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# Neuroscience

4th edition

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- Targets what you really need to know
- Student-tested and reviewed

Allan Siegel ■ Heidi Siegel


# Neuroscience

PreTest® Self-Assessment and Review

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# Neuroscience

## PreTest® Self-Assessment and Review
## Fourth Edition

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0-07-138978-4

The material in this eBook also appears in the print version of this title: 0-07-137350-0.



DOI: 10.1036/0071389784

To Carla, wife and mother, whose patience, support, and understanding made this book possible and to David Eliahu, Tzipporah Hannah, Matan Dov, Nadav David, and Adi Hila.

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# Contents

# Preface

The study of the neurosciences has undergone remarkable growth over the past two decades. To a large extent, such advancements have been made possible through the development of new methodologies, especially in the fields of neuropharmacology, molecular biology, and neuroanatomy. Neuroscience courses presented in medical schools and related schools of health professions generally are unable to cover all the material that has evolved in recent years. For this reason, *Neuroscience: PreTest® Self-Assessment and Review* was written for medical students preparing for licensing examinations as well as for undergraduate students in the health professions.

The subject matter of this book is mainly anatomy and physiology of the nervous system. Also, an attempt was made to encompass the subjects of molecular and biophysical properties of membranes, neuropharmacology, and higher functions of the nervous system. Moreover, clinical correlations for each part of the central nervous system, often using MRI and CT scans, are presented. Although it is virtually impossible to cover all aspects of neuroscience, the objective of this book is to include its most significant components as we currently understand them.

The authors wish to express their gratitude to Leo Wolansky, M.D., and Alan Zimmer, M.D., of blessed memory, for providing the MRI and CT scans.

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# Introduction

Each *PreTest® Self-Assessment and Review* allows medical students to comprehensively and conveniently assess and review their knowledge of a particular basic science, in this instance neuroscience. The 500 questions parallel the format and degree of difficulty of the questions found in the United States Medical Licensing Examination (USMLE) Step 1. Practicing physicians who want to hone their skills before USMLE Step 3 or recertification may find this to be a good beginning in their review process.

Each question is accompanied by an answer, a paragraph explanation, and a specific page reference to an appropriate textbook or journal article. A bibliography listing the sources can be found following the last chapter of this text.

An effective way to use this PreTest® is to allow yourself one minute to answer each question in a given chapter. As you proceed, indicate your answer beside each question. By following this suggestion, you approximate the time limits imposed by the step.

After you finish going through the questions in the section, spend as much time as you need verifying your answers and carefully reading the explanations provided. Pay special attention to the explanations for the questions you answered incorrectly, but read every explanation. The authors of this material have designed the explanations to reinforce and supplement the information tested by the questions. If you feel you need further information about the material covered, consult and study the references indicated.

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# Neuroscience

PreTest® Self-Assessment and Review

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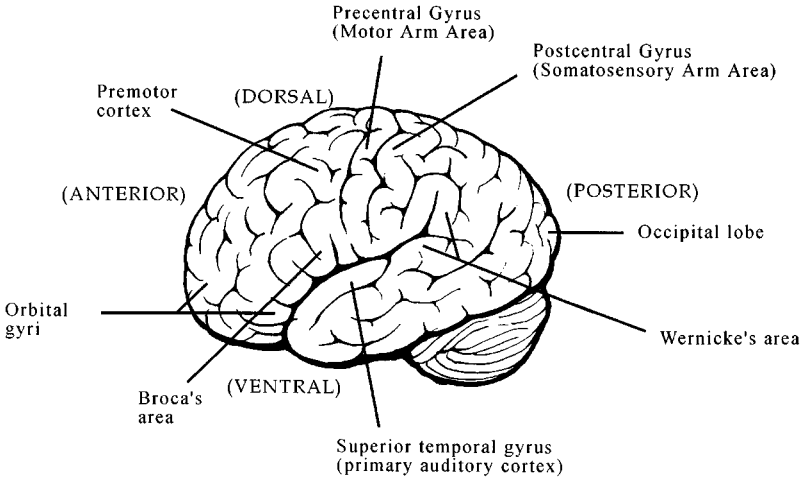
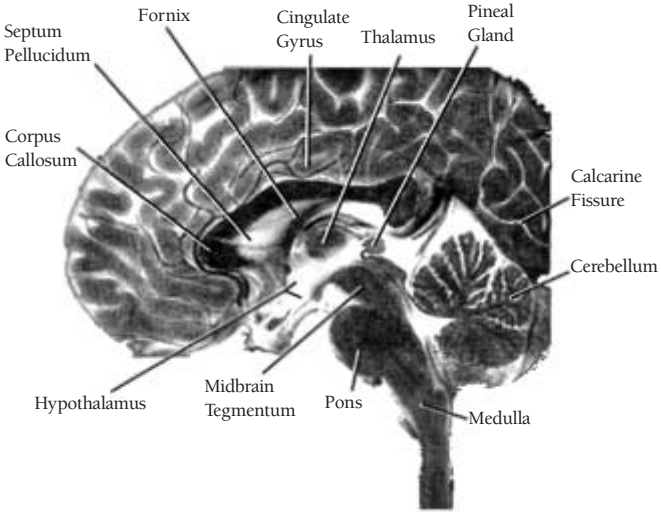
# High-Yield Facts

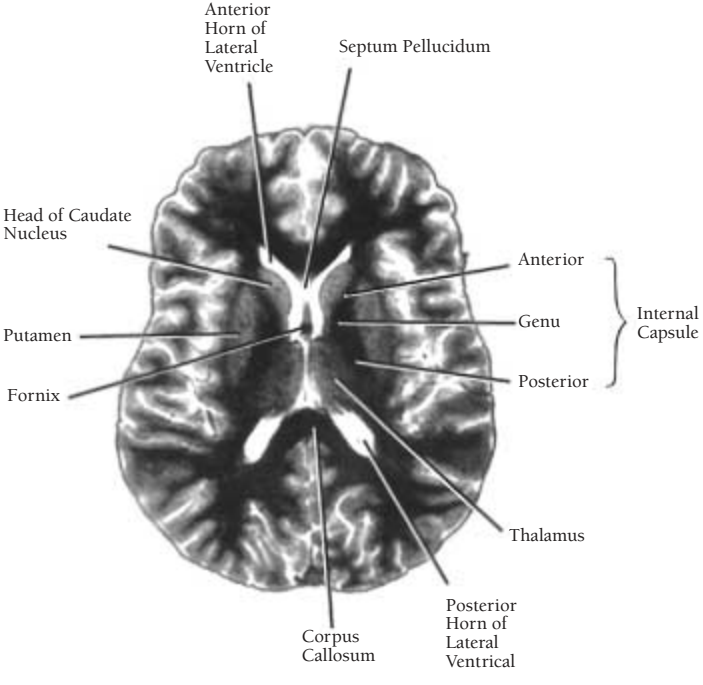
## GROSS ANATOMY OF THE BRAIN

1. Lateral view of the brain. The loci of key motor and sensory structures of the cerebral cortex are indicated in this figure. Anatomical definitions: anterior—toward the front (rostral end) of the forebrain; posterior—toward the back (caudal end) of the forebrain; dorsal—toward the superior surface of the forebrain; ventral—toward the inferior surface of the forebrain. Note that with respect to the brainstem and spinal cord, the terms *anterior* and *ventral* are synonymous; likewise, *posterior* and *dorsal* are also synonymous. Here, the term *rostral* means toward the midbrain, and the term *caudal* means toward the sacral aspect of spinal cord.
2. Midsagittal view of the brain. Magnetic resonance image: T2-weighted, high-resolution, fast spin echo image.
3. Horizontal (transaxial) view of the brain. Magnetic resonance image: Fast inversion recovery for myelin suppression image.
4. Frontal view of the brain. Magnetic resonance image: Fast inversion recovery for myelin suppression image.






*(Courtesy of Leo J. Wolansky, M.D.)*

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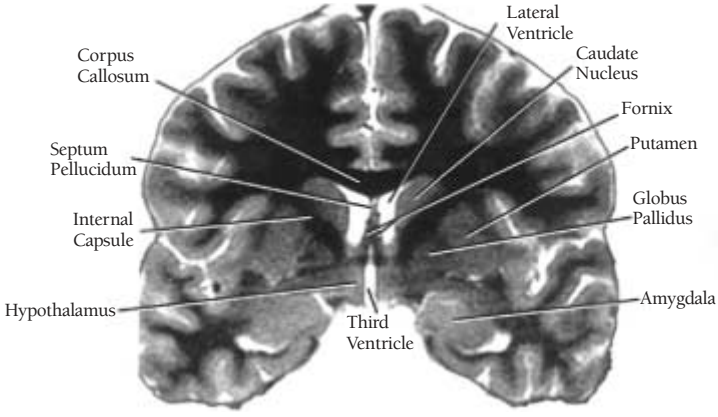



*(Courtesy of Leo J. Wolansky, M.D.)*

## DEVELOPMENT

5. The sulcus limitans divides the alar plate, from which sensory regions of the spinal cord and brainstem are formed, and a basal plate, from which motor regions of the spinal cord and brainstem are formed.

## THE NEURON

6. The neuron consists of a cell body, dendrites (which extend from the cell body), and an axon. Activation of sodium channels is associated with membrane depolarization, while activation of potassium and chloride channels is associated with membrane hyperpolarization. After information is received from a presynaptic neuron, depolarization occurs in the postsynaptic neuron; then, the action potential is initiated and propagated down the axon from the initial segment.
7. Myelin formation is produced in the peripheral nervous system by numerous Schwann cells, while a similar function in the central nervous system is carried out by an oligodendrocyte, which can wrap itself around numbers of neurons. Myelination in the nervous system allows for rapid conduction of action potentials by a process of saltatory conduction, in which the signals skip along openings in the myelin called *nodes of Ranvier.* Neurons that are myelinated (e.g., the pyramidal tracts and dorsal column-medial lemniscal system) are rapidly conducting, whereas those that are poorly or nonmyelinated (e.g., certain pain-afferent fibers to the spinal cord) are slowly conducting. Damage to such myelinated neurons typically disrupts the transmission of neural signals and is frequently seen in autoimmune diseases such as multiple sclerosis, in which sensory and motor functions are severely compromised.

## THE SYNAPSE AND NEUROTRANSMITTERS

8. The binding of the neurotransmitter to the receptor molecule is determined by the postsynaptic receptor, which serves a gating function for particular ions. The receptor is responsible for opening or closing ligand-gated channels, which are regulated by noncovalent binding of compounds such as neurotransmitters. The neurotransmitter, which is contained in presynaptic vesicles and released onto the postsynaptic terminal, causes activation of the receptor, which in turn, produces postsynaptic potentials.
9. The sequence of events in synaptic transmission is: transmitter synthesis → release of transmitter into synaptic cleft → binding of transmitter to postsynaptic receptor → removal of transmitter.
10. Major excitatory transmitters include: Substance P, acetylcholine, and excitatory amino acids; major inhibitory transmitters include: GABA, enkephalin, and glycine. Disruption of neurotransmitter function can lead to different diseases of the nervous system. One such example involves the role of acetylcholine at the neuromuscular junction. When antibodies are formed against the acetylcholine receptor at the neuromuscular junction, transmission is disrupted and the autoimmune disease called *myasthenia gravis* occurs. This disorder includes symptoms such as weakness and fatigue of the muscles.

## SPINAL CORD

11. Major ascending tracts of the spinal cord and their functions include:

    *Dorsal columns.* Mediates conscious proprioception, two-point discrimination, and some tactile sensation ipsilaterally to the dorsal column nuclei and then contralaterally from the dorsal column nuclei to the postcentral gyrus from the VPL of the thalamus.

    *Lateral spinothalamic tract.* Mediates pain and temperature inputs contralaterally to the VPL and posterior complex of the thalamic nuclei and then to the postcentral gyrus.

    *Anterior spinothalamic tract.* Mediates tactile impulses contralaterally to the VPL and then to the postcentral gyrus.

    *Posterior spinocerebellar tract.* Mediates unconscious proprioception from muscle spindles and Golgi tendon organs of the lower limbs through the inferior cerebellar peduncle ipsilaterally to the anterior lobe of the cerebellar cortex.

    *Cuneocerebellar tract.* Mediates unconscious proprioception from muscle spindles and Golgi tendon organs of the upper limbs from the accessory cuneate nucleus through the inferior cerebellar peduncle to the anterior lobe of the cerebellar cortex.

*Anterior spinocerebellar tract.* Mediates unconscious proprioception from the Golgi tendon organs of the lower limbs bilaterally to the anterior lobe of the cerebellar cortex. This tract initially crosses in the spinal cord and then crosses again through the superior cerebellar peduncle.

Major descending tracts of the spinal cord and their functions include:

*Lateral corticospinal tract.* Mediates voluntary control of motor functions from the contralateral cerebral cortex to all levels of the spinal cord.

*Rubrospinal tracts.* Mediates descending excitation of flexor motor neurons at both the cervical and lumbar levels of the contralateral spinal cord.

*Reticulospinal tracts.* The lateral reticulospinal tract arises from the medulla and descends bilaterally to the cervical and lumbar levels of the spinal cord, mediating inhibition upon the spinal reflexes, mainly of extensors; the medial reticulospinal tract arises from the pons and descends mainly ipsilaterally to the cervical and lumbar levels of the spinal cord and facilitates extensor reflexes.

*Vestibulospinal tracts.* The lateral vestibulospinal tract arises from the lateral vestibular nucleus and descends ipsilaterally to the cervical and lumbar levels of the spinal cord, mediating powerful excitation of the extensor motor neurons; the medial vestibulospinal tract arises from the medial vestibular nucleus and descends mainly to the cervical levels of the spinal cord, mediating postural reflexes of the head and neck.

12. Major disorders of the spinal cord include:

*Brown-Séquard's syndrome.* Hemisection of the spinal cord often due to a bullet or knife wound—contralateral loss of pain and temperature below the level of the lesion; bilateral segmental loss of pain and temperature at the level of the lesion; ipsilateral loss of conscious proprioception below the level of the lesion; ipsilateral upper motor neuron paralysis below the level of the lesion; ipsilateral lower motor neuron paralysis at the level of the lesion.

*Tabes dorsalis.* Damage to the dorsal root ganglion and dorsal columns resulting from syphilis—ipsilateral loss of conscious proprioception and tendon reflexes.

*Amyotrophic lateral sclerosis (ALS).* A disease whose etiology is not yet known that destroys both corticospinal fibers and ventral horn cells, causing abnormal reflexes, muscle weakness, atrophy, and ultimately death.

*Syringomyelia.* Caused by abnormal closure of the central canal during development, by trauma, or by a tumor, the result of which is an enlargement of the central canal, causing a segmental bilateral loss of pain and temperature due to damage to the decussating spinothalamic fibers.

*Combined systems disease.* Results from pernicious anemia associated with a deficiency in vitamin $B_{12}$; there is degeneration of both the dorsal columns and corticospinal tracts, resulting in a loss of conscious proprioception, position sense, upper motor neuron symptoms, and muscle weakness.

## AUTONOMIC NERVOUS SYSTEM

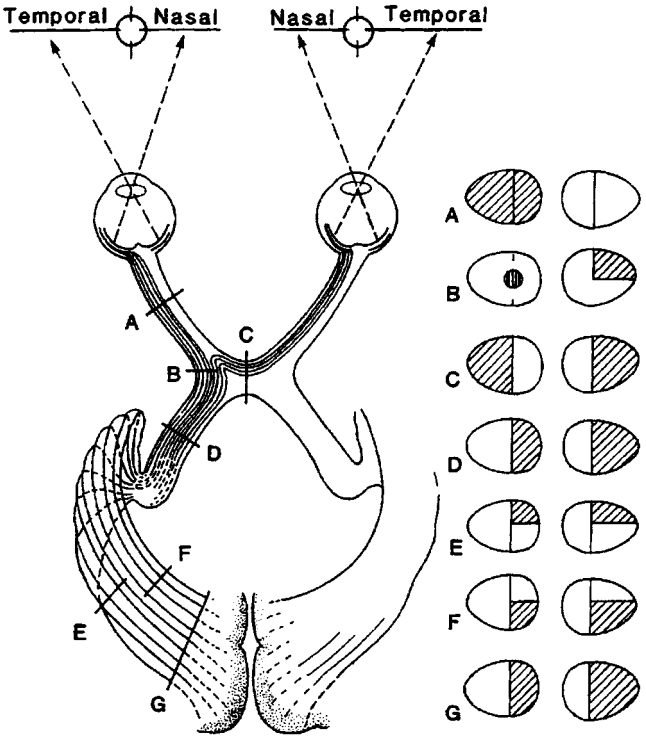
13. The sympathetic nervous system arises from the thoracic and lumbar cords ($T_1$–$L_2$), and the parasympathetic nervous system arises from $S_2$–$S_4$ and cranial nerves III, VII, IX, and X. All preganglionic neurons are cholinergic as well as parasympathetic postganglionic neurons. In addition, sympathetic postganglionic innervation of sweat glands and blood vessels in skeletal muscle is also cholinergic. Most other postganglionic sympathetic endings are adrenergic. Examples of functions of the sympathetic nervous system include: pupillary dilation, acceleration of heart rate, constriction of blood vessels of the trunk and extremities, and inhibition of gastric motility. Examples of functions of the parasympathetic nervous system include: pupillary constriction, decrease in heart rate, secretion of the salivary and lacrimal glands, and stimulation of gastric motility.

## THE BRAINSTEM AND CRANIAL NERVES

14. *Lateral medullary syndrome (Wallenberg's syndrome).* Lesions of the lateral aspect of the lower half of the brainstem, due to occlusion of the inferior cerebellar arteries, produce loss of pain and temperature on the same side of the face and opposite side of the body, as well as Horner's syndrome (i.e., myosis, ptosis, and decreased sweating on one side of the face due to disruption of the sympathetic supply to the orbit and pupil, or, as applies in the present context, to disruption of descending sympathetic fibers through the brainstem to the spinal cord).
15. *Medial medullary syndrome.* Lesions of the medial aspect of the medulla typically resulting from occlusion of the anterior spinal artery produce contralateral loss of conscious proprioception, contralateral hemiparesis, and weakness of tongue muscles, which are protruded to the side of the lesion. Body paralysis, which involves the side contralateral to the lesion, coupled with cranial nerve weakness, which involves the side ipsilateral to the lesion, is called *alternating hypoglossal hemiplegia.*
16. Cranial nerves mediate multiple functions in the nervous system: motor nuclei—general somatic efferent (NIII, NIV, NVI, and NXII), special visceral efferent (NV, NVII, NIX, NX, and NXI), general visceral efferent (NIII, NVII, NIX, and NX), general somatic afferent (NV, NIX, and NX), special sensory afferent (NII and NVIII), or special visceral afferent (NI, NVII, NIX, and NX).

## SENSORY SYSTEMS

17. Loss of partial or total aspects of the visual field can be understood in terms of damage to the retinal pathways, including their targets in the lateral geniculate nucleus and visual cortex. The schematic diagram depicts the kinds of field deficits that occur following lesions of different aspects of the visual pathway. Key: (A) optic nerve lesion producing total blindness in the left eye; (B) lesion that disrupts the right retinal nasal fibers that project from the base of the left optic nerve producing right upper quadrantanopia and left scotoma; (C) lesion of optic chiasm producing bitemporal hemianopsia; (D) unilateral (left) optic tract lesion producing a right homonymous hemianopsia; (E) interruption of left visual radiations that pass ventrally through the temporal lobe to the lower bank of the visual cortex (i.e., the loop of Meyer) producing an upper right




*(With permission of Adams et al.)*

quadrantanopia; (F) interruption of left visual radiations that pass more dorsally through the occipital lobe to the upper bank of the visual cortex, producing a lower right quadrantanopia; and (G) lesion of the left visual cortex that produces a right homonymous hemianopsia.

18. The principles of an excitatory focus-and-surround inhibition, as well as that of a somatotropic organization are present within a given receptor system and form the functional basis for discriminative functions in a number of the sensory systems, including the auditory circuit. The auditory pathways are complex and involve the following synaptic connections: first-order root fibers of the spiral ganglion, which originate in the cochlea (organ of Corti), synapse in the cochlear nuclei of the upper medulla; second-order neurons, which project through lateral lemnisci, terminate bilaterally in the inferior colliculus; third-order neurons project to the medial geniculate nucleus; and fourth-order neurons project to the superior temporal gyrus (primary auditory cortex).
19. *Vestibular pathway.* First-order neurons originate from vestibular ganglia and have peripheral processes located in specialized receptors in the utricle, saccule, and semicircular canals. The central branches of this neuron reach the brain and terminate in the vestibular nuclei. Second-order neurons may pass to the cerebellar cortex (flocculonodular lobe) or project directly into the medial longitudinal fasciculus, where the fibers may run in a rostral or caudal direction, terminating in the NIII, NIV, or NVI of the midbrain and pons or spinal cord, respectively. Damage to these fibers, especially within the medial longitudinal fasciculus or cerebellum, produces nystagmus (i.e., involuntary movement of the eyes, in the horizontal or vertical plane, first slowly and then followed by a rapid jerking return).

## MOTOR SYSTEMS

20. Voluntary motor control affecting mainly the flexor system is expressed through the descending pyramidal tracts plus the rubrospinal tract, and control of functions associated with posture is mediated through such descending pathways as the vestibulo- and reticulospinal tracts. Modulation of motor functions is mediated by the basal ganglia and cerebellum. Involuntary motor disturbances at rest (called *dyskinesias*) are associated with the disruption of functions of the basal ganglia, and motor disturbances occurring during attempts at movement are frequently associated with damage to the cerebellum or its afferent or efferent pathways.
    A. The basal ganglia consist of the neostriatum (caudate nucleus and putamen), the paleostriatum (globus pallidus), and two additional structures that are anatomically and functionally related to the basal ganglia—the substantia nigra and subthalamic nucleus.

1. Disorders of the basal ganglia
   a. *Parkinson's disease.* Characterized by "pill rolling" tremor, akinesia (poverty of movement), and rigidity. This disorder is due to a reduction in striatal dopamine following a loss of dopamine neurons in the pars compacta of the substantia nigra, which project to the neostriatum.
   b. *Chorea.* Characterized by short, jerky movements of the distal extremities at rest. It is associated with lesions of the striatum. One form of chorea, called *Huntington's chorea,* is a genetic disorder that is associated with a chromosomal mutation. It results in destruction of GABAergic and cholinergic neurons in the caudate nucleus, and there is concomitant loss of neurons in the prefrontal regions of the neocortex.
   c. *Athetosis.* Characterized by slow writhing movements of the extremities and muscles of the neck. The lesion may involve the striatum.
   d. *Hemiballism.* Characterized by wild (flailing) movements of the limbs on one side of the body. It is due to damage of the subthalamic nucleus on the contralateral side.
2. Disorders of the cerebellum
   a. *Anterior lobe (paleocerebellum).* Characterized by a wide, staggering gait ataxia resulting primarily from damage that affects the vermal and paravermal regions of the anterior lobe.
   b. *Posterior lobe (neocerebellum).* Characterized most frequently by loss of coordination while executing voluntary movements.
   c. *Flocculonodular lobe (archicerebellum).* Characterized by a loss of equilibrium with the patient displaying a wide, staggering ataxic gait. Lesions of this region also produce eye movement disorders, including nystagmus.

## HIGHER AUTONOMIC AND BEHAVIORAL FUNCTIONS

21. Control of autonomic and endocrine functions as well as emotional behavior are mediated by the limbic system, hypothalamus, and midbrain periaqueductal gray matter. Interrelationships among these three groups of structures are as follows:
    A. For the expression of emotional behavior, such as rage and autonomic functions, these are mediated from the medial hypothalamus → midbrain periaqueductal gray → autonomic (i.e., neurons in lower medulla that regulate heart rate, blood pressure, and respiration) and somatomotor neurons (i.e., neurons of the trigeminal nerve that control vocalization), autonomic, and

somatomotor neurons of the spinal cord. These processes are further regulated by different groups of neurons within the limbic system (i.e., hippocampal formation, amygdala, septal area), which produce their effects by projecting directly or indirectly to the hypothalamus or midbrain periaqueductal gray.

B. For the regulation of endocrine functions, these are mediated from the supraoptic and paraventricular nuclei of the hypothalamus → posterior lobe of the pituitary, and from the medial hypothalamus → anterior lobe of the pituitary (via the vascular system). Limbic projections to the hypothalamus enable structures such as the hippocampal formation, amygdala, and septal area to modulate endocrine functions of the hypothalamus. Disruption of hypothalamic neurons may alter the mechanism for the expression of rage behavior and, likewise, affect temperature regulation, sexual behavior, feeding, drinking, and endocrine functions. Damage to neurons of the limbic system frequently leads to temporal lobe epilepsy and changes in the threshold for the expression of rage behavior [i.e., when different groups of neurons in the amygdala are damaged, heightened aggressiveness or a reduction in aggression (such as the Klüver-Bucy syndrome) may ensue]. Damage to the hippocampal formation can result in temporal lobe epilepsy and short-term memory deficits.

## CEREBRAL CORTEX

22. Dysfunctions associated with cerebrovascular accidents and tumors can be understood in terms of the principles of cortical localization and cerebral dominance. Following is a list of common disorders, their descriptions, and the cortical regions most closely associated with each disorder.

    A. *Upper motor neuron paralysis.* Damage to the precentral, premotor, and supplementary motor areas (as well as the internal capsule, crus cerebri, or corticospinal tracts), resulting in a loss of voluntary control of the upper and lower limbs, depending upon the extent of the lesion. This disorder is also associated with hyperreflexia, hypertonicity, and a positive Babinski's sign.

    B. *Broca's aphasia.* Damage to the inferior frontal gyrus of the dominant hemisphere. The patient cannot name simple objects but has no difficulty in comprehending spoken language.

    C. *Wernicke's aphasia.* Damage to the region of the superior temporal gyrus and/or adjoining regions. The patient has difficulty in comprehending language but speech appears fluent.

    D. *Astereognosia.* Damage to the parietal cortex of the contralateral side results in a failure of tactile recognition of objects (e.g., a blackboard eraser, a pack of cigarettes).

E. *Unilateral sensory neglect.* Damage to the parietal lobe (usually of the right hemisphere) can cause this disorder. The patient typically ignores stimuli on the opposite (i.e., left) side of body space, which includes visual, somatosensory, and auditory stimuli. The individual will neglect the opposite side of the body by neglecting to shave that side of the face and by denying that there is anything wrong with that side of the body, which may include a motor paralysis. The patient may further draw a picture of flowers or of a clock in which the petals on the flowers or the numbers on the clock are limited to the right side of each of the figures.

F. *Apraxia.* Damage to the posterior parietal cortex can prevent an individual from conceptualizing the sequence of events necessary to carry out a task, even though the basic sensory and motor pathways necessary to produce the required movements are intact. In effect, the patient is thus unable to carry out the task.

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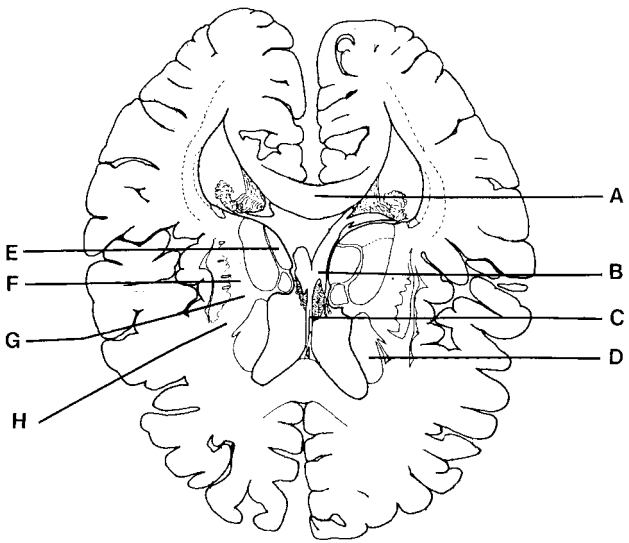
# Gross Anatomy of the Brain

## *Questions*

**DIRECTIONS:** Each group of questions below consists of lettered options followed by a set of numbered items. For each numbered item, select the **one** lettered option with which it is **most** closely associated. Each lettered option may be used once, more than once, or not at all.

**Questions 1–7**




**1.** This area contains fibers that arise from the leg region of the precentral gyrus

**2.** This region contains fibers that project primarily, if not exclusively, to the pons

**3.** A lesion at this site could produce weakness of muscles that mediate swallowing, chewing, breathing, and speaking

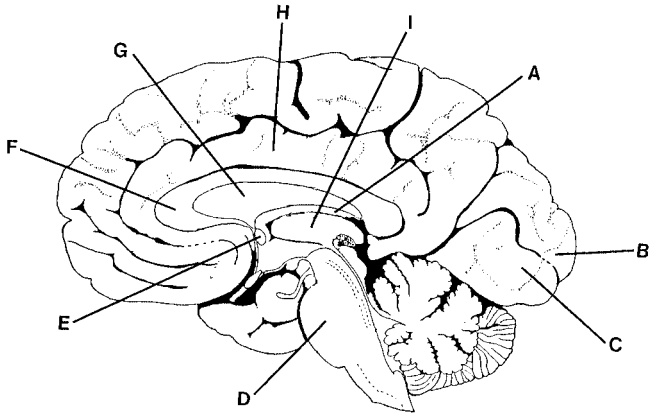
**4.** Fibers in this region project to the mamillary bodies

**5.** This structure is a major receiving area of the basal ganglia for afferent fibers arising from the cerebral cortex

**6.** This structure has extensive projections to the rostral aspect of the frontal lobe, including the prefrontal cortex

**7.** This region receives major dopaminergic inputs from the substantia nigra

### Questions 8–15




**8.** This commissure of the brain conveys olfactory information

**9.** This structure forms the medial wall of the lateral ventricle

**10.** This cortical structure is considered part of the limbic lobe and receives a significant input from the anteroventral thalamic nucleus

**11.** Loss of cells in this region results in a loss of vision in the lower visual field

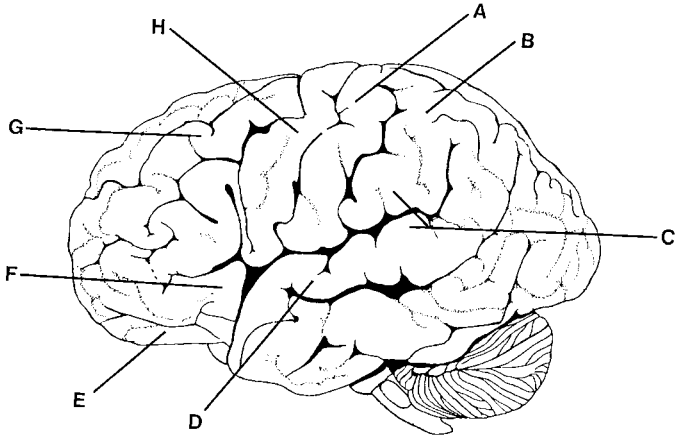
**12.** This bundle constitutes a major fiber pathway of the hippocampal formation

**13.** Cells in this region constitute the second-order neurons of a fiber pathway from the cerebral cortex to the cerebellar cortex

**14.** Damage to this region unilaterally will produce an upper quadrantanopia

**15.** Communication between the frontal lobes of each side of the brain is mediated through this structure

**Questions 16–24**




**16.** Cells in this region give rise to fibers that supply the cervical cord

**17.** This region receives somatosensory inputs from the lower limb

**18.** A lesion of this region will likely result in receptive aphasia

**19.** This region receives auditory inputs from the medial geniculate nucleus

**20.** A lesion at this site will produce a speech deficit, referred to as *expressive* aphasia

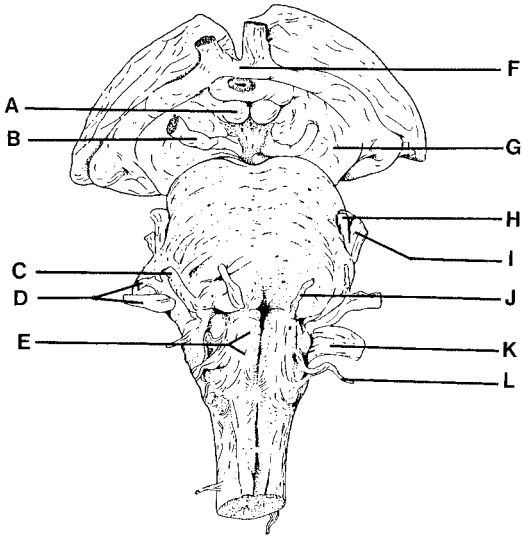
**21.** This region of the brain is associated with higher intellectual functions

**22.** This region of the cortex sends significant projections to the pretectal region and superior colliculus

**23.** A lesion of this region will typically produce a disorder involving negligence of the opposite body half and visual space

**24.** A lesion at this site would typically produce an upper motor neuron (UMN) paralysis

## Questions 25–35



**25.** This fiber bundle contains axons from neurons in the nucleus ambiguus

**26.** This structure controls muscles of mastication

**27.** The axons from neurons in this structure project to the anteroventral thalamic nucleus

**28.** This nerve controls the muscles of facial expression

**29.** Fibers in this region may terminate in the midbrain, pons, medulla, or spinal cord

**30.** First-order neurons from this area mediate somatosensory signals from the face

**31.** Damage to this structure could produce both a medial gaze paralysis and failure of pupillary constriction

**32.** Damage to this structure produces a lateral gaze paralysis

**33.** Disruption of fibers at this site resulting from a tumor would likely produce a bitemporal hemianopsia

**34.** These fibers mediate impulses that signal changes in the position of the head

**35.** A lesion of this nerve will cause the tongue to deviate to the side of the lesion

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response.

**36.** The walls that form the cisterns encasing the brain include

a. Ependyma and nerve cells
b. Dura mater and ependyma
c. Pia mater and arachnoid
d. Arachnoid and ependyma
e. Pia mater, arachnoid, and dura mater

**37.** Which of the following statements about the blood-brain barrier is correct?

a. It has well-developed capillary pores that allow for selective diffusion of substances
b. It is selectively permeable to certain compounds such as biogenic amines
c. It is found within all structures enclosed by the meninges, including the pineal gland
d. Tight junctions associated with the blood-brain barrier are formed exclusively by neuronal or glial processes
e. The blood-brain barrier is generally limited to highly vascular regions of the brain, such as those present at the level of the ventromedial hypothalamus

# Gross Anatomy of the Brain

## *Answers*

**1–7. The answers are 1-F, 2-H, 3-G, 4-B, 5-D, 6-E, 7-D.** *(Nolte, pp 65–69, 375–390, 451–453, 513–524, 559–561, 565–582.)* This figure is a horizontal view of the brain at the level of the head of the caudate nucleus and the internal capsule. The posterior limb of the internal capsule (F) contains fibers that arise from the leg region of the cerebral cortex and project to lumbar levels of the spinal cord, thus serving as UMNs for the elicitation of voluntary movement of the contralateral leg. Fibers in the anterior limb of the internal capsule (H) project in large numbers to deep pontine nuclei and represent first-order neurons in a pathway linking the cerebral cortex with the cerebellum. Pseudobulbar palsy is characterized in part by a weakness of the muscles controlling swallowing, chewing, breathing, and speaking. It results from a lesion of the UMNs associated with the head region of the cortex, which pass through the genu of the internal capsule (G) en route to brainstem cranial nerve nuclei upon which they synapse. The descending column of the fornix (B), situated along the midline of the brain, contains fibers that arise from the hippocampal formation and project in large part to the mamillary bodies.

The head of the caudate nucleus (D) is part of an important element of the motor systems called the *basal ganglia.* It receives significant inputs from several regions associated with motor functions. These include the cerebral cortex and the dopamine-containing region of the substantia nigra (i.e., the pars compacta). The mediodorsal thalamic nucleus (E) projects large quantities of axons to extensive regions of the rostral half of the frontal lobe, including the prefrontal cortex. It also receives significant projections from the prefrontal region of the cortex.

**8–15. The answers are 8-E, 9-G, 10-H, 11-B, 12-A, 13-D, 14-C, 15-F.** *(Afifi, pp 47–58, 422–424, 426–430, 476–477.)* This figure is a midsagittal section of the brain. A major portion of the anterior commissure (E) contains fibers that arise from the olfactory bulb and decussate to the contralateral olfactory bulb. The septum pellucidum (G) forms the medial wall

of the lateral ventricle, which in fact separates the lateral ventricle on one side from that on the opposite side. The cingulate gyrus (H) is a prominent structure on the medial aspect of the cerebral cortex and constitutes a component of the limbic lobe. It receives a significant input from the anteroventral thalamic nucleus.

