Chapter 1: Studying the Nervous System

1. Is the number of genes in an organism’s genome an accurate predictor of the organism’s complexity? Explain.

2. Diagram a neuron and label its components. In what ways are neurons specialized for communication? Do these specializations distinguish neurons from other types of cells?

3. What did Golgi and Cajal disagree about?

4. What are the main types of glial cells, and what is the main function of each? Glia outnumber neurons in the brain, yet neurons tend to be the focus of studies. Why?

5. Diagram the myotatic (knee-jerk) spinal reflex. Show the afferent and efferent neurons and the interneuron (local circuit neuron).

6. Distinguish between the central nervous system (CNS) and the peripheral nervous system (PNS).

7. What technique(s) would you use if you wanted to (a) find the boundaries between layers of the cortex; (b) determine where the axons from nucleus X project; (c) visualize neurons that make protein Y; (d) examine the detailed dendritic structure of neurons in region Z?

Key Terms

- action potential
- afferent neuron
- anterograde tracing
- associational systems
- astrocyte
- autonomic ganglia
- autonomic motor division (system)
- axon
- axon hillock
- brain
- central nervous system (CNS)
- chemical synapse
- cognitive neuroscience
- commissure
- convergence
- cortex
- cranial nerve ganglia

- dendrite
- divergence
- dorsal root ganglia
- efferent neuron
- electrical synapse
- enteric system
- exon
- extracellular recording
- functional brain imaging
- functional magnetic resonance imaging (fMRI)
- ganglion
- gene
- glia
- gray matter
- interneuron
- intracellular recording
- intron
- lesion studies
- local circuit neuron
- magnetic resonance imaging (MRI)
- microglial cell
- motor neuron
- motor system
- myelin
- myotatic spinal reflex
- nerve cell
- nerve
- neural circuit
- neural system
- neuroglia
- neuron
- neuropil
- neurotransmitter molecule
- neurotransmitter receptor
- nucleus
- oligodendrocyte
- parasympathetic division (system)
- peripheral nervous system (PNS)
- positron emission tomography (PET)
- postsynaptic specialization
- presynaptic terminal
- projection neuron
- receptive field
- receptor potential
- retrograde tracing
- Schwann cell
Chapter 2: Electrical Signals of Nerve Cells

1. Draw a basic experimental setup for recording membrane potentials.

2. Draw a recording of a typical action potential. Label the axes and the key features of the action potential. Identify the underlying events for each of the following:
   Rising phase
   Overshoot
   Peak
   Falling phase
   Undershoot

3. Suppose a water-filled aquarium is divided into two compartments by a membrane that is impermeable to all ions. If KCl is added to one compartment, what will happen to the distribution of ions? Is there a potential difference between the two compartments? What will happen to the membrane potential if the membrane suddenly becomes selectively permeable to K⁺ (but not to Cl⁻)? What would happen if you then added NaCl to one compartment only?

4. What is the magnitude of a typical neuron’s resting membrane potential? Why do neurons and other cells have a negative resting membrane potential?

5. What is meant by the statement: “Ion channels and ion pumps have complementary functions?”

6. Explain the difference between action potentials (all-or-none) and synaptic potentials (graded).

7. Distinguish between hyperpolarization and depolarization.

8. What is meant by electrochemical equilibrium?

9. Write out the Nernst equation. Explain how it could be used to determine the equilibrium potential for K⁺. Why is it useful to know the K⁺ equilibrium potential?

10. In what situation would you use the Goldman equation as opposed to the Nernst equation?

11. Suppose you are recording a neuron’s resting membrane potential. If you added KCl to the external medium, what would happen to the resting potential? Compare this to what would happen if you had added the same amount of NaCl. What can you conclude from this comparison?

Key Terms
1. What is the voltage clamp technique? Explain how it allowed Hodgkin and Huxley to determine the contribution of Na\(^+\) and K\(^+\) conductances to the action potential.

2. Does current flow from positive to negative, or negative to positive? Which way does current flow across the membrane during the rising phase of the action potential? During the falling phase?

3. Suppose you are recording action potentials from a neuron. How would the action potential be affected if you remove Na\(^+\) from the external medium? What if you remove external K\(^-\) instead?

4. How does the voltage sensitivity of K\(^-\) conductance contribute to the action potential?

5. Do unmyelinated axons carry action potentials? Draw a diagram to help explain the regenerative property of the action potential, using the concepts of active and passive current flow.

