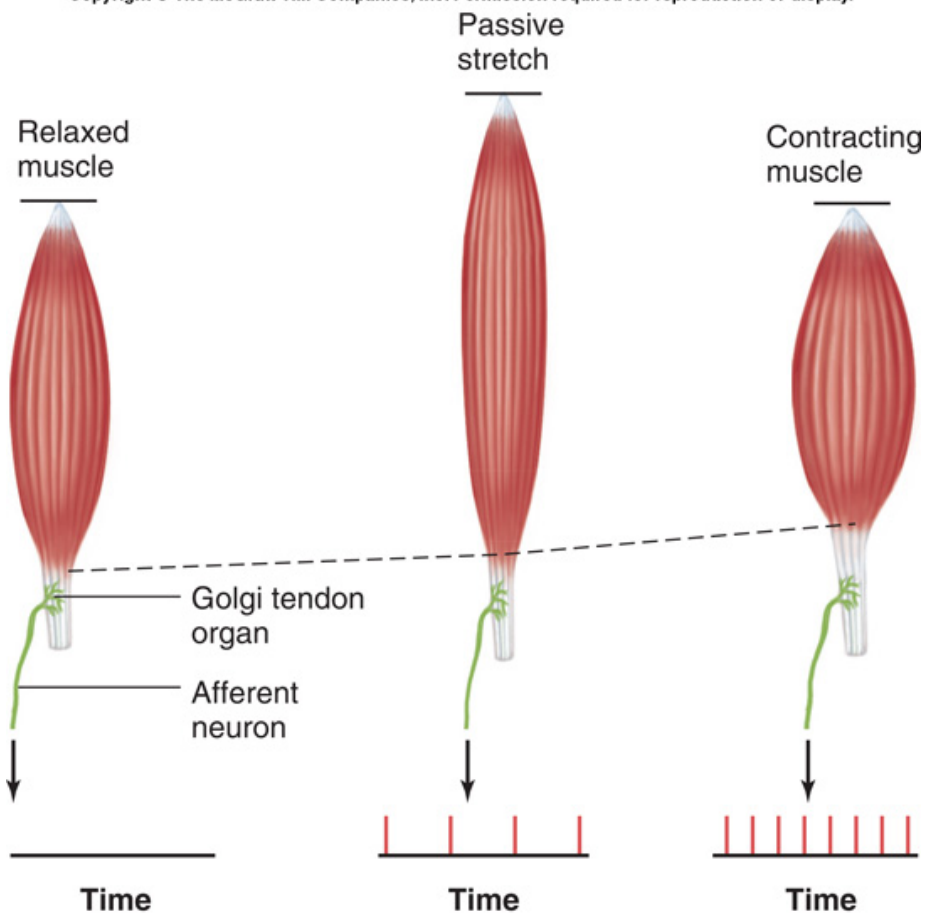
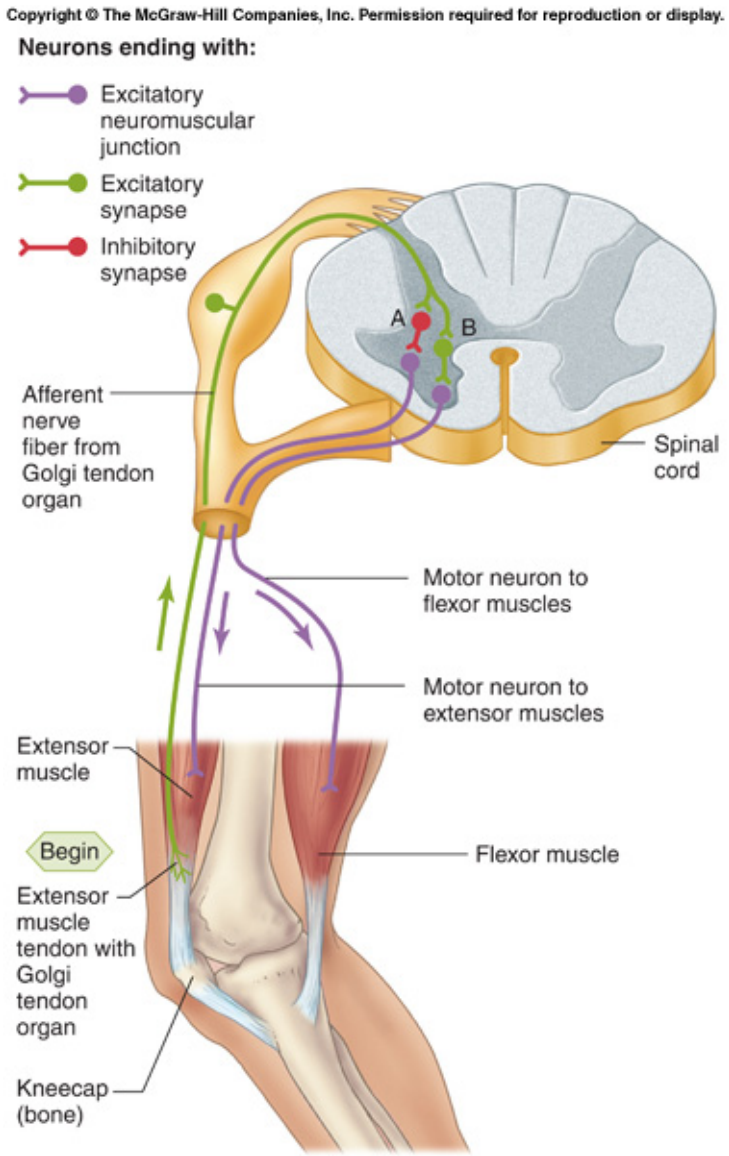