The major output pathway of the hippocampal formation is the fornix system of fibers (A), which arises from cells in its subicular cortex and adjoining regions of hippocampus. These fibers are then distributed to the anterior thalamic nucleus, mamillary bodies, and septal area. The basilar portion of the pons (D) lies in the ventral half of this region of the brainstem. It receives inputs from each of the lobes of the cerebral cortex, which it then relays to the cerebellar cortex. The primary visual cortex lies on both banks of the calcarine fissure. Cells located on the lower bank receive inputs from the lateral geniculate nucleus that relate to either the nasal or temporal upper visual fields. Therefore, a lesion of this region would produce an upper quadrantanopia (i.e., loss of one-quarter of the visual field). The corpus callosum constitutes the major channel by which the cerebral cortex on one side can communicate with the cortex of the opposite side. The genu of the corpus callosum (F) contains fibers that pass from the frontal lobe of one side to that of the other.

**16–24. The answers are 16-H, 17-A, 18-C, 19-D, 20-F, 21-E, 22-G, 23-B, 24-H.** *(Nolte, pp 52–60, 62–69, 375–394, 440–445, 507–524.)* This figure is a lateral view of the cerebral cortex. Cells in the "arm" area of the primary motor cortex (H) project their axons to the cervical level of the spinal cord. This area receives major input from the ventrolateral nucleus of the thalamus. The leg region of the primary somatosensory cortex (A) lies immediately caudal to the central sulcus, is almost devoid of pyramidal cells, and is referred to as a *granulous cortex.* Damage to the cells situated in the region of the dorsal border of the superior temporal gyrus and the adjoining area of the inferior parietal lobule (Wernicke's area) (C) causes impairment in the appreciation of the meanings of written or spoken words.

The primary, secondary, and tertiary auditory receiving areas in the cortex are located mainly in the superior temporal gyrus (D). It is the final receiving area for inputs from the medial geniculate nucleus, which represents an important relay in the transmission of auditory signals to the cortex. An additional area of the cortex governing speech (F) is called the

*motor speech area,* or *Broca's area.* It is situated in the inferior aspect of the frontal lobe immediately rostral and slightly ventral to the precentral gyrus. Lesions of this region produce impairment of the ability to express words in a meaningful way or to use words correctly. The orbital frontal cortex (E) lies in a position inferior and rostral to Broca's motor speech area. This region governs higher-order intellectual functions and some aspects of emotional behavior.

The caudal aspect of the middle frontal gyrus (G) contains cells that, when activated, produce conjugate deviation of the eyes. This action is believed to be accomplished, in part, by virtue of descending projections to the superior colliculus, pretectal region, and horizontal gaze center of the pons. Lesions of the posterior parietal lobe (B) of the nondominant hemisphere will produce a disorder of body image, referred to as *sensory neglect.* The patient will frequently fail to recognize or neglect to shave or wash those body parts. The patient may even fail to recognize the presence of a hemiparesis involving that part of the body as well. The precentral gyrus (H) constitutes the primary motor cortex. Lesions of this region produce a UMN paralysis involving a contralateral limb.

**25–35. The answers are 25-K, 26-H, 27-A, 28-C, 29-G, 30-I, 31-B, 32-J, 33-F, 34-D, 35-L.** *(Nolte, pp 255–280, 284–287.)* This figure is a ventral view of the brainstem. Fibers that arise from the nucleus ambiguus exit the brain on the lateral side of the medulla as part of the vagus nerve (K) and innervate the muscles of the larynx and pharynx as special visceral efferents. The motor root (H) lies medial to the sensory root and innervates the muscles of mastication. The mamillary bodies (A), which lie on the ventral surface of the brain at the caudal aspect of the hypothalamus, project many of their axons to the anteroventral thalamic nucleus as the mamillothalamic tract. The facial nerve (C) exits the brain at the level of the ventrolateral aspect of the caudal pons and its special visceral efferent component innervates the muscles of facial expression.

The cerebral peduncle (G) is situated in the ventrolateral aspect of the midbrain and contains fibers of cortical origin that project to all levels of the neuraxis of the brainstem and spinal cord. Note that the selection of choice E, the pyramids, would not have been a correct choice since the fibers present at this level can only terminate within the medulla or spinal cord. First-order somatosensory fibers from the region of the face (I) enter the brain laterally at the level of the middle of the pons as the sensory root

of the trigeminal nerve. The oculomotor nerve (B) exits the brain at the level of the ventromedial aspect of the midbrain and some fibers of the general somatic efferent component of this nerve innervate the medial rectus. Damage to this component results in a loss of ability for medial gaze. Another component of the oculomotor nerve, the GVE component, constitutes the preganglionic parasympathetic neuron in a disynaptic pathway whose postganglionic division innervates the pupillary constrictor muscles. Accordingly, damage to the preganglionic division results in loss of pupillary constriction, which normally occurs in the presence of light as well as in accommodation. The abducens nerve (J) exits the brain at a ventromedial position at the level of the medulla-pontine border, and its fibers innervate the lateral rectus muscle. Damage to this nerve results in a lateral gaze paralysis.

The optic chiasm (F) contains fibers that cross over to reach the lateral geniculate nucleus on the side contralateral to the retina from which they originated. Such fibers are associated with the temporal (i.e., lateral) visual fields. Therefore, damage to the optic chiasm will cause blindness in the lateral half of each of the visual fields. Such a deficit is referred to as *bitemporal hemianopsia.* First-order neurons from the labyrinth organs (i.e., semicircular canals, saccule, and utricle) convey information concerning the position of the head in space along the vestibular component of the eighth nerve into the central nervous system. This nerve enters the brain laterally at the level of the upper medulla. The hypoglossal nerve (L) exits the brain at the level of the middle of the medulla between the pyramid and the olive. These fibers innervate muscles that move the tongue toward the opposite side. For this reason, a lesion of the hypoglossal nucleus or its nerve will result in a deviation of the tongue to the side of the lesion because of the unopposed action of the contralateral hypoglossal nerve, which remains intact.

**36. The answer is c.** *(Nolte, pp 76–94–47, 96–115.)* The meninges of the brain include the pia mater, the arachnoid, and the dura mater. The pia mater and the arachnoid are situated closest to the brain, and the dura mater is in an external position. Normally, the space between the pia mater and the arachnoid is called the *subarachnoid space* and is filled with cerebrospinal fluid. Surrounding the brain, the subarachnoid space shows local variations. At places where bulges are present, they are referred to as *cisterns.*

**37. The answer is b.** *(Kandel, pp 1288–1294. Nolte, 3/e, pp 136–139.)* The blood-brain barrier is selectively permeable to certain types of substances, such as biogenic amines, and not to others. The barrier is formed by tight junctions consisting of capillary endothelial cells that are frequently in contact with the glial end-feet of astrocytes. The barrier does not contain well-developed capillary pores. It is not found within circumventricular organs such as the subfornical organ and the pineal gland, but it is applied to all other brain tissues.

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# Development

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**38.** Alpha motor neurons are derived from the

a. Alar plate
b. Basal plate
c. Sulcus limitans
d. Neural crest
e. Roof plate
f. Mesencephalon
g. Floor plate
h. Rathke's pouch
i. Rhombic lips

**39.** The proper sensory nucleus is derived from the

a. Alar plate
b. Basal plate
c. Sulcus limitans
d. Neural crest
e. Roof plate
f. Mesencephalon
g. Floor plate
h. Rathke's pouch
i. Rhombic lips

**40.** The dorsal root ganglia are derived from the

a. Alar plate
b. Basal plate
c. Sulcus limitans
d. Neural crest
e. Roof plate
f. Mesencephalon
g. Floor plate
h. Rathke's pouch
i. Rhombic lips

**41.** The spinal trigeminal nucleus is derived from the

a. Alar plate
b. Basal plate
c. Sulcus limitans
d. Neural crest
e. Roof plate
f. Mesencephalon
g. Floor plate
h. Rathke's pouch
i. Rhombic lips

**42.** The choroid plexus is derived from the

a. Alar plate
b. Basal plate
c. Sulcus limitans
d. Neural crest
e. Roof plate
f. Mesencephalon
g. Floor plate
h. Rathke's pouch
i. Rhombic lips

**43.** The cerebellum is derived from

a. Neural crest cells
b. Rhombic lips
c. Mesencephalon
d. Sulcus limitans
e. Telencephalon
f. Myelencephalon
g. Floor plate
h. Rathke's pouch

**44.** The amygdala is derived from

a. Neural crest cells
b. Rhombic lips
c. Mesencephalon
d. Sulcus limitans
e. Telencephalon
f. Myelencephalon
g. Floor plate
h. Rathke's pouch

**45.** The anterior pituitary is derived from

a. Neural crest cells
b. Rhombic lips
c. Mesencephalon
d. Sulcus limitans
e. Telencephalon
f. Myelencephalon
g. Floor plate
h. Rathke's pouch

**46.** Sympathetic ganglia are derived from

a. Neural crest cells
b. Rhombic lips
c. Mesencephalon
d. Sulcus limitans
e. Telencephalon
f. Myelencephalon
g. Floor plate
h. Rathke's pouch

**47.** A young child is brought into the hospital emergency room because he has episodes of vomiting, headaches, problems in acquisition of motor skills, cranial nerve dysfunction, and problems in breathing. This combination of syndromes most closely relates to which of the following disorders?

a. Cleft palate
b. Hydrocephalus
c. Anencephaly
d. Syringomyelia
e. Congenital aneurysm

**48.** A brain MRI scan taken from a 6-month-old baby revealed that while the overall size of the cerebral cortex was normal, the size of the pyramidal tracts was considerably smaller than normal. The most likely explanation for this defect is that there is a

a. Reduction in the numbers of cortical neurons giving rise to pyramidal tract fibers
b. Reduction in the numbers of synaptic contacts made by pyramidal tract neurons
c. Reduction in the extent of myelin found on pyramidal tract neurons
d. Reduction in the amount of neurotransmitter released by pyramidal tract neurons
e. Reduction in the numbers of glial cells attached to pyramidal tract neurons

**49.** Apoptosis is likely to occur following

a. Stimulation of an afferent nerve fiber
b. Severing of an afferent nerve fiber
c. The beginning of myelin formation
d. Elimination of nerve growth factor
e. Reduction in brain serotonin levels

# Development

## *Answers*

**38. The answer is b.** *(Nolte, pp 36–44.)* Structures associated with motor functions, such as alpha motor neurons, are derived from the basal plate.

**39. The answer is a.** *(Nolte, pp 36–44.)* Structures associated with sensory functions, such as the proper sensory nucleus and the spinal nucleus of cranial nerve V, are derived from the alar plate.

**40. The answer is d.** *(Nolte, pp 36–44.)* A number of structures, such as the dorsal root ganglia, sympathetic ganglia, and chromaffin cells of the adrenal medulla, are derived from neural crest cells.

**41. The answer is a.** *(Nolte, pp 36–44.)* As noted above in the answer to question 39, structures associated with sensory functions, such as the spinal nucleus of the trigeminal nerve, are derived from the alar plate.

**42. The answer is e.** *(Nolte, pp 36–44, 100, 102–104, 376, 471.)* The choroid plexus is attached to the roof of the ventricles and is thus derived from the roof plate.

**43. The answer is b.** *(Nolte, pp 38–43.)* The cerebellum is formed from the dorsolateral aspects of the alar plates, which bend medially and posteriorly to form the rhombic lips.

**44. The answer is e.** *(Nolte, pp 36–44.)* The amygdala is derived from the telencephalon, a part of the forebrain.

**45. The answer is h.** *(Martin, p 423; Nolte, pp 36–43.)* The anterior lobe of the pituitary is formed as an in-pocket derivative of the ectodermal stomodeum, called *Rathke's pouch.*

**46. The answer is a.** *(Nolte, pp 36–41.)* The sympathetic ganglia, as well as other structures (e.g., chromaffin cells of the adrenal medulla and dorsal root ganglia), are derived from neural crest cells.

**47. The answer is b.** *(Afifi, pp 513–521.)* The symptoms described are characteristic of hydrocephalus. Hydrocephalus may come about as a result of defects such as the failure of formation of the cerebellar vermis, foramens of Magendie and Luschka, or of the corpus callosum. There is an enlarged cranium as a result of the buildup of cerebrospinal fluid (CSF), causing brain damage. Several of the symptoms may also be caused by a compression of the posterior fossa and the absence of a cerebellar vermis. Cleft palate is a fissure of the medial aspect of the lip and would not result in the symptoms described previously. Anencephaly is the complete or partial absence of the brain and is not compatible with life. Syringomyelia is associated with bilateral segmental loss of pain and temperature. A congenital aneurysm can occur in a variety of places within the CNS and is typically associated with stroke in the adult.

**48. The answer is c.** *(Afifi, pp 505–508.)* Extensive myelination occurs in postnatal development. The failure of the pyramidal tracts to form myelin would account for the reduction in their size. In this particular situation, the size of the cerebral cortex was approximately normal, suggesting that there was no significant decrease in cortical cells. Variation in the numbers of synaptic contacts, transmitter formation, and glial cells would not account for a reduction in the size of the pyramidal tract.

**49. The answer is d.** *(Purves, pp 503–506.)* When nerve growth factor is eliminated, cell death results and involves fragmentation, shrinkage, and ultimate phagocytosis of the cell. Apoptosis is believed to be triggered by a biochemical process that causes transcription of a variety of genes. Nerve growth factor blocks the activation of this process. It should also be noted that this form of cell death differs from that occurring after nerve injury or trauma to the nerve. The other choices listed are unrelated to the process of apoptosis.

# The Neuron

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

### Questions 50–51

The methods involving microinjections of Fluoro-Gold or horseradish peroxidase (HRP) have been employed over the past few decades by many investigators.

**50.** These methods have been used to identify:

a. Cell bodies
b. Metabolic activity of neurons
c. Sensory endings of nerve fibers
d. Central nervous system receptors
e. Degenerating axons

**51.** These methods take advantage of the principle of:

a. Metabolic mapping of central nervous system (CNS) pathways
b. Anterograde labeling of degenerating axons
c. Retrograde transport
d. Anterograde transport
e. Visualization of demyelinating peripheral nerves

### Questions 52–53

Substances such as tritiated amino acids and phaseolus vulgaris agglutinin microinjected into specific regions of the brain have also been employed by many investigators for the study of the nervous system.

**52.** These methods are specific in that they label:

a. Cell bodies
b. Glial cells
c. Sensory receptors
d. Motor end plates
e. Axons and axon preterminals

**53.** These methods take advantage of the principle of:

a. Metabolic mapping of CNS pathways
b. Staining of degenerating axons
c. Retrograde transport
d. Anterograde transport
e. Retrograde degeneration

**54.** Magnetic resonance imaging (MRI) and 2-deoxyglucose (2-DG) autoradiography are methods developed to

a. Label dopamine receptors
b. Label serotonin receptors
c. Label metabolically active structures
d. Stain individual cell bodies, dendrites, and axons
e. Stain astrocytes

**55.** Which of the following procedures would be utilized in order to show positive staining of groups of serotonin neurons?

a. Electrical brain stimulation
b. Glutamate stimulation of the brain
c. HRP staining of neurons
d. Immunocytochemical labeling
e. Metabolic staining of neurons

**56.** An individual sustained a severe knife wound, damaging a spinal nerve adjoining its entry to the spinal cord. If one could examine this peripheral nerve and its cell body, which of the following events would he or she most likely observe?

a. A displacement of the nucleus toward the periphery of the cell
b. A mitotic division of the neuronal cell body
c. A more intense staining of the cell body
d. Degeneration of processes along the axon proximal but not distal to the lesion
e. An initial loss of mitochondria in the axoplasm at Ranvier's node

**57.** A 65-year-old man is diagnosed with a form of a peripheral neuropathy. This individual will likely display

a. A loss in motor function, but sensory functions will remain largely intact
b. A reduction in conduction velocity of the affected nerve
c. An increase in the number of Ranvier's nodes
d. Degeneration of myelin but the axon will typically remain intact
e. Signs of an upper motor neuron (UMN) paralysis

**58.** Which of the following statements about nerve cells is correct?

a. Typically, one copy of the same peptide is cut from the same precursor molecule
b. It is generally recognized that the cytosol provides the source of selective protein synthesis limited to neurotransmitters
c. Cytosolic proteins show significant modification or processing following their translation
d. Nuclear and mitochondrial proteins that are encoded by the cell's nucleus are targeted to their proper organelle by a process called *posttranslational importation*
e. Secretory proteins undergo little or no modification or processing after translation

**59.** Which of the following statements concerning axoplasmic transport is correct?

a. Large membranous organelles are transported by slow axonal transport
b. Cytosolic proteins are transported by fast transport
c. Retrograde transport is generally limited to a fixed rate of movement of particles
d. Anterograde transport is dependent upon microtubules

**60.** Which of the following statements correctly characterizes ion channels?

a. The passage of ions through ion channels typically requires an active mechanism
b. A common stimulus serves as the basis for opening ion channels
c. Exposure of a ligand-gated channel to continuous high concentrations of its ligand is the necessary and sufficient stimulus for opening that channel
d. The opening or closing of an ion channel may be affected by the use of drugs

**61.** Which of the following statements concerning the resting membrane potential is correct?

a. Passive fluxes of $Na^+$ and $K^+$ are balanced by an active pump that derives energy from enzymatic hydrolysis of adenosine 5′-triphosphate (ATP)
b. A membrane is depolarized when the separation of the charge across the membrane is increased
c. As the inside of the cell is made more negative with respect to the outside, the cell becomes depolarized
d. In a cell whose membrane possesses only $K^+$ channels, the membrane potential cannot be determined
e. The resting membrane potential is unrelated to the separation of the charge across the membrane

**62.** Which of the following statements concerning the length constant is correct?

a. The length constant is the distance along a dendrite where the change in membrane potential produced by a current becomes stable
b. Length constant increases as the membrane resistance decreases
c. Length constant increases as the axial resistance increases
d. Length constant is greater in unmyelinated than in myelinated fibers
e. As the length constant increases in a postsynaptic neuron, the efficiency of electronic conduction of synaptic potentials (at that synapse) decreases

**63.** Which of the following statements concerning ligand gating of neuronal membrane channels is true?

a. The normal triggering mechanism for gating involves nonspecific binding by large classes of molecules
b. Channels are opened when a given molecule selectively binds with the gating molecule
c. Ligand gating is triggered by changes in the electrical potential across the membrane
d. The channels are constructed of a mixture of proteins and lipids
e. The gating molecule shows no conformational change during the gating process

**64.** After the occurrence of an action potential, there is a repolarization of the membrane. The principal explanation for this event is that

a. Potassium channels have been opened
b. Sodium channels have been opened
c. Potassium channels have been inactivated
d. The membrane becomes impermeable to all ions
e. There has been a sudden influx of calcium

**65.** During an in vitro experiment, the membrane potential of a nerve cell is hyperpolarized to −120 mV. At that time, a transmitter, known to be inhibitory in function, is applied to the preparation and results in a depolarization of the membrane. The most likely reason for this occurrence is that

a. Inhibitory transmitters normally depolarize the postsynaptic membrane
b. The normal response of the postsynaptic membrane to any transmitter is depolarization
c. The inhibitory transmitter activates ligand-gated potassium channels
d. Sodium channels become inactivated
e. Calcium channels become activated

## Questions 66–73

The neurophysiologist, Kuffler, studied the electrophysiology of glial cells, using the optic nerve and its surrounding glial sheath. He found that the mean value of the resting potential of these cells, as recorded by intracellular microelectrodes, was 89.6 mV. The potassium concentration in the bathing solution was 3 meq/L. Assume that $RT/F = 61$.

**66.** Assuming that the resting potential is equivalent to the potassium equilibrium potential, calculate the approximate intracellular potassium concentration (in meq/L).

a. 11
b. 33
c. 88
d. 140
e. 155

**67.** What would be the concentration of potassium (meq/L) in the bathing fluid in order to depolarize the membrane potential to zero?

a. 11
b. 33
c. 88
d. 140
e. 155

**68.** A probable explanation for the depolarization of glial cells following stimulation of nerve fibers is that it is a result of

a. A delayed increase in potassium conductance
b. An early sodium influx
c. A large efflux of sodium ions
d. A temporal summation that results in a long-lasting depolarization
e. An influx of chloride ions

**69.** Stimulation of the optic nerve with a volley of impulses caused a slow and long-lasting depolarization of the associated glial cells. The mean value of the depolarization was 12.1 mV. If this depolarization was due solely to an increase in potassium ion concentration in the intracellular clefts, calculate the change in the concentration of potassium in the extracellular environment (in meq/L).

a. 1.79
b. 36.30
c. 137.00
d. 140.50
e. $5.35 \times 10^{-6}$

**70.** The trigger zone that integrates incoming signals from other cells and initiates the signal that the neuron sends to another neuron or muscle cell is the

a. Cell body
b. Dendritic trunk
c. Dendritic spines
d. Axon hillock and initial segment
e. Axon trunk

**71.** Which of the following statements concerning the membrane time constant is correct?

a. The time constant is a function of the membrane's resistance and capacitance
b. The time constant is unrelated to the membrane's capacitance
c. The time course of the rising phase of a synaptic potential is specifically dependent upon the time constant for that cell
d. The falling phase of a synaptic potential is dependent upon active and passive membrane properties
e. The integration of synaptic potentials is unrelated to the length of the time constant

**72.** Which of the following statements concerning sodium channels is true?

a. They are opened when the membrane is hyperpolarized
b. They display a high conductance in the resting membrane
c. They open rapidly following depolarization of the membrane
d. They are rapidly inactivated by tetraethylammonium
e. They are rapidly activated by tetrodotoxin

**73.** The equilibrium potential for potassium, as determined by the Nernst equation, differs from the resting potential of the neuron because

a. An active sodium-potassium pump makes an important contribution to the regulation of the resting potential
b. The membrane is permeable to ions other than potassium
c. The Nernst equation basically considers only the relative distribution of potassium ions across the membrane
d. The resting potential is basically dependent upon the concentration of sodium but not potassium ions across the membrane
e. The Nernst equation fails to account for local changes in temperature that influence the resting membrane potential

**74.** Based upon one's knowledge of the typical distribution of ions across a cell membrane, one could predict that an appropriate resting membrane potential would be

a. +70 mV
b. +30 mV
c. 0 mV
d. −70 mV
e. −100 V

**75.** If a membrane is permeable only to sodium ions and the concentration of sodium ions on one side of the membrane is the same as that on the other side, then which of the following statements would best characterize the resting membrane potential for that cell?

a. A pump mechanism will cause the cell to become hyperpolarized
b. The membrane potential would be zero
c. The tendency would be for current to be directed inwardly
d. The membrane potential could not be predicted from the Nernst equation
e. There would be an initial decrease followed by an increase in membrane potential

**76.** Which of the items listed below best characterizes the following statement: A graded, fast potential, lasting from several milliseconds to seconds, resulting from a chemical transmitter binding to a receptor to produce either an excitatory postsynaptic potential (EPSP) that depends upon a single class of channels for sodium and potassium or an inhibitory postsynaptic potential (IPSP) that is dependent upon chloride or potassium conductance?

a. Receptor potentials
b. Electrical postsynaptic potentials
c. Increased-conductance postsynaptic potentials
d. Decreased-conductance postsynaptic potentials
e. Decreased-conductance postsynaptic potentials

**77.** The term *all-or-none response* is most closely related to

a. The resting potential
b. Increased-conductance presynaptic potentials
c. Increased-conductance postsynaptic potentials
d. The generator potential
e. The action potential

**78.** The passive spread of a presynaptic current across a gap junction that is activated by changes in voltage, pH, or calcium ion levels is most closely associated with

a. The resting potential
b. The action potential
c. Electrical presynaptic potentials
d. Electrical postsynaptic potentials
e. Receptor potentials

# The Neuron

## *Answers*

**50–51. The answers are 50-a, 51-c.** *(Martin, pp 23–27.)* With HRP histochemistry, the glycoprotein enzyme HRP is injected into the region of the terminal endings of the neuronal pathway under examination and is incorporated into the axons through a process of micropinocytosis. Horseradish peroxidase is then retrogradely transported back to the cell bodies of origin of that pathway, where it is then degraded. By reacting the tissue with an appropriate substrate, the labeled cells can be visualized under light microscopy.

**52–53. The answers are 52-e, 53-d.** *(Martin, pp. 23–27.)* The mapping of pathways utilizing anterograde tracing of fibers depends upon the process of axonal transport. For example, if a tritiated amino acid such as $^{3}$H-leucine is microinjected into a region of the brain, it gets synthesized into protein in the cell bodies and transported down the respective axons to their terminals. By utilizing autoradiographic methods, one can identify the loci of the label contained in the protein that has been transported to the axon terminals. The application of phaseolus vulgaris agglutinin also utilizes the principle of anterograde transport to map the distribution of pathways from cell bodies injected with this substance.

**54. The answer is c.** *(Martin, pp 23–27.)* Magnetic resonance imaging permits the visualization of the degenerative process that results in demyelination of axons. 2-Deoxyglucose autoradiography is used to metabolically map pathways and structures that are functionally active as a result of sensory, chemical, or electrical stimulation of nervous tissue. Local variations in energy metabolism can be visualized because the glucose analog $^{14}$C-2-deoxyglucose (2-DG) is phosphorylated to 2-DG-6-phosphate where it is not further metabolized and is retained in the neuron. The rate of incorporation into neurons is related to the rate of glucose utilization, which is, itself, a function of energy metabolism.

**55. The answer is d.** *(Martin, pp 23–27; Kandel, pp 891–895.)* Immunocytochemical methods, including in situ hybridization, have been used to

identify the presence and localization of specific neurotransmitters and receptors, such as serotonin.

**56. The answer is a.** *(Afifi, pp 30–34; Nolte, pp 18–21.)* Damage to a nerve fiber proximal to its cell body will cause, among other changes, retrograde degeneration of the cell body. A number of changes occur in the neuron during the process of retrograde degeneration. The cell body initially shows some swelling and becomes distended. At the beginning of the degenerative process, there is an accumulation of mitochondria in the axoplasm at Ranvier's nodes. The nucleus is then displaced toward the periphery of the cell. The Nissl granules break down, first in the center of the cell; later, the breakdown spreads outward. In addition, the axonal process distal to the site of the lesion will undergo degeneration. It should be noted that retrograde degeneration procedures were used experimentally prior to the advent of histochemical methods for identifying cell bodies of origin of given pathways in the CNS.

**57. The answer is b.** *(Kandel, pp 82–83, 700–704.)* In a peripheral neuropathy, there may be damage to either the myelin or the axon directly, although, more often, there is damage to the myelin. Because of myelin (or axonal) damage, there is a reduction (or loss) of conduction velocity. The disorder may affect both sensory and motor components of the peripheral nerve, thereby causing dysfunction in both the sensory and the motor processes associated with that nerve. Because there is peripheral neuronal damage, the motor loss will be reflected in a weakness, paralysis, or reflex activity associated with the affected muscle, as well as impairment of sensation.

**58. The answer is d.** *(Kandel, pp 88–98.)* Nuclear and mitochondrial proteins are encoded by the nucleus and are formed on free polysomes. The mechanism by which they are targeted to their proper organelle is called *posttranslational importation.* Specific receptors bind and translocate these proteins, and it is the recognition of the structural features of these proteins that allows for transport into the nucleus from the cytoplasm. In the processing of large proteins such as opioid peptides, more than one copy and different peptides are produced from the same precursor molecule. This precursor is referred to as a *polyprotein* because more than one active peptide is present. All protein synthesis begins in the cytosol.

Cytosolic proteins are the most extensive type of protein in the cell and include those that make up the cytoskeleton and enzymes that catalyze the different metabolic reactions of the cell. Messenger RNAs (mRNAs) for these proteins pass through nuclear pores, become associated with ribosomes, and ultimately form free polysomes in the cytoplasm of the cell. Cytosolic proteins display little modification or processing compared with proteins that remain attached to the membranes of endoplasmic reticulum or the Golgi apparatus. Messenger RNA that encodes protein that will become a constituent of organelles or secretory products is formed on polysomes that are attached to endoplasmic reticulum. Such sheets of membrane in association with ribosomes are called *rough endoplasmic reticulum.* Secretory products typically undergo significant modification after translation. For example, neuropeptide transmitters are cleaved from polypeptide chains, in part, in the endoplasmic reticulum and the Golgi apparatus.

**59. The answer is d.** *(Kandel, pp 100–104.)* Transport in either direction utilizes microtubules as a vehicle or track by which the particles are transported. Among the particles transported down the axon from the cell body are newly synthesized membranous organelles, including synaptic vesicles or their precursors, which ultimately reach the axon terminals. In retrograde transport, the particles transported include endosomes generated from the nerve terminal, mitochondria, and components of endoplasmic reticulum. Large membranous organelles are transported along the axon both anterogradely and retrogradely by fast axonal transport. In contrast, cytosolic proteins and components of the matrix of the cytoskeleton are transported by slow axonal transport. There are different rates of retrograde transport in which the faster component is approximately twice as fast as the slow component.

**60. The answer is d.** *(Kandel, pp 107–116.)* Ion flux through ion channels is considered to be passive in nature and functions in the absence of any mechanism that requires energy metabolism. Cation channels are generally associated with membranes that are semipermeable to selective ions such as $Na^+$, $K^+$, or $Cl^-$. The electrochemical gradient is a function of two forces: (1) the chemical concentration gradient, which is derived from the relative differences in the distributions of ions across the membrane, and

(2) the electrical potential difference between the two sides of the membrane as a function of the distribution of the ionic charges. When ions may flow through channels, current varies as a function of concentration. However, at high ionic concentration differences, a saturation phenomenon is observed that is due to resistance to flow through the channels. It is believed that different kinds of stimuli can function to open or close channels. For example, mechanical activation may lead to the opening of channels. Some channels (ligand-gated) are regulated by the noncovalent binding of chemical ligands such as neurotransmitters; others (electrically gated) are affected by changes in membrane voltage that cause a change in the conformation of the channel. Alternatively, relatively long-lasting changes may result when second messengers bind to the channel at which time there is protein phosphorylation mediated by protein kinases. Such modification of the channel can be reversed by dephosphorylation. In contrast, when a ligand-gated channel is exposed to prolonged, high concentrations of its ligand, it tends to become refractory (i.e., desensitized to the presence of that ligand).

**61. The answer is a.** *(Kandel, pp 125–138.)* The potential difference across the membrane is a result of the separation of charge and is called the *resting membrane potential.* Accordingly, the potential difference across the membrane is a direct function of the numbers of positive and negative charges on either side of the membrane. As the separation of charge across the membrane is reduced, the membrane is said to be depolarized. Conversely, as the separation of charge is increased, the membrane becomes hyperpolarized. In the latter case, the inside of the cell is made more negative with respect to the outside. If a cell has only a single channel in its membrane (such as for $K^+$), the gradients for the other ions become irrelevant and the membrane potential will approach the equilibrium potential for the single ion ($K^+$ in this example). There is a tendency for ions to leak down their electrochemical gradients from one side of the membrane to the other. For there to be a steady resting membrane potential, the gradients across the membrane must be held constant. Changes in ionic gradients are avoided, in spite of the leak, by the presence of an active $Na^+/K^+$ pump (a membrane protein) that moves $Na^+$ out of the cell and at the same time brings $K^+$ into the cell. Such a pumping mechanism requires energy because it is working against the electrochemical gradients of the two ions. The energy is derived from the hydrolysis of ATP.

**62. The answer is b.** *(Kandel, pp 222–223.)* The length constant is defined as $R_m/R_a$, where $R_m$ equals membrane resistance and $R_a$ equals axial resistance. It is the distance along a fiber where a change in membrane potential produced by a given current decays to a value of approximately one-third of its original value. As can be seen from the definition, the length constant is directly proportional to the membrane resistance and inversely related to the axial resistance (i.e., the resistance of the cytoplasm within the fiber). The membrane resistance is increased significantly through the process of myelination, which thus produces an increase in the value of the length constant. When the length constant along a dendrite is relatively large, it has the effect of increasing the efficiency of electrotonic conduction along the dendritic process as compared with a similar dendrite with a smaller length constant. In this manner, the synaptic potential along the dendrite distal to the synapse will be relatively larger in a dendrite that has a larger length constant than one that has a smaller length constant.

**63. The answer is b.** *(Kandel, pp 105–119, 185, 196, 240.)* The triggering mechanism for ligand gating involves the selective binding of a particular molecule with the protein channel. This binding causes a conformational change of the channel protein that results in the movement of the channel back and forth, which, in effect, opens or closes the channel. Neurotransmitters can regulate channels as a result of their binding properties. An example is the action of acetylcholine at the neuromuscular junction, which is capable of activating channels in the membrane of skeletal muscle. Most cation channels are selective for sodium, potassium, or calcium. Ion channels are composed of large-membrane glycoproteins that vary widely in their molecular weights. In contrast to ligand gating, other types of channels may be activated by changes in the electrical potential across the cell membrane, a process referred to as *voltage gating.*

**64. The answer is a.** *(Kandel, pp 150–164.)* In the late phase of the action potential, potassium channels become opened and potassium efflux produces a hyperpolarization of the membrane. During the repolarization of the membrane, sodium channels are closed (sodium inactivation). Recall that activation of sodium channels is associated with the generation of the action potential. Calcium has a strong electrochemical gradient that drives it into the cell; this coincides with the upstroke of the action potential. A number of different types of calcium-gated potassium channels have been

described that are activated during the action potential. Thus, it would appear that calcium influx during the action potential could generate opposing effects. On the one hand, calcium influx carries a positive charge into the cell, which contributes to the depolarization of the membrane. On the other hand, calcium influx may help to open up more potassium channels, which contributes to an outward ionic flow of potassium that causes repolarization of the membrane.

**65. The answer is c.** *(Kandel, pp 125–138.)* To understand how an inhibitory transmitter can actually cause a partial depolarization of the membrane, refer to the Goldman equation. The release (or application) of an inhibitory transmitter will serve to open specific ion channels, notably those of potassium. If the membrane is artificially hyperpolarized to −120 mV, the opening of the potassium channel will lead to a redistribution of the ions across the membrane to a normal level. If the normal equilibrium potential for potassium is approximately −75 mV, then, application of an inhibitory transmitter (that typically functions by opening potassium channels) will result in a redistribution of potassium ions toward the potassium equilibrium potential (i.e., −75 mV). Consequently, the membrane potential will be reduced (i.e., depolarized) from −120 mV to a value close to −75 mV. Other possible answers are clearly incorrect. Inhibitory transmitters normally function to hyperpolarize the membrane. Postsynaptic membranes may either be depolarized or hyperpolarized, depending upon the nature of the transmitter and receptor complex present at the synapse. Since the influx of calcium during the depolarization phase of the action potential leads to opposing effects, activation of this channel cannot account for the observed effects. Inactivation of sodium channels would not result in a depolarization of the membrane, but, instead, may contribute to the hyperpolarization of the membrane.

**66. The answer is c.** *(Kandel, pp 132–138.)* To solve the problem, use the Goldman equation, which reduces to the Nernst equation:

$$\begin{aligned}
\text{Equilibrium potential} &= (RT/F) \times \ln [K_i] / [K_o] \\
-89.6 &= 61 (\ln [K_i] - \ln [3]) \\
-89.6/61 &= \ln [K_i] - 0.48 \\
1.47 &= \ln [K_i] - 0.48 \\
1.95 &= \ln [K_i] \\
88.54 &= K_i
\end{aligned}$$

**67. The answer is c.** *(Kandel, pp 132–138.)* If the bathing solution is brought to 88 meq/L, the ionic concentrations outside and inside the membrane would be equal and, therefore, the membrane potential would be depolarized to zero.

**68. The answer is a.** *(Kandel, pp 132–138.)* In this situation, the roles of sodium and chloride ions were not of central importance. Temporal summation also cannot account for these findings and is thus irrelevant to the question at hand. The depolarization of 12.1 mV can be attributed to an increase in the concentration of potassium in the intracellular cleft.