6. What is the purpose of myelin? Explain how myelin speeds the conduction of the action potential.

7. What prevents action potentials from turning around and going back up the axon?

**Key Terms**

activate
cell membrane conductance
depolarization
depolarization electrochemical equilibrium
equilibrium potential
Goldman equation
hyperpolarization
ion channel
Nernst equation
overshoot phase
receptor potential
resting membrane potential
rising phase
synaptic potential
threshold potential
undershoot

Chapter 3: Voltage-Dependent Membrane Permeability

1. What is the voltage clamp technique? Explain how it allowed Hodgkin and Huxley to determine the contribution of Na\(^+\) and K\(^+\) conductances to the action potential.

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**Key Terms**

activate
cell membrane conductance
depolarization
depolarization electrochemical equilibrium
equilibrium potential
Goldman equation
hyperpolarization
ion channel
Nernst equation
overshoot phase
receptor potential
resting membrane potential
rising phase
synaptic potential
threshold potential
undershoot
Chapter 4: Channels and Transporters

1. Why did Hodgkin and Huxley surmise that neuronal membranes must have ion channels? What properties did they think ion channels would have? What properties did they not anticipate?

2. What is patch clamping useful for?

3. What makes the frog oocyte a useful expression system for studying proteins such as ion channels?

4. Compare the responses of voltage-gated Na⁺ and K⁺ channels to depolarization. How would you expect these channel properties to affect the shape, duration and frequency of action potentials?

5. Compare ion channels and active transporters with regard to structure and function.

6. What must all active transporters be able to do? Distinguish between the two classes of active transporters: ATPase pumps and ion exchangers. Give an example of each.

7. What experimental approaches can be used to determine which ions can pass through a particular ion channel?

8. There are nearly 100 genes for K⁺ channels. Why so many? Wouldn’t one or two be enough?

9. List major stimulus types that can gate (open or close) various kinds of ion channels.

10. Describe briefly how each of the following can be used to learn about ion channels:
    - X-ray crystallography
    - Expression of mRNA in *Xenopus* oocyte
    - Patch clamping
    - Mutagenesis
    - Toxins

11. What do Cl⁻, Ca²⁺, N⁺ and K⁺ channels have in common structurally? How are they different?

12. Does the Na⁺/K⁺ pump make a major contribution to a neuron’s resting potential? Explain.

Key Terms

- active transporters
- ATPase pump
- channelopathies
- cyclic nucleotide gated channel
- electrogenic pump
- inactivation
- ion exchanger
- ion selectivity
- ligand-gated ion channel
- macroscopic current
- microscopic current
- mutagenesis
- Na⁺ pump
- ouabain
- pore
- selectivity filter
- voltage sensor
- voltage-gated
- X-ray crystallography
Chapter 5: Synaptic Transmission

1. Compare the pros and cons of electrical versus chemical synapses.

2. If someone challenged the existence of chemical synapses as “only a theory,” how would you reply?

3. What criteria define a neurotransmitter?

4. Are synaptic vesicles delivered to the nerve terminal by slow or fast axonal transport? Explain.

5. List the steps involved in chemical neurotransmission.

6. What is the significance of the quantal nature of MEPPs? What is the evidence that EPPs are composed of MEPPs?

7. What lines of evidence suggest that neurotransmitters are released from synaptic vesicles?

8. Summarize the experimental evidence that synaptic vesicles are recycled in the axon terminal.

9. It has been demonstrated that a rise in presynaptic Ca²⁺ is necessary and sufficient for neurotransmitter release. What experimental evidence supports the claim that Ca²⁺ is necessary? Sufficient?

10. Indicate how each of the following are involved in neurotransmitter secretion:
    - NSF
    - SNAPs
    - SNAREs (synaptobrevin, synaptotagmin, SNAP-25)
    - Synaptotagmin
    - Synapsin
    Which one is key in the regulation of transmitter release by Ca²⁺?

Key Terms

- acetylcholine (ACh)
- auxilin
- botulinum toxin
- Ca²⁺/calmodulin-dependent protein kinase, type II (CaMKII)
- clathrin
- co-transmitter
- dynamin
- end plate
- end plate current (EPC)
- end plate potential (EPP)
- EPSP
- excitatory
- fast axonal transport
- gap junction
- G-protein–coupled receptor
- G-protein
- Hsc-70
- inhibitory
- ionotropic receptor
- IPSP
- large dense-core vesicles
- ligand-gated ion channel
- metabotropic receptor
- miniature end plate potential (MEPP)
- neurotransmitter
- NSF
- postsynaptic
- postsynaptic current (PSC)
- postsynaptic potential (PSP)
- presynaptic
- receptor molecule
- reversal potential
- slow axonal transport
- small clear-core vesicle
- SNAP-25
- SNAPs
- SNAREs
- summation
- synapsin
- synaptic cleft
- synaptic vesicle
- synaptic vesicle cycle
- synaptobrevin
- synaptojanin
- synaptotagmin
- syntaxin
- tetanus toxin
Chapter 6: Neurotransmitters and Their Receptors

1. Compare peptide and classical small-molecule neurotransmitters with regard to synthesis and removal from the synaptic cleft.