Action potentials in afferent neurons

Contraction of the extensor muscle on the thigh tenses the Golgi tendon organ and activates it to fire action potentials. Responses include:

Inhibition of the motor neurons that innervate this muscle (A), and excitation in the opposing flexor's motor neurons (B).

Note: NMJ = neuromuscular junction



The neural components of the pain-withdrawal reflex in this example proceed as follows:

1. Pain sensory afferents detect pain in foot and send action potentials via dorsal horn of spinal cord.
2. Interneurons in the cord activate extensor muscles on the "pained" side of the body and flexor muscles on the opposite side of the body.
3. Muscles move body away from painful stimulus.

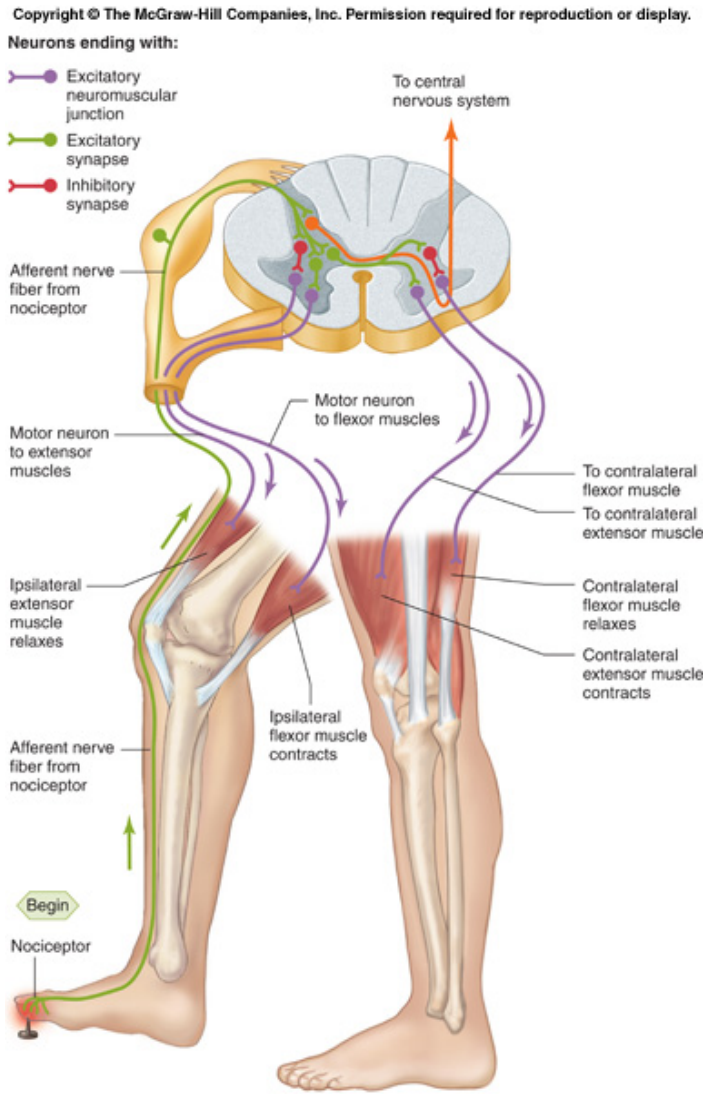


Figure 10-9

Extensive neural networks between the major "motor areas" of the cerebral cortex permit fine control of movement, utilizing sensory and intentional signals to activate the appropriate motor neurons at an appropriate level of stimulation.