**69. The answer is a.** *(Kandel, pp 132–138.)* Use the Goldman equation reduced to the Nernst equation. The resting membrane potential is −89.6 mV, $RT/F = 51$, the potassium concentration is 3 meq/L, and $K_i$ is calculated to be 88.54 mV.

$$\begin{aligned}
\text{Equilibrium potential} &= (RT/F) \times 61 \ln (K_o/K_i) \\
-89.6 - 12.1 &= 61 \times \ln (K_o - 88.54) \\
-77.5 &= 61 \times (\ln K_o - \ln 88.54) \\
-1.27 &= \ln K_o - \ln 88.54 \\
-1.27 - (-1.95) &= \ln K_o \\
+0.68 &= \ln K_o \\
4.79 \text{ meq/L} &= K_o
\end{aligned}$$

Therefore, the change in extracellular potassium would be

$$4.79 - 3.00 = 1.79 \text{ meq/L}$$

**70. The answer is d.** *(Kandel, pp 222–223.)* The trigger zone for the initiation of impulses from a neuron includes a specialized region of the cell body—the *axon hillock*—together with the section of the axon that adjoins this region—the *initial segment*. Other components of the neuron, such as the dendrites and cell body, receive inputs from afferent sources but are not capable of initiating impulses at these sites. The same is true concerning more distal aspects of the axon over which the impulse is conducted.

**71. The answer is a.** *(Kandel, pp 140–149.)* The time and space constants represent passive properties of a neuron. The electrical equivalent circuit utilizes the concept that a membrane has both capacitive and resistive

properties in parallel, in which case, the rising phase of a potential change is governed in part by the product of the resistance and capacitance of the membrane. The rising phase of a synaptic potential is governed by both active and passive properties of the membrane; however, the falling phase is regulated solely by the passive properties. As the time constant is increased, the probability of integration of converging synaptic signals is increased because such signals will be more likely to overlap in time (temporal summation).

**72. The answer is c.** *(Kandel, pp 105–123, 154–169.)* Sodium channels are rapidly opened following depolarization of the membrane. The rapid influx of ions results in a further depolarization of the membrane, which, in turn, can lead to an action potential. When the membrane is hyperpolarized, sodium channels are closed. Moreover, in the resting membrane, sodium channels are not activated. Tetraethylammonium is a drug that selectively blocks only potassium channels. Tetrodotoxin blocks sodium channels.

**73. The answer is b.** *(Kandel, pp 125–148.)* Because the membrane is a leaky one, the sodium-potassium pump serves an important function in actively transporting ions from one side of the membrane to the other. The membrane is permeable to ions other than potassium, such as sodium and chloride. This fact is taken into consideration in the Goldman equation. This equation includes the distribution of all of these other ions in its formula for determining the value of membrane potential. Accordingly, the resting membrane potential is dependent upon the concentration of these other ions as well as potassium. While it is true that the Nernst equation considers the relative distribution of potassium ions across the membrane, this statement, in itself, does not explain why the equilibrium potential for potassium differs from the resting potential of the neuron. The statement that the Nernst equation does not take into account differences in temperature is false. But, again, even if that statement were true, it would nevertheless not account for the differences between the equilibrium potential for potassium and the resting potential of the neuron.

**74. The answer is d.** *(Purves, pp 42–46.)* At rest, the cell typically generates a constant voltage across the membrane. The voltage is negative (inside), varying from approximately −40 to −90 mV, which is determined

by the relative concentrations of the different ions inside and outside the membrane.

**75. The answer is b.** *(Kandel, pp 175–295; Purves, pp 44–45, 99–114.)* If the cell membrane is permeable to only one ion such as sodium, and the concentration of this ion is equal on both sides of the membrane, then there will be no membrane potential recorded across the membrane. This is intuitively so as determined from the Nernst equation where both the numerator and denominator are the same, therefore generating the logarithm of 1, whose value is 0.

**76. The answer is c.** *(Kandel, pp 175–295; Purves, pp 44–45, 85–97, 99–114.)* Increased-conductance postsynaptic potentials are fast, graded potentials, lasting from several milliseconds to several seconds. If the potential is an EPSP, it depends upon a single class of ligand-gated channels for sodium and potassium. If the response is an IPSP, then it depends upon ligand-gated channels for potassium and chloride. Decreased-conductance postsynaptic potentials are mediated by a chemical transmitter or intracellular messenger to produce a graded, slow potential, lasting from seconds to minutes. This response is related to a closure of sodium, potassium, or chloride channels. Receptor potentials result from the application of a sensory stimulus that produces a fast, graded potential that involves a single class of channels for both sodium and potassium.

**77. The answer is e.** *(Purves, pp 43–74.)* The action potential is characterized by an all-or-none response in which the overshoot may reach an amplitude of up to 100 mV. The mechanism involves separate ion channels for sodium and potassium. The resting potential is characterized by a relatively steady potential, usually in the region of 270 mV, but which may range from 235 to 270 mV. This potential is mainly dependent upon potassium and chloride channels.

**78. The answer is d.** *(Kandel, pp 140–148.)* Electrical postsynaptic potentials involve the passive spread of current across a gap junction that is permeable to a variety of small ions. The stimulus for such activation may be a change in either voltage, pH, or intracellular calcium. Answers and explanations for questions 74 to 77 relate to the other choices presented.

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# The Synapse

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**79.** *Gap junctions* are characteristic of

a. Axodendritic synapses
b. Axoaxonic synapses
c. Axosomatic synapses
d. Dendrodendritic synapses
e. Electrical synapses

**80.** Which of the following characterizes a principal feature of axosomatic synapses?

a. It is referred to as a type I synapse
b. They have an electrical continuity linking the pre- and postsynaptic cells
c. They are typically inhibitory
d. Synaptic transmission is mediated by glutamate
e. They form the predominant synapse of cortical projections to the neostriatum

**81.** In a typical chemical synapse, which of the following constitutes the correct sequence of events involved in neurotransmission?

a. The action potential stimulates the presynaptic terminal → the presynaptic terminal is depolarized, opening voltage-gated $Ca^{2+}$ ion channels, causing an influx of these ions into the presynaptic terminal → release of the transmitter into the synaptic cleft by exocytosis → a postsynaptic current produces an excitatory postsynaptic potential (EPSP) or an inhibitory postsynaptic potential (IPSP), changing the excitability of the postsynaptic cell
b. Release of the transmitter into the synaptic cleft by exocytosis → an influx of $Ca^{2+}$ through channels, causing the vesicles to fuse with the presynaptic membrane → opening of the postsynaptic channels → binding of the transmitter to the receptor molecules in postsynaptic membrane
c. The vesicular membrane is retrieved from the plasma membrane → release of the transmitter into the synaptic cleft by exocytosis → the action potential stimulates the presynaptic terminal → binding of the transmitter to the receptor molecules in the postsynaptic membrane
d. Opening or closing of the postsynaptic channels → depolarization of the presynaptic terminal causing an opening of $Ca^{2+}$ ion channels → release of the transmitter into the synaptic cleft by exocytosis → synthesis and storage of the transmitter in the presynaptic terminal
e. The presynaptic terminal is depolarized, opening voltage-gated $Ca^{2+}$ ion channels, causing an influx of these ions into the presynaptic terminal → opening or closing of the postsynaptic channels → release of the transmitter into the synaptic cleft by exocytosis → the vesicular membrane is retrieved from the plasma membrane

**82.** Which of the following statements correctly characterizes synapses?

a. Synaptic vesicles constitute important features for transmission in both chemical and electrical synapses
b. A postsynaptic neuron typically receives input from different presynaptic axons that are either excitatory or inhibitory, but it cannot receive inputs from both types
c. Synaptic delay is approximately the same for both chemical and electrical synapses
d. Receptors can provide a gating function with respect to a given ion channel
e. The mechanism of indirect gating of ions normally does not involve the activation of G proteins

**83.** Which of the following properties or characteristics are correct in comparing γ-aminobutyric acid (GABA) with glycine?

a. Both are known to have inhibitory as well as excitatory properties
b. Both utilize a similar mechanism of gating of the chloride channel
c. GABA but not glycine utilizes receptors that are transmembrane proteins
d. Glycine is associated with the generation of seizure activity, but the effects of GABA are to block such activity
e. Glycine generates its most significant effects in the cerebral cortex while the effects of GABA are more restricted to the basal ganglia, brainstem, and spinal cord

**84.** Which of the following correctly describes the *N*-methyl-D-aspartate (NMDA) receptor?

a. It controls a high-conductance anion channel
b. The NMDA channel is easily blocked by the presence of magnesium
c. NMDA is selective for ionotropic receptors
d. Insufficient amounts of glutamate, acting through NMDA receptors, may cause neuronal cell death
e. Current flow is blocked in the presence of glutamate, leading to hyperpolarization of the cell

**85.** Which of the following is a second messenger system directly activated by the binding of norepinephrine to a beta-adrenergic receptor?

a. Inositol 1,4,5-triphosphate ($IP_3$)
b. Adenosine 3′,5′-cyclic phosphate (cAMP)
c. Diacylglycerol (DAG)
d. Arachidonic acid
e. Prostaglandins

**86.** Hyperpolarization of the neuron is governed by

a. Chloride and sodium
b. Chloride and potassium
c. Potassium and sodium
d. Sodium and calcium
e. Sodium only

**87.** The release of the transmitter is directly governed by

a. Sodium influx
b. Sodium efflux
c. Potassium influx
d. Potassium efflux
e. Calcium influx

**88.** Which of the following statements is appropriate to second messengers within neurons?

a. They have little effect upon receptors
b. They regulate gene expression that leads to neuronal growth and synthesis of new proteins
c. They generally do not interact in the opening or closing of ion channels
d. Glutamate always has excitatory effects upon metabotropic receptors
e. They are directly involved in the gating of sodium channels by NMDA receptors

**89.** *N*-methyl-D-aspartate (NMDA), kainate, and quisqualate all act on which of the following receptors?

a. GABA receptors
b. Excitatory amino acid receptors
c. Adrenergic receptors
d. Opioid receptors
e. Dopamine receptors

# The Synapse

## Answers

**79. The answer is e.** *(Kandel, pp 178–180; Purves, pp 99–101.)* Electrical synapses are less common than chemical synapses but can be found in the nervous systems of different species. A unique feature of electrical synapses is that two neurons communicate with each other by having the membranes of each neuron lie very close together. The contact between the neurons is called a *gap junction.* These junctions contain aligned paired channels so that each paired channel forms a pore larger than those observed in ligand-gated channels and which allows for the bidirectional transmission.

**80. The answer is c.** *(Kandel, pp 209–217; Purves, pp 1–8, 415–417.)* Axon terminals that make synaptic contact with the soma of postsynaptic cells are frequently observed to be inhibitory and are referred to as a *type II synapse.* A classic example of this is in the cerebellar cortex, where an interneuron (basket cell) makes synaptic contact with the soma of the Purkinje cell. These are chemical and not electrical synapses, and their actions are frequently mediated by GABA. Activation of the basket cell results in subsequent inhibition of the Purkinje cell. The overwhelming number of excitatory synapses are observed to be axodendritic. They are referred to as a *type I synapse* and are frequently characterized by specialized extensions of the dendrites called *spines.* These synapses also display a dense basement membrane and a prominent presynaptic density. Cortical projections to the neostriatum have been shown to be excitatory and their functions mediated by glutamate.

**81. The answer is a.** *(Purves, pp 101–104.)* The sequence of events that occur in the transmission of a chemical synapse is as follows: The transmitter is synthesized and stored in the presynaptic vesicles. The action potential is propagated down the presynaptic axon to its presynaptic terminal. The presynaptic terminal is then depolarized, which causes the opening of voltage-gated $Ca^{2+}$ channels. Then, there is $Ca^{2+}$ through these channels, causing the vesicles to fuse with the presynaptic membrane. The transmitter is then released into the presynaptic cleft (by exocytosis) and binds to recep-

tor molecules in the postsynaptic membrane. This leads to the opening or closing of postsynaptic channels. The resultant current results in an EPSP or IPSP, which causes a change in excitability of the postsynaptic cell. The vesicular membrane is then retrieved from the plasma membrane.

**82. The answer is d.** *(Kandel, pp 207–219; Purves, pp 99–114.)* Perhaps the most significant feature of the receptor is that it serves a gating function for particular ions. It can do this either directly, if it is part of the ion channel, or indirectly, by activating a G protein that, in turn, activates a second messenger system. This process results in a modulation of the ion channel's activity. In particular, the G protein stimulates adenylate cyclase, converting ATP to cAMP. In turn, cAMP induces activation of cAMP-dependent protein kinase, which modulates channels by phosphorylating the channel protein or some other protein that works on that channel. The synaptic vesicles may be round or flat, and filled or empty. They are typically filled with a neurotransmitter that is released onto the synaptic cleft. The receptive process on the postsynaptic region (i.e., the postsynaptic receptor) takes on a very important function. The binding of the transmitter to the receptor molecule is determined by this receptor, which is a membrane-spanning protein. When the transmitter is released onto the postsynaptic membrane, it leads to an action potential in the postsynaptic neuron (see the answer to question 81 for further details). In contrast, transmission at electrical synapses are mediated through gap junctions. Because the presynaptic and postsynaptic membranes of (gap junctions of) electrical synapses are connected by gap junction channels, electrical synapses function by means of the passive flow of ionic current through the gap junction from one neuron to the next. Postsynaptic neurons can receive both excitatory and inhibitory inputs. A classic example is a ventral horn motor neuron, which may receive an excitatory sensory input emanating from the same side of the body and an inhibitory input from the contralateral side. (See the chapter entitled "The Spinal Cord" for further discussion of this point.) Because of the nature of the difference in mechanisms for synaptic transmission and the relative sizes of the synaptic gaps, which are much smaller for electrical synapses, the synaptic delay for electrical synapses is much shorter than that of chemical synapses.

**83. The answer is b.** *(Kandel, pp 214–221; Purves, pp 128–131.)* Both GABA and glycine are inhibitory transmitters found in the spinal cord and

elsewhere in the central nervous system (CNS). Glycine was originally shown to be present in the spinal cord, but more recent studies have shown it to be present in the brain as well. However, its effects in the brain are believed to be much weaker than in the spinal cord. Both are inhibitory and act on a similar chloride channel, which, when activated, permits this ion to enter the cell and make it more negative (i.e., hyperpolarize the cell). Since both transmitters are inhibitory, it is assumed that their actions would be to inhibit seizure activity, although this has only been shown for GABA. Each of the channels is formed from a transmembrane protein. It contains a transmitter-binding site on the outer side of the membrane, and its conducting pore is embedded in the cell membrane. Another feature, that both channels produce electrical signals as a result of the movement of ions down their electrochemical gradients within their channels, is a feature common to both excitatory and inhibitory transmitters.

**84. The answer is b.** *(Kandel, pp 212–215; Purves, pp 127–129.)* The NMDA receptor regulates a channel permeable to several cations, which include calcium, sodium, and potassium. This channel, however, is easily blocked by magnesium. In fact, it requires a significant depolarization of the membrane in order for magnesium to be exuded from the channel so that sodium and calcium can enter the cell. Glutamate receptors can be divided into two categories: (1) metabotropic receptors that gate channels indirectly through second messengers and (2) ionotropic receptors that gate channels directly. One of the unusual features of this transmitter-gated channel is that it is also gated by voltage. Thus, conductance reaches its peak when both glutamate is present and the cell is depolarized. High concentrations of glutamate could result in death of the cell. This may be due to an unusually large influx of calcium through NMDA-activated channels. The calcium might activate proteases, resulting in the formation of free radicals that could be toxic to the cell. (See the discussion below for further discussion of NMDA receptors.)

**85. The answer is b.** *(Kandel, pp 181–185, 281–294; Purves, pp 103–111, 117–137.)* When norepinephrine reaches a β-adrenergic receptor, a G protein activates adenyl cyclase, which generates a second messenger, cAMP, from ATP. cAMP activates a cAMP-dependent kinase that alters the conformation of regulatory subunits of other kinases. This frees catalytic subunits to phosphorylate specific proteins, which, in turn, leads to the cellular

response. $IP_3$ and DAG are associated with the transmitter acetylcholine, which binds to muscarinic receptors, and arachidonic acid is linked to histamine, which binds to histamine receptors. Prostaglandins are metabolites of arachidonic acid.

**86. The answer is b.** *(Kandel, pp 181–185, 281–294; Purves, pp 103–111, 117–137.)* In neurons within the CNS, an inhibitory transmitter will open chloride channels. In addition, second messengers may also mediate inhibition. It is likely that they do so by opening potassium channels. When a chloride channel is opened, it will lead to movement of this ion down its concentration gradient and into the cell. This will make the cell more negative (i.e., hyperpolarized). At the same time, there will be an efflux of potassium, which will also produce hyperpolarization of the cell because positive charges are now being removed. On the other hand, sodium and calcium influx are associated with depolarization of the cell.

**87. The answer is e.** *(Kandel, pp 208–226, 253–276; Purves, pp 103–111.)* Experimental methods permit evaluation of the relative contributions of different ions in the regulation of transmitter release. Neither tetrodotoxin, which blocks voltage-gated sodium channels, nor tetraethylammonium, which blocks voltage-gated potassium channels, will block the generation of a postsynaptic potential when the presynaptic cell is artificially depolarized. In contrast, presynaptic calcium influx triggers the release of the transmitter and results in a postsynaptic potential. Moreover, when presynaptic calcium influx is blocked, no postsynaptic potential is produced. Action potentials at the presynaptic axon terminals open up calcium channels, permitting calcium influx. This event helps move synaptic vesicles to active sites as actin filaments (which anchor the vesicles) are dissolved.

**88. The answer is b.** *(Kandel, pp 182–185, 208–226, 253–276.)* Second-messenger kinases can lead to the phosphorylation of ion channel proteins. Such a process can lead to either the closing of a previously open ion channel or to the opening of a previously closed channel. For example, norepinephrine acts through cAMP to close the potassium channel, resulting in an increase in excitability. Second messengers can phosphorylate transcriptional regulatory proteins and thus alter gene expression. In particular, existing proteins may be altered and new proteins may be synthesized. Moreover, such effects may generate other alterations, such as the induc-

tion of neuronal growth. Second messengers can also interact directly with an ion channel to cause it to open or close (in the absence of a protein kinase). They also can produce a level of desensitization in receptors, which is a function of the extent of phosphorylation. While glutamate excites ionotropic receptors, it has a more diverse modulatory effect upon metabotropic receptors, which could be expressed in either receptor excitation or inhibition. The direct gating of ion channels by NMDA receptors is an example of a process that does not immediately involve a second messenger.

**89. The answer is b.** *(Kandel, pp 212–214; Purves, pp 153–159.)* NMDA, kainate, and quisqualate act upon excitatory amino acid receptors. The NMDA receptor differs from the other types of receptors in that it is blocked by $Mg^{2+}$ and controls a cation channel permeable to calcium, sodium, and potassium. Pharmacologically, NMDA receptors can be blocked by 2-amino-5-phosphonovaleric acid. The quisqualate receptor is activated by quisqualic acid; it has a high affinity for L-glutamate and α-amino-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA). The kainate receptor is activated by kainic acid. It regulates a channel that is permeable to sodium and potassium, binds AMPA, and is important in the process of excitotoxicity.

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# Neurochemistry/ Neurotransmitters

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**90.** In the biosynthesis of dopamine, the immediate precursor of dopamine is

a. Tyrosine
b. Tyrosine hydroxylase
c. Tryptophan
d. L-Dihydroxyphenylalanine (L-DOPA)
e. Dopamine β-hydroxylase

**91.** The rate-limiting step in the biosynthesis of dopamine is

a. Tryptophan hydroxylase
b. Tyrosine hydroxylase
c. Dopamine β-hydroxylase
d. Phenylethanolamine-*N*-methyl transferase
e. Choline acetyltransferase

**92.** A baby is born with an inherited autosomal recessive trait in which there is a delay in development, resulting in the occurrence of seizures and mental retardation. The child was diagnosed as having phenylketonuria (PKU). The likely neurochemical locus of this genetic defect is

a. Tyrosine
b. Tryptophan
c. Tryptophan hydroxylase
d. Dopamine
e. Phenylalanine (Phe) hydroxylase

**93.** Which of the following statements concerning the end-plate potential is correct?

a. It is dependent upon the release of dopamine from the nerve ending
b. The amplitude of this potential is much higher than central nervous system (CNS) postsynaptic potentials
c. It is an all-or-none response
d. It is unrelated to the concentration of transmitter released from the presynaptic terminals
e. It is selectively associated with the opening of chloride channels

**94.** The channel at the neuromuscular junction associated with the end-plate potential is

a. Blocked by a noradrenergic β-receptor antagonist
b. Blocked by an *N*-methyl-D-aspartate– (NMDA-) receptor antagonist
c. Blocked by an α-amino-hydroxy-5-methyl-4-isoxazolepropionic acid– (AMPA-) receptor antagonist
d. Nicotinic gated
e. Muscarinic gated

## Questions 95–96

The following case relates to the next two questions. After an individual is admitted to the hospital, it is determined that he displays a variable weakness of cranial nerve and limb muscles but shows no clinical signs of denervation from tests, which include electromyogram (EMG) recordings. This disorder was partially reversed by the administration of drugs that inhibit acetylcholinesterase.

**95.** The individual is likely to be suffering from

a. Multiple sclerosis (MS)
b. Amyotrophic lateral sclerosis (ALS)
c. Myasthenia gravis
d. Combined system disease
e. Muscular dystrophy (MD)

**96.** The likely basis for this disorder is the result of

a. The production of excessive quantities of acetylcholine (ACh)
b. The production of antibodies that act against nicotinic ACh receptors
c. A minor stroke involving the motor strip of the cerebral cortex
d. A vitamin B deficiency
e. Viral encephalitis

**97.** The neurotoxin, α-bungarotoxin, has been utilized as a valuable experimental tool because it binds to the

a. $GABA_A$ receptor
b. $GABA_B$ receptor
c. Nicotinic receptor
d. NMDA receptor
e. Histamine receptor

### Questions 98–99

The following case relates to the next two questions. An individual who complains about disruption in limb muscle function is diagnosed with a disorder in which the transmitter released at the neuromuscular junction is not removed from the synaptic cleft.

**98.** The primary mechanism involved in removal of the transmitter at the neuromuscular junction is

a. Enzymatic degradation
b. Diffusion
c. Reuptake
d. Actions of antibodies
e. Distribution of sodium and potassium ions along muscle membrane

**99.** The enzyme required for the metabolism of the transmitter at the neuromuscular junction is

a. Choline acetyltransferase
b. Glutaminase
c. Glutamine synthetase
d. Acetylcholinesterase
e. Serine hydroxymethyltransferase

## Questions 100–102

The following case relates to the next three questions. An individual is admitted to the emergency room of a hospital after taking a drug of abuse that destroyed selective groups of neurons in the brainstem. After the individual became ambulatory, he became chronically depressed.

**100.** Which of the following neuronal groups in the brainstem might be related, either directly or indirectly, to this person's condition?

a. Vestibular nuclei
b. Nucleus ambiguus
c. Trigeminal spinal nucleus
d. Dorsal column nuclei
e. Raphe nuclei

**101.** The neurotransmitter loss most likely linked to the symptoms is

a. Enkephalin
b. Dopamine
c. Norepinephrine
d. Serotonin
e. Glycine

**102.** An accepted approach toward the treatment of this disorder would be to administer

a. A serotonin reuptake inhibitor (SSRI)
b. A CNS depressant
c. A dopaminergic antagonist
d. A noradrenergic antagonist
e. An NMDA blocker

**103.** The immediate precursor of epinephrine is

a. Tyrosine
b. Dopamine
c. Norepinephrine
d. Melatonin
e. Phe

**104.** The rate-limiting step in the biosynthesis of serotonin is:

a. Tyrosine hydroxylase
b. Tryptophan hydroxylase
c. Phenylethanolamine-*N*-methyl transferase
d. Dopamine β-hydroxylase
e. Glutamic acid decarboxylase

## Questions 105–108

The following case relates to the next four questions. An elderly individual is admitted to a hospital after a long period in which the family had complained that he had showed increasing incidences of disorientation coupled with memory loss. The patient was diagnosed with Alzheimer's disease and a few years later, after further physical and mental deterioration, the patient died. An autopsy was taken of his brain and regional brain chemistry and neuropathology identified.

**105.** The likely sites where neuropathology was identified include

a. Cerebellar cortex, hypothalamus, red nucleus
b. Substantia nigra, midbrain periaqueductal gray, ventrolateral thalamus
c. Nucleus gracilis, deep pontine nuclei, vestibular nuclei
d. Cerebral cortex, basal nucleus of Meynert, hippocampus
e. Fastigial nucleus, subthalamic nucleus, superior colliculus

**106.** One neurotransmitter most often implicated in this disorder with respect to the affected brain regions is

a. Histamine
b. Substance P
c. ACh
d. Enkephalin
e. Dopamine

**107.** Upon examination of the brain, it was shown that the affected regions displayed

a. A decrease in substance P in the hypothalamus and brainstem reticular formation
b. Marked degeneration of most myelinated pathways
c. Amyloid deposits and neurofibrillary tangles
d. Marked retrograde degeneration in sensory neurons of the brainstem
e. Glial loss associated with the medial lemniscus and spinothalamic pathways

**108.** A therapeutic strategy for treatment of this disorder that shows promise is

a. Surgical removal of selective regions of the cerebral cortex
b. Administration of serotonergic agonists that act specifically on cerebral cortical neurons
c. Administration of cholinergic antagonists directed against nicotinic receptors in the cerebral cortex
d. Administration of noradrenergic agonists directed against $\alpha_2$ receptors in the cerebral cortex
e. Administration of compounds that slow aggregation of amyloid-$\beta$ peptide into its fibrillar form

**109.** The most ubiquitous excitatory neurotransmitter in the brain is

a. ACh
b. Glutamate
c. Norepinephrine
d. Dopamine
e. Substance P

## Questions 110–111

The following case relates to the next two questions. An individual is admitted to the emergency room and is diagnosed as having cortical damage and resultant neuronal degeneration due to an ischemic insult. The neurologist concluded that the brain damage involved neurotoxicity of those cells.

**110.** The neurotransmitter change associated with neurotoxicity in this case is believed to involve

a. Extracellular accumulation of norepinephrine
b. Extracellular accumulation of ACh
c. Extracellular accumulation of glutamate
d. Extracellular loss of serotonin
e. Extracellular loss of GABA

**111.** The likely mechanism underlying neurotoxicity as a result of ischemia involves

a. Entry of $Ca^{2+}$ into the cell
b. Reduction of extracellular chloride
c. Delayed removal of norepinephrine from the synapse
d. Hypersensitivity of the postsynaptic membrane to GABA
e. Failure of degradation of ACh

### Questions 112–113

The following case relates to the next two questions. A 50-year-old man suffers from anxiety attacks.

**112.** To treat this disorder, the patient would most likely be treated with

a. Picrotoxin
b. Naloxone
c. Chlordiazepoxide
d. Bicuculline
e. Dopamine

**113.** The mechanism underlying the action of this drug of choice is

a. Blockade of chloride channel permeability
b. Opioid receptor blockade
c. Binding of the drug to the GABA benzodiazepine site
d. Activation of muscarinic cholinergic receptor
e. Competitive binding of the $GABA_A$-receptor site

### Questions 114–115

The following case relates to the next two questions. A 55-year-old female patient is admitted to the hospital for treatment of hypertension.

**114.** To treat this disorder, the patient was administered

a. Yohimbine
b. Clonidine
c. Sodium lactate
d. Cholecystokinin
e. Carbon dioxide

**115.** The treatment of this combination of symptoms by the appropriate drug (listed in question 114) is mediated by its actions on

a. Muscarinic ACh receptors
b. Dopaminergic receptors
c. $\alpha_2$-adrenergic receptors
d. Serotonergic receptors
e. GABA receptors

### Questions 116–117

The following case relates to the next two questions. As a result of a leg injury, a 30-year-old male developed chronic pain and was subsequently treated with morphine. Consequently, he developed an addiction to morphine.

**116.** The predominant site where this effect is mediated is the

a. Opioid nociceptin receptor
b. Opioid $\mu$ receptor
c. Opioid $\delta$ receptor
d. Opioid $\kappa$ receptor
e. Dopamine $D_2$-receptor

**117.** A region of the brain where this receptor has been shown through extensive research to be heavily concentrated is the

a. Mammillary bodies
b. Precentral gyrus
c. Midbrain periaqueductal gray
d. Inferior olivary nucleus
e. Deep pontine nuclei

### Questions 118–119

The following case relates to the next two questions. A 65-year-old male was admitted to the emergency room and was diagnosed with a stroke.

**118.** A drug that might be given to the patient in order to reduce the deleterious effects of the stroke would be a

a. GABA antagonist
b. Dopamine antagonist
c. Norepinephrine antagonist
d. NMDA antagonist
e. Serotonin antagonist

**119.** Drug treatment suggested from the answer to the previous question could be effective by

a. Raising the blood pressure
b. Attempting to decrease disruption of the blood-brain barrier
c. Reducing seizure activity in the cerebral cortex and limbic system
d. Stabilizing body temperature
e. Reducing brain serotonin levels in the brainstem and cerebral cortex

### Questions 120–121

The following case relates to the next two questions. A patient is admitted to the hospital after experiencing increasing episodes of temporal lobe seizure activity.

**120.** To treat this disorder, which of the following drugs should be administered?

a. Physostigmine
b. Bicuculline
c. Pilocarpine
d. Kainate
e. Vigabatrin

**121.** The reason why the drug (selected from among the choices in the previous question) can be effective is

a. It blocks NMDA receptors
b. It activates noradrenergic receptors
c. It activates GABA receptors
d. It activates cholinergic receptors
e. It blocks dopamine receptors

**122.** Nitric oxide is synthesized from

a. Glutamate
b. Choline
c. L-arginine
d. Tyrosine
e. Tryptophan

**123.** Nitric acid differs from other "classical" neurotransmitters in that

a. Nitric acid is a gaseous transmitter
b. Nitric acid has both excitatory and inhibitory functions
c. Nitric acid occurs only in response to injury
d. The distribution of nitric acid is limited to the peripheral nervous system
e. Nitric acid is packaged in vesicles

**124.** A 60-year-old male has high blood pressure and the diagnosis indicates that it is due in part to retention of water. Which of the following compounds would most likely relate to this process?

a. Oxytocin
b. Serotonin
c. Histamine
d. Vasopressin
e. Somatostatin

**125.** A 65-year-old man has been experiencing considerable pain due to a chronic back problem. If the patient is administered morphine to alleviate the problem, a possible mechanism by which morphine would provide effective action includes

a. Release of somatostatin
b. Release of histamine
c. Release of vasopressin
d. Release of ACh
e. Release of substance P

**126.** A receptor that requires the simultaneous binding of two different agonists for activation is

a. L-AP4 receptor
b. Kainate receptor
c. NMDA receptor
d. AMPA receptor
e. $GABA_A$ receptor

**127.** A 16-year-old boy takes the drug of abuse, phencyclidine (PCP). The deleterious effects of the drug are due in part to

a. Blockade of NMDA receptors
b. Blockade of AMPA receptors
c. Blockade of cholinergic receptors
d. Blockade of $GABA_A$ receptors
e. Blockade of $GABA_B$ receptors

**128.** Epileptiform activity is believed to include the activation of

a. GABA receptors
b. Glutamate receptors
c. Nicotinic receptors
d. Serotonin receptors
e. Glycine receptors

**129.** Monoamines differ from neuroactive peptides in which of the following ways?

a. Monoamines are synthesized only in the cell body of neurons
b. Synthesis of monoamines is governed by messenger RNA (mRNA) on ribosomes, which is not true for neuroactive peptides
c. Monoamines are generally synthesized as part of a larger precursor molecule, called a *prohormone*
d. Monoamine neurons are generally regarded as having only excitatory properties, while peptides are inhibitory
e. Monoamine neurons are principally found within brainstem nuclei, while peptide-containing neurons are found throughout the brain

**130.** An enzyme that is directly responsible for the degradation of norepinephrine is

a. Tryptophan hydroxylase
b. Tyrosine hydroxylase
c. Dopamine β-hydroxylase
d. Catechol-*O*-methyltransferase
e. Choline acetyltransferase

**131.** A 50-year-old woman has been treated over the past 6 months with lithium for an ongoing disorder. The woman was most likely suffering from

a. Panic attacks
b. Schizophrenia
c. Epilepsy
d. Bipolar disorder
e. Anxiety

**132.** A long-lasting depletion of norepinephrine can be produced by administration of

a. Amphetamine
b. Apomorphine
c. Clonidine
d. Reserpine
e. Yohimbine

**133.** In a recent study, attenuation of release of catecholamine was noted when an agonist was administered to the experimental preparation. These results are best understood in terms of

a. The presence of a GABAergic neuron at the synapse
b. Postsynaptic inhibition
c. The presence of presynaptic autoreceptors
d. Destruction of the catecholamine cell body
e. Collateral inhibition

**134.** Removal of norepinephrine from the region of the synaptic cleft may be achieved by which of the following mechanisms?

a. Reuptake
b. Enzymatic degradation
c. Diffusion
d. A combination of enzymatic degradation and diffusion
e. A combination of enzymatic degradation, diffusion, and reuptake

# Neurochemistry/ Neurotransmitters

## Answers

**90. The answer is d.** *(Siegel et al., pp 244–246; Kandel, pp 282–284.)* The biosynthesis of catecholamines includes the following steps: tyrosine is converted into L-dihydroxyphenylalanine (L-DOPA) by tyrosine hydroxylase. L-DOPA is then decarboxylated by a decarboxylase to form dopamine (and $CO_2$). The conversion of dopamine to norepinephrine comes about by the action of the enzyme dopamine β-hydroxylase. The rate-limiting enzyme in the biosynthesis of serotonin is tryptophan hydroxylase. In this process, tryptophan is converted to 5-hydroxytryptophan by tryptophan hydroxylase and by 5-hydroxytryptophan decarboxylase into serotonin.

**91. The answer is b.** *(Kandel, pp 282–284; Siegel et al., pp 284–285.)* As indicated above (answer to question 90), the rate-limiting step in the biosynthesis of dopamine is tyrosine hydroxylase, which converts tyrosine into L-DOPA. The rate-limiting step in the biosynthesis of serotonin is tryptophan hydroxylase. The enzyme, dopamine β-hydroxylase, converts dopamine to norepinephrine. Phenylethanolamine-*N*-methyl transferase is involved in the conversion of norepinephrine to epinephrine. Choline acetyltransferase is involved in the biosynthesis of ACh.

**92. The answer is e.** *(Kandel, pp 36–37; Siegel et al., pp 244–245; Gilroy, pp 370–371.)* Phenylketonuria results in severe mental retardation and is caused by a defect in the gene that provides the code for Phe hydroxylase, the enzyme that converts Phe to tyrosine. As a result of this defective gene, there is an abundance of Phe in the brain, which produces a toxic metabolite, thus interfering in brain development and maturation.

**93. The answer is b.** *(Kandel, pp 187–197.)* The amplitude of the end-plate potential differs from that of postsynaptic potentials observed in the CNS in that end-plate potentials can be as great as 70 times larger than CNS postsynaptic potentials. The end-plate potential is dependent upon the release of ACh from the nerve endings. Dopamine is not released at the

neuromuscular junction. The end-plate potential is a graded potential, and is not an all-or-none response. Another important feature of this potential is that it is directly related to the quantity of neurotransmitter (ACh) released from the presynaptic terminals. The release of ACh onto the muscle membrane is associated with the opening of sodium and potassium channels, and not chloride channels.

**94. The answer is d.** *(Kandel, pp 196–198.)* The transmitter at the neuromuscular junction is ACh and its actions are mediated by the nicotinic ACh-gated channel. As noted above, it produces the end-plate potential by permitting the passage of both sodium and potassium ions. Noradrenergic, muscarinic, and excitatory amino acid receptors are not known to function at the neuromuscular junction.

**95–96. The answers are 95-c, 96-b.** *(Kandel, pp 298–304; Gilroy, pp 623–639, 640–642; Siegel et al., pp 871–883.)* Myasthenia gravis is an autoimmune disease that causes cranial nerve and limb muscle weakness by producing antibodies that act against the nicotinic receptor at the neuromuscular junction. The result is that the action of nerve fibers that innervate skeletal muscle are affected, producing loss of the effects of ACh at the neuromuscular junction. The net result is a reduction of the size of the action potential in the muscle, producing a weakness in the affected muscle. This disorder is reversed by administration of drugs that inhibit the enzyme, acetylcholinesterase, that degrades ACh. Multiple sclerosis, ALS, and combined system disease (see the chapter entitled "The Spinal Cord") involve damage to axons and/or nerve cells within the CNS, producing much more profound damage to motor functions and, in the case of combined system disease, damage to both motor and sensory systems. Muscular dystrophy is typically characterized, in part, by progressive weakness of muscles and degeneration of the muscle fibers. The other disorders listed all involve disorders affecting the CNS, and thus, the symptoms associated with these disorders differ significantly from those described in this case. Excessive release of ACh is not a realistic event that is likely to occur (except from the bite of a black widow spider). In theory, if it were to occur, there is no reason to believe that muscular weakness would be a symptom. Instead, there would be some rigidity and muscle spasms.