2. List the precursor(s), rate-limiting enzyme of synthesis, and mechanism(s) of removal from the synaptic cleft for the following:
   - GABA
   - Glutamate
   - 5-HT
   - ACh
   - DA
   - NE

3. What are the main structural and functional differences between ionotropic and metabotropic receptors?

4. Give examples of neurotransmitters in each of the following categories:
   - Purinergic
   - Biogenic amine
   - Amino acid
   - Peptide

5. Is a serotonin reuptake inhibitor an agonist or an antagonist? Explain.

6. Which neurotransmitter systems are particularly associated with (a) depression, (b) anxiety, (c) pain, and (d) addiction?

7. What are the two major inhibitory neurotransmitters in the brain?

8. What is the glutamate-glutamine cycle?

9. What is excitotoxicity and why is it clinically important?

10. What are the three major types of ionotropic glutamate receptors, and why were they given these names?

11. What features make nitric oxide (NO) such an unusual neurotransmitter?

Key Terms

- adrenaline
- AMPA receptor
- atropine
- biogenic amine
- bicuculline
- α-bungarotoxin
- catecholamine
dopamine
- endocannabinoid
- epinephrine
- glutamate-glutamine cycle
- histamine
- kainate receptor
- muscarine
- muscimol
- myasthenia gravis
- neuropeptide
- nicotine
- NMDA receptor
- noradrenaline
- norepinephrine
- pre-propeptide
- propeptide
- serotonin
- small-molecule neurotransmitter
- strychnine
- vesicular transporter
Chapter 7: Molecular Signaling within Neurons

1. Two major second messenger systems linked to metabotropic neurotransmitter receptors are the cAMP system and the phosphoinositide system. Draw a table comparing the main steps in these second messenger pathways. (Refer to Figure 7.6.)

2. Define the following terms and give examples of each:
   - Cell signaling molecules
   - Receptors
   - Effector proteins
   - Second messengers
   - Later effectors
   - Heterotrimeric G-proteins
   - Transcription factors
   - Immediate early genes

3. Why is it so important to keep Ca\(^{2+}\) levels low inside the cell, and how is this accomplished?

4. Protein kinases and phosphatases are major targets of second messenger systems. Why is it so important to regulate protein phosphorylation?

5. The nervous system is known for its plasticity (modifiability)—its ability to show enduring modifications in response to environmental changes. This typically involves changes in gene expression. Draw a diagram illustrating how neurotransmission can lead to changes in gene expression.

6. What are some potential points of intersection between second messenger systems? With so many points of intersection, how could a neuron keep track of individual signals?

7. How do second messenger systems “turn off” again after they have been turned on?

Key Terms
- adenyl cyclase
- CaMKII
- calbindin
- calcineurin
- calcium pump
- calmodulin
- cell-associated signaling molecule
- cell-impermeant signaling molecule
- cell-permeant signaling molecule
- c-fos
- channel-linked receptor
- CRE
- CREB
- cAMP
- cGMP
- cyclic nucleotide
- DAG
- delayed response gene
- endocrine
- enzyme-linked receptor
- G-protein–coupled receptor
- G-protein
- GTP-binding protein
- guanylyl cyclase
- heterotrimeric G-protein
- immediate early gene
- inositol trisphosphate (IP\(_3\)) receptor
- intracellular receptor
- intracellular signal transduction
- MAP kinase
- monomeric G-protein
- Na\(^+/Ca^{2+}\) exchanger
- nerve growth factor
- NGF
- paracrine
- PIP\(_2\)
- PKC
- promoter
- protein kinase
- protein phosphatase
- ras
- ryanodine receptor
- signal amplification
- signal transduction
- small G-protein
- synaptic transmission
- transcription factor
- transcriptional activator protein
Chapter 8: Synaptic Plasticity

1. What is plasticity?

2. Explain why changes in Ca\(^{2+}\) levels are fundamental in short-term synaptic changes such as facilitation, synaptic depression, augmentation, and posttetanic potentiation. What can these short-term changes contribute to the study of learning and memory?

3. What is the synaptic basis for short-term sensitization in *Aplysia*? What additional changes underlie long-term sensitization?