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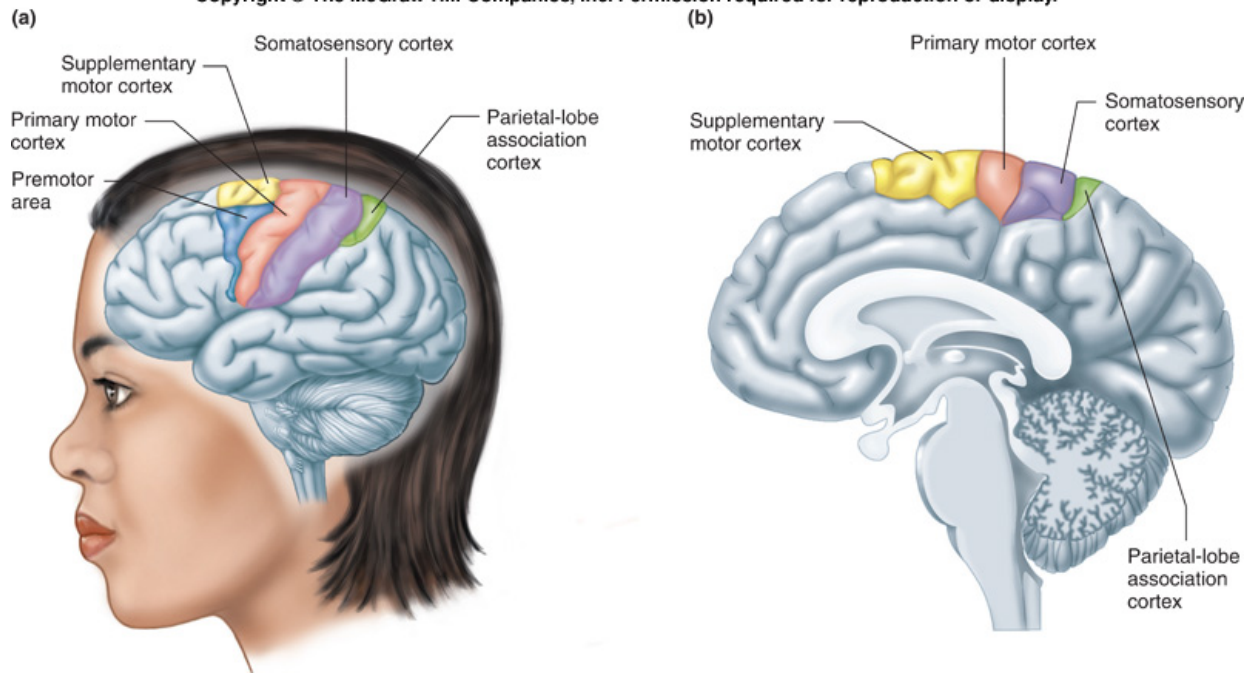

Somatotopic Map

The location and relative size of the cartoon body-shapes represent the location and relative number of motor-related neurons in the cerebral cortex.