**97. The answer is c.** *(Siegel et al., pp 874–877.)* The α-toxins, including α-bungarotoxin, can produce postsynaptic effects similar to that observed with curare, by binding specifically to the α-subunits of the nicotinic ACh receptor. In the case of the neuromuscular junction, the binding is to α-subunit of the nicotinic ACh receptor. Because of the selective actions of α-bungarotoxin upon the ACh receptor, it has been used effectively as an experimental tool to study the properties and actions of ACh and its associated receptors.

**98–99. The answers are 98-a, 99-d.** *(Kandel, pp 107–112, 294–295; Siegel et al., pp 342–343, 877–879.)* There are three basic mechanisms by which the transmitter is removed from the synaptic cleft: (1) enzymatic degradation, (2) reuptake, and (3) diffusion. In the case of the neuromuscular junction, ACh (and not glutamate) is the neurotransmitter and the primary mechanism involves enzymatic degradation. The enzyme involved is acetylcholinesterase, which helps break down ACh into acetate and choline. Choline is then taken up by the presynaptic terminal. Concerning the other choices, choline acetyltransferase is the enzyme involved in the synthesis of ACh, glutaminase, and glutamine synthetase are involved in the formation of glutamate from glutamine and glutamine from glutamate, respectively. Serine hydroxymethyltransferase is the enzyme that converts serine into glycine.

**100–102. The answers are 100-e, 101-d, 102-a.** *(Siegel et al., pp 264–287; Kandel, pp 280–295.)* There is an increasing body of evidence that reductions in serotonin levels play an important role in depressive disorders. The raphe neurons, located along the midline of the brainstem, provide the basic sites of serotonergic neurons that project to all parts of the brain and spinal cord. The other choices refer to structures that concern motor and/or sensory functions mainly associated with cranial nerves. Since the raphe neurons were damaged, the neurotransmitter most likely responsible for the onset of depression in this instance is serotonin. It is possible that other transmitter systems, such as the catecholamines, may also play a role in this disorder; however, they would not be chiefly responsible for the disorder in this instance because of the restricted locus of the lesion. Recent practice has been to treat depression with serotonin reuptake inhibitors such as fluoxetine (Prozac), which has been found to be effective

after several weeks of treatment. The other choices for question 102 would be inappropriate because they would likely have a depressant effect on CNS functions.

**103. The answer is c.** *(Kandel, pp 282–284; Siegel et al., pp 244–246, 268–270.)* Tyrosine is the amino acid substrate from which dopamine, norepinephrine, and epinephrine are formed. Tyrosine is converted into L-DOPA by tyrosine hydroxylase. L-DOPA is decarboxylated by a decarboxylase into dopamine (and $CO_2$). Dopamine is converted into norepinephrine by dopamine β-hydroxylase. Norepinephrine is converted into epinephrine by phenylethanolamine-*N*-methyl transferase. Melatonin is formed from serotonin.

**104. The answer is b.** *(Kandel, pp 281–287.)* The rate-limiting step or controlling reaction in the biosynthesis of serotonin is tryptophan hydroxylase, which converts tryptophan into 5-hydroxytryptophan. Three of the other enzymes listed as choices—tyrosine hydroxylase, phenylethanolamine-*N*-methyl transferase, and dopamine β-hydroxylase—are, as noted in the explanation to the previous question, involved in the biosynthetic pathways for norepinephrine and epinephrine. Glutamic acid decarboxylase is utilized in the synthesis of GABA.

**105–108. The answers are 105-d, 106-c, 107-c, 108-e.** *(Kandel, pp 1151–1157; Siegel et al., pp 950–965.)* The primary regions shown to be affected by Alzheimer's disease include the basal nucleus of Meynert, (which contains cholinergic neurons that project widely to the forebrain, including the cerebral cortex), the hippocampal formation, and the cerebral cortex. The other choices included structures that have not been significantly implicated in this disorder. The neurotransmitter that has been most implicated in this disorder is ACh. Alzheimer's brains have been shown to have reduced levels of ACh and cholinergic markers, especially after damage to cholinergic neurons of the basal nucleus of Meynert. While reductions in other neurotransmitter levels may also occur, the other choices of neurotransmitters presented have not been clearly implicated in this disorder. One of the clearest neuropathological characteristics of Alzheimer's disease is the presence of amyloid deposits and neurofibrillary tangles in the cerebral cortex. In fact, there has been a new and promising strategy that has been applied for the treatment of Alzheimer's disease. It

involves the attempt to administer small molecules that retard the aggregation of amyloid-β peptides that form fibrillar amyloid plaques, which affect the normal functions of neurons.

**109. The answer is b.** *(Purves, pp 153–155; Siegel et al., pp 328–332.)* The largest numbers of excitatory synapses in the CNS are mediated by glutamate as it is believed that approximately half of the synapses in the brain release glutamate. For example, functions mediated by fibers that originate from the cerebral cortex and descend to such regions as the neostriatum, thalamus, brainstem, and spinal cord are generally believed to be mediated by glutamate. Many other neuronal systems throughout the brain and spinal cord utilize glutamate as well. Dopaminergic and noradrenergic neurons, while mostly excitatory, can also be inhibitory at some synapses and are less numerous than glutamate. Cholinergic and substance P synapses are also excitatory, but are likewise less numerous than glutamate.

**110–111. The answers are 110-c, 111-a.** *(Purves, p 130; Siegel et al., pp 328–332.)* It has been discovered that one mechanism of neurodegeneration involves prolonged activation of neurons by glutamate. It is believed that if glutamate accumulates in the extracellular space and is not removed, the presence of glutamate will effectively stimulate the neuron to death. It has been shown that neurotoxicity is linked to cell death after a stroke, which causes brain ischemia and oxygen deprivation. Glutamate receptors are involved in ischemic cell damage in the following way: Glutamate released from the presynaptic terminal would normally activate NMDA and AMPA receptors in the postsynaptic membrane. This results in an increase in the intracellular concentration of $Ca^{2+}$, which remains long after the initial stimulus is removed, and thus prevents the cell from reestablishing a resting membrane potential. The net effect here is to produce injury (or death) to the cell.

**112–113. The answers are 112-c, 113-c.** *(Siegel et al., pp 336–340.)* One of the strategies used effectively for the treatment of anxiety disorders is to use classes of drugs that suppress CNS activity. One such class includes benzodiazepine agonists, such as chlordiazepoxide. This drug enhances GABA transmission by binding to the benzodiazepine site on the $GABA_A$-receptor benzodiazepine chloride ionophore complex. In this manner, it acts as a GABA agonist, producing anxiolytic, sedative, and anticon-

vulsant effects. The other choices for both questions relate to drugs that have opposite effects, namely, ones with excitatory effects on CNS neurons.

**114–115. The answers are 114-b, 115-c.** *(Kandel, pp 1214–1222; Siegel et al., pp 1080–1086.)* Clonidine has long been used effectively for the treatment of hypertension. The other choices listed are compounds that can induce panic attacks and are, therefore, inappropriate for the treatment for this patient. Clonidine is an adrenergic agonist whose functions are mediated by its actions upon $\alpha_2$ receptors. Administration of this drug results in an overall decrease in noradrenergic transmission. Although the precise mechanism by which the effects of clonidine become manifest is unknown, it may be that it reduces noradrenergic transmission by acting upon $\alpha_2$ presynaptic receptors, which typically produce autoinhibition of the noradrenergic pathways.

**116–117. The answers are 116-b, 117-c.** *(Kandel, pp 480–484; Siegel et al., pp 1096–1098.)* Research conducted over the past 2 decades has shown that the actions of morphine are mediated through opioid $\mu$ receptors, while other opioid receptors appear not to play a significant role. Likewise, dopamine receptors are not involved in this process. The region of the brain where concentrations of opioid receptors are very heavily concentrated is the midbrain periaqueductal gray. This region plays an important role in the modulation of pain and is particularly responsive to opioid activation by morphine. While other areas of the brain indicated in question 117 may also contain opioid receptors, concentrations of this receptor are not known to be high (including a structure such as the mammillary bodies for which no known functions have been identified). Moreover, none of these regions are known to play any role in the regulation of pain.

**118–119. The answers are 118-d, 119-b.** *(Siegel et al., pp 715–721.)* As mentioned earlier in the explanations for questions 110 and 111, glutamate has been implicated in ischemia-induced brain damage following brain trauma such as a stroke. It has also been shown that administration of NMDA-receptor antagonists following a stroke is effective in the treatment of stroke by reducing tissue infarction and neuronal cell death. The other choices listed in question 118 are not known to relate to the reversal of the deleterious effects of stroke. The NMDA-receptor antagonist is effective, in part, by decreasing disruption of the blood-brain barrier. It has

been suggested that this becomes manifest by a blockade of the neuronal production of reactive oxygen species that occurs as a result of activation of NMDA receptors. Again, the other choices listed for question 119 have no known relationship to the process in question.

**120–121. The answers are 120-e, 121-c.** *(Siegel et al., pp 755–763.)* One of the drugs that has been used effectively for the treatment of epilepsy, especially complex partial seizures involving the temporal lobe, has been vigabatrin. The other choices of drugs are ones that enhance convulsive activity either by facilitating excitatory transmitter function or by inhibiting inhibitory transmitter functions. Vigabatrin functions by enhancing GABA-mediated inhibition of neurons, perhaps by the inhibition of GABA-transaminase.

**122. The answer is c.** *(Kandel, p 295; Purves, pp 173–175, 460, 465; Siegel et al., pp 442–443.)* Nitric oxide is synthesized from L-arginine when stimulated by nitric oxide synthase. Choline is a precursor of ACh, tyrosine of dopamine and norepinephrine, and tryptophan of serotonin. Glutamate, a neurotransmitter, is synthesized from glutamine, and can be converted into GABA by glutamic acid decarboxylase.

**123. The answer is a.** *(Purves, pp 173–175; Kandel, p 295; Siegel et al., pp 442–444, 725–727.)* Nitric acid differs from more classical, or traditional, neurotransmitters in that, in addition to acting as a neurotransmitter, it is a gas and also acts as a second messenger. After nitric oxide is formed, it diffuses locally and interacts with specific molecules such as the enzyme catalyzing cyclic guanosine 5′-monophosphate (cGMP) synthesis, guanylyl. A number of different neurotransmitters can have either excitatory or inhibitory effects, depending upon the receptors with which they interact. Since nitric acid is coupled to a variety of neurotransmitter systems, it is likely that it is also involved in both excitatory and inhibitory processes. Recent studies have shown that nitric acid is likely involved in a wide variety of processes and is not limited to a single function. Moreover, it is widely distributed throughout both the CNS as well as in the peripheral nervous system. While many transmitters are packaged in synaptic vesicles, nitric acid differs by diffusing widely without being packaged in synaptic vesicles.

**124. The answer is d.** *(Kandel, pp 978–980.)* Vasopressin is produced mainly from the magnocellular neurons of the hypothalamus. The hormone is released into the capillaries of the posterior pituitary. When it is released into the vascular system, it stimulates the kidneys to conserve water. The action of oxytocin is related to functions of the uterus and breasts. This hormone plays a role in the expulsion of the fetus at birth and in the milk ejection reflex following suckling. Substance P, histamine, and somatostatin are not known to relate specifically to this process.

**125. The answer is b.** *(Siegel et al., pp 294–310.)* When an opioid compound, especially a μ-receptor agonist (such as morphine), is administered in response to chronic pain, this causes the release of histamine in neurons. This leads to the activation of histamine $H_2$ receptors, which play a role in the relief of pain. In fact, there are ongoing attempts now to develop drugs, such as histamine $H_3$-receptor compounds, which have been shown to mediate antinociception and have anti-inflammatory properties as well. The other choices listed in this question are not known to relate to the alleviation of pain, in particular, with respect to morphine administration. In fact, substance P is associated with the elicitation of pain impulses.

**126. The answer is c.** *(Siegel et al., pp 315–325.)* NMDA receptors are unique among receptors in that they require the simultaneous binding of two different agonists for their activation. NMDA ion channels are opened after such compounds as glutamate and glycine are applied to the membranes that include NMDA receptors. Recent evidence has shown that a metabotropic glutamate receptor, L-AP4, is present in the retina. Activation of this receptor may serve to hyperpolarize bipolar neurons within the retina. Glutamate activation (of this receptor) constitutes an unusual action because most neurons in the CNS are depolarized by glutamate. AMPA is one of several classes of ionotropic glutamate receptors and functions as a synaptic receptor for fast excitatory synaptic transmission mediated through glutamate. The other choices, kainate and GABA receptors, do not have this property.

**127. The answer is a.** *(Siegel et al., pp 319–321.)* NMDA ion channels are opened by both glutamate and glycine. On the other hand, $Mg^2$ generates a voltage-dependent block of this ion channel. The drug of abuse, phencyclidine (PCP), also utilizes a similar mechanism to block NMDA-receptor

channels. The other choices do not relate to this mechanism with respect to PCP.

**128. The answer is b.** *(Siegel et al., pp 326–333.)* Excitatory amino acids and, in particular, the glutamate family of compounds have long been thought to play an important role in epileptiform activity. Epileptiform activity typically includes AMPA-receptor activation. However, as the seizure becomes more intense, there is increased involvement of NMDA receptors. This is evidenced by the facts that NMDA antagonists can reduce the intensity and length of the seizure activity and that, following removal of human epileptic hippocampal tissue, there is an up-regulation of both AMPA and NMDA receptors. Metabotropic glutamate receptors have been shown to be present in the retina but have not yet been demonstrated to be present in regions of the brain that are typically epileptogenic. GABA and glycine are inhibitory transmitters; therefore, seizures would logically block such receptor activation. There has been no substantive evidence concerning the role of cortical nicotinic receptors in epilepsy.

**129. The answer is e.** *(Purves, pp 117–137.)* Peptides differ from other neurotransmitters in several ways. Monoamines can be formed in all parts of the neuron with the completion of synthesis in the nerve terminal. In contrast, peptides are formed as a result of mRNA that is directed upon ribosomes, thus limiting the site of synthesis to the cell body where the processing is accomplished by the endoplasmic reticulum and Golgi apparatus. Typically, different neuroactive peptides are cleaved from a single, much larger molecule (a prohormone) that has no biologic activity. The active peptide is cleaved by specific peptidases and is ultimately transported down the axon to the nerve terminal. In addition, the overwhelming majority of monoamine neurons is situated in the brainstem, while neuroactive peptides can be found over widespread regions of both the brainstem and forebrain, and, in particular, the limbic structures. Both monoamines and peptides may display inhibitory as well as excitatory properties. For example, enkephalins are generally inhibitory, while substance P neurons are excitatory. Monoamine neurons may have excitatory effects in one region of the brain and inhibitory effects in another region.

**130. The answer is d.** *(Kandel, pp 280–286; Purves, pp 127–137.)* Tryptophan hydroxylase, tyrosine hydroxylase, and choline acetyltransferase are

enzymes that are critical for the biosynthesis of serotonin, catecholamines, and ACh, respectively. Dopamine β-hydroxylase converts dopamine to norepinephrine. Catechol-*O*-methyltransferase and monoamine oxidase are critical for the metabolic degradation of catecholamines.

**131. The answer is d.** *(Siegel et al., pp 1080–1082; Kandel, pp 1213–1216.)* Lithium has been used for a number of years as an effective drug for the treatment of bipolar disorders. It has been shown to decrease the length, severity, and recurrence of manic states as well as the depressive components of this disorder. The mechanism of action of lithium in effectively combating bipolar disorder is not absolutely clear since it has a wide variety of biological effects. In part, these include: changes in the expression of some G proteins and subtypes of adenyl cyclase, alteration of the coupling of G proteins to neurotransmitter receptors, alterations of monoamine levels and receptors, and effects upon ion channels. Monoaminergic drugs are generally used for the treatment of panic disorders and, to some extent, to treat anxiety. Anxiety attacks are also treated with benzodiazepine drugs. Drugs for the treatment of epilepsy generally include those that increase or maintain GABA levels or decrease glutamate levels. For schizophrenia, a wide range of drugs has been used; these include those which affect monoaminergic, cholinergic, and GABAergic systems. See question 453 and its answer for further discussion.

**132. The answer is d.** *(Kandel, pp 280–286; Purves, pp 127–137.)* Reserpine interferes with the uptake-storage mechanism associated with amine granules, which results in destruction of these granules. Administration of this drug will produce long-lasting depletion of norepinephrine. Amphetamine blocks the reuptake mechanism and, thus, produces a net increase in the release of norepinephrine. Apomorphine is a nonspecific dopamine agonist; clonidine is an $\alpha_2$-receptor agonist, and yohimbine is an $\alpha_2$-receptor antagonist.

**133. The answer is c.** *(Kandel, pp 280–286; Siegel et al., pp 247, 1078; Purves, pp 127–137.)* These findings can best be explained in terms of a mechanism that involves presynaptic autoreceptors. These receptors modulate the release of a catecholamine by responding to the concentration of this transmitter within the synapse. It thus represents a specific negative feedback mechanism. For example, if the concentration of transmitter in

the synapse is high, then release will likely be inhibited. Less inhibition (i.e., more transmitter release) will occur if concentrations are low. Other choices are obviously incorrect. The presence of a GABAergic neuron at the synapse, postsynaptic inhibition, and collateral inhibition are unrelated since they refer to events that are associated with the postsynaptic neuron, not the catecholamine (presynaptic) neuron. As a result of the phasic nature of this phenomenon, destruction of the catecholamine cell body would produce events that were not phasic; indeed, there would be permanent loss of the neuron's capacity to release transmitter.

**134. The answer is e.** *(Kandel, pp 281–286; Purves, pp 120–135.)* There are three mechanisms by which a transmitter is removed from the region of the synaptic cleft. The most common one is reuptake, in which transporter molecules mediate high-affinity reuptake that is specific for the transmitter in question. Other mechanisms include diffusion, which removes some components of the transmitter substance, and enzymatic degradation of the amine achieved by the enzymes monoamine oxidase and catechol-*O*-methyltransferase.

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# The Spinal Cord

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**135.** A college student received an injury as a result of being tackled in a football game. After the game, the student was treated at a local hospital and was found to be unable to abduct and rotate the left arm at the shoulder, flex the elbow, and extend the wrist of the left side. Upon further examination, testing revealed depression of the biceps reflex of this limb, but the reflex activity involving the other limbs was normal. The most likely site of the injury is

a. Precentral gyrus
b. Basilar pons
c. Ventral horn cells at C1
d. Nerve roots of C5-C6
e. Triceps muscle

**136.** A neurological examination of a 75-year-old male reveals that when the abdominal wall is stroked, the muscles of the abdominal wall of the side of the body stimulated failed to contract. Other neurological tests appeared normal. The likely region affected includes

a. C1–C5 spinal segments
b. C6–T1 spinal segments
c. T2–T7 spinal segments
d. T8–T12 spinal segments
e. L1–L5 spinal segments

**137.** A 65-year-old female finds that she has weakness in attempting to flex her left knee and extend the hip. Neurophysiological analysis of the affected regions revealed a reduced number of motor units firing with fasciculations and slowed conduction velocity. There was no depression of tendon reflexes or muscle wasting. Likewise, plantar and abdominal reflexes were normal, and there was little sensory loss nor any signs of sphincter disturbances. The disturbances experienced by this woman are probably due to

a. Peripheral neuropathy of nerves on the left side of the body that exit the spinal cord at L4–S1
b. Damage of the neuromuscular junctions associated with nerves that exit the left side of spinal cord between T8–L3
c. Degeneration of nerve cells in the ventral horn of the left side of the spinal cord between T8–T12
d. Degeneration of fibers contained in the lateral funiculus of the left side of the thoracic spinal cord
e. Damage to the dorsal horn of the spinal cord of the left side between L1–L4

**138.** A 55-year-old man discovered that he had pain in the neck and right arm and weakness in extending his fingers of his right hand with loss of sensation in the right thumb and middle fingers. A neurological examination further revealed a weakness of the right biceps reflex, but other neurological signs could not be detected. The most likely diagnosis of this individual is

a. Syringomyelia involving the cervical cord
b. A knife wound of the right arm completely severing nerves innervating the biceps muscle
c. Prolapse of a cervical disk
d. Poliomyelitis involving the cervical cord
e. AIDS

**139.** A 60-year-old woman was hospitalized with a severe respiratory infection for several weeks. Afterward, she displayed symptoms of myalgia and weakness of the lower limbs. In addition, she also showed loss of muscle tone and some flaccidity with loss of tendon reflexes. Examination also revealed a weakness of facial muscles. This constellation of symptoms progressed for approximately 2 weeks and persisted for more than a year, at which time, recovery took place at a slow rate. There was also some demyelination coupled with lymphatic inflammation at the site of demyelination. The most likely cause of this patient's condition is

a. Myasthenia gravis
b. Muscular dystrophy (MD)
c. Multiple sclerosis (MS)
d. Guillain-Barré syndrome
e. Lumbar disk prolapse

### Questions 140–141

The following case relates to the next two questions. A 46-year-old man finds that, over a period of time, he has progressive bilateral weakness of both upper and lower limbs beginning with the muscles of the hands. However, testing reveals that sensory functions appear normal. Eventually, this individual is found to have wasting of muscles, fasciculations, and evidence of upper motor neuron (UMN) dysfunction together with an increase in tendon reflexes. After a few additional months, the patient develops facial weakness and an inability to swallow (dysphagia). Further analysis reveals abnormalities in the electromyegram (EMG) of the upper and lower extremities, denervation atrophy. However, the cerebrospinal fluid (CSF) remains normal.

**140.** This patient is most likely suffering from

a. MS
b. Amyotrophic lateral sclerosis (ALS)
c. Poliomyelitis
d. Myasthenia gravis
e. A cerebral cortical stroke

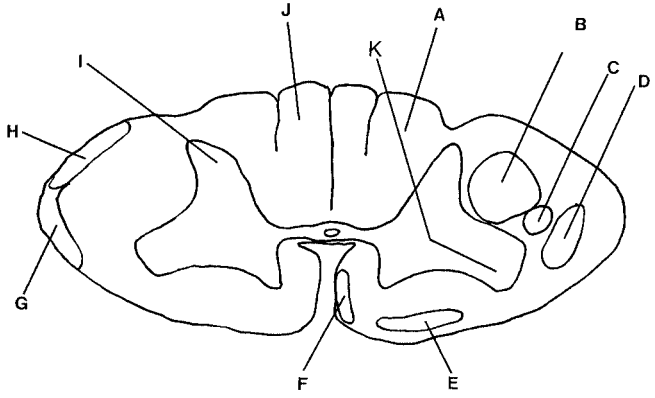
**141.** The neuronal regions affected include

a. Dorsal horns of the spinal cord
b. Lateral columns of the spinal cord
c. Ventral horns of the spinal cord
d. Dorsal columns and ventral horns of the spinal cord
e. Ventral horns and lateral columns of the spinal cord

**142.** A 38-year-old woman is referred to a neurologist because she complained of visual loss and muscle weakness. Subsequent examination revealed additional signs: impairment of other sensations, which included tingling and burning sensations; weakness of the lower limbs; paralysis of the upper limbs; progressive impairment of gait; signs of UMN involvement (i.e., spasticity and increased tendon reflexes); and bladder disturbances. No signs of infection were detected as measured by blood analysis, cultures, and chest x-ray. However, elevations in CSF protein were noted as well an abnormal IgG synthesis. Consequently, the neurologist's diagnosis of the patient was that she was suffering from

a. Diffuse cerebellar degeneration
b. ALS
c. Multiple sclerosis MS
d. A peripheral neuropathy
e. A prefrontal cortical brain tumor

**DIRECTIONS:** Each group of questions below consists of lettered options followed by a set of numbered items. For each numbered item, select the **one** lettered option with which it is **most** closely associated. Each lettered option may be used once, more than once, or not at all.




### Questions 143–151

Match each description with the appropriate structure on the cross section of the spinal cord.

**143.** First-order neurons that transmit sensory information from the upper limbs to the medulla

**144.** Pathway that transmits sensory signals directly to the thalamus

**145.** Fibers that originate from the contralateral cerebral cortex

**146.** Fibers that arise from the midbrain and facilitate flexor motor neurons

**147.** Fibers that facilitate extensor motor neurons

**148.** Second-order neurons that transmit information from muscle spindles to the cerebellum

**149.** Region of the spinal cord that receives direct sensory inputs from fibers mediating pain and temperature sensation

**150.** Fibers that arise from the ipsilateral cerebral cortex

**151.** Fibers that reach the cerebellum via the superior cerebellar peduncle

### Questions 152–155

Each effect below may be caused by a lesion at a particular site. Use the previous diagram to locate the site of each causative lesion.

**152.** Lower motor neuron (LMN) deficit

**153.** UMN deficit

**154.** Alleviation of pain associated with the lower limb

**155.** Ataxia of movement

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**156.** The level of the section of the spinal cord depicted on the previous diagram is

a. Sacral
b. Lower lumbar
c. Upper lumbar
d. Thoracic
e. Cervical

**157.** The polar regions of the muscle spindle are excited by

a. Unmyelinated C fibers
b. 1A fibers
c. Gamma motor neurons
d. Alpha motor neurons
e. General visceral efferent fibers

**158.** First-order sensory neurons that terminate in laminae I and II of the spinal cord convey mainly

a. Tactile sensation
b. Pain and temperature sensation
c. Unconscious proprioception limited to inputs from muscle spindles
d. Unconscious proprioception limited to inputs from Golgi tendon organs
e. Inputs associated with pressure receptors

**159.** It has been established that the transmitter released by the axon terminals of first-order pain and temperature fibers is most likely

a. Enkephalins
b. Acetylcholine (ACh)
c. Substance P
d. γ-aminobutyric acid (GABA)
e. Serotonin

**160.** Which of the following statements concerning Lissauer's marginal zone is true?

a. Many fibers that convey unconscious proprioception enter this zone
b. This zone is composed of coarse, heavily myelinated fibers
c. Fibers within Lissauer's marginal zone may ascend or descend several segments
d. These fibers synapse with alpha motor neurons of extensor muscles
e. Cells in this zone typically project to thalamic nuclei

**161.** Which of the following statements concerning Clarke's nucleus dorsalis is correct?

a. It is generally regarded as a nucleus associated with autonomic functions
b. It contains second-order neurons for the transmission of unconscious proprioceptive information
c. It contains second-order neurons for the transmission of information from pain receptors
d. Fibers originating from this nucleus cross in the spinal cord
e. This nucleus is found principally at cervical levels of the spinal cord

**162.** Which of the following arrangements best describes the somatotopic organization of the neurons situated in the ventral horn of the spinal cord?

a. Neurons innervating flexor muscles lie ventral to those innervating extensors, and neurons innervating the muscles of the hand lie lateral to those innervating the trunk
b. Neurons innervating flexor muscles lie dorsal to those innervating extensors, and neurons innervating the muscles of the hand lie medial to those innervating the trunk
c. Neurons innervating flexor muscles lie dorsal to those innervating extensors, and neurons innervating the muscles of the hand lie lateral to those innervating the trunk
d. Neurons innervating the muscles of the hand lie lateral to those innervating the trunk, but those innervating flexor and extensor muscles are not topographically segregated
e. Neurons innervating the muscles of the hand lie dorsal to those innervating the trunk, and those innervating flexor muscles lie medial to those innervating extensors

**163.** Which of the following statements correctly characterizes the descending component of the medial longitudinal fasciculus (MLF)?

a. The descending component of the MLF contains fibers arising from the inferior and lateral vestibular nuclei
b. The descending component of the MLF contains fibers that originate in large part in the medial vestibular nucleus and play a role in the regulation of labyrinthine modulation of head position
c. The descending fibers of the MLF are contained within the ventrolateral aspect of the white matter of the spinal cord in a position just lateral to the lateral vestibulospinal and lateral reticulospinal tracts
d. The descending fibers of the MLF suppress extensor reflex activity of the lower limbs of the contralateral side
e. The descending component of the MLF relays impulses from several forebrain nuclei to the intermediolateral cell column of the spinal cord for the regulation of blood pressure

**164.** Which of the following pathways all cross in the spinal cord?

a. Lateral spinothalamic tract, anterior spinothalamic tract, posterior spinocerebellar tract
b. Anterior spinothalamic tract, lateral spinothalamic tract, anterior corticospinal tract
c. Anterior spinocerebellar tract, posterior spinocerebellar tract, lateral vestibulospinal tract
d. Anterior corticospinal tract, lateral spinothalamic tract, dorsal columns
e. Medial vestibulospinal tract, lateral spinothalamic tract, anterior spinothalamic tract

**165.** Which of the following statements concerning muscle spindles is true?

a. They detect the rate of change of muscle length
b. They are high-threshold receptors
c. They are arranged in series with the extrafusal muscle fibers
d. They contain a single type of intrafusal fiber
e. They are primarily tension detectors

**166.** The posterior spinocerebellar and anterior spinocerebellar tracts differ in which of the following ways?

a. Fibers from the posterior spinocerebellar tract enter the cerebellum via the superior cerebellar peduncle, while those from the anterior spinocerebellar tract enter the cerebellum via the inferior cerebellar peduncle
b. Fibers of the posterior spinocerebellar tract mediate impulses from the Golgi tendon organs, while those of the anterior spinocerebellar tract mediate impulses arising from the muscle spindles
c. Fibers associated with the posterior spinocerebellar tract signal whole-limb movement, while those associated with the anterior spinocerebellar tract signal information concerning the activity of individual muscles
d. Fibers of the posterior spinocerebellar tract arise from all levels of the spinal cord, while those of the anterior spinocerebellar tract arise only from cervical levels
e. The posterior spinocerebellar tract arises mainly from thoracic levels, while the anterior spinocerebellar tract arises mainly from lumbar levels

**167.** An injury to a patient results in a hemisection of the right half of the spinal cord that extends from T8 to T12. It is probable that the patient will experience

a. Loss of pain and temperature sensation from the right leg; loss of conscious proprioception from the left leg; UMN paralysis of the left leg
b. Loss of pain and temperature sensation from the left leg; loss of conscious proprioception from the right leg; UMN paralysis of the left leg
c. Loss of pain and temperature sensation from the left arm and leg; loss of conscious proprioception from the right leg and arm; flaccid paralysis of the right leg
d. Loss of pain and temperature sensation from the left leg and loss of conscious proprioception from the right leg; UMN paralysis of the right leg
e. Bilateral loss of pain and temperature sensation and conscious proprioception, both from the lower half of the body; UMN paralysis of the left leg and flaccid paralysis of the right leg

## Questions 168–172

The next five questions relate to this case history. Audrey is a 45-year-old woman who was brought to her local hospital's emergency room by her husband because of several days of progressive weakness and numbness in her arms and legs. Her symptoms had begun with tingling in her toes, which she assumed to be her feet "falling asleep." However, this feeling did not disappear, and she began to feel numb, first in her toes on both feet, then ascending to her calves and knees. Two days later, Audrey began to feel numb in her fingertips, and had difficulty lifting her legs. When she finally was unable to climb the stairs of her house because of her leg weakness, difficulty gripping the banister, and shortness of breath, her husband urged her to go to the emergency room. The neurologist who examined Audrey in the emergency room noticed that she was short of breath while sitting in bed. He asked the respiratory therapist to measure her vital capacity (the greatest volume of air that can be exhaled from the lungs after a maximal inspiration), and the value for this was far lower than was expected for her age and weight. Her neurologic examination showed that her arms and legs were very weak, so that she had difficulty lifting them against gravity. She was unable to feel a pin or a vibrating tuning fork at all on her legs and below her elbows, but was able to feel the pin on her upper chest. The neurologist could not elicit any reflexes from her ankles or knees. He subsequently advised the emergency room staff that Audrey needed to have a spinal tap and be admitted to the intensive care unit immediately.

**168.** Where in the nervous system is the damage?

a. Frontal lobe
b. Temporal lobe
c. Peripheral nerves and nerve roots
d. Spinal cord
e. Muscle

**169.** Audrey can't feel a pinprick in certain locations. Which receptor carries this information?

a. Merkel's tactile disk
b. Ruffini's corpuscle
c. Pacinian corpuscle
d. Cδ and Aδ fibers
e. Meissner's corpuscle

**170.** Which receptor should be activated by the tuning fork?

a. Cδ and Aδ fibers
b. Merkel's tactile corpuscle
c. Pacinian corpuscle
d. Ruffini's corpuscle
e. Meissner's corpuscle

**171.** The absent reflexes are a sign of a lesion of which portion of the nervous system?

a. The frontal lobe
b. The dorsal horn of the spinal cord or any point distal to this structure
c. The brainstem
d. The cervical corticospinal tract
e. Any point that is proximal to the upper cervical spinal cord

**172.** Damage to which nervous system structure caused the difficulty breathing?

a. Medullary respiratory center
b. Diencephalon
c. Pons
d. Phrenic nerve innervating the diaphragm
e. Trigeminal nerve

## Questions 173–177

Gary is a 35-year-old man who was previously healthy until one day, when he noticed that his right leg was weak. As the day progressed, he found that he was dragging the leg behind him when he walked, and he finally asked a friend to drive him home from work because he was unable to lift his right foot up enough to place it on the gas peddle. He also noticed that his left leg felt a little bit numb. Finally, his wife convinced him to go to the emergency room of his local hospital.

When Gary arrived at the emergency room, he was having a great deal of difficulty walking. The physician who examined him asked him when this began, and when Gary thought about it in more depth, he realized that perhaps this had started slowly several days before, and he had ignored the symptoms. Gary's language function, cranial nerves, and motor and sensory examinations of his arms were within normal limits. When the physician examined Gary's right leg, it was markedly weak, with very brisk reflexes in the knee and ankle. Vibration and position sense in the right leg were absent. Pain and temperature testing were normal in the right leg, but these sensations were absent on the left leg and abdomen to the level of his umbilicus. Reflexes in the left leg were normal, but when the physician scratched the lateral portion of the plantar surface on the bottom side of Gary's right foot, the great toe moved up. The remainder of Gary's examination was normal.

**173.** What area of the nervous system is damaged?

a. Brainstem
b. Cervical spinal cord
c. Thoracic spinal cord
d. Frontal lobe
e. Peripheral nerves

**174.** Damage to which tract could give Gary the loss of vibration and position sense on the right side?

a. Right fasciculus cuneatus
b. Right fasciculus gracilis
c. Left fasciculus cuneatus
d. Left fasciculus gracilis
e. Right Lissauer's tract

**175.** Gary's loss of left-sided pain and temperature sensation could be due to damage to which tract?

a. Right fasciculus cuneatus
b. Right fasciculus gracilis
c. Right spinothalamic tract
d. Left spinothalamic tract
e. Left corticospinal tract

**176.** Why is Gary's right leg weak?

a. There is muscle damage in the right leg
b. There is damage in his left frontal lobe
c. There is damage to the right corticospinal tract
d. The dorsal root is damaged
e. There is damage to the right femoral nerve

**177.** The upward movement of Gary's toe when the plantar surface of his foot was scratched is indicative of a lesion in which portion of the nervous system?

a. UMN
b. LMN
c. Peripheral nerves
d. Muscles
e. Sural nerve

# The Spinal Cord

## Answers

**135. The answer is d.** *(Simon et al., pp 164–168; Gilroy, pp 592–594.)* In this case, disruption of the root fibers of C5–C6 involve components of the brachial plexus and affect muscle groups such as the deltoid, supraspinatus, intraspinatus, biceps, and flexor carpi radialis. These muscles govern abduction of the arm, rotation of the arm at the shoulder, flexion of the elbow and wrist. Reflex activity would also be affected due to disturbance of both alpha and gamma motor neurons serving the biceps muscle. Lesions involving the cerebral cortex or pons, especially the region of the pyramidal tracts, would produce a UMN paralysis, which would include hyperreflexia and hypertonia. An LMN paralysis involving the ventral horn cells at C1 would not affect the brachial plexus and the muscle groups indicated in this question. The triceps muscle is not involved in producing the movements affected by the injury.

**136. The answer is d.** *(Simon et al., pp 163–164.)* In this case, there is a loss of superficial abdominal reflexes, which require that spinal segments T8–T12 are intact. The test for these reflexes is to stroke a quadrant of the abdominal wall with an object such as a wooden stick. The normal response is for the muscle of the quadrant stimulated to contract and for movement of the umbilicus in the direction of the stimulus.