4. What is long-term potentiation (LTP), and how is it obtained experimentally? Use LTP in CA1 of the hippocampus as an example.

5. What is long-term depression (LTD), and how is it obtained experimentally?

6. Compare cellular mechanisms involved in LTP versus LTD.

7. Is LTP a good model for learning and memory? Include in your answer associativity and input specificity.

8. How were silent synapses discovered? How are silent synapses converted to active excitatory synapses?

9. What is the basis for spike timing-dependent plasticity (STDP)? How does STDP help explain the frequency dependency of LTP and LTD?

10. What might LTP and epilepsy have in common?

**Key Terms**

associativity
augmentation
habitation
long-term depression (LTD)
long-term potentiation (LTP)
posttetanic potentiation (PTP)
sensitization
spike timing-dependent plasticity (STDP)
synaptic depression
synaptic facilitation

Chapter 9: The Somatic Sensory System: Touch and Proprioception

1. What are the relative merits of phasic (rapidly adapting) versus tonic (slowly adapting) receptors?

2. What is proprioception? Name three kinds of proprioceptors.

3. If you were asked to modify the somatic sensory system to improve its two-point discrimination, what changes would you suggest?

4. What is a somatosensory receptive field?

5. Where are the gracile and cuneate nuclei? What is the equivalent of the dorsal column nuclei for somatosensory input from the face?

6. Where is the primary somatic sensory cortex (SI)? Are there differences between the four Brodmann’s areas that comprise SI?

7. What are some afferent and efferent connections of SI?

8. Compare the dorsal column (medial lemniscal) and spinothalamic (anterolateral) pathways with respect to anatomy and sensory modality. For each pathway, where are the cell bodies and axon terminals of the first-, second- and third-order neurons? Where does each pathway decussate? Is the left side of the body represented in the right or left SI cortex?

9. The perceived somatosensory stimulus is really a highly filtered and distorted representation of the actual physical stimulus. Do you agree? Explain.

**Key Terms**

Aβ afferent
Aγ fiber
Brodman’s area 1
Brodman’s area 2
Brodman’s area 3a
Brodman’s area 3b
C fiber
Clark’s nucleus
cranial nerve ganglia
cutaneous mechanoreceptor
dermatome
dorsal column nuclei
dorsal column
dorsal root ganglia
extrafusal fiber
fasciculus cuneatus
fasciculus gracilis
first-order neuron
free nerve ending
Golgi tendon organ
group Ia afferents
group Ib afferents
group II afferents
haptics
homunculus
internal arcuate fiber
internal capsule
intrafusal muscle fiber
joint receptor
mandibular branch
maxillary branch
mechanoreceptor
medial lemniscus
Meissner afferent
Merkel cell afferent
mesencephalic trigeminal nucleus
muscle spindle
nucleus cuneatus
nucleus gracilis
ophthalmic branch
Pacinian afferents
parallel pathway
postcentral gyrus
posterior funiculi
primary somatic sensory cortex
primary somatosensory cortex (SI)
principal nucleus
proprioceptors
pseudo-unipolar
rapidly adapting afferent
receptive field
receptor (or generator) potential
Ruffini afferent
secondary somatosensory cortex (SII)
second-order neuron
sensory transduction
slowly adapting afferent
somatotopic map
somatotopy
spinal nucleus
stereognosis
subcutaneous mechanoreceptor
third-order neuron
trigeminal (cranial nerve V) ganglion
trigeminal brainstem complex
trigeminal lemniscus
trigeminal nerve
trigeminothalamic tract
two-point discrimination
ventral posterior complex
ventral posterior lateral (VPL) nucleus
ventral posterior medial (VPM) nucleus
\( \gamma \) motor neuron
Chapter 10: Pain

1. What is the evidence that nociception is mediated by specific nociceptors rather than by strongly stimulated tactile receptors or warm receptors?

2. Are hyperalgesia and allodynia beneficial? Summarize the contributions of peripheral and central sensitization.

3. Due to a spinal injury, a patient has lost pain and temperature sensation on the left half of his body from the waist down. Where is his injury? Where would you expect loss of tactile sensation in this patient?