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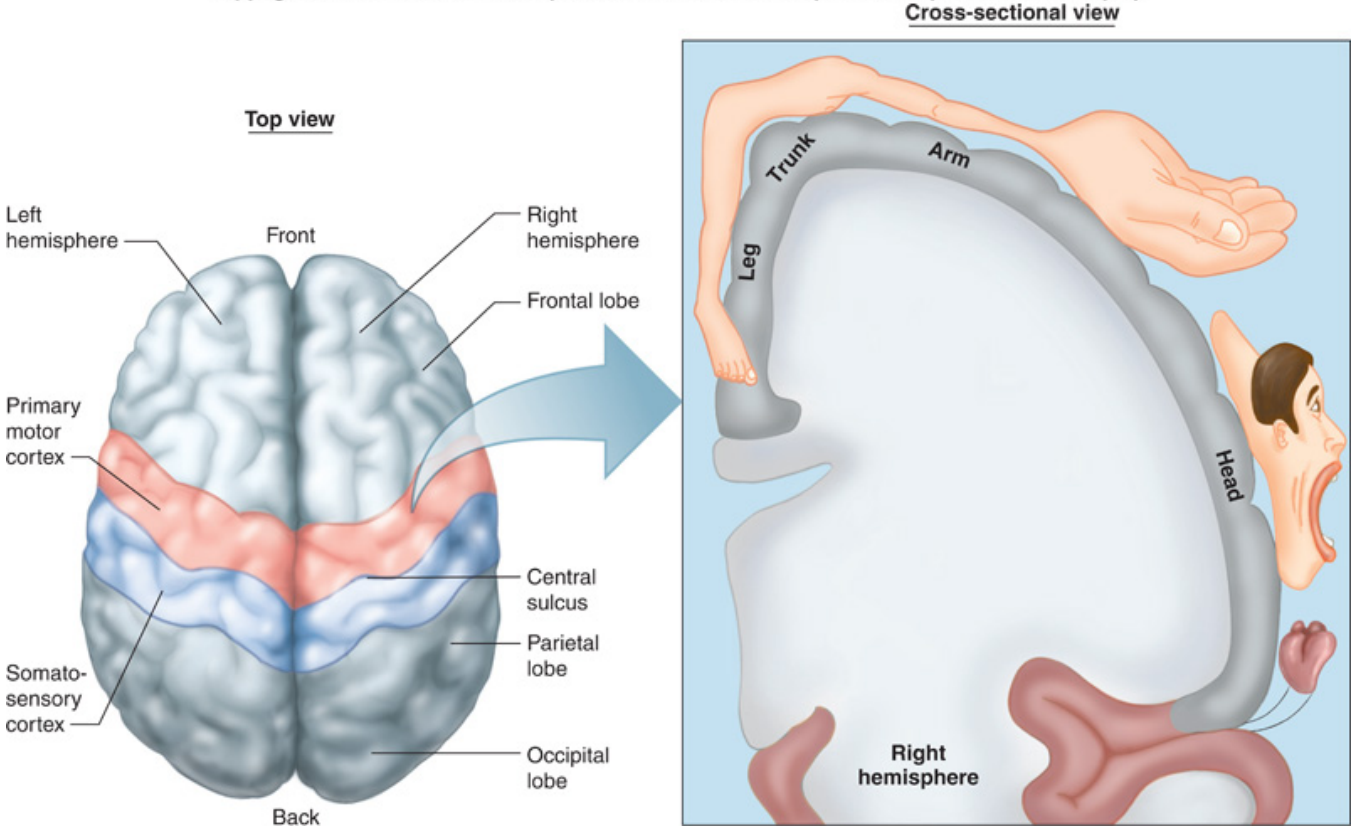
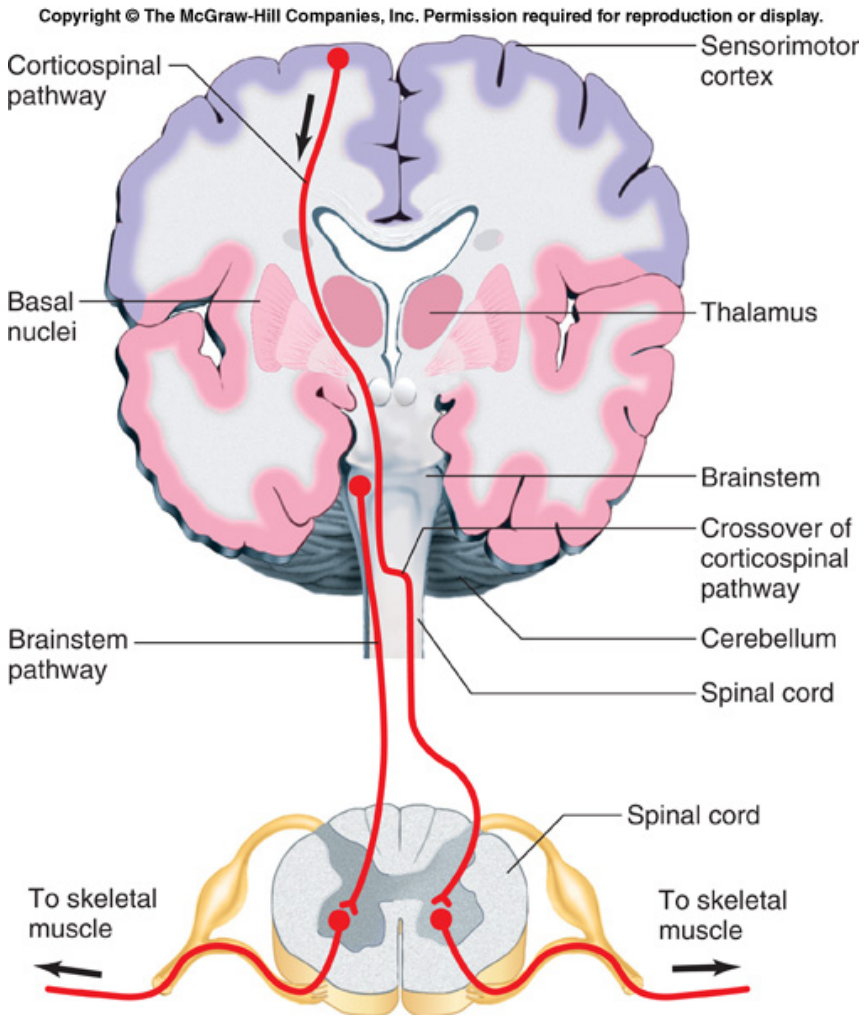


Efferent motor commands from the cerebral cortex are contralateral or "crossed," meaning that the left cortex controls the muscles on the right side of the body (and vice versa), whereas the brainstem influences ipsilateral (same side) motor activity.



Motor activity must be informed about the body's center of gravity in order to make adjustments in the level of stimulation to muscles whose contraction prevents unstable conditions (falling).