**137. The answer is a.** *(Simon, pp 168–187.)* The nerves innervating the knee and hip exit the spinal cord between L4–S1. Typical characteristics of a peripheral neuropathy include muscle weakness directed in a more pronounced manner upon the proximal muscles. Depression of tendon reflexes is generally not seen, and muscle wasting might occur only at a very late stage of the disease. Damage to the neuromuscular junction, such as myasthenia gravis, produces a different constellation of deficits. These include muscle fatigue and weakness that is fluctuating. This disorder also typically affects cranial nerves. In addition, the spinal segments indicated (T8–L3) are not associated with the muscle groups in question. Damage to the ventral horn would produce an LMN (flaccid) paralysis, which is not characteristic of the muscle weakness of this patient. Likewise, damage to

the lateral funiculus would produce a UMN (spastic) paralysis, and dorsal horn damage would produce sensory deficits as well as affect muscle tone. In addition, the spinal segments indicated in this last choice (e) do not relate to the muscle groups affected in the patient.

**138. The answer is c.** *(Simon, pp 173–183.)* The most likely cause of the condition in this patient is a cervical disk prolapse. This disorder would produce pain in the neck and arm, which increases with movement of the head. It would also cause loss of some sensation in the thumb and other fingers, as well as weakness in both finger extension and of the biceps reflex. Syringomyelia would produce bilateral segmental loss of pain and temperature. A knife wound completely severing the nerve would result in a functional loss similar to that experienced with an LMN paralysis. Polio results in loss of LMNs, thus also producing an LMN paralysis. One of the effects of AIDS is that it produces damage to the lateral and dorsal columns, resulting in the appearance of a UMN disorder.

**139. The answer is d.** *(Gilroy, pp 612–628; Simon et al., pp 182–184.)* Guillain-Barré syndrome is an acute polyneuropathy whose occurrence frequently follows a respiratory infection. It results in myalgia of the lower limbs, loss of muscle tone and tendon reflexes, and some flaccidity. The disorder can also affect the seventh cranial nerve. The disorder can produce diffuse demyelination of the peripheral nerves with an increase in lymphocytes present at the sites of demyelination. The other disorders listed are generally progressive where eventual recovery without intervention is not known to occur. Myasthenia gravis and lumbar disk prolapse would not show demyelination and lymphocyte increases near the sites of demyelination. Multiple sclerosis involves central nervous system (CNS) structures; therefore, the constellation of symptoms would be different. As indicated earlier, MD is progressive with effects upon both proximal muscles and later in distal muscles.

**140–141. The answers are 140-b, 141-e.** *(Gilroy, pp 201–215, 357–362; Simon, pp 170–171, 179.)* Amyotrophic lateral sclerosis is characterized by a progressive loss of motor functions, first seen as weakness in limb muscles, especially those of the fingers, and later of the other limbs. Sensory functions are not significantly affected. Over time, there is wasting, atrophy, and fasciculations of limb muscles followed by UMN signs. Elec-

tromyogram abnormalities can also be observed of the upper and lower extremities. In MS, there is also sensory loss, such as loss or blurring of vision, as well as bladder problems. Poliomyelitis and myasthenia gravis involve LMN symptoms, while a cerebral cortical stroke would result in a UMN disorder without LMN signs. In ALS, there is damage initially to ventral horn cells of the spinal cord, producing LMN signs. As the disease progresses, there is involvement of UMNs located in the lateral columns of the spinal cord (i.e., corticospinal dysfunction), thereby producing UMN signs such as an increase in tendon reflexes and the presence of an extensor plantar response. Sensory neurons are not involved in this disorder.

**142. The answer is c.** *(Gilroy, pp 199–211.)* Multiple sclerosis is a demyelinating autoimmune disease that affects CNS function. This disorder produces a wide variety of symptoms, including sudden sensory dysfunction and loss, which affect vision and the somatosensory system, causing tingling, pain, and hypesthesia. Broad functional motor disturbances also occur, including weakness of the upper or lower limbs, UMN signs, and gait impairment. There is also bladder dysfunction as well as an increase in CSF protein and IgG synthesis. Diffuse cerebellar degeneration would produce gait ataxia and deficits in the accuracy of intentional movements. As noted earlier, ALS would produce both a UMN and an LMN paralysis, which typically does not extend to sensory functions. Likewise, a peripheral neuropathy would not produce UMN signs, visual deficits, and extensive motor disturbances as described in this case. A tumor of the prefrontal cortex would affect some cognitive and emotional functions, but it would not affect sensory processes such as vision and somatosensation, nor would it produce signs of a UMN disorder or muscle weakness.

**143–151. The answers are 143-A, 144-D, 145-B, 146-C, 147-E, 148-H, 149-I, 150-F, 151-G.** *(Afifi, pp 59–83; Nolte, pp 220–235.)* Sensory fibers that terminate in the medulla are located in the dorsal columns. Fibers mediating conscious proprioception from the upper limb are contained in the fasciculus cuneatus (A). The lateral spinothalamic tract (D) transmits pain and temperature information directly to the thalamus. The lateral corticospinal tract (B) originates in the contralateral cortex and crosses over at the level of the lower medulla. This important pathway mediates control over volitional movements. When these fibers are cut, there is a clear loss of ability to produce volitional movements. The

rubrospinal tract (C), situated adjacent to the lateral corticospinal tract, originates from the red nucleus of the midbrain and facilitates the actions of flexor motor neurons. The lateral vestibulospinal tract (E) powerfully facilitates alpha motor neurons of extensor muscles. This tract is located in the ventral funiculus adjacent to the gray matter. The axons of the cells situated in this part of the gray matter (i.e., ventral horn) innervate extensor motor neurons.

The posterior (or dorsal) spinocerebellar tract (H) transmits information from muscle spindles to the cerebellum via the inferior cerebellar peduncle. This tract is located on the lateral aspect of the lateral funiculus of the cord, just above the anterior (or ventral) spinocerebellar tract. Pain and temperature fibers from the periphery terminate directly in the region of the dorsal horn, called the *substantia gelatinosa* (I). A smaller component of the corticospinal tract, the anterior corticospinal tract (F), originates from the cerebral cortex and passes ipsilaterally to the spinal cord. In its ventromedial position, the fibers are ipsilateral to their cortical origin. Just prior to their termination, many of the fibers are distributed to the contralateral side of the cord. The anterior (or ventral) spinocerebellar tract (G) arises from wide regions of the gray matter of the cord. These fibers pass contralaterally to the lateral aspect of the lateral funiculus to reach a position just below the dorsal spinocerebellar tract. These fibers then ascend to the cerebellum via the superior cerebellar peduncle, conveying information from Golgi tendon organs located in the lower limbs.

**152–155. The answers are 152-K, 153-B, 154-D, 155-J.** *(Afifi, pp 59–83, 91–104; Nolte, pp 255–259.)* Ventral horn cells (K) constitute the final common path for descending motor pathways controlling movement since they directly innervate skeletal muscle. Therefore, they are referred to as *lower motor neurons* (LMNs), and lesions involving any component of these neurons result in an LMN deficit. The deficit is characterized by a flaccid paralysis of the muscle groups innervated by these neurons. In contrast, neurons from the cerebral cortex (and elsewhere in the brain) that pass in the lateral funiculus of the cord (B) and innervate ventral horn cells rather than skeletal muscle are referred to as *upper motor neurons* (UMNs). Lesions of these fibers produce a UMN syndrome, which is characterized by a spastic paralysis. The lateral spinothalamic tract (D) conveys pain and temperature signals to the thalamus. Surgical (or other) damage to these fibers will disrupt the transmission of pain signals and alleviate pain from

the lower (as well as upper) limbs. The fasciculus gracilis (J) conveys, in part, information from joint capsules to the brain. Disruption of these fibers will block the transmission to the cerebral cortex of these signals that indicate the position of the limb following or preceding movement of that limb. Such loss will prevent the necessary feedback signals concerning one's position in space to reach the cortex. As a result, there will be a compensatory motor response characterized by a wide ataxic gait.

**156. The answer is e.** *(Afifi, pp 64–66; Nolte, pp 221–224.)* The cervical level of the spinal cord can be distinguished from other levels of the cord by the following characteristics: the presence of a well-defined fasciculus cuneatus medullae spinalis, situated immediately lateral to the fasciculus gracilis medullae spinalis; the presence of well-defined motor nuclei that are clumped into six different groups, three of which can be distinguished; an absence of an intermediolateral cell column; and relatively extensive quantities of both white and gray matter.

**157. The answer is c.** *(Kandel, pp 715–724.)* Gamma motor neurons innervate the polar regions of the muscle spindle and, when excited, cause resetting of the spindle by stretching it, resulting in a lowering of the threshold for activation of that receptor by an external force. Unmyelinated C fibers mediate nociceptive sensations from the periphery to the spinal cord and thus do not relate to this question. 1A fibers arise from the nuclear region of the spindle and mediate spindle activity to the spinal cord and thus form the afferent limb of the monosynaptic stretch reflex. Alpha motor neurons arise in the ventral horn of spinal cord and innervate extrafusal muscle fibers, causing movement of the limb when excited. It does not innervate the polar regions of the spindle. General visceral afferent fibers exit from the intermediolateral cell columns (at T1–L3 for sympathetics and S2–S4 for parasympathetics) of the spinal cord and innervate postganglionic neurons for these respective autonomic systems. Such fibers, therefore, do not relate to muscle spindles, including their polar regions.

**158. The answer is b.** *(Nolte, pp 225–232; Afifi, pp 59–89.)* First-order neurons that convey pain and temperature sensations to the spinal cord terminate principally in laminae I and II upon dendrites of cells located in other laminae. For the most part, tactile and pressure sensations are carried

by the dorsal column–medial lemniscal systems, which terminate in the lower medulla. Fibers that mediate unconscious proprioception terminate in Clarke's nucleus dorsalis.

**159. The answer is c.** *(Afifi, pp 59–89, 126–130.)* Immunocytochemical studies have demonstrated that the sensory neurons that terminate in laminae I and II of the dorsal horn of the spinal cord stain intensely for substance P. These neurons are believed to mediate pain impulses. Other transmitter substances, while present within the spinal cord, have not been associated directly with first-order sensory afferent fibers.

**160. The answer is c.** *(Nolte, pp 234–237; Afifi, pp 59–89.)* Lissauer's marginal zone, located on the dorsolateral margin of the dorsal horn of the spinal cord, receives many incoming fibers that are either unmyelinated or finely myelinated. These fibers principally mediate pain and temperature sensations. The fibers contained in this bundle may ascend or descend several segments, serving to integrate different levels of the substantia gelatinosa, which receives these inputs. These fibers are not known to make synaptic contact with motor neurons. Neurons in the substantia gelatinosa do not generally ascend beyond the spinal cord.

**161. The answer is b.** *(Afifi, pp 59–89.)* Clarke's nucleus dorsalis is situated in the medial aspect of lamina VII of the cord at thoracic and lumbar levels, but does extend up to C8. It receives first-order inputs from fibers that convey muscle spindle and Golgi tendon organ information. Fibers from Clarke's nucleus dorsalis run laterally to form the dorsal spinocerebellar tract on the ipsilateral side, which terminates mainly in the anterior lobe of the cerebellum.

**162. The answer is c.** *(Afifi, pp 59–89.)* The neurons situated in the ventral horn of the gray matter of the cord are somatotopically organized. This relationship is most clearly seen at cervical levels of the cord. The neurons innervating flexors lie dorsal to those innervating extensors, and the neurons innervating the muscles of the trunk are situated medial to those innervating the hand. These relationships take on added significance when one considers the nature of the descending motor pathways that synapse with these cells. For example, fibers associated mainly with the control of the flexor musculature, such as the corticospinal and rubrospinal tracts, are

situated at relatively dorsal levels of the lateral funiculus of the cord. Similarly, fibers associated with the regulation of antigravity muscles (i.e., generally the extensor musculature) are situated in a more ventral position. Thus, the somatotopic organization is maintained throughout the brainstem as well as the spinal cord.

**163. The answer is b.** *(Afifi, pp 59–89; Nolte, p 351.)* This pathway originates, in large measure, in the medial vestibular nucleus, although other regions such as the interstitial nucleus of Cajal of the midbrain, superior colliculus (by virtue of the tectospinal tract), and reticular formation also contribute fibers to this bundle. Fibers from the inferior vestibular nucleus project, instead, to the cerebellum and contribute a few fibers to the ascending component of the MLF; the lateral vestibular nucleus is the origin of the lateral vestibulospinal tract and this cell group also contributes fibers to the ascending component of the MLF. A principal descending component of the MLF arises from the medial vestibular nucleus, and, accordingly, this bundle is sometimes referred to as the *medial vestibulospinal tract*. The overall function of the MLF is to help coordinate changes in position or balance with the position of the head and eyes. The descending fibers of the MLF provide the anatomic substrate by which the inputs from the vestibular apparatus can influence the manner in which the head will be positioned. It accomplishes this by modulating upper cervical neurons that innervate muscles of the neck that control the position of the head. Since the projection is to the cervical cord, it would not likely have any direct effect upon extensor reflex activity of the lower limbs. Likewise, these descending fibers do not affect any structures that would cause alterations in blood pressure.

**164. The answer is b.** *(Afifi, pp 59–89.)* Both the lateral and anterior spinothalamic tracts cross over to the contralateral white matter of the cord relatively close to their cell bodies of origin and ascend to the thalamus. Similarly, the ventral spinocerebellar tract crosses over to the contralateral side and ascends as a distinct fiber pathway in the far lateral aspect of the white matter immediately below the position occupied by the dorsal spinocerebellar tract. The anterior corticospinal tract represents approximately 10 percent of the fibers descending from the cortex as corticospinal fibers. These fibers pass ipsilaterally through the brainstem to the spinal cord, reaching the anterior funiculus of the cord. Near the level at which these fibers terminate, most anterior corticospinal fibers cross over in the com-

missure of the spinal cord to supply the intermediate gray of the ventral horn. Posterior spinocerebellar fibers, which arise from Clarke's nucleus dorsalis, do not cross in the spinal cord. Instead, they pass laterally from their cell of origin and ascend within the dorsal half of the far lateral aspect of the white matter to the cerebellum. Lateral vestibulospinal fibers arise from the lateral vestibular nucleus and descend ipsilaterally within the ventral funiculus to all levels of the spinal cord, where they terminate upon neurons in the ventral horn. Dorsal column fibers are first-order neurons that arise from the periphery and enter the spinal cord at all levels. They ascend ipsilaterally in the fasciculus gracilis and cuneatus to the level of the dorsal column nuclei of the medulla, where they terminate.

**165. The answer is a.** *(Kandel, pp 715–724.)* In contrast to Golgi tendon organs, which detect tension, muscle spindles respond to the rate of change in the length of the muscle and are referred to as *velocity detectors.* They are low-threshold detectors and are connected in parallel with the extrafusal muscle fibers. Stretching the muscle results in an elongation of intrafusal fibers, which stretches the sensory nerve endings in the spindle, producing an increase in the discharge rate. The muscle spindle actually contains three different types of intrafusal fibers—dynamic nuclear bag, static nuclear bag, and nuclear chain fibers—all of which are innervated by a single 1A afferent fiber. Static nuclear bag fibers and nuclear chain fibers are innervated by group II afferent fibers. The various properties of these intrafusal fibers combine in generating the firing patterns of the spindle.

**166. The answer is e.** *(Afifi, pp 64–66; Kandel, pp 715–724, 841–844; Nolte, pp 237–239.)* The posterior spinocerebellar tract carries impulses from both muscle spindles and Golgi tendon organs to the cerebellum via the inferior cerebellar peduncle. The anterior spinocerebellar tract supplies inputs to the cerebellum via the superior cerebellar peduncle. The two tracts differ anatomically: the posterior spinocerebellar tract arises mainly from thoracic levels (C8 to L2 or L3), whereas the anterior spinocerebellar tract arises mainly from lumbar levels. The inputs from the anterior spinocerebellar tract are from the Golgi tendon organ. Thus, these tracts also differ functionally in that the dorsal spinocerebellar tract signals information associated with individual muscles, whereas the anterior spinocerebellar tract signals information associated with groups of muscles (i.e., whole-limb movements).

**167. The answer is d.** *(Afifi, pp 94–97.)* Hemisection of the right side of the spinal cord that involves segments T8 to T12 will result in contralateral loss of pain and temperature sensation below the level of the lesion and ipsilateral loss of conscious proprioception below the level of the lesion. Thus, this patient will experience loss of pain and temperature in the left leg and loss of conscious proprioception in the right leg. In addition, there will be damage to the descending corticospinal fibers that normally are essential for activation of the LMNs that control muscles of the right leg (i.e., UMN paralysis of the right leg). However, since the lesion is situated below the entry of sensory fibers as well as the origin of anterior horn cells that innervate the upper limbs, no loss of sensation to the upper limbs will ensue, nor will there be an LMN or UMN paralysis of the upper limbs. The pain and temperature fibers ipsilateral to the site of the lesion are unaffected because the second-order neurons decussate at the approximate level of their cell bodies of origin and ascend on the side contralateral to the lesion, leaving this system intact.

**168. The answer is c.** *(Adams, pp 43–48, 1312–1318.)* This patient does not have a UMN lesion (spinal cord or above) because of the absent reflexes and ascending paralysis bilaterally involving all of the extremities. Lesions in the brain almost always give unilateral findings, and spinal cord lesions give a distinct level. The damage cannot be in the muscle, because the patient has sensory involvement, as well. This case is an example of Guillain-Barré syndrome, or an inflammatory disease of the peripheral nerve resulting from demyelination. Inflammatory cells are found within the nerves, as well as segmental demyelination and some degree of wallerian degeneration. This damage can cause an ascending paralysis and sensory loss, affecting the arms, face, and legs. The CSF often has a high protein level, making a spinal tap a useful test for the diagnosis of Guillain-Barré syndrome. Nerve conduction studies are also helpful in making the diagnosis. Most neurologists believe Guillain-Barré syndrome to be an immunologic reaction directed against the peripheral nerve, and some patients have a history of having had some type of infection prior to developing Guillain-Barré syndrome. However, a clear-cut cause is rarely found. Despite a known cause, most patients recover from Guillain-Barré syndrome, although the speed of recovery varies. Treatment is currently available (administration of gamma globulin), and, if instituted early in the course of the disease, decrease in the length of the illness is possible.

**169. The answer is d.** *(Kandel, pp 430–440.)* Pain is mediated by CS and AS fibers in the skin.

**170. The answer is c.** *(Kandel, pp 430–440.)* Pacinian corpuscles best mediate vibration.

**171. The answer is b.** *(Adams, pp 43–48.)* The reflexes are lost because the LMNs, which are affected by this process, are unable to participate in the reflex arc necessary for a knee or ankle jerk to take place. These LMNs originate with stretch receptors in the tendons. Answers a, c, d, and e are all examples of UMN lesions, usually characterized by hyperactive reflexes.

**172. The answer is d.** *(Adams, pp 43–48.)* This is an example of a LMN problem. Answers a, b, and c are UMN structures. The trigeminal nerve is a cranial nerve that mediates sensation on the face and the muscles of mastication. Loss of diaphragmatic function causes respiratory distress.

**173. The answer is c.** *(Afifi, pp 91–103.)* Gary has a spinal cord syndrome called *Brown-Séquard's syndrome,* or hemisection of the spinal cord. The lesion is not at the cervical level because motor functions of the upper limbs were considered normal. The examiner can pinpoint the location of the lesion by using the "sensory level," or level at which the loss of pain and temperature begin, by remembering that the lesion affects fibers that have entered the spinal cord one or two levels below it, and then cross to the contralateral side. Therefore, a loss of sensory function at the $T_{10}$ level indicates a lesion at the $T_8$ or $T_9$ level. A level at which motor deficits begin can be helpful as well, but in lesions of the thoracic spinal cord, muscles innervated by thoracic nerves are difficult to test. The examiner still expects weakness in the lower extremities, and this helps to make the diagnosis. Brown-Séquard's syndrome may occur as a result of different types of tumors or infections of the spinal cord.

**174. The answer is b.** *(Afifi, pp 91–103.)* Because one-half of the spinal cord is damaged, the dorsal columns are damaged, and the patient will have loss of proprioception and vibration ipsilateral to and below the level of the lesion. The loss must be ipsilateral because fibers mediating this type of sensation cross above the level of the lesion. The fasciculus gracilis carries fibers originating from the sacral, lumbar, and lower thoracic levels,

and the fasciculus carries those from the upper thoracic and cervical levels. Lissauer's tract carries pain and temperature fibers via the dorsal root entry zone. Brown-Séquard's syndrome may occur as a result of different types of tumors or infections of the spinal cord.

**175. The answer is c.** *(Afifi, pp 91–103.)* The spinothalamic tract carries fibers mediating pain and temperature. The primary pain fibers enter the spinal cord and pass one or two segments in Lissauer's marginal zone before making a synapse with neurons that form the lateral spinothalamic tract. Fibers of the lateral spinothalamic tract then cross to the contralateral side one or two segments above or before where the primary afferent fibers have entered the cord. Accordingly, pain and temperature are lost below the lesion on the contralateral side. The cuneate and gracile fasciculi mediate proprioception and vibration, and the corticospinal tract mediates voluntary motor function.

**176. The answer is c.** *(Afifi, pp 91–103.)* The corticospinal tract mediates voluntary motor function. The fibers cross in the medullary pyramids, thus lesions below this structure cause ipsilateral weakness. The reflexes are brisk, since in a UMN lesion, there is a loss of inhibition to spinal reflexes. Muscle, dorsal root, and femoral nerve damage are all examples of lesions distal to the spinal cord. A frontal lobe lesion would not cause a sensory or motor level, and would probably cause problems more proximally, such as slurred speech.

**177. The answer is a.** *(Afifi, pp 91–103.)* A positive Babinski's sign, or dorsiflexion of the great toe when the lateral portion of the plantar surface of the foot is scratched, is a sign of corticospinal tract dysfunction, a tract consisting of UMNs. Peripheral nerve (including the sural nerve) lesions are LMN lesions.

# The Autonomic Nervous System

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**178.** Synaptic transmission in autonomic ganglia is primarily

a. Cholinergic
b. Noradrenergic
c. Serotonergic
d. GABAergic
e. Peptidergic

**179.** Which of the following statements concerning the function of peptides in the autonomic nervous system is true?

a. They are present only at preganglionic axon terminals of the parasympathetic nervous system
b. They are present only at postganglionic axon terminals of the parasympathetic nervous system
c. They are present in sympathetic ganglia where they function primarily as neurotransmitters
d. They are present in sympathetic ganglia where they function primarily as neuromodulators
e. They have not been localized in any of the autonomic ganglia

**180.** The carotid sinus reflex involves

a. Baroreceptor afferent fibers from cranial nerve XI
b. Glossopharyngeal efferent fibers
c. Interneurons within the nucleus ambiguus of the medulla
d. Efferent fibers contained in the intermediate component of the facial nerve
e. Vagal efferent fibers

**181.** Calcium currents present in heart muscle cells are

a. Reduced by norepinephrine acting through beta receptors
b. Increased by norepinephrine acting through beta receptors
c. Increased by acetylcholine acting on muscarinic receptors
d. Increased by acetylcholine acting on nicotinic receptors
e. Increased by serotonin acting on serotonin $1_A$ receptors

**182.** Bladder functions are regulated by which of the following combinations of inputs?

a. Vagal and sacral efferent fibers only
b. Vagal, sacral, and descending fibers from the cerebral cortex
c. Lumbar and sacral efferent fibers only
d. Lumbar, sacral, and descending fibers from the cerebral cortex
e. Lumbar, thoracic, and cervical fibers only

**183.** Synthesis and storage of norepinephrine can be prevented by

a. Guanethidine sulfate
b. Reserpine
c. Phenoxybenzamine hydrochloride
d. Hexamethonium chloride
e. Metoprolol

**184.** The hypothalamus and amygdala are able to modulate the output of the autonomic nervous system by virtue of their connections with the

a. Ventrolateral nucleus of the thalamus
b. Nucleus accumbens
c. Solitary nucleus
d. Red nucleus
e. Ventral horn cells at the level of C8–T12 of the spinal cord

# The Autonomic Nervous System

## Answers

**178. The answer is a.** *(Kandel, pp 970–974.)* The transmitter released from preganglionic endings of both sympathetic and parasympathetic fibers is acetylcholine (ACh). The other transmitters listed are not involved at this synapse. Evidence in support of this view is derived, in part, from studies that demonstrated that drugs that block nicotinic receptors (e.g., hexamethonium chloride, curare) also block the output of these systems.

**179. The answer is d.** *(Kandel, pp 970–972.)* Recent studies demonstrate that a wide variety of peptides are found within most sympathetic ganglia. Evidence further suggests that these peptides do not act as transmitters, but instead, serve as neuromodulators. In this manner, the action of peptides in autonomic ganglia is to alter the efficiency of neuronal excitability and the effectiveness of cholinergic transmission at autonomic synapses.

**180. The answer is e.** *(Kandel, pp 879–880.)* The carotid sinus reflex involves several neuronal elements. The afferent side of the reflex begins with stretch receptors in the walls of the carotid sinus. These receptors signal pressure as a result of stretch of the low-capacitance vessel. This causes an afferent volley of action potentials to pass along the glossopharyngeal nerve into the medulla, where the fibers synapse with neurons in the solitary nucleus. These neurons, in turn, synapse upon neurons in the dorsal motor nucleus of the vagus nerve whose axons innervate the heart. Activation of this reflex results in a decrease in heart rate and force of contraction. As a consequence of the decrease in cardiac output, there is an ensuing decrease in blood pressure as well.

**181. The answer is b.** *(Kandel, pp 964–973.)* The calcium current of heart muscle cells is enhanced by the release of norepinephrine, which acts on β-adrenergic receptors. This effect is additionally mediated by the modulation of the potassium current, which serves to keep the action potential of the muscle cells constant. The pacemaker current is also affected by this

process since its threshold is decreased as a result of activation of the β receptors (which further involves the second-messenger system–cAMP-dependent protein kinase). Lowering the threshold of the pacemaker current serves to increase heart rate. Serotonin is not involved in postsynaptic regulation of the heart. Acetylcholine has an inhibitory effect upon heart muscle by acting through different mechanisms.

**182. The answer is d.** *(Kandel, pp 963–972.)* The smooth muscle of the bladder is innervated by postganglionic fibers of the sympathetic nervous system that arise from the inferior mesenteric ganglion. This ganglion, in turn, receives its inputs from T12–L2 of the intermediolateral cell column of the spinal cord. The smooth muscle of the bladder also receives inputs from postganglionic parasympathetic fibers that are innervated by preganglionic fibers arising from S2–S4. The external sphincter of the bladder (striated muscle) is innervated by ventral horn cells from the spinal cord. These ventral horn cells, in turn, receive inputs from supraspinal neurons that arise, in part, from the cerebral cortex. It is these neurons that form a part of the substrate for voluntary control over bladder functions.

**183. The answer is b.** *(Cooper et al, pp 197–222, 227–290. Siegel et al, pp 221–240, 243–259.)* Noradrenergic activity can be blocked by a number of mechanisms. Reserpine, for example, prevents the synthesis and storage of norepinephrine in sympathetic nerve terminals. Guanethidine sulfate affects noradrenergic transmission by blocking the release of norepinephrine at the sympathetic endings. Competitive α-receptor blockers include phenoxybenzamine hydrochloride and phentolamine, whereas metoprolol blocks $\beta_1$ receptors. Since ACh is the transmitter at preganglionic synapses of both the parasympathetic and sympathetic nervous systems, hexamethonium chloride is an effective ganglionic blocker at these synapses.

**184. The answer is c.** *(Kandel, pp 965–967.)* The solitary nucleus of the medulla plays a significant role in the neural control of autonomic functions because it receives input from several different regions of the brain that regulate such functions. These inputs include fibers that arise from the hypothalamus, central nucleus of the amygdala, midbrain periaqueductal gray, and sensory processes (i.e., visceral afferents) of the glossopharyngeal and vagus nerves. The last signal changes in blood pressure and levels of

oxygen and carbon dioxide in the blood. The ventrolateral nucleus of the thalamus, red nucleus of the midbrain, and ventral horn cells of the spinal cord are associated with somatomotor rather than autonomic function. The nucleus accumbens is believed to be associated with motivational processes.

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# The Brainstem and Cranial Nerves

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

### Questions 185–186

The following test is administered to a patient: A cotton applicator is gently applied to the cornea of the eye as the patient is asked to look upward. The patient did not blink in response to stimulation of the cornea.

**185.** Which cranial nerves are normally involved in this reflex?

a. Nerves II and III
b. Nerves III and IV
c. Nerves III and V
d. Nerves V and VII
e. Nerves VII and IX

**186.** The nerves involved in this reflex are characterized as

a. Special sensory afferent and general somatic efferent
b. General somatic efferent alone
c. General somatic afferent and special visceral efferent
d. General somatic afferent and general somatic efferent
e. Special visceral afferent and special visceral efferent

### Questions 187–189

A 56-year-old woman experiences a loss of taste affecting the front of her tongue and the ability to smile as a result of an infection.

**187.** If the sensory loss involves damage of cell bodies, the specific group of neurons so affected would be the

a. Otic ganglion
b. Nodose (inferior) ganglion
c. Pterygopalatine ganglion
d. Geniculate ganglion
e. Trigeminal ganglion

**188.** The cranial nerve most immediately affected is

a. Nerve V
b. Nerve VI
c. Nerve VII
d. Nerve IX
e. Nerve X

**189.** The components of the nerve that is affected include

a. General somatic afferent and general somatic efferent
b. Special visceral afferent and special visceral efferent
c. General visceral afferent and general somatic efferent
d. General somatic afferent and general visceral efferent
e. Special visceral afferent and general visceral efferent

## Questions 190–193

A 55-year-old man, who has been suffering from hypertension for the past 8 years, experiences attacks of pain in the regions of the pharynx and ear, which are usually preceded by swallowing and coughing spells. Each attack, which lasted for an average of 1 minute, occurred a number of times; ultimately, this condition showed remission. Although the neurological examination was basically normal, a subsequent MRI was taken and revealed an abnormality at the base of the skull.

**190.** The most likely cranial nerve involved in this disorder is

a. Nerve V
b. Nerve VII
c. Nerve IX
d. Nerve XI
e. Nerve XII

**191.** The motor component of this cranial nerve arises from the

a. Otic ganglion
b. Nodose ganglion
c. Nucleus ambiguus
d. Inferior salivatory nucleus
e. Lateral reticular nucleus

**192.** The cell bodies of the sensory component of the affected nerve are located in the

a. Solitary nucleus
b. Superior ganglion
c. Geniculate ganglion
d. Vestibular nuclei
e. Trigeminal ganglion

**193.** The motor and sensory components of this nerve that were affected are respectively characterized as

a. General visceral efferent and general visceral afferent
b. Special visceral efferent and general somatic afferent
c. General somatic efferent and special visceral afferent
d. Special visceral efferent and special visceral afferent
e. General somatic efferent and general somatic afferent

### Questions 194–195

In a classic experiment performed by Sherrington in the cat, marked rigidity was demonstrated in a decerebrate preparation. Similarly, an 80-year-old woman displayed rigidity, which resembled that shown in the cat after having a stroke.

**194.** The likely location of the stroke is in the

a. Thalamus
b. Hypothalamus
c. Upper midbrain
d. Pons
e. Spinal cord

**195.** The rigidity can be accounted in part by the unopposed action of the

a. Rubrospinal tract
b. Lateral vestibulospinal tract
c. Corticospinal tract
d. Medial vestibulospinal tract
e. Lateral reticulospinal tract

### Questions 196–198

A 43-year-old male is recovering from an infectious disease and experiences a marked instability in his blood pressure with episodes of spiking of blood pressure. After a series of extensive examinations, it was concluded that this disorder was due to the effects of the infectious agent upon a component of the peripheral nervous system.

**196.** Logical sites where an infectious agent could produce such an effect include the

a. Superior ganglia of cranial nerves IX and X
b. Geniculate and trigeminal ganglia
c. Otic and superior salivatory ganglia
d. Carotid sinus and aortic arch
e. Carotid and aortic bodies

**197.** The (appropriate) receptors situated in the sites listed in question 196 respond best to:

a. Stretch
b. Change in chloride ion concentration
c. Contractions of the gut
d. Decrease in oxygen concentration
e. Increase in carbon dioxide concentration

**198.** Neurons situated in the (appropriate) sites indicated in question 196 mediate their effects by projecting directly to the:

a. Trigeminal spinal nucleus
b. Fastigial nucleus
c. Midbrain reticular formation
d. Solitary nucleus
e. Autonomic nuclei of the facial nucleus (cranial nerve VII)

**199.** An individual has difficulty in adjusting his head, especially after he changes his posture. The most likely pathway affected that might cause this deficit is the

a. Lateral vestibulospinal tract
b. Medial vestibulospinal tract
c. Medial reticulospinal tract
d. Lateral reticulospinal tract
e. Rubrospinal tract

### Questions 200–202

An individual experiences an ipsilateral paralysis of the soft palate and pharynx, producing hoarseness and dysphagia (inability to swallow) and, in addition, displays a loss of the carotid sinus reflex.

**200.** The nerve group most likely affected is the

a. Cranial nerve XII
b. Cranial nerve XI
c. Cranial nerve X
d. Cranial nerve VII
e. Ventral horn cells of the cervical cord

**201.** The most probable nuclei damaged in this case include the

a. Solitary and lateral reticular nuclei
b. Deep pontine and facial nuclei
c. Dorsal motor nucleus and nucleus ambiguus
d. Ventral horn of the cervical segment of the spinal cord
e. Inferior salivatory and medial vestibular nuclei

**202.** The neurons associated with the loss of functions described in this case can be characterized as

a. General somatic efferent and special visceral efferent
b. General visceral efferent and special visceral efferent
c. General somatic efferent and general visceral efferent
d. General visceral efferent and general visceral afferent
e. Special visceral efferent and special visceral afferent

**203.** A patient complains that he cannot move his right eye to the right and that the right side of his face is expressionless. The likely locus of the lesion is the

a. Dorsal aspect of the medulla
b. Ventromedial medulla
c. Dorsal pons
d. Ventromedial pons
e. Medial midbrain

**204.** Upon examination, the patient is unable to move his right eye medially. The lesion is likely to be located in the

a. Dorsal medulla
b. Ventromedial medulla
c. Dorsal pons
d. Ventromedial pons
e. Medial midbrain

### Questions 205–207

The patient experiences difficulty in walking down stairs and reports some double vision as well.

**205.** In this instance, the lesion is most likely located in the

a. Medulla
b. Dorsal pons
c. Ventromedial pons
d. Midbrain
e. Spinal cord

**206.** The lesion involved the

a. Cervical spinal cord ventral horn cells
b. Cranial nerve VII
c. Cranial nerve VI
d. Cranial nerve IV
e. Cranial nerve III

**207.** This nerve is classified as a

a. General somatic efferent
b. Special visceral efferent
c. General visceral efferent
d. Combined general visceral and somatic efferent
e. Combined general visceral and special visceral efferent

**208.** Principal afferent fiber systems that supply the inferior olivary nucleus include the

a. Hypothalamus and amygdala
b. Caudate nucleus and subthalamic nucleus
c. Solitary nucleus and nucleus of the ventrolateral medulla
d. Red nucleus and spinal cord
e. Deep pontine nuclei and vestibular nuclei

**209.** The principal projection target of the inferior olivary nucleus is the

a. Cerebral cortex
b. Midbrain periaqueductal gray
c. Vestibular nuclei
d. Dorsal column nuclei
e. Cerebellar cortex

## Questions 210–211

An elderly female patient complains that she cannot taste the food that she eats. A careful neurological examination reveals no evidence of peripheral damage of the taste receptors. The evidence suggests, instead, that there was selective damage of certain regions of the brainstem.

**210.** One of the sites where damage could result in the selective loss of taste includes the

a. Superior olivary nucleus
b. Inferior salivatory nucleus
c. Solitary nucleus
d. Spinal nucleus of the trigeminal nerve
e. Reticular tegmental nucleus of the pons

**211.** A principal target of the brainstem structure (referring to the answer to the previous question) is the

a. Anterior thalamic nucleus
b. Reticular thalamic nucleus
c. Ventral posteromedial thalamic nucleus
d. Ventrolateral thalamic nucleus
e. Dorsomedial thalamic nucleus

## Questions 212–215

A 68-year-old woman had suffered from an infectious disorder for several weeks. Following recovery from this disorder, she experienced some loss of taste and an increase in salivation, together with pain spasms in the region of the pharynx, which extended into the ear. She also experienced some bradycardia and cardiac arrhythmia, as well as deviation of the uvula to the unaffected side.