4. Give an example of referred pain and offer a possible explanation.

5. What is the recently discovered major pathway for visceral pain?

6. Is phantom limb pain less “real” than pain from an intact limb?

7. Why do chili peppers seem hot?

8. The placebo effect on pain can be blocked by naloxone. What does this observation reveal about the pain system?

9. Are there any practical applications of the gate control theory of pain?


11. What are the three major groups of endogenous opioids?

12. Why, do you think, is severe chronic pain not eliminated by ablating the primary somatosensory cortex?

Key Terms

- affective–motivational
- allodynia
- anterolateral system
- Aδ and C fiber nociceptor
- Aδ mechanosensitive nociceptor
- Aδ mechanothermal nociceptor
- central sensitization
- dissociated sensory loss
- dorsolateral tract of Lissauer
- dynorphin
- endogenous opioid
- endorphin
- enkephalin
- exteroception
- first pain
- gate theory of pain
- hyperalgesia
- interoception
- neuropathic pain
- nociceptor
- peripheral sensitization
- polymodal nociceptor
- second pain
- sensory discriminative
- spinothalamic tract
- TRP channel
- vanilloid receptor
Chapter 11: Vision: The Eye

1. Why do you have both rods and cones instead of just one type of photoreceptor?

2. Do you have more rods or cones in your retina? In your fovea? What accounts for the fact that your rods do not contribute to vision in daylight?

3. Draw a simplified diagram of the retina and label the five types of retinal neurons. Which layer is the outer nuclear layer?

4. Is the retina part of the central nervous system? Explain.

5. Photoreceptors are atypical in that they are depolarized (~40 mV) in darkness and are hyperpolarized by light stimuli. What components of photoreceptors account for this?

6. What is the evidence that color vision is trichromatic?

7. What observations led Kuffler to define two types of retinal ganglion cells, off-center and on-center? Explain how this receptive field organization is useful in detecting luminance contrast and changes in light intensity.

8. List the key steps in phototransduction in a rod, from absorption of a photon to decreased cGMP concentration and closure of ion channels.

9. Why is light adaptation in the retina so important, and what does it involve?

10. What is the role of horizontal cells?

Key Terms

accommodation
age-related macular degeneration (AMD)
amacrine cell
ametropia
anterior chamber
aqueous humor
arrestin
bipolar cell
cataract
choroid
ciliary body
ciliary muscle
cones
cornea
cyclic guanosine monophosphate
dichromacy
diabetic retinopathy
dioptrometry
fovea
foveola
fundus
ganglion cell
glaucoma
horizontal cell
hyperopia
interphotoreceptor retinoid binding protein
IRBP
iris
lens
light adaptation
limbus
luminance
luminance contrast
macula lutea
macular degeneration
mesopic vision
myopia
off-center ganglion cell
on-center ganglion cell
opsin
optic disc
optic nerve
optic papilla
outer segment
pigment epithelium
photopic vision
photoreceptor
phototransduction
plexiform layer
posterior chamber
presbyopia
pupil
retina
retinal
retinal pigment epithelium
retinitis pigmentosa
retinoid cycle
rhodopsin
rhodopsin kinase
rods
sclera
Chapter 12: Central Visual Pathways

1. What percentage of the axons in your optic nerve crosses at the optic chiasm?

2. Draw a sketch of the primary visual pathway.

3. The retina sends information to the dorsal lateral geniculate nucleus (dLGN) for pattern vision. Name three other targets of retinal ganglion cells and indicate what each pathway is specialized for.

4. If your right visual cortex stopped functioning, what part of your visual field would be lost?

5. What part of the retina has the largest proportional representation?

6. Is the world mapped upside down on the retina? On V1?

7. Explain how Hubel and Wiesel mapped visual receptive fields. How do receptive field characteristics of neurons in V1 compare with those in the dLGN?

8. Are binocular neurons found in the LGN? In layer IV of primary visual cortex? Where does input from both eyes first converge?

9. What are ocular dominance columns and orientation columns?

10. What lines of evidence suggest that the magnocellular and parvocellular streams are two parallel anatomical pathways with functionally distinct characteristics?

Key Terms

anopsia
autostereogram
binocular field
bitemporal hemianopsia
blind spot
Brodman’s area 17
cerebral achromatopsia
cerebral achromatopsia
cerebral akinetopsia
dorsal lateral geniculate nucleus
Edinger-Westphal nucleus
far cell
heteronomous hemianopsia
homonymous hemianopsia
Chapter 13: The Auditory System

1. What perceptual qualities are based on frequency and amplitude of sound waves?

2. What is the audible frequency range of humans (in Hertz)? What is the approximate range for human speech sounds?

3. Describe the tonotopy of the basilar membrane.

4. List the steps in stimulus transduction, from the physical sound stimulus to the electrical signals of inner hair cells. Indicate which steps take place in the external, middle, and inner ear.