**212.** The cranial nerve most directly involved in this deficit is

a. Cranial nerve VII
b. Cranial nerve IX
c. Cranial nerve X
d. Cranial nerve XI
e. Cranial nerve XII

**213.** The cell bodies of origin of the nerve fibers that directly innervate the organ responsible for an increase in salivation are called the

a. Inferior salivatory nuclei
b. Superior salivatory nuclei
c. Otic ganglion
d. Geniculate ganglion
e. Nucleus ambiguus

**214.** The origin of the fibers that were affected causing deviation of the uvula is the:

a. Solitary nucleus
b. Inferior salivatory nucleus
c. Facial nucleus
d. Nucleus ambiguus
e. Dorsal motor nucleus of the vagus

**215.** The likely site of the lesion affecting this nerve is in the:

a. Upper medulla
b. Lower medulla
c. Lower pons
d. Upper pons
e. Base of the skull

## Questions 216–218

A 40-year-old male who had been suffering from a disorder of unknown origin complains to his physician that he has difficulty in producing a smile from the left side of his face, and that he can't salivate or produce tears from the left eye. Further analysis showed some loss of taste and that the affected muscles were flaccid and the eyelids were open.

**216.** The cell bodies of origin within the central nervous system (CNS) whose peripheral innervation of skeletal muscles were affected by this disorder lie in the

a. Upper medulla
b. Lower pons
c. Upper pons
d. Lower midbrain
e. Upper midbrain

**217.** The preganglionic parasympathetic fibers of this nerve arise from the

a. Dorsal motor nucleus of the vagus
b. Nucleus ambiguus
c. Inferior salivatory nucleus
d. Superior salivatory nucleus
e. Edinger-Westphal nucleus of cranial nerve III

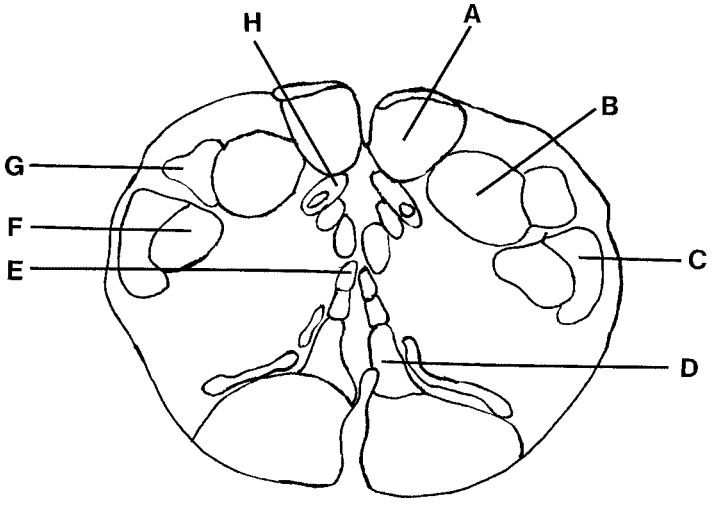
**218.** Where is the most likely locus of this lesion?

a. Nucleus of the facial nerve
b. Inferior and superior ganglia of cranial nerve IX
c. Geniculate ganglion
d. Cerebral cortex
e. Reticular formation

**DIRECTIONS:** Each group of questions below consists of lettered options followed by a set of numbered items. For each numbered item, select the **one** lettered option with which it is **most** closely associated. Each lettered option may be used once, more than once, or not at all.

### Questions 219–223

Match each type of cell or fiber with the appropriate site.




**219.** Cells in this structure respond to movement of the lower limb

**220.** Cells in this structure respond to a vibratory stimulus applied to the hand

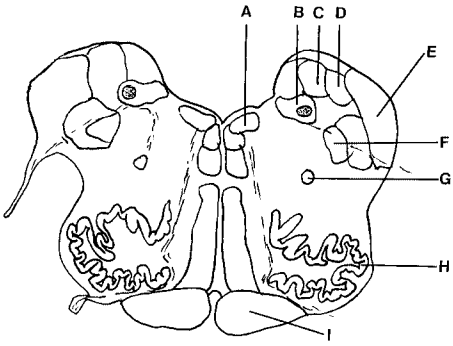
**221.** Fibers in this region mediate reflexes associated with the head

**222.** First-order pain and temperature fibers are found here

**223.** These fibers mediate conscious proprioception and two-point discrimination from the opposite side of the body

## Questions 224–232

Match each description with the appropriate site.




**224.** This nucleus responds to taste impulses

**225.** Neurons respond to vestibular inputs and project to the spinal cord

**226.** Neurons receive inputs from the red nucleus and spinal cord

**227.** Neurons respond to changes in blood pressure

**228.** Neurons contribute the largest number of fibers that are contained in the inferior cerebellar peduncle

**229.** This nucleus participates in the gag reflex

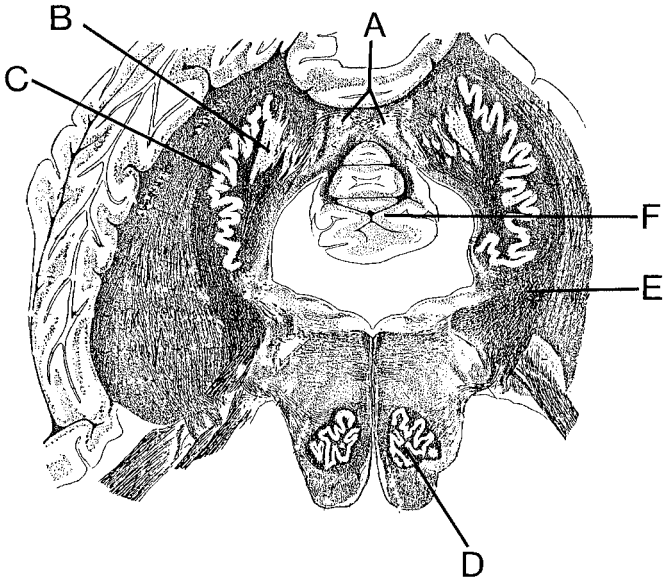
**230.** Neurons mediate voluntary control of motor functions

**231.** This nucleus innervates muscles of the tongue

**232.** Fibers in this bundle arise from the spinal cord and the brainstem and project directly to the cerebellum

### Questions 233–238

Match each description with the appropriate site.




**233.** Neurons that project to the reticular formation

**234.** Neurons that project to the vestibular nuclei

**235.** Neurons that project to the ventrolateral nucleus of the thalamus

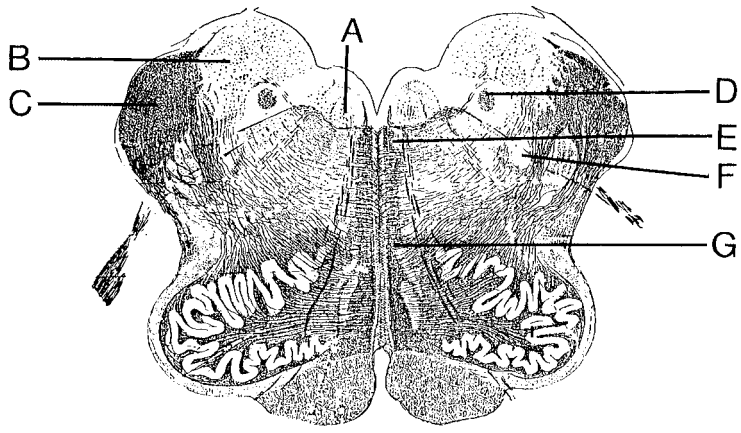
**236.** Neurons that project primarily to the red nucleus

**237.** Structure that receives inputs from the vermal region of the cerebellar cortex

**238.** Neurons that receive inputs from the lateral aspects of the cerebellar hemispheres

### Questions 239–245

Match each description with the correct site.




**239.** This structure receives direct inputs from the vestibular apparatus

**240.** These fibers arise from vestibular nuclei

**241.** This structure transmits taste impulses to the thalamus

**242.** This structure is a general somatic efferent nucleus

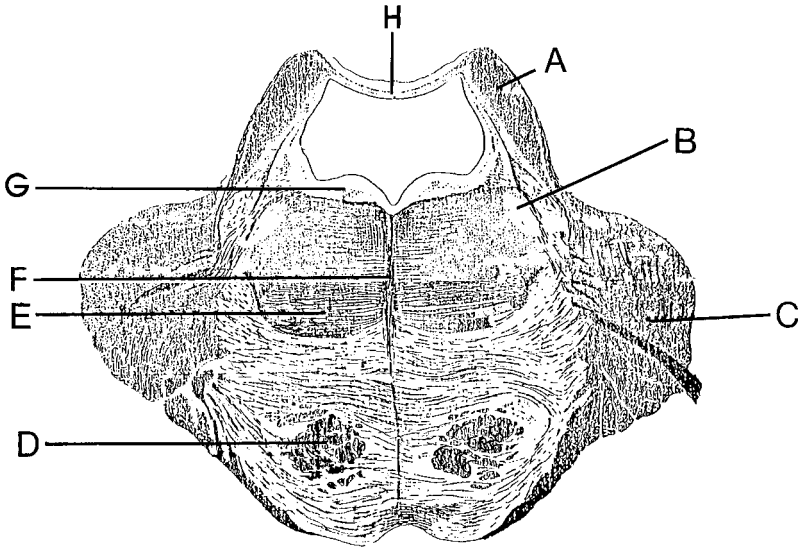
**243.** These fibers arise from the dorsal column nuclei

**244.** These fibers transmit pain and temperature signals from the region of the head to the thalamus

**245.** These fibers convey muscle spindle afferents to the cerebellum

### Questions 246–250

Match each structure with the correct site.




**246.** Axons that are second-order corticocerebellar neurons

**247.** Lower motor neurons (LMNs)

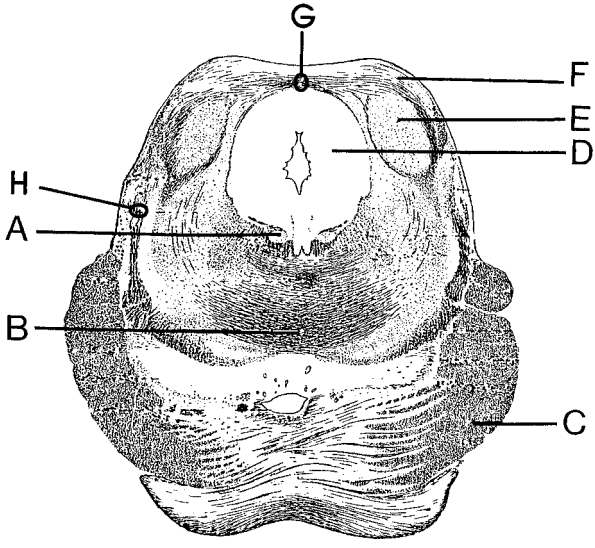
**248.** Upper motor neurons (UMNs)

**249.** Axons that terminate, in part, in the ventrolateral thalamic nucleus

**250.** Somatotopically organized sensory pathways

### Questions 251–255

Match each structure with the correct site.




**251.** Sensory relay nucleus

**252.** Fibers that arise from the contralateral dentate and interposed nuclei

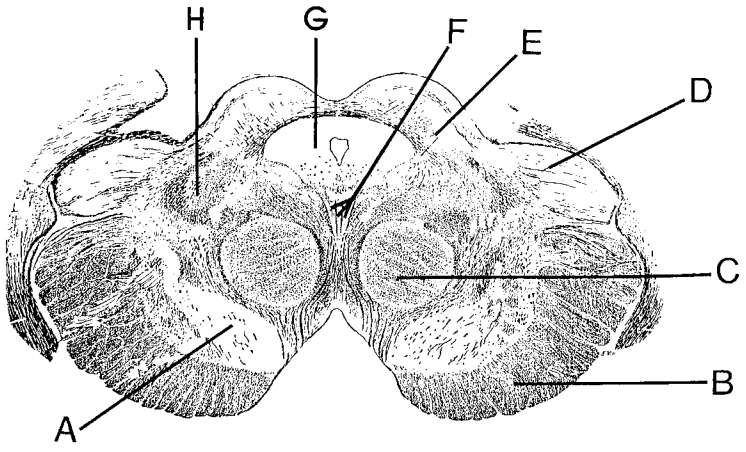
**253.** Fibers that arise from the cerebral cortex

**254.** Nucleus that receives inputs from vestibular structures

**255.** Structure that is rich in enkephalin-positive cells and nerve terminals

### Questions 256–260

Match each description with the appropriate site.




**256.** Site of neurons that respond to moving stimuli

**257.** Specific relay nucleus

**258.** Source of dopaminergic innervation of the striatum

**259.** Nucleus that receives direct inputs from the cerebellum and cerebral cortex

**260.** LMNs

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**261.** Which of the following statements concerning the spinal trigeminal nucleus is correct?

a. It receives direct inputs from first-order descending sensory fibers contained in the ipsilateral spinal tract of cranial nerve V
b. It projects its axons mainly contralaterally to the ventral posterolateral nucleus of the thalamus
c. Cells contained in the most caudal aspect of this nucleus respond mainly to mechanical and tactile stimuli
d. It receives inputs from primary afferent fibers entering the spinal cord at levels C3 and C4
e. It contains cells whose axons project to the hypothalamus

**262.** Which of the following features concerning the area postrema is true?

a. It is located in the ventral medulla at a position that is caudal to the fourth ventricle
b. It is considered part of the brain because the cells of this structure are protected by the blood-brain barrier
c. It plays a role in the regulation of emetic functions
d. The cells synthesize norepinephrine
e. It receives major inputs from the forebrain

**263.** The vagus nerve (cranial nerve X) includes which of the following components?

a. General somatic afferent, special visceral afferent, general visceral afferent, and general visceral efferent
b. Special visceral afferent, special sensory afferent, general visceral afferent, and general visceral efferent
c. General visceral afferent and general visceral efferent only
d. General visceral efferent and special visceral efferent only
e. Special visceral efferent, general visceral efferent, and general visceral afferent only

**264.** Lesions involving the dorsolateral medulla can produce

a. Loss of pain and thermal sensation on the contralateral half of the face
b. Loss of pain and temperature sensation on the ipsilateral side of the body
c. Dysphonia
d. Hemiparesis
e. Intention tremor

**265.** Which of the following statements concerning the olivocochlear bundle is correct?

a. It arises from the inferior olivary nucleus and projects to the cochlea
b. Stimulation of it inhibits acoustic fiber responses to auditory stimuli
c. It communicates directly with the medial lemniscus
d. It can be seen easily in brainstem sections taken from the upper pons
e. It is part of the ascending auditory pathway to the dorsal cochlea nucleus

**266.** Unilateral deafness may result from a lesion of

a. The auditory cortex of one side
b. The lateral lemniscus of one side
c. Cranial nerve VIII on one side
d. The medial geniculate
e. The medial lemniscus

**267.** Which of the following contains first-order sensory neurons with their cell bodies located within the CNS?

a. Geniculate ganglion
b. Spiral ganglion
c. Mesencephalic nucleus of cranial nerve V
d. Solitary nucleus
e. Scarpa's ganglia

**268.** In a lateral gaze paralysis, both eyes are conjugatively directed to the side opposite the lesion. In this condition, the locus of the lesion is the

a. Root fibers of cranial nerve III
b. Nucleus of cranial nerve III
c. Root fibers of cranial nerve VI
d. Nucleus of cranial nerve VI
e. Nucleus and root fibers of cranial nerve IV

**269.** Which of the following statements concerning the paramedian pontine reticular formation is true?

a. It projects fibers directly to the hypoglossal nucleus
b. Bilateral lesions cause a partial deafness
c. It projects its fibers to the basal ganglia
d. It is a critical site for the integration of impulses regulating vertical and horizontal gaze
e. It is a major site of noradrenergic fibers that project to the forebrain

**270.** A patient displays an ipsilateral paralysis of lateral gaze coupled with a contralateral hemiplegia. A lesion is most likely situated in the

a. Ventromedial medulla
b. Dorsomedial medulla
c. Ventrocaudal pons
d. Dorsorostral pons
e. Ventromedial midbrain

**271.** Which of the following cranial nerves all carry special visceral afferent fibers?

a. V, VII, and IX
b. III, VI, and XII
c. IX, X, and XI
d. II, VII, and VIII
e. I, VII, and IX

**272.** A patient displays the following constellation of symptoms: UMN paralysis of the left leg, paralysis of the lower half of the left side of the face, and a left homonymous hemianopsia. The lesion is most likely located in the

a. Medulla
b. Basilar pons
c. Pontine tegmentum
d. Midbrain
e. Forebrain

**273.** When a patient is asked to follow an object when it is placed in the right side of his visual field, he is unable to move his right eye either up or down. The lesion is most likely situated in the

a. Medulla
b. Basilar aspect of the pons
c. Pontine tegmentum
d. Midbrain
e. Cerebellum

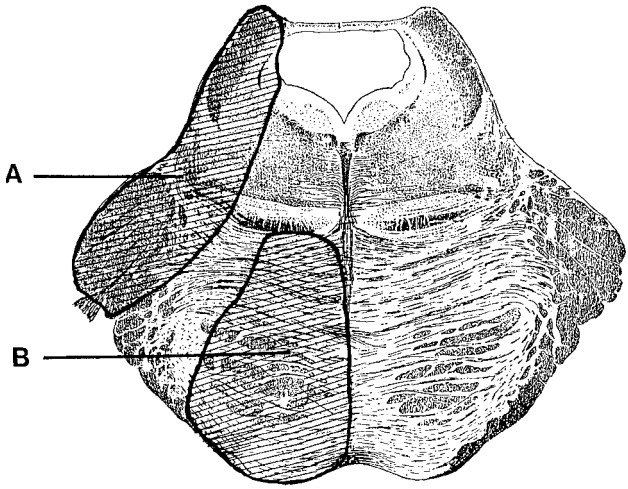
**274.** A patient is capable of displaying pupillary constriction during an accommodation reaction but not in response to a direct-light stimulus. The lesion is most likely present in the

a. Optic nerve
b. Ventral cell column of cranial nerve III
c. Pretectal area
d. Visual cortex
e. Edinger-Westphal nucleus of cranial nerve III

**275.** Structures associated with the taste pathway include the

a. Geniculate ganglion, chorda tympani, and medial lemniscus
b. Solitary nucleus, parabrachial nucleus, and ventral posteromedial nucleus
c. Solitary nucleus, ventral posterolateral nucleus, and postcentral gyrus
d. Solitary nucleus, ventral posteromedial nucleus, and superior parietal lobule
e. Geniculate ganglion and ventral posterolateral nucleus

## Questions 276–279



**276.** The lesion at A most likely resulted from an occlusion of the

a. Basilar artery
b. Superior cerebellar artery
c. Anterior spinal artery
d. Vertebral artery
e. Posterior inferior cerebellar artery

**277.** The lesion at B is most likely the result of an occlusion of the

a. Paramedian branch of the basilar artery
b. Circumferential branch of the basilar artery
c. Superior cerebellar artery
d. Anterior inferior cerebellar artery
e. Anterior spinal artery

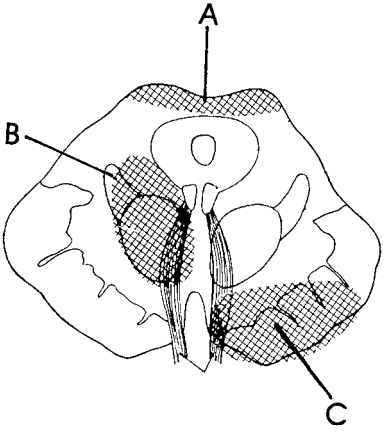
**278.** Structures affected by the lesion at B include

a. Medial lemniscus
b. Lateral lemniscus
c. Corticospinal tract
d. Medial longitudinal fasciculus
e. Tectospinal tract

**279.** The lesion at B would most likely result in which of the following deficits?

a. Paralysis of the contralateral limbs
b. Loss of conscious proprioception of the contralateral side of the body
c. Nystagmus
d. Lateral gaze paralysis
e. Facial paralysis

## Questions 280–282




**280.** A patient with the lesion at A will generally show which of the following deficits?

a. Partial blindness
b. Loss of ability to gaze medially
c. Loss of ability to show tracking movements
d. Loss of accommodation reflex
e. Nystagmus

**281.** Which of the following deficits is likely to occur as a result of the lesion at B?

a. Contralateral loss of conscious proprioception
b. Transient tremor of the ipsilateral limb
c. Ipsilateral fourth-nerve palsy
d. Hearing loss
e. Contralateral loss of taste sensation

**282.** The deficits associated with the lesion at *C* are the result of damage to the

a. Substantia nigra and crus cerebri
b. Red nucleus and crus cerebri
c. Crus cerebri and cranial nerve III
d. Red nucleus and substantia nigra
e. Substantia nigra and cranial nerve III

**283.** Which of the following statements best describes the regions of the ventral tegmental area and pars compacta of the substantia nigra?

a. Both regions contain dopaminergic neurons but project to different populations of forebrain structures
b. Both regions provide converging dopaminergic inputs to the hypothalamus
c. Both regions provide converging dopaminergic inputs to the neostriatum
d. Both regions are innervated by GABAergic fibers and project to the cerebral cortex
e. Lesions of either region result in the development of a parkinsonian-like syndrome

**284.** The principal ascending auditory pathway of the brainstem is the

a. Medial lemniscus
b. Lateral lemniscus
c. Trapezoid body
d. Trigeminal lemniscus
e. Brachium of the superior colliculus

### Questions 285–289

Emma is a 64-year-old woman who has had heart disease for many years. While carrying chemicals down the stairs of the dry-cleaning shop where she worked, she suddenly lost control of her right leg and arm. She fell down the stairs and was able to stand up with some assistance from a coworker. When attempting to walk on her own, she had a very unsteady gait, with a tendency to fall to the right side. Her supervisor asked her if she was all right, and noticed that her speech was very slurred when she tried to answer. He called an ambulance to take her to the nearest hospital. The physician who was called to see Emma in the emergency room noted that her speech was slurred as if she were intoxicated, but the grammar and meaning were intact. Her face appeared symmetric, but when asked to pro-

trude her tongue, it deviated toward the left. She was unable to tell if her right toe was moved up or down by the physician when she closed her eyes, and she couldn't feel the buzz of a tuning fork on her right arm and leg. In addition, her right arm and leg were markedly weak. The physician could find no other abnormalities on the remainder of Emma's general medical examination.

**285.** Where in the nervous system has the damage occurred?

a. Right lateral medulla
b. Occipital lobe
c. Left lateral medulla
d. Right cervical spinal cord
e. Left medial medulla

**286.** Where in the nervous system could a lesion occur that causes arm and leg weakness, but spares the face?

a. Right corticospinal tract in the cervical spinal cord
b. Left inferior frontal lobe
c. Left medullary pyramids
d. Occipital lobe
e. Both A and C are plausible sites

**287.** Other than the weakness on her right side, what type of deficit could cause Emma's gait problem, and where could a lesion causing this deficit occur?

a. Proprioceptive, left medial lemniscus
b. Sight, left eye
c. Descending component of the medial longitudinal fasciculus
d. Pain, left spinothalamic tract
e. Proprioceptive, right medial lemniscus

**288.** Deviation of the tongue to the left, away from the right hemiparesis, implies a lesion in which area of the nervous system?

a. Right hypoglossal nucleus
b. Left hypoglossal nucleus
c. Right inferior frontal lobe
d. Left inferior frontal lobe
e. Right cerebral peduncle

**289.** What type of speech problem does Emma have?

a. Broca's aphasia
b. Wernicke's aphasia
c. Mixed aphasia
d. Dysarthria
e. Agnosia

## Questions 290–294

Julie is a 29-year-old office worker with diabetes, who awoke one morning with the inability to close her left eye and a left facial droop. Her left eye felt a bit dry, as well. She had run out of sick days and, hoping that the problem would go away, went to work. After several coworkers noticed that her face was drooping and that she was especially sensitive to loud noises on her left side, they convinced her to go to the nearest emergency room in order to make sure that she did not have a stroke. She was examined right away in the emergency room because of her age. The doctor noted right away that her mouth drooped on the left side. Her left eye was slightly closed. He tested her speech and mental status, which were normal, other than some slight slurring of her speech. Her vision and eye movements were also normal. Sensation and jaw movement were also normal, but when she was asked to wiggle her eyebrows, only the right side of her forehead moved. When asked to close her eyes tightly, and not allow him to open her eyes, her right eye would not open, but her left eye could not oppose the force. She was not able to hold air in her cheeks when asked to hold her breath, and when asked to smile, only the right side of her mouth elevated. She was very sensitive to noise on her left side. When asked to protrude her tongue, it did not deviate to either side, but if she closed her eyes and sugar water was placed on the left side of the anterior portion of her tongue, she could not identify it. The remainder of her examination was normal. A nurse asked if a head CT should be ordered in order to look for a stroke or tumor, but the doctor said that it wasn't necessary. He told Julie that he would draw some blood and give her a medication to take for a while.

**290.** Assuming that the doctor was correct, and that this isn't a stroke, where in the nervous system has the damage occurred?

a. Buccinator muscle
b. Trigeminal nerve
c. Facial nerve
d. Glossopharyngeal nerve
e. Hypoglossal nerve

**291.** Julie's facial weakness is characteristic of

a. A muscle lesion
b. A lesion of the internal capsule
c. A superior brainstem lesion
d. A UMN seventh-nerve lesion
e. An LMN seventh-nerve lesion

**292.** Damage to which area may have produced the defect in taste in the anterior two-thirds of her tongue?

a. Intermediate nerve
b. Glossopharyngeal nerve
c. Lingual nerve
d. Facial nerve, distal to the chorda tympani nerve
e. Facial nerve, proximal to the chorda tympani nerve

**293.** Assuming that Julie had no prior problems with her ears or cochlear nerve, damage to the nerve supply of which muscle could cause the sensitivity to or distortion of noises?

a. Digastric
b. Platysma
c. Buccinator
d. Geniohyoid
e. Stapedius

**294.** If Julie did not have the loss of taste and noise sensitivity, but did have the inability to move her left eye to the left, which area would now be damaged?

a. The trigeminal and abducens nerves
b. The facial and trigeminal nerves, distal to their exit from the brainstem
c. The facial and abducens nerve nuclei within the pons
d. The facial nerve, distal to the chorda tympani nerve
e. The facial nerve, distal to the geniculate ganglion

## Questions 295–299

A second-year medical student was asked to see a nursing home patient as a requirement for a physical diagnosis course. The patient was a 79-year-old man who was apparently in a coma. The student wasn't certain of how to approach this case, so he asked the patient's wife, who was sitting at the bedside, why this patient was in a coma. The wife replied: "Oh, Paul isn't in a coma. But he did have a stroke." Slightly confused, the student leaned over and asked Paul to open his eyes. He opened his eyes immediately. However, when asked to lift his arm or speak, Paul did nothing. The student then asked Paul's wife if she was certain that his eye opening was not simply a coincidence, and that he really was in a coma, since he was unable to follow any commands. Paul's wife explained that he was unable to move or speak as a result of his stroke. However, she knew that he was awake, because he could communicate with her by blinking his eyes. The student appeared rather skeptical, so Paul's wife asked her husband to blink once for "yes" and twice for "no." She then asked him if he were at home and he blinked twice. When asked if he were in a nursing home, he blinked once. The student then asked him to move his eyes, and he was able to look in his direction. However, when the student asked him if he could move his arms or legs, he blinked twice. He also blinked twice when asked if he could smile. He did the same when asked if he could feel someone moving his arm. The student thanked Paul and his wife for their time, made notes of his findings, and returned to class.

**295.** Where in the nervous system could a lesion occur that can cause paralysis of the extremities bilaterally, as well as the face, but not of the eyes?

a. High cervical spinal cord bilaterally
b. Bilateral thalamus
c. Bilateral basal ganglia
d. Bilateral pontine tegmentum
e. Bilateral frontal lobe

**296.** An infarct in what vascular distribution could cause this lesion?

a. Anterior spinal artery
b. Vertebral artery
c. Basilar artery
d. Middle cerebral artery
e. Posterior cerebral artery

**297.** Damage to which tracts caused Paul's inability to move his arms and legs?

a. Corticospinal and corticobulbar tracts
b. Spinothalamic tract
c. Solitary tract
d. Superior cerebellar peduncle
e. Inferior cerebellar peduncle

**298.** Damage to which tract caused Paul's lack of perception of someone moving his arm?

a. Corticospinal and corticobulbar
b. Middle cerebellar peduncle
c. Spinothalamic tract
d. Rubrospinal tract
e. Medial lemniscus

**299.** What area is spared to preserve consciousness?

a. Deep frontal white matter
b. Pontine reticular formation
c. Temporal lobes
d. Prefrontal cortex
e. Occipital lobe

### Questions 300–304

Herb, a 62-year-old man who has smoked two packs of cigarettes per day for 35 years, was suffering from a chronic cough that was attributed to smoking habit by his physician. One day, Herb noticed that his right eyelid drooped slightly and that his right pupil was smaller than the left. He also noticed that the inner side of his right hand was numb and that he had begun to drop things from his right hand. He had no other symptoms. Herb consulted his physician who directed him to a neurologist.

The neurologist noted that although the right pupil was smaller than the left, it was still reactive to light. Although Herb's right eyelid drooped slightly, he could close his eyes tightly when asked to do so. The neurologist noted that Herb did not sweat on the right side of his face. He was unable to feel a pinprick on the inner surface of his right hand, and his right triceps and hand muscles were weak.

**300.** Where in the nervous system has damage occurred?

a. Left oculomotor nerve
b. Right oculomotor nerve
c. Edinger-Westphal nucleus
d. Sympathetic fibers coursing from the hypothalamus to the intermediolateral cell column
e. Parasympathetic fibers coursing from the Edinger-Westphal nucleus

**301.** Herb's small pupil is due to

a. Unopposed action of the muscles with parasympathetic innervation
b. Unopposed action of the muscles with sympathetic innervation
c. Both sympathetic and parasympathetic damage
d. A lesion in the nucleus of the third nerve
e. A lesion distal branches of the trochlear nerve

**302.** Why was Herb able to close his eye tightly, despite a drooping eyelid?

a. The facial nerve does not innervate muscles mediating eye closure
b. The facial nerve is only partially affected
c. The facial nerve is unaffected by this lesion
d. The trigeminal nerve compensates for eye closure
e. This lesion only affects involuntary eye closure

**303.** Which pair of neurotransmitters is involved in the pathway that has been damaged?

a. Substance P and acetylcholine (ACh)
b. Norepinephrine and epinephrine
c. 5-HT and GABA
d. GABA and ACh
e. ACh and norepinephrine

**304.** Damage to which fibers caused the numbness and weakness of his hand?

a. Damage to the ipsilateral cerebral peduncle
b. Damage to the corticospinal tract
c. Damage to the cervical spinal roots entering the brachial plexus
d. Infarction of the basilar artery
e. Damage to the median nerve

## Questions 305–309

Mike is a 35-year-old man who had optic neuritis (an inflammation of the optic nerve causing blurred vision) several years before. He was told that he had a 50% chance of eventually developing multiple sclerosis (MS), a degenerative disease of the CNS white matter. One day, he noticed that he had double vision and felt weak on his right side. Although he noted that the symptoms were becoming steadily worse throughout the day, he attributed this to stress from his job as a stockbroker, and in order to relax, he decided to take a drive in his car. While he was driving, his vision became steadily worse. As he was about to pull over to the side of the road, he saw two trees on the right side of the road. Uncertain which was the actual image, he attempted to place his right foot on the break pedal. Mike suddenly realized that he was unable to lift his right leg, and his car collided with the tree. A pedestrian on the side of the road called the EMS, and Mike was brought to a nearby emergency room.

A neurologist was called to see Mike because the emergency room physicians thought he may have had a stroke, despite his young age. The neurologist spoke to Mike, then examined him. He found that his left eye was deviated to the left and down. When he attempted to look to his right, his right eye moved normally, but his left eye was unable to move further to the right than the midline. His left pupil was dilated and did not contract to light from a penlight. His left eyelid drooped, and he had difficulty raising it. In addition, the right side of his mouth remained motionless when he attempted to smile, but his forehead was symmetric when he raised his eyebrows. Mike's right arm and leg were markedly weak. The neurologist told Mike that he wasn't certain that this was necessarily a stroke, but admitted him to the hospital for observation and tests.

**305.** A lesion in which nerve caused Mike's double vision?

a. Optic nerve
b. Oculomotor nerve
c. Cervical sympathetic fibers
d. Trochlear nerve
e. Abducens nerve

**306.** Damage to which nerve innervating which eye muscles caused Mike's eye to be deviated toward the left side and down?

a. Superior rectus, superior oblique, inferior rectus, inferior oblique
b. Superior rectus, inferior rectus, inferior oblique, lateral rectus
c. Superior rectus, inferior rectus, inferior oblique, medial rectus
d. Lateral rectus, superior oblique, medial rectus, inferior rectus
e. Lateral rectus, superior oblique, inferior oblique, medial rectus

**307.** Where in the nervous system did the damage occur?

a. Left frontal lobe
b. Right frontal lobe
c. Left eye
d. Cervical spinal cord
e. Midbrain

**308.** Damage to which fibers caused the enlarged, unreactive pupil on the left?

a. Medial longitudinal fasciculus
b. Frontal or pontine eye fields
c. Edinger-Westphal nucleus or preganglionic parasympathetic fibers
d. Trochlear nerve
e. Cervical sympathetic fibers

**309.** Damage to which area caused Mike's weakness?

a. Left precentral gyrus
b. Right precentral gyrus
c. Left cervical spinal cord
d. Right cervical spinal cord
e. Left cerebral peduncle

### Questions 310–314

A 17-year-old high school football player presented to a neurology clinic because his mother thought that he may have acquired neck problems during a game. A month before, he had sustained a concussion from a blow to his head from another player. Shortly after, she noted that he intermittently tilted his head to the side. When asked what was the matter, he simply said that sometimes he had double vision, and that the images were situated on top of each other vertically, making it difficult to go down stairs.

When examined, there was no neck pain or limitation of motion. He tended to keep his head tilted to the right side. When asked to follow the doctor's finger with his head in a straight position, his left eye would not move downward when his eyes were turned to the right, and tended to remain slightly deviated toward the left. At this point, he stated that he had double vision, and felt better if his head was tilted to the right. The remainder of his eye movements, as well as the remainder of his exam, was normal.

**310.** Where has the damage occurred?

a. The oculomotor nerve
b. The abducens nerve
c. The trochlear nerve
d. The trigeminal nerve
e. The facial nerve

**311.** Which muscle is weakened?

a. Superior rectus
b. Inferior rectus
c. Lateral rectus
d. Superior oblique
e. Inferior oblique

**312.** From which portion of the brainstem has the damaged nerve emerged?

a. Right ventral midbrain
b. Right dorsal midbrain
c. Left ventral midbrain
d. Left dorsal midbrain
e. Left ventral pons

**313.** What is the action of the weak muscle?

a. Outward and upward rotation of the orbit
b. Outward and downward rotation of the orbit
c. Inward and upward rotation of the orbit
d. Inward and downward rotation of the orbit
e. Deviation of the orbit laterally

**314.** How could the head trauma have caused the double vision?

a. Direct damage to the eye
b. Damage to the occipital lobes
c. Damage to the midbrain
d. Damage to the pons
e. Damage to the cranial nerve peripherally

# The Brainstem and Cranial Nerves

## *Answers*

**185–186. The answers are 185-d and 186-c.** *(Gilroy, p 33; Simon et al, p 347; Afifi, p 175.)* The reflex described in this question is the corneal reflex. It involves the reflex activation of the ophthalmic division of the sensory component of cranial nerve V in response to touching of the cornea and the motor division of the facial (cranial nerve VII), which produces the motor component (i.e., the blinking response). The sensory component of cranial nerve V is classified as a general somatic afferent fiber and the somatic motor component of cranial nerve VII is classified as a special visceral efferent fiber.

**187–189. The answers are 187-d, 188-c, 189-b.** *(Gilroy, pp 587–589; Afifi, pp 163–165.)* Taste associated with the anterior two-thirds of the tongue is mediated by the facial (cranial nerve VII) nerve. The geniculate ganglion contains the cell bodies associated with the sensory (gustatory) component of the seventh nerve. The somatic motor component of the seventh nerve mediates the muscles of facial expression. Thus, the sensory and motor components of the seventh nerve affected in this individual can be characterized as special visceral afferent (because this afferent contains chemoreceptors) and special visceral efferent (because the motor component innervates skeletal muscle and is derived from a branchial arch), respectively.

**190–193. The answers are 190-c, 191-c, 192-b, 193-b.** *(Gilroy, pp 590–591; Afifi, pp 131–132.)* Cranial nerve IX, the glossopharyngeal nerve, innervates the skeletal muscles of the pharynx. The motor component involved arises from the nucleus ambiguus of the medulla. This cranial nerve also contains afferents, a component of which arises from the superior ganglion. These sensory neurons convey somatosensory sensation, including pain afferents that ultimately synapse in the spinal trigeminal nucleus. The motor component of the glossopharyngeal nerve mediating swallowing and coughing constitutes a special visceral efferent (because it

is derived from a visceral arch), and the sensory component conveying pain is referred to as a general somatic afferent fiber.

**194–195. The answers are 194-d and 195-b.** *(Afifi, p 232; Kandel, pp 668–669, 817.)* Rigidity can occur by experimentally producing a decerebrate preparation (i.e., severing the brainstem at the level of the pons) and it appears clinically as well. In both situations, there has to be extensive destruction of brain tissue below the midbrain in the region of the pons, but which spares the medulla. In this way, the lateral vestibulospinal tract remains intact. This pathway powerfully facilitates extensor motor neurons and extensor reflexes, thus contributing significantly to the expression of decerebrate rigidity, in particular, when the descending inhibitory pathways, which arise from more rostral levels, are disrupted by the experimental procedure or stroke.

**196–198. The answers are 196-d, 197-a, 198-d.** *(Afifi, pp 133–135; Kandel, pp 972–975.)* Specialized peripheral receptors, which specifically respond to changes in blood pressure, include the carotid sinus (associated with cranial nerve IX) and the aortic arch (associated with cranial nerve X). If these receptors (or the cell bodies associated with these receptors) are damaged, then one of the fundamental regulatory mechanisms for the control of blood pressure would be disrupted. The results of such a disruption would likely lead to increases and instability in blood pressure with evidence of spiking of blood pressure. Because these sensory receptors in these structures respond to increases in blood pressure, they are, in effect, stretch receptors and are consequently referred to as *baroreceptors.* The principal projection of the axons associated with these baroreceptors is the solitary nucleus of the medulla, which in turn, projects to autonomic nuclei such as the dorsal motor nucleus of the vagus nerve, ventrolateral medulla, and higher regions associated with autonomic functions, which include the PAG, hypothalamus, and limbic system.

**199. The answer is b.** *(Afifi, pp 82–85.)* The medial vestibulospinal tract arises from the medial vestibular nucleus and descends in the medial longitudinal fasciculus to cervical levels where it controls LMNs, which innervate (flexor) muscles controlling the position of the head. The lateral vestibulospinal tract facilitates extensor motor neurons of the limbs; the rubrospinal tract facilitates flexor motor neurons of the limbs; and the reticulospinal tracts modulate muscle tone of the limbs.

**200–202. The answers are 200-c, 201-c, 202-b.** *(Martin, pp 42–44; Afifi, pp 125–133.)* This individual who suffers a paralysis of the soft palate and pharynx, as well as loss of the carotid sinus reflex, sustains damage that includes cranial nerve X. It should be noted that several of these symptoms could have been incurred by damage to cranial nerve IX as well. However, in this question, cranial nerve IX was not listed as a choice. The axons of the nucleus ambiguus of cranial nerve X innervate the soft palate and pharynx. Damage to these neurons would frequently cause dysphagia, hoarseness, and paralysis of the soft palate. In addition, damage to the dorsal motor nucleus of the vagus constitutes an efferent limb for expression of the carotid sinus reflex. Thus, damage to these two nuclear groups would produce the constellation of deficits described for this case. The nucleus ambiguus is classified as a special visceral efferent fiber because it innervates skeletal muscle and it is derived from a visceral arch, while the dorsal motor nucleus innervates thoracic and abdominal viscera and is therefore classified as a general visceral efferent fiber.

**203. The answer is c.** *(Martin, pp 42–49; Afifi, pp 148–153.)* The combined deficit in which the patient loses ability to (use his lateral rectus muscle) abduct his right eye and display facial expression on the right side of the face means that the lesion is located in the dorsal pons at the site where the facial nerve curves around (just above) the motor nucleus of cranial nerve VI. Thus, a lesion at this site will affect both cranial nerves, causing the combined deficits described previously.

**204. The answer is e.** *(Afifi, pp 209–213.)* The cranial nerve involved in adduction of the eye is cranial nerve III, in which the act of moving the eye medially is governed by the medial rectus muscle. Cranial nerve III is located near the midline of the rostral half of the midbrain just below the midbrain periaqueductal gray.

**205–207. The answers are 205-d, 206-d, 207-a.** *(Martin, pp 42–49; Afifi, pp 194–195.)* To walk down stairs, one has to have the ability to move the eyes down when they are in the medial position. This involves the use of cranial nerve IV (trochlear nerve), which innervates the superior oblique muscle (whose action is to pull the eye downward when in the medial position). If there is damage to this nerve on one side, the eyes will not be able to focus on the same visual field, thus producing double vision. Cranial

nerve IV is classified as a general somatic efferent fiber because it innervates skeletal muscle and it is derived from somites.

**208. The answer is d.** *(Afifi, pp 118–120.)* Two of the principal afferent fiber systems that project to the inferior olivary nucleus include the red nucleus and spinal cord. These inputs serve important functions of enabling the inferior olivary nucleus of transmitting information related to both sensory (spinal cord) and motor (red nucleus) processes to the cerebellar cortex. The other structures indicated in this question do not have known projections to the inferior olivary nucleus.

**209. The answer is e.** *(Afifi, pp 118–120.)* The primary projection pathway of the inferior olivary nucleus exits this nucleus, enters the inferior cerebellar peduncle of the contralateral side of the brain, and passes into the cerebellar cortex, terminating on apical dendrites of Purkinje cells throughout the cerebellar cortex in a somatotopic manner. As indicated previously in the explanation of the previous question, this pathway represents an important source of input to the cerebellum from significant regions mediating sensory and motor information (via the inferior olivary nucleus). Other suggested answers do not include known projection targets of the inferior olivary nucleus.

**210–211. The answers are 210-c and 211-c.** *(Afifi, pp 133–134.)* The central pathways mediating taste include the following: primary afferent taste fibers associated with taste receptors of cranial nerves VII, IX, and X synapse in the solitary nucleus. Many fibers from the solitary nucleus project to the ventral posteromedial nucleus of the thalamus, which, in turn, project to the ventrolateral aspect of the postcentral gyrus.

**212–215. The answers are 212-b, 213-c, 214-d, 215-e.** *(Afifi, pp 130–134; Gilroy, pp 590–591; Simon et al, pp 92–93.)* The cranial nerve that was directly affected was the glossopharyngeal nerve (cranial nerve IX). This is a mixed and complex nerve containing: (1) special visceral efferents from the nucleus ambiguus that supply the stylopharyngeus muscle (for elevation of pharynx in speech); (2) special visceral afferent fibers that transmit taste impulses from the posterior third of the tongue, general visceral afferent fibers associated with the inferior ganglion whose receptors lie in the carotid sinus that regulates cardiovascular functions; (3) general

somatic afferents whose cell bodies lie in the superior ganglion of cranial nerve IX, and which mediate somatosensory information, including pain from the pharynx; and (4) general visceral efferent fibers that originate in the inferior salivatory nucleus, which are preganglionic and synapse in the otic ganglion. The postganglionic fiber from the otic ganglion innervates the parotid gland and mediates, in part, salivation. Thus, when this nerve is affected by an infectious agent, it results in the constellation of symptoms presented earlier in this case. Since the cell bodies of motor (or visceral motor) fibers (mediating motor and visceral effects) as well as the terminals of sensory afferents (mediating pain from the pharynx) lie in different regions of the medulla, it is very unlikely that such an effect could be the result of damage centrally. A much more likely occurrence is that the infectious agent produced disruption of the glossopharyngeal nerve peripherally, such as at the base of the skull or jugular foramen, where all the components run together and can be more easily affected.

**216–218. The answers are 216-b, 217-d, 218-c.** *(Afifi, pp 163–166; Gilroy, pp 588–589; Simon et al, pp 347–348.)* The nerve affected by this disorder is cranial nerve VII (facial nerve). The cell bodies of origin, which innervate the muscles of facial expression (special visceral efferents), arise from the facial nucleus, which are located in the ventrolateral aspect of the lower pons. The preganglionic parasympathetic neurons, which synapse with postganglionic neurons in the submandibular and pterygopalatine ganglia, arise from the superior salivatory nucleus of the lower pons. Based on the same reasoning as indicated in the answer to question 215, the most likely locus of the defect is the geniculate ganglion. The region of the geniculate ganglion and regions adjacent to it contain sensory, skeletal, and visceral motor components of this nerve. Therefore, disruption of this nerve in the region of the geniculate ganglion will produce the constellation of deficits described in this case. The other choices are not appropriate. Cranial nerve IX is not involved. Neither are the regions of the reticular formation and facial nucleus, because lesions at either of these locations could not account for the totality of deficits described in this case. The lesion could not have involved the cerebral cortex because the motor effects were described as a flaccid facial paralysis. A cortical lesion does not produce flaccidity of these muscles.

**219–223. The answers are 219-A, 220-B, 221-E, 222-C, 223-D.** *(Afifi, pp 104–117.)* The nucleus gracilis (A) contains cells that respond to

movement of the lower limb as a result of joint capsule activation. The nucleus cuneatus (B) contains cells that respond to a variety of stimuli applied to the upper limb, including vibratory stimuli. One component of the descending medial longitudinal fasciculus (E) contains fibers that arise from the medial vestibular nucleus that project to cervical levels and contribute to reflex activity associated with the position of the head. Fibers of the medial lemniscus (D) arise from the contralateral dorsal column nuclei and ascend to the ventral posterolateral nucleus of the thalamus. These fibers transmit the same information noted earlier for the dorsal column nuclei, which includes two-point discrimination and conscious proprioception from the opposite side of the body.

**224–232. The answers are 224-B, 225-C, 226-H, 227-B, 228-H, 229-G, 230-I, 231-A, 232-E.** *(Nolte, pp 254–276; Afifi, 117–135.)* Different groups of neurons of the solitary complex (B) respond to taste stimuli and to inputs that signal sudden changes in blood pressure. The medial vestibular nucleus (C) receives direct vestibular inputs from the otolith organ and semicircular canals. Axons of medial vestibular neurons descend to the spinal cord in the medial longitudinal fasciculus and serve to regulate reflexes associated with the head. The inferior vestibular nucleus (D) also receives vestibular inputs, but does not project its axons to the spinal cord. The inferior olivary nucleus (H) receives inputs from the red nucleus and spinal cord, and it projects its axons through the inferior cerebellar peduncle (where it constitutes its largest component) to the contralateral cerebellar cortex, where they synapse with the dendrites of Purkinje cells.

The nucleus ambiguus (G) is a special visceral efferent nucleus that is situated in a position ventrolateral to that of the hypoglossal nucleus. Its axons innervate the muscles of the larynx and pharynx and, therefore, are essential for the occurrence of such responses as the gag reflex. The pyramids (I), located on the ventromedial aspect of the brainstem, contain fibers that arise from the sensorimotor cortex. These neurons serve as essential upper motor neurons that mediate voluntary control of motor functions. The hypoglossal nucleus (A), a general somatic efferent nucleus, is located in the dorsomedial aspect of the medulla. Its axons innervate the muscles of the tongue and cause extrusion of the tongue toward the opposite side. Fibers contained in the inferior cerebellar peduncle (E) arise from cells located in both the spinal cord and brainstem.

**233–238. The answers are 233-A, 234-A, 235-C, 236-B, 237-A, 238-C.** *(Afifi, pp 147–167, 303–320; Nolte, pp 470–484.)* The efferent projections of the cerebellum arise from three distinct groups of nuclei called *deep cerebellar nuclei.* The nucleus located in the most medial position is the fastigial nucleus (A). It gives rise to at least two important projections: one that is distributed to the reticular formation and another that is distributed to the vestibular nuclei. The nucleus situated most laterally is the dentate nucleus (C). It projects through the superior cerebellar peduncle and its axons innervate principally the ventrolateral nucleus of the thalamus.

Neurons of the emboliform (B) and globose (not labeled) nuclei lie in an intermediate position between the fastigial and dentate nuclei and, therefore, are often referred to as the *interposed nuclei.* Their axons project through the superior cerebellar peduncle principally to the red nucleus. The projections from the cerebellar cortex to the deep cerebellar nuclei are topographically organized. Cells located in the far medial aspect of the cerebellar cortex, the vermal region, project (via Purkinje cell axons) to the fastigial nucleus (A). In contrast, the lateral aspects of the cerebellar hemispheres project to the dentate nucleus (C), which is the most lateral of the deep cerebellar nuclei.

**239–245. The answers are 239-B, 240-E, 241-D, 242-A, 243-G, 244-F, 245-C.** *(Afifi, 118–139; Nolte, pp 230–239, 255–274, 284–307.)* The inferior vestibular nucleus (B) lies immediately medial to the inferior cerebellar peduncle (shown at C) and receives direct inputs from first-order vestibular fibers that arise from the vestibular apparatus. The medial longitudinal fasciculus (E) contains second-order vestibular fibers, the majority of which ascend in the brainstem to innervate cranial nerve nuclei III, IV, and VI. A small component of this bundle also descends to cervical levels of the spinal cord from the medial vestibular nucleus. The solitary nucleus (D) receives inputs from first-order taste fibers and is thus a special visceral afferent nucleus that transmits taste signals to the ventral posteromedial nucleus of the thalamus. The solitary nucleus also receives cardiovascular inputs from cranial nerve IX and, for this reason, has properties of a general visceral afferent nucleus as well. The hypoglossal nucleus (A) innervates the muscles of the tongue. It is classified as a general somatic efferent nucleus because it is derived from somites rather than branchial arches and innervates skeletal muscle.

The medial lemniscus (G) ascends to the thalamus and transmits information associated with conscious proprioception. This bundle constitutes a second-order neuron that arises from the dorsal column nuclei of the lower medulla. The dorsal column nuclei receive first-order signals that mediate conscious proprioception from fibers contained within the dorsal columns of the spinal cord. The spinal nucleus of cranial nerve V (F) receives pain and temperature fibers from first-order trigeminal neurons that arise from the head. The inferior cerebellar peduncle (C) is one of two principal cerebellar afferent pathways. One major fiber group contained within the inferior cerebellar peduncle arises from brainstem structures such as the contralateral inferior olivary nucleus and reticular formation. The other groups of fibers contained within this bundle arise from the spinal cord. Of the fibers that ascend in this bundle from the spinal cord, many constitute second-order muscle spindle afferents that arise from Clarke's nucleus dorsalis.

**246–250. The answers are 246-C, 247-B, 248-D, 249-A, 250-E.** *(Afifi, pp 147–167, 303–320; Nolte, pp 230–239, 256–259, 470–478.)* The middle cerebellar peduncle (C) serves as a relay nucleus for the transmission of information from the cerebral cortex to the cerebellum. Fibers in this peduncle arise from the contralateral deep pontine nucleus, which receives its principal afferents from the cerebral cortex. The motor nucleus of cranial nerve V (B) is an LMN (special visceral efferent) because it innervates the muscles of mastication. Corticobulbar and corticospinal fibers (D) are situated in the ventral aspect of the basilar pons. Fibers of the superior cerebellar peduncle (A) project to both the red nucleus and ventrolateral nucleus of the thalamus. The medial lemniscus (E) is a somatotopically organized pathway that arises from the dorsal column nuclei and projects to the ventral posterolateral nucleus of the thalamus. Fibers of this pathway that arise from the nucleus gracilis (associated with the leg) project to more dorsolateral aspects of the ventral posterolateral nucleus. Fibers arising from the nucleus cuneatus (associated with the arm) project to more ventromedial aspects of the ventral posterolateral nucleus.

**251–255. The answers are 251-E, 252-B, 253-C, 254-A, 255-D.** *(Afifi, pp 187–213; Nolte, pp 266–277.)* The inferior colliculus (E) is situated in the caudal aspect of the tectum and is an important relay nucleus for the

transmission of auditory information to the cortex from lower levels of the brainstem. The decussation of the superior cerebellar peduncle (B) is also present at caudal levels of the midbrain and is usually seen together with the inferior colliculus. These crossing fibers arise from the dentate and interposed nuclei and terminate in the contralateral red nucleus and ventrolateral nucleus of the thalamus. The crus cerebri (C) contains fibers that arise from all regions of the cortex and project to all the levels of the brainstem and the spinal cord. The trochlear nucleus (cranial nerve IV) (A), which is situated just below the periaqueductal gray at the level of the inferior colliculus, receives direct inputs from ascending fibers of the medial longitudinal fasciculus that arise from vestibular nuclei. The midbrain periaqueductal gray (D) contains dense quantities of enkephalin-positive cells and nerve terminals. The transmitter (or neuromodulator) enkephalin plays an important role in the regulation of pain and emotional behavior.

**256–260. The answers are 256-E, 257-D, 258-A, 259-C, 260-F.** *(Afifi, pp 187–213; Nolte, pp 266–277.)* The superior colliculus (E), situated at a more rostral level of the tectum, plays an important role in tracking or pursuit of moving stimuli. The medial geniculate nucleus (D), which is part of the forebrain, actually sits over the lateral aspect of the midbrain and can be seen at rostral levels of the midbrain. It is part of an auditory relay system and receives its inputs from the inferior colliculus via fibers of the brachium of the inferior colliculus. The pars compacta is situated in the medial aspect of the substantia nigra (A) and contains dopamine neurons whose axons innervate the striatum. The red nucleus (C), a structure associated with motor functions, receives direct inputs from both the cerebral cortex and the cerebellum. The oculomotor nerve (cranial nerve III) (F), located at the level of the superior colliculus, contains general somatic efferent components that innervate extraocular eye muscles and general visceral efferent components whose postganglionic fibers innervate smooth muscles associated with pupillary constriction and bulging of the lens.

**261. The answer is a.** *(Afifi, pp 171–177; Nolte, pp 294–301.)* The spinal trigeminal nucleus receives its sensory inputs from first-order neurons contained in the ipsilateral descending tract of cranial nerve V. A central property of the spinal trigeminal nucleus is that it is uniquely associated with pain inputs (to the exclusion of the main sensory nucleus and mesen-

cephalic nucleus). Fibers from this nucleus mainly project contralaterally to the ventral posteromedial nucleus of the thalamus.

**262. The answer is c.** *(Kandel, pp 1292–1294; Nolte, pp 136, 138–139.)* The area postrema is of interest because it is a circumventricular organ associated with emetic functions. As a circumventricular organ, the area postrema constitutes a part of the ependymal lining of the brain's ventricular system (in this case, the fourth ventricle). The area postrema contains both fenestrated and nonfenestrated capillaries that allow for enhanced transport, which possibly accounts for the fact that it lies outside the blood-brain barrier. Axons and dendrites from neighboring structures (but not from the forebrain) innervate this structure, which is composed of astroblast-like cells, arterioles, sinusoids, and some neurons. Various peptides (but not monoamine-containing neurons) have also been shown to be present in this structure. Experimental evidence has strongly implicated the area postrema as a chemoreceptor trigger zone for emesis. It responds to digitalis glycosides and apomorphine.

**263. The answer is a.** *(Afifi, pp 129–131.)* Cranial nerve X is a highly complex nerve. It contains a few general somatic afferents from the back of the ear that enter the brain as cranial nerve X but terminate in the trigeminal complex. Special visceral afferents include fibers from chemoreceptors for taste associated with the epiglottis and chemoreceptors in the aortic bodies that sense changes in $O_2$-$CO_2$ levels in the blood. General visceral afferent fibers arise from the trachea, pharynx, larynx, and esophagus and signal changes in blood pressure to the brainstem. Special visceral efferent fibers innervate the constrictor muscles of the pharynx and the intrinsic muscles of the larynx. General visceral efferent fibers constitute part of the cranial aspect of the parasympathetic nervous system; thus, they are preganglionic parasympathetic fibers that innervate the heart, lung, esophagus, and stomach.

**264. The answer is c.** *(Afifi, pp 142–144; Nolte, p 280.)* A primary characteristic of a lesion of the dorsolateral medulla is loss of pain and temperature sensation on the contralateral side of the body and ipsilateral half of the face. Damage to the descending tract of the trigeminal nerve and to the spinal nucleus of cranial nerve V will produce loss of sensation on the ipsilateral side of the face. There also will be damage to the lateral spinothala-

mic tract, which has already crossed at the level of the spinal cord and which conveys pain and temperature sensation from the contralateral side of the body. In addition, fibers arising from the nucleus ambiguus exit laterally from the medulla, and these fibers, which innervate the larynx and pharynx, would also be affected, causing dysphonia. Hemiparesis would not result from this lesion since the pyramidal tract would remain intact. The cerebellum would also be spared and intention tremor associated with cerebellar damage would not occur.

**265. The answer is b.** *(Martin, pp 212–213; Nolte, pp 326–342.)* The olivocochlear bundle is a most interesting pathway because it arises from the region immediately dorsal to the superior olivary nucleus and projects contralaterally back to the hair cells of the cochlea. Stimulation of this bundle results in inhibition or reduction of responses to auditory signals by auditory nerve fibers. There is no evidence that the olivocochlear bundle bears any anatomic or functional relationship to the medial lemniscus. Since the pathway arises from the superior olivary nucleus, which is present at the level of the lower pons, it would not be visible in a section taken from the upper pons.

**266. The answer is c.** *(Afifi, pp 481–484; Nolte, pp 326–342.)* Since the auditory relay system is a highly complex pathway in which auditory signals are bilaterally represented at all levels beyond the receptor level, lesions at these levels would not produce a solely unilateral deafness. Such a loss could only result when the lesion involves either the receptor or the first-order neurons of the nerve (i.e., cranial nerve VIII itself). The medial lemniscus is not related to the auditory system.

**267. The answer is c.** *(Afifi, pp 171–176; Nolte, pp 295–301.)* In general, first-order sensory neurons form ganglia outside the CNS. There is one exception, the mesencephalic nucleus of cranial nerve V, which transmits unconscious proprioception (i.e., muscle spindle activity) from jaw muscles. These inputs serve as the first-order neurons for a disynaptic pathway to the cerebellum, as well as for a monosynaptic pathway with the motor nucleus of cranial nerve V for the jaw-closing reflex.

**268. The answer is d.** *(Afifi, pp 168–171; Nolte, pp 290–294.)* Conjugate lateral gaze requires the simultaneous contractions of the lateral rectus

muscle of one eye and the medial rectus of the other eye. Recent studies have indicated that there is a region that integrates and coordinates such movements and that the site is part of the nucleus of cranial nerve VI. It is likely that it accomplishes this phenomenon, in part, because ascending axons from the abducens nucleus pass through the medial longitudinal fasciculus to the contralateral nuclei of cranial nerve III. Thus, the abducens nucleus serves not only to innervate the lateral rectus muscle but also to integrate signals necessary for conjugate deviation of the eyes. The abducens nucleus appears to be the only cranial nerve structure where lesions of the root fibers and nucleus fail to display identical effects.

**269. The answer is d.** *(Afifi, pp 169–171; Nolte, pp 288–294, 351–355, 359–373.)* The paramedian pontine reticular formation is an important integrating structure controlling the position of the eyes. It receives inputs from the cerebral cortex (presumably the region of the frontal eye fields) and fibers from the cerebellum, spinal cord, and vestibular complex. Its efferent fibers project to the cerebellum, vestibular complex, pretectal region, interstitial nucleus of Cajal, and nucleus of Darkschewitsch of the rostral midbrain. These all are nuclei concerned with the regulation of eye position and movements. It is not related to any other known motor or auditory functions, nor has it been shown to contain ascending noradrenergic neurons.

**270. The answer is c.** *(Afifi, pp 227–230; Nolte, pp 260–262, 290–294.)* For a lesion to produce both an ipsilateral gaze paralysis and contralateral hemiplegia, it must be situated in a location where fibers regulating both lateral gaze and movements of the contralateral limbs lie close to each other. The only such location is the ventrocaudal aspect of the pons, where fibers of cranial nerve VI descend toward the ventral surface of the brainstem and where corticospinal fibers are descending toward the spinal cord. The other regions listed in the question do not meet this condition.

**271. The answer is e.** *(Purves, pp 263–282.)* The group called special visceral afferent fibers is limited to those cranial nerves that convey impulses to the brain associated with olfaction (I) and taste (VII, IX, and X). Since olfaction and taste involve chemical senses, some authors also include cranial nerves IX and X in the group because these nerves contain components involved in signaling changes in $O_2$ and $CO_2$ levels in the blood.

**272. The answer is e.** *(Purves, pp 258–261; Nolte, pp 381–385.)* Because the deficit includes a homonymous hemianopsia, the lesion has to be located somewhere in the forebrain, such as in the region that includes the optic tract and internal capsule on the right side of the brain. The motor neurons of cranial nerve VII, as well as spinal cord motor neurons, receive cortical fibers that are crossed, which accounts for the fact that motor dysfunctions of the lower face and body involve lesions on the same side.

**273. The answer is d.** *(Afifi, pp 211, 227–231.)* Inability to move the eyes up or down when they are displaced laterally would result from a lesion of the midbrain involving cranial nerve III. Because the somatomotor neurons of cranial nerve III supply, in part, the superior and inferior recti muscles as well as the inferior oblique muscle, cranial nerve III is responsible for up-and-down movements of the eye when they are positioned laterally. Recall that when the eye is positioned medially, it is the superior oblique that is innervated by cranial nerve IV that pulls the eye downward.

**274. The answer is c.** *(Afifi, p 218; Nolte, pp 431–432.)* This disorder is referred to as the *Argyll Robertson pupil* and occurs with CNS syphilis (tertiary). Although the precise site of the lesion has never been fully established, it is believed to be in the pretectal area. The reasoning is as follows: In the pupillary light reflex, many optic fibers terminate in the pretectal area and superior colliculus region and are then relayed to the autonomic nuclei of cranial nerve III. Impulses from this component of cranial nerve III then synapse with postganglionic parasympathetics that innervate the pupillary constrictor muscles, thus producing pupillary constriction. In the case of the accommodation reflex, retinal impulses first reach the cortex and are then relayed through corticofugal fibers to the brainstem. Some of these fibers are then relayed directly or indirectly to both motor and autonomic components of cranial nerve III, thus activating the muscles required for the accommodation reaction to occur, which includes pupillary constriction.

**275. The answer is b.** *(Kandel, pp 642–644; Nolte, pp 305–306, 313–316.)* The solitary nucleus receives first-order neurons from the taste system and thus serves as a critical relay nucleus for the taste pathway. Axons arising from the solitary nucleus project to the ventral posteromedial nucleus of the thalamus and may also synapse in the parabrachial

nuclei of the upper pons. Structures such as the ventral posterolateral nucleus, medial lemniscus, and superior parietal lobule are not associated with the taste pathway.

**276. The answer is b.** *(Kandel, pp 1309–1313; Nolte, pp 259–267, 280.)* The superior cerebellar artery supplies the dorsolateral aspect of the upper pons. The basilar artery supplies the medial aspect of the pons. The other arteries (vertebral, anterior spinal, and posterior inferior cerebellar) supply different parts of the medulla. The lateral aspect of the upper pons contains spinothalamic fibers, the lateral lemniscus, and the locus ceruleus (situated just dorsal to the motor nucleus of cranial nerve V, which is also affected by the lesion). This lateral pontine lesion produces a syndrome that includes (1) loss of pain and temperature sensation from the contralateral side of the body (damage to the lateral spinothalamic tract), (2) ipsilateral loss of masticatory reflexes (damage to the motor nucleus of cranial nerve V), (3) diminution of hearing (disruption of secondary auditory pathways), and (4) Horner's syndrome (disruption of descending fibers from the hypothalamus and midbrain that mediate autonomic functions).

**277. The answer is a.** *(Kandel, pp 1309–1313.)* The paramedian branch of the basilar artery supplies the ventromedial pons (i.e., medial basilar pons). The circumferential branch supplies more lateral regions of the pons as does the superior cerebellar artery. The anterior spinal and anterior inferior cerebellar arteries supply different parts of the medulla.

**278. The answer is c.** *(Afifi, pp 180–184; Kandel, pp 1309–1313.)* The lesion is restricted to the basilar pons. Thus, the only structure affected by this lesion among the choices given is the corticospinal tract. The other structures listed are situated in the tegmentum of the pons.

**279. The answer is a.** *(Afifi, pp 180–184; Kandel, pp 1309–1313.)* Since the lesion is restricted to the medial aspect of the basilar part of the pons, the corticospinal tract would be affected, producing paralysis of the contralateral limbs. Although other structures would also be affected and could produce additional deficits, such deficits are not listed in this question. The other dysfunctions listed would not occur because they are associated with structures situated in the pontine tegmentum, which is not included in this lesion.

**280. The answer is c.** *(Afifi, pp 227–229; Kandel, pp 1312–1314.)* The lesion involves the superior colliculus. This structure receives inputs from the cerebral cortex and optic tract and its neurons respond to moving objects in the visual field. It is considered essential for the regulation of tracking movements. Lesions of the superior colliculus have not been shown to produce any of the other deficits listed in this question. Nystagmus is not likely to occur because the lesion does not involve the medial longitudinal fasciculus or the pontine gaze center.

**281. The answer is a.** *(Afifi, pp 227–229; Kandel, pp 1312–1314.)* The lesion will disrupt fibers of the medial lemniscus (lateral aspect of the lesion) and thus produce contralateral loss of conscious proprioception. It will also disrupt fibers passing from the cerebellum to the red nucleus and ventrolateral nucleus of the thalamus, which could account for a tremor of the contralateral limb. Note that there would be no ipsilateral motor loss because functions associated with the red nucleus are expressed on the contralateral side. Oculomotor palsy would also be present because the lesion disrupts root fibers of cranial nerve III. However, the lesion is too rostral to affect cranial nerve IV. There is no hearing loss because the auditory fibers are situated too far laterally. Since the taste pathway is essentially ipsilateral, if any fibers are damaged by the lesion, deficits in taste sensation would be ipsilateral.

**282. The answer is c.** *(Afifi, pp 227–229; Kandel, pp 1312–1314.)* The primary structures damaged by this lesion include the crus cerebri, which results in a UMN paralysis of the contralateral limbs, as well as a paresis of the lower facial and tongue muscles. The other outstanding syndrome present from this lesion is a paralysis that results from damage to cranial nerve III. Other structures may be marginally affected.

**283. The answer is a.** *(Siegel et al, pp 251–252; Afifi, pp 199–201.)* The pars compacta of the substantia nigra contains dopamine neurons whose axons project to the neostriatum. In contrast, the dopaminergic neurons of the ventral tegmental area project to other areas of the forebrain, such as the hypothalamus, limbic system, and cerebral cortex. There is no known overlap in the distribution of these dopaminergic projection systems. A parkinsonian-like syndrome results from damage to the pars compacta of the substantia nigra, but not from a lesion restricted to the ventral tegmental region.

**284. The answer is b.** *(Afifi, pp 154–156; Nolte, pp 340–342.)* The principal ascending pathway of the auditory system listed in this question is the lateral lemniscus. It transmits information from the cochlear nuclei to the inferior colliculus. The trapezoid body is a commissure that contains some of the fibers of the lateral lemniscus that cross from the cochlear nuclei of one side of the brainstem en route to the inferior colliculus of the other side. The trapezoid body is present at the level of the caudal pons. The brachium of the superior colliculus, trigeminal lemniscus, and medial lemniscus do not transmit auditory sensory information.

**285–289. The answers are 285-e, 286-e, 287-a, 288-b, 289-d.** *(Adams, pp 799, 1383; Afifi, pp 125–127, 141–143.)* Emma has had a stroke resulting from occlusion of medial branches of the left vertebral artery, presumably secondary to atherosclerosis (i.e., cholesterol deposits within the artery, which eventually occlude it). The resulting syndrome is called the *medial medullary syndrome,* because the affected structures are located in the medial portion of the medulla. These structures include: the pyramids, the medial lemniscus, the medial longitudinal fasciculus, and the nucleus of the hypoglossal nerve and its outflow tract. Emma's symptoms result from damage to the aforementioned structures, and may have been caused by the same process (atherosclerosis) that resulted in her heart disease. The weakness of her right side was caused by damage to the medullary pyramid on the left side. Her face was spared because fibers supplying the face exited above the level of infarct. However, a lesion in the corticospinal tract of the cervical spinal cord above C5 could cause arm and leg weakness, and spare the face, because facial fibers exit in the rostral medulla. A lesion in the inferior portion of the precentral gyrus of the left frontal lobe would cause right-sided weakness, but would include the face, because this area is represented more inferiorly than are the extremities. Her unsteady gait was a result of the weakness of her right side, but may also have been the result of the loss of position and vibration sense on that side from damage to the medial lemniscus (as demonstrated by the inability to identify the position of her toe with her eyes closed, and the inability to feel the vibrations of a tuning fork). Without position sense, walking becomes unsteady because it is necessary to feel the position of one's feet on the floor during normal gait. Damage to both the medial lemniscus and pyramids at this level causes problems on the contralateral side because this lesion is located rostral to the level where both of these fiber bundles cross to the

opposite side of the brain. Damage to the descending component of the medial longitudinal fasciculus could only affect head and neck reflexes, but not gait. Gait is also unaffected by pain inputs. Deviation of the tongue occurs because fibers from the hypoglossal nucleus innervate the genioglossus muscle on the ipsilateral side of the tongue. This muscle normally protrudes the tongue toward the contralateral side. Therefore, if one side is weak, the tongue will deviate toward the side ipsilateral to the lesion when protruded. A lesion in the precentral gyrus causes protrusion of the tongue toward the side that is contralateral to the lesion, because it is rostral to the crossing of fibers into the hypoglossal nucleus. Emma's speech was dysarthric (slurred) because her tongue was weak on the left side. The physician saw this during the exam when her tongue deviated to the left when protruded. Since the weakness of the tongue is purely a motor problem, rather than an effect that is manifested by a lesion to higher centers in the cortex (which mediate the structure and function of speech), the grammar, content, and meaning of Emma's speech remained intact, as would be expected with an aphasia or agnosia.

**290–294. The answers are 290-c, 291-e, 292-e, 293-e, 294-c.** *(Adams, pp 1376–1377; Afifi, pp 163–168, 179–181.)* This is an example of Bell's palsy, or damage to the facial nerve distal to its nucleus in the pons. The motor weakness is LMN because of the involvement of the upper one-third of the face (this has bilateral innervation within the CNS). The loss of taste on the anterior two-thirds of the tongue and the hyperacusis (sensitivity to noise) point to damage that is distal to the brainstem because these are functions whose nerves join the facial nerve distal to its exit from the pons. This type of palsy may be caused by a virus and is more common among people with diabetes. This type of facial paralysis, involving the upper one-third of the facial muscles, is characteristic of an LMN facial nerve lesion. Since there is bilateral innervation within the CNS, from the prefrontal gyrus bilaterally until their synapse at the facial nerve nucleus, all UMN facial weakness spares the forehead. Since there is motor weakness of the face and since the chorda tympani nerve (which subserves taste) joins the facial nerve, it is likely that the lesion exists proximal to where the chorda tympani joins the facial nerve. A lesion in the lingual nerve (a branch of the trigeminal) would result in a loss of taste as well, but would also result in a loss of sensation to the face, not motor weakness. If the lesion occurred distal to the chorda tympani nerve, taste would have been

spared. The facial nerve sends a branch to the stapedius muscle distal to the geniculate ganglion, but proximal to the chorda tympani nerve. Lesions proximal to this branch will cause weakness of the stapedius muscle. Contraction of this muscle normally serves as a mechanism for dampening the motion of the ossicles, thus lowering the amount of stimulation reaching the organ of Corti. If this muscle is paralyzed, hyperacusis, or increased acuity, as well as hypersensitivity to low tones will occur. Since the genu of the facial nerve is in close proximity to the nucleus of the abducens nerve, the pons is a likely location for this particular type of combination of findings. Since the damage to the facial nerve has occurred distal to the facial nerve nucleus, an LMN facial palsy is present. The lack of hyperacusis and the presence of normal taste imply that the lesion is proximal to the geniculate ganglion. Therefore, the lesion must be between the facial nerve nucleus and the geniculate ganglion, and the location in the pons is the most likely choice. If this clinical picture is present, then an infarct or tumor in the pons must be suspected, and, in this case, an imaging study would be more appropriate.