5. There are more outer hair cells than inner hair cells. What do the outer hair cells do?

6. Draw a tuning curve for a neuron in the auditory system, showing the neuron’s characteristic frequency. Explain how this curve would be determined experimentally.

7. What two strategies does the auditory system use to code sound frequency?

8. Where does the auditory nerve project?

9. Compare the strategies for sound localization used by neurons in the MSO versus LSO/MNTB.

10. Briefly compare the functions of the inferior colliculus, medial geniculate complex, and primary auditory cortex. What kinds of experimental approaches are used to elucidate these functions?

11. The primary visual and somatosensory cortices have a topographic map of sensory space. Does the primary auditory cortex have a map of auditory space?

Key Terms

amplitude
auditory meatus
auditory nerve fiber
auditory space map
basilar membrane
belt area
characteristic frequency
cochlea
coincidence detector
concha
Chapter 14: The Vestibular System

1. In what ways are the vestibular and auditory sense organs similar?

2. Explain how the semicircular canals are specialized to assess rotational acceleration of the head, while the otolith organs are specialized to detect linear acceleration and static position of the head relative to the gravitational axis.

3. As your head turns horizontally to the left, what happens to activity levels in your left and right vestibular nerve? The imbalance in neural activity would lead to physiological nystagmus, with the slow component in which direction? What purpose would these eye movements serve? A similar imbalance of activity would be obtained by irrigating your left ear with _______ (warm or cold?) water.

4. If you lost your vestibulo-ocular reflex (VOR), what symptoms would you experience?

5. Which cranial nerve serves both vestibular and auditory hair cells? Where does the vestibular nerve project?

6. What purposes do the vestibulo-cervical and vestibulo-spinal reflexes serve?

7. How does vestibular information reach the cortex?

Key Terms
ampulla
crista
cupula
kinocilium
labyrinth
macula
medial longitudinal fasciculus
ocellus
otoconia
otolith organ
otolithic membrane
saccule
Scarpa’s ganglion
semicircular canal
stereocilia
striola
utricle
vestibular nerve ganglion
vestibular nuclei
vestibulo-ocular reflex (VOR)
Chapter 16: Lower Motor Neuron Circuits and Motor Control

1. Why are lower motor neurons referred to as the “final common path” for movements? Are the motor neurons that innervate head muscles upper or lower motor neurons?

2. List the three sources of direct synaptic input to \( \alpha \) motor neurons. Which is the major input?

3. Do neurons in the cerebellum and basal ganglia synapse on \( \alpha \) motor neurons?

4. As you try to lift a heavy box, which type of motor unit do you recruit first and which do you recruit last, according to the size principle?

5. What prevents muscle spindles from being useless when their muscle contracts?

6. Diagram the muscle stretch reflex. Explain how the antagonist muscle is inhibited via reciprocal innervation. What is the stretch reflex good for in everyday life?

7. Diagram the clasp-knife reflex. Explain how it works and what it is good for.

8. What happens to activity levels in muscle spindle afferents versus Golgi tendon organ afferents when a muscle contracts? When a muscle is passively stretched? Is this consistent with the proposal that muscle spindle and Golgi tendon organ feedback systems monitor and maintain muscle length and force, respectively?

9. What are central pattern generators in the spinal cord, and what would life be like without them?

10. As you begin a weight-lifting program, what changes can you expect in your muscles at the level of motor units?

Key Terms

- amyotrophic lateral sclerosis (ALS)
- basal ganglia
- central pattern generator
- cerebellum
- crossed extension reflex
- extrafusal muscle fiber
- fast fatigable (FF) motor unit
- fast fatigue-resistant (FR) motor unit
- flexion reflex
- Golgi tendon organ
- intrafusal muscle fiber
- local circuit neuron
- lower motor neuron
- motor neuron pool
- motor unit
- muscle spindle
- muscle tone
- premotor cortex
- primary motor cortex
- size principle
- slow (S) motor unit
- stretch reflex
- upper motor neuron
- \( \alpha \) motor neuron
- \( \gamma \) motor neuron
Chapter 17: Upper Motor Neuron Control of the Brainstem and Spinal Cord

1. Medial versus lateral spinal cord interneurons differ in location and pattern of connections. Describe how these anatomical distinctions correspond to functional differences.

2. The major subcortical sources of upper motor neurons are the vestibular nuclei, superior colliculus, red nucleus, and reticular formation. For each region, (a) briefly describe its role in motor control; (b) indicate whether it is part of the midbrain, pons, and/or medulla; and (c) name the pathway that projects from each of these regions to the spinal cord.

3. The primary motor cortex “controls movements, not individual muscles.” What does this mean, and what evidence supports it?

4. Contrast the roles of the primary motor cortex and premotor cortex. What types of experimental evidence support your answer?

5. How would mirror motor neurons help you learn new skills in a gymnastics class?

6. Give an example of feedforward and feedback in postural control.

7. Does the pyramidal tract originate only in the motor cortex? Does it terminate only in the spinal cord?

8. Compare the uncrossed ventral corticospinal tract with the crossed lateral corticospinal tract with regard to origin, location, and function.

9. What is the head’s equivalent of the body’s corticospinal tract?

10. How is it that the motor cortex can direct precise movements even though single neurons are broadly tuned?

11. What is the basis for muscle tone?

12. What is spinal shock?

Key Terms

- anterior corticospinal tract
- Broca’s area
- colliculospinal tract
- corticobulbar tract
Chapter 18: Modulation of Movement by the Basal Ganglia

1. What structures are included in the basal ganglia? List the main receiving areas and output areas of the basal ganglia.

2. Summarize the role of the basal ganglia in movement.

3. What is the corpus striatum?

4. What is largest source of neural input to the basal ganglia?

5. Would you expect your putamen neurons to fire as you are reaching for a doughnut, or in anticipation of your reach? Would their firing correspond better with the position of the doughnut or with the starting position of your arm? What does this suggest about the role of the putamen?

6. Parallel loops involving the basal ganglia each handle information from different cortical areas. Diagram the motor loop, indicating whether each of the pathways is excitatory or inhibitory. What are the other loops?

7. What is disinhibition? Briefly describe how inhibition and disinhibition operate in the control of saccades.

8. In Huntington’s disease, which neurons in the basal ganglia are the main ones that degenerate? Explain in terms of basal ganglia circuitry how this would lead to hyperkinetic symptoms.

9. In Parkinson’s disease, which neurons degenerate? Explain the hypokinetic symptoms of this disease in terms of the circuitry of the basal ganglia. What kinds of treatments have been used to alleviate Parkinson’s symptoms?

Key Terms
caudate nucleus
corpus striatum
corticostriatal pathway
external segment
globus pallidus
hemiballismus
internal segment
medium spiny neurons
pallidum
Parkinson’s disease
putamen
spiny neurons
substantia nigra pars compacta
substantia nigra pars reticulata
subthalamic nucleus
trinucleotide repeats
ventral anterior nuclei
ventral lateral nuclei
Chapter 19: Modulation of Movement by the Cerebellum

1. What are the functional differences between the cerebrocerebellum, vestibulocerebellum, and spinocerebellum?

2. Name the three cerebellar peduncles. Which contain cerebellar afferents and which contain efferents?

3. What is the largest source of input to the cerebellum?

4. What is meant by "fractured" somatotopy in the cerebellar cortex?

5. What kinds of sensory information do you think the cerebellum might need in order to compare intended movements with actual movements? How does the cerebellum get its sensory input?

6. Diagram the basic circuit of the cerebellum, showing a Purkinje cell, granule cell, parallel fiber, mossy fiber, climbing fiber, and a neuron in a deep cerebellar nucleus. Label the three layers of the cerebellar cortex.

7. What type of neurons carry the output of the cerebellar cortex? Is it true that all of the output of the cerebellar cortex is inhibitory? Do you think this is inconsistent with the complex tasks of the cerebellum?

8. What does cerebellar ataxia reveal about normal functioning of the cerebellum?

9. Neither the cerebellum nor the basal ganglia project directly to the spinal cord. How then does their activity influence motor neurons? Briefly compare their roles.

Key Terms

- action tremor
- basket cells
- brachium conjunctivum
- brachium pontis
- cerebellar ataxia
- cerebellar hemispheres
- cerebellar peduncles
- cerebrocerebellum
- climbing fibers
- Creutzfeldt-Jakob disease (CJD)
- cuneate nucleus
- decussation of the superior cerebellar peduncle
- dentate nucleus
- dorsal nucleus of Clarke
- dysdiadochokinesia
- dysmetria
- external nucleus
- fastigial nucleus
- flocculus
- folia
- Golgi cells
- granule cells
- inferior cerebellar peduncle
- inferior olive
- intention tremors
- interposed nuclei
- mesencephalic nucleus
- middle cerebellar peduncle
- molecular layer
- mossy fibers
- nodulus
- nystagmus
- parallel fibers
- pontine nuclei
- prions
- Purkinje cell
- red nucleus
- reeler mutant
- restiform body
- spinocerebellum
- stellate cell
- superior cerebellar peduncle
- transverse pontine fibers
- vermis
- vestibulocerebellum
- vestibulo-ocular reflex
- weaver mutant
Chapter 24: Modification of Brain Circuits as a Result of Experience

1. What is a critical period? Give several examples. How would you test for the presence of a critical period? What developmental changes might be responsible for the end of a critical period?

2. How might Hebb’s postulate explain the formation of ocular dominance columns? How does the three-eyed frog research support the involvement of Hebb synapses?

3. Monocular deprivation during a critical period affects the development of ocular dominance columns. Describe the effects on anatomy, physiology, and perception. What experimental methods were used to make these observations? What would you conclude from the observation that ocular dominance is less affected by binocular than monocular deprivation?

4. How does strabismus during the critical period affect the development of ocular dominance in the visual cortex? What are the consequences for visual perception?

5. What are the clinical implications of Hubel and Wiesel’s research on the effects of monocular deprivation and strabismus?

6. Describe the parallels between the learning of speech in humans and courtship songs in birds.

7. Explain how Ca\(^{2+}\) could be involved in Hebbian strengthening of synapses.

Key Terms

amblyopia
competitive interaction
CREB
critical periods
esotropia
ethology
exotropia
imprinting
Konrad Lorenz
ocular dominance columns
strabismus

Chapter 26: The Association Cortices

1. Describe the basic organizational features of neocortex, shared by association cortices and sensory and motor cortices.

2. What features distinguish association cortices from sensory and motor cortices? Consider thalamic input and corticocortical connections.

3. On what basis did Brodmann decide where to put the boundaries between Brodmann’s areas?

4. What are the main function(s) of (a) parietal association cortex, (b) temporal association cortex, and (c) frontal association cortex? What techniques and approaches have been used to reveal these functions?

5. What does the study of agnosias contribute to cognitive neuroscience?

6. What does contralateral neglect syndrome suggest about the neuroanatomy of attention? Why does contralateral neglect result from damage to the right, but not left, parietal lobe cortex?

7. Where and what are “recognition neurons,” “planning neurons,” and “attention neurons”?

8. What cortical region is particularly critical for the delayed response task?


Key Terms

agnosias
apraxia
association cortices
cognition
contralateral neglect syndrome
corticocortical connections
cytoarchitectonic areas
delayed response task
interhemispheric connections
medial dorsal nuclei
neocortex
prosopagnosia
pulvinar
Chapter 27: Speech and Language


2. Compare the language functions of the right and left hemispheres. What techniques have been used to investigate cerebral lateralization (hemispheric specialization)?

3. Where is Broca’s area? Wernicke’s area? Compare Wernicke’s aphasia and Broca’s aphasia. What can the variety of aphasias tell us about the neural basis of language?

4. What similarities between sign language and spoken language suggest that they have common neural substrates?

5. Is hemispheric specialization in language functions unique to humans? Discuss.

6. What is the relationship between handedness, lateralization of language, and anatomical hemispheric asymmetry?

7. What evidence suggests the importance of biological constraints or predispositions in language learning?

8. If a split-brain patient is briefly shown a pencil in her left visual field, will she be able to describe the pencil? Which hand would she use to select the pencil by feel from a set of test objects? Explain with the aid of a diagram.

Key Terms
aphasias
aprosodias
Broca’s aphasia
Broca’s area
conduction aphasia
expressive aphasia
grammar
motor aphasia
phoneme
planum temporale
prosodic elements
receptive aphasia
sensory aphasia
split-brain patients
syntax
vocal folds
Wernicke’s aphasia
Wernicke’s area

Chapter 31: Memory

1. Distinguish between declarative memory and nondeclarative (procedural) memory. Give examples of each.

2. What lines of evidence support the proposal that declarative memory and procedural memory involve different brain mechanisms?

3. What evidence shows that short-term and long-term memory involve different brain mechanisms?

4. Would you want to have a perfect memory? Consider the advantages of forgetting.

5. What has patient H.M. taught us about human memory? In addition to studying people with brain damage, what approaches could be used to assess hippocampal involvement in consolidation?

6. Where would you expect to find engrams (stored representations of memories) for (a) declarative and (b) procedural memories? What types of evidence support your answer?

7. What are the brain and behavioral symptoms of Alzheimer’s disease? What is known about the causes of Alzheimer’s disease?

8. Based on what you know about human memory, what advice can you offer to students studying for final exams?

Key Terms
amnesia
anterograde amnesia
consolidation
declarative memory
engram
immediate memory
Korsakoff’s syndrome
long-term memory
nondeclarative memory
priming
retrograde amnesia
savant syndrome
working memory