**295–299. The answers are 295-d, 296-c, 297-a, 298-e, 299-b.** *(Adams, pp 802–805; Afifi, pp 163–168, 180–182.)* This is an example of the *locked-in syndrome,* or pseudocoma, caused by an infarction of the pontine tegmentum. Because the tracts mediating movement of the limbs and face run through this region, the patient is unable to move the face, as well as both arms and legs. Consciousness and eye movements are preserved. The pontine tegmentum is mainly supplied by the basilar artery. Complete occlusion of this artery causes deficits on both sides since this artery supplies both sides of the pons. Basilar artery occlusion causes damage to the basilar pons, where the corticospinal and corticobulbar tracts run. These tracts contain motor fibers mediating movement of the limb and face, respectively. This results in complete paralysis to both sides of the body and the face. None of the tracts in the other choices mediate conscious movement. Sensory loss, including loss of proprioception (feeling the movement of a limb), also occurs as a result of damage to the medial lemniscus bilaterally. This tract contains fibers from the dorsal columns and also runs through the pontine tegmentum. Patients with the locked-in syndrome are often mistaken for comatose patients due to their inability to move or speak. If the lesion spares the reticular formation, an area mediating consciousness in the pons, the patient will remain alert.

**300–304. The answers are 300-d, 301-a, 302-c, 303-e, 304-c.** *(Adams, p 280, 538; Afifi, pp 163–168, 179–184, 209–220, 227–229; Kandel, pp 962–974.)* Herb's drooping eyelid, small pupil, and lack of sweating on the right side are examples of Horner's syndrome. This is caused by the interruption of sympathetic fibers anywhere along their course from the hypothalamus and brainstem, to the intermediolateral cell column in the upper thoracic levels of the spinal cord where neurons, supplying sympathetic innervation to the pupil, the levator palpebrae superioris muscle of the eyelid and sweat glands of the face, are located. Interruption of this sympathetic innervation will result in the drooping of the upper eyelid (ptosis), pupillary constriction (miosis; due to unopposed action of the parasympathetic innervation of the circular muscles of the iris), and lack of sweating on the face. Parasympathetic or oculomotor damage causes pupillary dilation, rather than constriction. Herb could close his eyes tightly because this function is mediated by the seventh nerve, which is not damaged by this lesion. Preganglionic sympathetic neurons are predominantly cholinergic, and postganglionic sympathetic neurons are predominantly noradrenergic. Horner's syndrome may be caused by either a preganglionic or postganglionic lesion. The location may be determined by the use of eyedrops specifically targeted at a particular neurotransmitter. One cause of interruption of the sympathetic fibers is a tumor of the apex (top portion) of the lung, called a *Pancoast tumor.* Because the apex of the lung is in close proximity to the spine, a Pancoast tumor may compress the upper thoracic spinal cord where the sympathetic fibers exit from it. Compression of the adjacent spinal nerves between C8 and T2, entering the brachial plexus, also interrupts the nerve supply to the hand and triceps muscle, causing numbness and weakness in these areas. Pancoast tumors do not often cause respiratory symptoms early on in their course because they are located far from the mainstem bronchi. Because these tumors have this unique location, the neurological abnormalities often predate the respiratory problems. The neurologist suspected that Herb may have a Pancoast tumor in the lung because of his long history of smoking.

**305–309. The answers are 305-b, 306-c, 307-e, 308-c, 309-e.** *(Afifi, pp 168–169, 194–196, 209–222, 227–232.)* The third cranial nerve (oculomotor) controls four of the six extraocular muscles that move the eye. When this nerve fails to function, the eye remains deviated laterally due to the unopposed action of the other two extraocular muscles. When the eyes

no longer move together, patients have double vision because the visual cortex now receives two different images. In addition, fibers originating in the third nerve nucleus innervate the levator palpebrae superioris, a muscle that helps to lift the eyelid. Damage to the optic nerve causes loss of vision, blurred vision, and a central scotoma (blind spot in the center of the visual field). Damage to the cervical sympathetic fibers causes Horner's syndrome, consisting of ptosis (drooping of the eyelid), miosis (constriction of the pupil), and anhydrosis (loss of sweating), not eye movement abnormalities. The actions of the superior oblique, the muscle innervated by the trochlear nerve, include intorsion, depression, and abduction. The abducens nerve mediates the lateral rectus muscle, which abducts the eye. The eye is depressed and abducted due to the unopposed actions of the superior oblique and lateral rectus muscles, which together move the eye downward and abduct it (see earlier discussion for the actions of these muscles). The other four muscles are innervated by the oculomotor nerve, which presumably has been damaged. This is an example of Weber's syndrome, or a lesion involving the third cranial nerve outflow tract, and the corticospinal and corticobulbar tracts in the cerebral peduncles of the midbrain. Weber's syndrome may occur as a result of an occlusion of the interpeduncular branches of the posterior cerebral artery (which supply this portion of the midbrain), a tumor pressing on this area, an aneurysm (circumscribed dilation of an artery) of the posterior communicating artery, or a plaque (lesion) related to multiple sclerosis. Fibers from the Edinger-Westphal nucleus are affected by lesions of the midbrain as well, and because they are instrumental in constricting the pupil, this lesion causes the patient to have a dilated pupil. If there is a mass that is external to the midbrain, but pressing on the oculomotor nerve, then the preganglionic parasympathetic fibers traveling to the ciliary ganglion, which, in turn, innervate the pupillary constrictor muscles, can be damaged, also causing a dilated pupil. Cervical sympathetic fibers cause pupillary dilatation, so damage to these fibers causes pupillary constriction (see Horner's syndrome, earlier). Involvement of the cerebral peduncle causes damage to the corticospinal and corticobulbar tracts, resulting in weakness of the contralateral face, arm, and leg. The motor deficit is contralateral because the corticospinal tracts cross in the medullary pyramids, below the level of the lesion. The upper portion of Mike's face was spared in this case (as well as in any other UMN lesion) because the face is innervated bilaterally until the level of the caudal pons, so a unilateral lesion results in sparing of this por-

tion of the face. The combination of a third-nerve palsy and contralateral hemiparesis can only occur in the midbrain. The observed effects relating to cranial nerve III could not be accounted for by cortical damage. Likewise, damage to the cervical cord would not affect the third nerve.

**310–314. The answers are 310-c, 311-d, 312-b, 313-d, 314-e.** *(Afifi, pp 194–203; Adams, p 270.)* Damage to the trochlear nerve causes weakness of the superior oblique muscle, resulting in the inability of the orbit to deviate downward when the eye is intorted. To compensate for the classically vertical double vision, the patient tends to tilt his head to the contralateral side, causing the contralateral eye to intort. The trochlear nerve supplies the superior oblique muscle. The trochlear nerve is the only nerve to decussate peripherally, and also to emerge from the dorsal aspect of the brainstem. In this case, the damaged nerve emerged from the right (contralateral) dorsal midbrain. The action of the superior oblique muscle is to rotate the orbit medially and downward. Because the trochlear nerve is not only the smallest cranial nerve but also has the longest course of any cranial nerve, it is especially vulnerable to trauma. One of the most common causes of trochlear nerve palsy is trauma.

# Sensory Systems

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**315.** The output of the retina is mediated by

a. Bipolar cells
b. Horizontal cells
c. Rods
d. Cones
e. Ganglion cells

**316.** A direct interneuron linking the receptor and ganglion cells is the

a. Bipolar cell
b. Horizontal cell
c. Golgi cell
d. Amacrine cell
e. Optic nerve cell

**317.** Cones differ from rods in that cones have

a. A higher sensitivity to light than rods
b. More photopigment than rods
c. Pigments that are sensitive to different parts of the light spectrum
d. A lower temporal resolution with a long integration time and slow response relative to rods
e. Lower acuity and are present in fewer numbers in the fovea than rods

**318.** An individual is diagnosed with retinitis pigmentosa, which produces a defective opsin. This defect will most likely result in

a. Degeneration of area 17 of the cerebral cortex
b. Degeneration of cone cells
c. Loss of central vision
d. Total loss of vision
e. Reduced response to light

**319.** Action potentials can be produced from

a. Amacrine cells
b. Rods
c. Cones
d. Ganglion cells
e. Horizontal cells

### Questions 320–321

An individual is diagnosed as being nearsighted by an optometrist.

**320.** To correct this person's defect, the doctor would recommend that he receive eyeglasses containing lenses that are

a. Cylindrical
b. Concave
c. Convex
d. Neutral
e. Rectangular

**321.** In this case, the corrective lens is applied because

a. Of retinal damage
b. The eyeball is too long
c. The eyeball is too short
d. The eyeball is oblong
e. The lens resists change

### Questions 322–323

Another individual is diagnosed as being farsighted by the same optometrist.

**322.** To correct this person's defect, the doctor would recommend that she receive eyeglasses containing lenses that are

a. Cylindrical
b. Concave
c. Convex
d. Neutral
e. Spherical

**323.** In this case, the corrective lens is applied because

a. Of corneal damage
b. The eyeball is too long
c. The eyeball is too short
d. The eyeball is oblong
e. The lens resists change

**324.** A person is told that he has astigmatism. To correct this defect, the optometrist prescribes a

a. Cylindrical lens because the cornea or lens is oblong
b. Concave lens because the eyeball is too long
c. Convex lens because the lens is too short
d. Neutral lens because the eyeball is normal but the cornea is too thin
e. Concave lens because the cornea is opaque

**325.** A patient complains of having constant headaches involving the frontal region. Further examination reveals increased intraocular pressure. The pupil is dilated but, at the time of examination, there is little evidence of visual deficits. The likely diagnosis given to this person is that she has

a. Cataracts
b. A tumor of the visual cortex or lateral geniculate nucleus
c. A tumor at the base of the brain impinging upon the optic chiasm
d. Glaucoma
e. Color blindness

**326.** A routine eye examination reveals the presence of inflammation limited to the left optic disk, probably due to neuritis of this region. The likely visual deficit resulting from this disorder is

a. Total blindness of the left eye
b. Left homonymous hemianopsia
c. Left heteronymous hemianopsia
d. Left enlargement of the blind spot
e. Left upper quadrantanopia

**327.** As a result of calcification of the internal carotid artery, which impinges upon the lateral half of the right optic nerve prior to its entrance to the brain of a 68-year-old woman, resulting in certain visual deficits. The most likely visual deficits will be

a. Total blindness of the right eye
b. Right nasal hemianopsia
c. Right homonymous hemianopsia
d. Right bitemporal hemianopsia
e. Right upper homonymous quadrantanopia

**328.** It is discovered that a 29-year-old male has a tumor pressing on the base of the brain where it is impinging upon the optic chiasm. He discovers that his field of vision is now seriously affected. The defect present in this individual is

a. Total blindness of both eyes
b. Bitemporal hemianopsia
c. Right homonymous hemianopsia
d. Binasal hemianopsia
e. Right lower homonymous quadrantanopia

**329.** A routine magnetic resonance imaging (MRI) reveals the presence of a tumor situated in the left optic tract proximal to the lateral geniculate nucleus. The patient complained of having a reduction in his field of vision. The likely visual deficit can be characterized as

a. Total blindness of the left eye
b. Bitemporal hemianopsia
c. Right homonymous hemianopsia
d. Left homonymous hemianopsia
e. Left homonymous quadrantanopia

**330.** A 70-year-old male is admitted to the emergency room and a subsequent MRI reveals the presence of a tumor involving parts of the left temporal lobe. In addition to certain short-term memory deficits, visual deficits are noted as well. The most likely deficits will include

a. Left homonymous hemianopsia
b. Right homonymous hemianopsia
c. Left upper quadrantanopia
d. Right upper quadrantanopia
e. Left lower quadrantanopia

**331.** A 55-year-old woman complains of headaches and is subsequently diagnosed as having a tumor localized to the left parietal lobe. In addition to a variety of sensory deficits, further examination also reveals a reduction in her visual fields. The most likely visual deficit would include

a. Left homonymous hemianopsia
b. Right homonymous hemianopsia
c. Left upper quadrantanopia
d. Right upper quadrantanopia
e. Right lower quadrantanopia

**332.** The conscious perception of movement is mediated by which of the following receptors?

a. Meissner's corpuscles
b. Free nerve endings
c. Merkel's receptors
d. Joint capsules
e. Pacinian corpuscles

**333.** Which of the following types of inhibition have been identified within the dorsal column nuclei?

a. Feed-forward inhibition utilizing local interneurons only
b. Feedback inhibition utilizing local interneurons only
c. Distal inhibition from fibers arising in the cerebral cortex only
d. Feed-forward, feedback, and distal inhibition
e. Feed-forward and distal inhibition only

**334.** Neurons capable of responding to the direction or orientation of a given stimulus moved along a receptive field are located in the

a. Spinal cord
b. Medulla
c. Pons
d. Thalamus
e. Cerebral cortex

**335.** Differentiate rapidly adapting and slowly adapting receptors

a. A rapidly adapting receptor responds continuously to the presence of a stimulus, while a slowly adapting receptor responds only at the onset of the stimulus
b. A rapidly adapting receptor responds only at the onset of the stimulus and to any step change in the stimulus position, while the slowly adapting receptor displays a persistent response to the presence of the stimulus
c. A rapidly adapting receptor will not respond to any subsequent stimulus following the initial stimulus, while the slowly adapting receptor responds quite readily to a second stimulus
d. A rapidly adapting receptor will discharge at a high frequency to the initial stimulus and then continue to discharge but at a somewhat lower rate, while a slowly adapting receptor will discharge at a high frequency throughout the period of the duration of the stimulus
e. Rapidly adapting receptors are limited to muscle spindles, while slowly adapting receptors include those associated with pain and temperature pathways

**336.** Referred pain is the result of

a. Inhibitory fibers that block transmission of pain impulses along a given pathway and then transfer the impulses to a different pathway associated with a different part of the body
b. A massive discharge along a given pathway that results in the activation of a separate pathway because of the principle of divergence
c. A convergence of primary afferent fibers from a given region onto second-order neurons that normally receive primary afferents from a different body part
d. The disruption of lateral spinothalamic fibers
e. The blockade of substance P from primary afferent terminals

**337.** The terminals of different classes of primary nociceptive afferents have been shown to release which of the following transmitters onto dorsal horn neurons of the spinal cord?

a. Enkephalins alone
b. Glutamate alone
c. Substance P alone
d. Glutamate and substance P
e. Enkephalins, substance P, and glutamate

**338.** Stimulation of gray matter around the cerebral aqueduct and fourth ventricle can produce analgesia. This phenomenon is explained in terms of

a. Activation of a pathway that ascends directly to the cortex and mediates analgesia
b. A descending pathway that blocks nociceptive inputs at the level of the dorsal horn
c. Activation of local interneurons that block ascending nociceptive signals at the level of the midbrain
d. Activation of an ascending inhibitory pathway that projects to the ventral posterolateral nucleus of the thalamus
e. Activation of cholinergic neurons in the basal forebrain

**339.** A cell that responds with an *on-center* and *off-surround* to generate contrast within the receptive field can be identified in

a. Retina (ganglion cell)
b. Lateral geniculate nucleus
c. Retina (ganglion cell) and lateral geniculate nucleus
d. Layer IV of the primary visual cortex (area 17)
e. Retina (ganglion cell), lateral geniculate nucleus, and area 18

**340.** The descending pathway for central control of nociception includes

a. Fibers from the periaqueductal gray that synapse directly on dorsal horn cells
b. Fibers from the periaqueductal gray that synapse on neurons of the nucleus raphe magnus that then synapse on dorsal horn cells
c. Fibers from the periaqueductal gray that synapse upon inferior olivary neurons that then synapse upon dorsal horn cells
d. Hypothalamic fibers that synapse upon neurons of the nucleus solitarius that then synapse upon neurons of the dorsal horn
e. Hypothalamic fibers that synapse directly upon dorsal horn neurons

**341.** Fibers in each optic tract synapse in

a. The lateral geniculate nucleus only
b. The lateral geniculate nucleus and the pretectal area
c. The lateral geniculate nucleus, the pretectal area, and the superior colliculus
d. The lateral geniculate nucleus, the pretectal area, the superior colliculus, and the suprachiasmatic nucleus
e. The lateral geniculate nucleus, the pretectal area, the superior colliculus, the suprachiasmatic nucleus, and the nuclei of cranial nerves III and IV

**342.** At the level of the dorsal horn of the spinal cord, nociceptive transmission may be blocked when descending fibers are

a. Opioidergic and only contact dendrites of postsynaptic neurons that contain opiate receptors
b. Opioidergic and only contact opiate receptors located presynaptically on nociceptive terminals
c. Opioidergic and contact both dendrites of postsynaptic neurons and presynaptic terminals, both of which contain opiate receptors
d. Serotonergic and only contact dendrites of postsynaptic neurons that contain 5-HT receptors
e. Cholinergic and contact both dendrites of postsynaptic neurons and presynaptic terminals, both of which contain muscarinic receptors

**343.** In the olfactory glomerulus, primary afferent fibers terminate principally upon

a. Granule cell dendrites forming axodendritic synapses
b. Granule cell axon terminals forming axoaxonic synapses
c. Mitral cell dendrites forming axodendritic synapses
d. Mitral cell axon terminals forming axoaxonic synapses
e. Axon terminals of fibers arising from the olfactory tubercle, forming axoaxonic synapses

**344.** When a cone is hyperpolarized by light

a. The on-center bipolar cell is excited and the off-center bipolar cell is inhibited
b. The on-center bipolar cell will inhibit the ganglion cell with which it makes synaptic contact
c. The ganglion cell that receives its input from an off-center bipolar cell will discharge because the bipolar cell is excited during the presence of the stimulus
d. An on-center bipolar cell excites a neighboring ganglion cell that receives its input from an off-center bipolar cell
e. A transmitter released from a cone cell has the same effect upon all processes with which it synapses

**345.** The region of the cortex most closely associated with the conscious perception of smell is the

a. Temporal neocortex
b. Posterior parietal lobule
c. Cingulate gyrus
d. Prefrontal cortex
e. Precentral gyrus

**346.** Lateral inhibition within the retina is most effectively achieved through the action of

a. Rod cells
b. Cone cells
c. Bipolar cells
d. Ganglion cells
e. Horizontal cells

**347.** Cells that respond to an image in a specific position and have discrete excitatory and inhibitory zones and a specific axis of orientation in which a response occurs are classified as

a. M cells of the lateral geniculate nucleus
b. P cells of the lateral geniculate nucleus
c. Simple cells of the visual cortex
d. Complex cells of the visual cortex
e. Hypercomplex cells of the visual cortex

**348.** The part of the olfactory receptor mechanism that initially responds to an olfactory stimulus is the

a. Mitral cell
b. Granule cell
c. Sustentacular cell
d. Basal cell
e. Olfactory cilia

**349.** The neural basis of olfactory discrimination is believed to utilize

a. Specific activation of different cell groups within the amygdala
b. Specific activation of different groups of olfactory glomeruli that are spatially organized and segregated within the olfactory bulb
c. Specific activation of different groups of cells within the olfactory tubercle
d. Temporal summation of olfactory signals in the anterior olfactory nucleus
e. Temporal summation of olfactory signals in the mediodorsal thalamic nucleus

**350.** The principal efferent pathway of the olfactory bulb arises from

a. Granule cells
b. Golgi cells
c. Receptor cells
d. Mitral cells
e. Periglomerular cells

**351.** Direct efferent projections of the olfactory bulb supply the

a. Hypothalamus and prefrontal cortex
b. Amygdala and pyriform cortex
c. Hippocampus and amygdala
d. Prefrontal cortex and medial thalamus
e. Septal area and prefrontal cortex

**352.** Which of the following statements concerning uncinate fits is correct?

a. They are characterized by olfactory hallucinations that occur as a result of an irritating lesion of the uncus, adjoining region of the parahippocampal gyrus, or region adjoining the amygdala
b. They are characterized by olfactory hallucinations that occur as a result of an irritating lesion of the mediodorsal thalamic nucleus
c. They are an epileptic disorder whose focus is the prefrontal cortex, and they result in a failure to discriminate odors
d. They are a partial complex seizure disorder of the amygdala and hippocampus that results in the expression of violent, uncontrolled behavior
e. They are an epileptogenic disturbance caused by the formation of a tumor that is limited to the olfactory bulb, and they result in a failure to discriminate odors

**353.** Which of the following statements about the taste system is correct?

a. Receptors for specific taste stimuli are positioned on specific regions of the tongue
b. A given taste bud will only respond to a single taste modality
c. All taste afferent fibers are contained within the facial nerve
d. The cellular mechanism for transduction is essentially the same for each of the categories of taste stimuli
e. Single primary taste fibers in the chorda tympani respond to more than one taste stimulus

**354.** Which of the following sensory systems is able to utilize a circuit that bypasses the thalamus for the transmission of sensory information from the periphery to the cerebral cortex?

a. Conscious proprioception
b. Taste
c. Olfaction
d. Vision
e. Audition

# Sensory Systems

## Answers

**315. The answer is e.** *(Kandel, pp 516–520.)* The output of the retina is mediated by the ganglion cells. Ganglion cells receive inputs from photoreceptor and bipolar cells. In turn, ganglion cells give rise to optic nerve fibers, which project through the optic chiasm and optic tracts to the lateral geniculate nucleus of the thalamus. Other cells mentioned in this question only produce local connections within the retina.

**316. The answer is a.** *(Kandel, pp 514–520.)* The bipolar cell receives inputs from the receptor cells (i.e., rods and cones). The response of the bipolar cell to the receptor cell input is then mediated to the ganglion cell. (See the discussion that follows for further consideration of the physiology of the retina.) Horizontal and amacrine cells connect neighboring receptor or bipolar cells; Golgi cells are not present in the retina; and optic nerve cells project out of the retina as indicated earlier.

**317. The answer is c.** *(Kandel, pp 508–515.)* Cones differ from rods in that cone cells contain pigments that are sensitive to different parts of the light spectrum, while rod cells are achromatic. The other choices are incorrect: rods have a greater sensitivity to light than rods and have more photopigment than cones. Cones also have a higher temporal resolution with a shorter integration time and more rapid response than cones. Cones also have greater acuity and are present in greater quantities in the fovea than rods.

**318. The answer is e.** *(Adams, pp 246–247.)* In one form of retinitis pigmentosa, there is a genetic defect with respect to rhodopsin. The result of this defect is the production of defective opsin. As a consequence, rod cells are affected, leading to a reduced response to light. However, central vision is spared as are cone cells. Central nervous system (CNS) neurons such as those located in area 17 are not directly affected and vision is not totally lost.

**319. The answer is d.** *(Kandel, pp 512–521.)* The only cell in the retina that is capable of producing an action potential is the ganglion cell. As indicated earlier, the ganglion cell gives rise to optic nerve fibers, which termi-

nate as optic tract fibers in the lateral geniculate nucleus. As a result of action potentials generated in the ganglion cells, volleys of impulses are transmitted over these fibers, resulting in the appropriate responses in the neurons of the lateral geniculate nucleus.

**320–321. The answers are 320-b, 321-b.** *(Kingsley, pp 436–439; Purves, pp 225–227.)* To correct for myopia, a person is prescribed a concave (or flat) lens, because objects focus in front of the retina. The concave lens helps to refocus the object onto the retina. The reason that the focus of the object is in front of the retina is because the eyeball is too long.

**322–323. The answers are 322-c, 323-c.** *(Kingsley, pp 436–439; Purves, pp 225–227.)* To correct for farsightedness, a person is prescribed a convex lens, because objects focus in front of the retina. The convex lens helps to refocus the object onto the retina. The reason that the focus of the object is in front of the retina is because the eyeball is too short.

**324. The answer is a.** *(Kingsley, pp 436–439; Purves, pp 225–227.)* In astigmatism, the shapes of the cornea and possibly the lens become oblong, resulting in differences in the curvature of the lens along the long and short axes. Thus, astigmatism is corrected with a cylindrical lens.

**325. The answer is d.** *(Gilroy, pp 142–143.)* Glaucoma is a condition of elevated intraocular pressure caused (perhaps by infection) when debris accumulates in the spaces that lead to Schlemm's canal. If not treated, it can rapidly lead to blindness because the pressure can block conduction along the optic nerve. In addition, glaucoma can also be associated with frontal headaches; the diagnosis can be identified by determining the intraocular pressure.

**326. The answer is d.** *(Simon, pp 138–145.)* A neuritis involving the optic disk would affect the size of the visual field loss around the optic disk, which corresponds to the blind spot. In general, this kind of neuritis would expand somewhat the size of the blind spot but would cause no further visual loss.

**327. The answer is b.** *(Simon, pp 138–145.)* Calcification of the internal carotid artery could serve to disrupt nerve fibers proximal to it. One such

group of fibers includes parts of the optic nerve. In this case, the component of the right optic nerve affected includes the lateral aspect, or those fibers that mediate vision associated with the nasal visual field of the right eye. If the damage were more extensive and if it involved the entire nerve, then total blindness of the right eye would have occurred.

**328. The answer is b.** *(Simon, pp 138–145.)* A tumor pressing on the optic chiasm will disrupt the optic nerve (and tract) fibers that cross to the opposite side. These fibers mediate vision associated with fibers arising from the nasal retina of each eye. Since the nasal retina of each eye is associated with the temporal visual field for each eye, the visual loss is referred to as a *bitemporal hemianopsia.*

**329. The answer is c.** *(Simon, pp 138–145.)* Disruption of optic tract fibers destined for the lateral geniculate nucleus will cause a homonymous hemianopsia because it affects fibers arising from the temporal retina of the ipsilateral side and from the nasal retina of the contralateral side. Since the damage occurred in the left optic tract, the loss of vision is reflected on the right visual field [i.e., the left temporal retina is associated with the nasal (or right) visual field of the left eye, and the right nasal retina is associated with the temporal (or right) visual field of the right eye]. Therefore, such a lesion would result in a right homonymous hemianopsia.

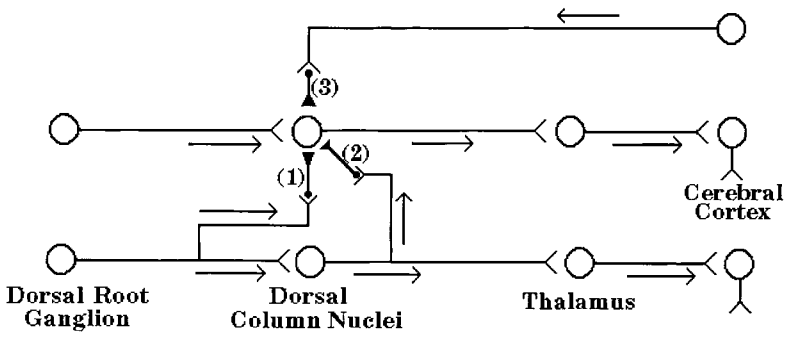
**330. The answer is d.** *(Simon, pp 138–145.)* From the lateral geniculate nucleus, there are two trajectories that the fiber pathways take en route to the visual cortex. One pathway passes dorsally through the parietal lobe and terminates in the upper bank of the calcarine fissure in the ipsilateral primary visual cortex. The second pathway takes a more circuitous (ventral) route—called the *Meyer-Archambault loop*—through the temporal lobe and terminates in the lower bank of the calcarine fissure in the ipsilateral primary visual cortex. The lower bank of the calcarine fissure is associated with the upper visual quadrants of the contralateral visual fields for both eyes, while the upper bank of the calcarine fissure is associated with the lower quadrants of the contralateral visual fields for both eyes. Thus, if there is a lesion of the left temporal lobe affecting the Meyer-Archambault loop, then the right upper quadrant for each eye will be affected. This deficit is referred to as a *right upper quadrantanopia.*

**331. The answer is e.** *(Simon, pp 138–145.)* The reasoning underlying the answer to this question is exactly as presented previously in the explanation of the answer to question #330. In brief, fibers from the left lateral geniculate destined for the upper bank of the calcarine fissure will mediate visual impulses associated with lower quadrants of the right visual fields for both eyes. This deficit is referred to as a *right lower quadrantanopia.*

**332. The answer is d.** *(Nolte, pp 192–212.)* Meissner's corpuscles, Merkel's receptors, and pacinian corpuscles respond to tactile, pressure, or possibly vibratory stimuli, while free nerve endings are associated with nociceptive stimuli. Joint capsules respond to movement of the limb, and the axons of these receptors contribute to the dorsal column–medial lemniscal system mediating the conscious perception of movement.

**333. The answer is d.** *(Kandel, pp 433–440, 451–457.)* To generate an excitatory focus with an inhibitory surround, three types of inhibition are present in the dorsal column nuclei. First-order neurons ascending in the dorsal columns make synaptic contact with different cells in the dorsal column nuclei and excite those cells. One such cell may be an inhibitory interneuron that makes synaptic contact with a neighboring dorsal column nuclear cell, thus inhibiting that cell (i.e., feed-forward inhibition). In addition, the dorsal column cell that is excited by the first-order neuron may make synaptic contact with another inhibitory interneuron (in addition to its classical ascending projection to the ventral posterolateral nucleus of the thalamus). This inhibitory interneuron makes synaptic contact with an adjacent dorsal column cell and inhibits that cell (i.e., feedback inhibition). Finally, a descending fiber from the postcentral gyrus can make synaptic contact with inhibitory interneurons that inhibit dorsal column cells. The figure below illustrates feedback, feed forward, and descending inhibition. Inhibitory neurons are depicted in black.

**334. The answer is e.** *(Kandel, pp 456–468.)* As a general rule, neurons that are situated in the cortex in association with any of the sensory systems take on a much higher level of complexity than neurons that are situated at lower levels of the relay network. In the case of the somatosensory system, direction-sensitive cells in the somatosensory cortex will respond to one direction of movement of a stimulus along the receptive field and not to another direction. Orientation-sensitive neurons respond best to move-




**(1) Feed-Forward Inhibition**
**(2) Feedback Inhibition**
**(3) Descending Inhibition**

ment along one axis of the receptive field. This is not true of neurons that are situated in lower levels of the somatosensory pathway.

**335. The answer is b.** *(Kandel, pp 430–444.)* A rapidly adapting receptor is one that discharges initially to the presence of a stimulus and to any stepwise change in the position or intensity of that stimulus (such as when the stimulus is terminated). A slowly adapting receptor responds continuously (perhaps with a decrease in its frequency) to the presence of the stimulus. A pacinian corpuscle is an example of a rapidly adapting receptor; axons from this receptor contribute to the dorsal column–medial lemniscal system.

**336. The answer is c.** *(Kandel, pp 472–485.)* Referred pain is a phenomenon in which pain impulses, arising from primary afferent fibers from one part of the body (such as from deep visceral structures), terminate on dorsal horn projection neurons that normally receive cutaneous afferents from a different part of the body (such as the arm). In this situation, a person who is suffering a heart attack experiences pain that appears to be coming from the arm. It is the convergence of these distinctly different inputs onto the same projection neurons that provides the basis for this phenomenon. None of the other possible mechanisms listed in this question have an anatomic or physiologic basis.

**337. The answer is d.** *(Kandel, pp 472–485.)* Primary nociceptive afferent fibers would have to release an excitatory transmitter in order for normal transmission to take place. Two excitatory transmitters have been identified in association with different classes of primary nociceptive afferents: (1) substance P and (2) excitatory amino acids. The best candidate as an excitatory amino acid is glutamate. Since enkephalins have been shown to be inhibitory transmitters in the pain system, they are not likely to be released from the primary afferents. Instead, other CNS neurons impinge upon the primary afferents, and enkephalins are released from those neurons.

**338. The answer is b.** *(Kandel, pp 472–485.)* Perhaps one of the most important discoveries in pain research made over the past 15 years is of a descending pathway that originates in the midbrain periaqueductal gray and makes synaptic contacts in the medulla. From the medulla, this pathway descends to the dorsal horn, where these fibers provide the anatomic substrate for suppression of pain inputs that enter the spinal cord from the periphery. There are no known inputs to the cortex that directly produce analgesia. The mechanism governing analgesia appears to operate at lower brainstem and spinal cord levels. The ascending fibers for transmission of pain impulses reach thalamic nuclei directly and, thus, local interneurons within the midbrain would not be able to interfere with such transmission. The pathway to the ventral posterolateral nucleus of the thalamus is an excitatory one and is not known to have any inhibitory properties. Cholinergic neurons in the basal forebrain have been implicated in memory functions and are not known to have any role in the regulation of pain sensation.

**339. The answer is c.** *(Kandel, pp 512–533.)* Both retina ganglion cells and lateral geniculate neurons exhibit an on-center and off-surround with respect to objects in the receptive field. Cells in area 18 of the visual cortex are not known to possess these characteristics. Cells in layer IV of the primary visual cortex do not have circular receptive fields. Instead, these cells respond to such stimuli as lines and bars.

**340. The answer is b.** *(Kandel, pp 485–487.)* The descending pathway for central inhibition of nociception involves the following: Fibers that originate in the midbrain periaqueductal gray matter project caudally to

the level of the nucleus raphe magnus, upon whose neurons they synapse. Fibers from the nucleus raphe magnus then project further caudally, where they synapse in the dorsal horn of the spinal cord.

**341. The answer is d.** *(Afifi, pp 474–476, 216–218, 404–406.)* Fibers of the optic tract synapse in a number of regions associated with the processing of visual information or visual reflex activity. These include the lateral geniculate nuclei (part of the classical visual pathway for relaying visual information to the visual cortex), the pretectal area (for elicitation of the pupillary light reflex and reflex movements of the eyes), the superior colliculus (for bilateral control of rapid eye movements), and the suprachiasmatic nucleus (which relates to the control of circadian rhythms). There are no known monosynaptic projections from the retina to the nuclei of cranial nerves III and IV.

**342. The answer is c.** *(Kandel, pp 485–487.)* Evidence indicates that within the dorsal horn of the spinal cord, descending pain inhibitory fibers from the lower brainstem (serotonergic and noradrenergic fibers) synapse upon interneurons that are enkephalinergic. These enkephalinergic neurons then synapse upon both presynaptic terminals of primary pain afferent fibers and the dendrites of dorsal horn projection neurons (which also receive inputs from the primary nociceptive afferent fibers).

**343. The answer is c.** *(Kandel, pp 629–632; Nolte, pp 316–323.)* The olfactory receptor and its primary afferent fiber terminate upon dendrites of mitral cells. This relationship is of importance because it is the axon of the mitral cell that projects out of the olfactory bulb (forming the major component of the lateral olfactory stria). The granule cell processes make synaptic contact with dendrites of mitral cells, forming dendrodendritic synapses, but are not known to make synaptic contact with primary afferent terminals. Cells arising in the olfactory tubercle are not known to project to the olfactory bulb. Instead, projections of cells situated in the olfactory tubercle contribute fibers to the medial forebrain bundle and stria medullaris.

**344. The answer is a.** *(Kandel, pp 510–521.)* When a cone is hyperpolarized by light, there is a reduction in the release of transmitter substance (glutamate). This reduced amount of transmitter results in excitation of the

on-center bipolar cell and inhibition of the off-center bipolar cell (presumably because the two types of bipolar cell contain different postsynaptic receptors). Since bipolar cells excite the ganglion cells, an off-center bipolar cell will be inhibited when light is present and, thus, will be unable to excite the ganglion cell to which it is connected. On-center bipolar cells are excited when light is present and so are the ganglion cells to which they are connected. In addition, on-center bipolar cells inhibit ganglion cells that receive their primary input from off-center bipolar cells. This serves to increase the likelihood that these ganglion cells will remain inhibited when the light stimulus is present. A cone cell may make synaptic contact with two types of bipolar cells (on-center or off-center). Because they possess different postsynaptic receptor mechanisms, the two types of bipolar cells will respond differently to input from cones.

**345. The answer is d.** *(Kandel, pp 63–635; Afifi, pp 461–462.)* Experimental evidence indicates the prefrontal cortex is a key region for the conscious perception of smell. This conclusion is based upon two observations. First, the prefrontal cortex receives major inputs from the olfactory bulb by following routes: olfactory bulb to pyriform cortex to prefrontal cortex; or olfactory bulb to pyriform cortex (and olfactory tubercle) to mediodorsal thalamic nucleus to prefrontal cortex. Second, lesions of the prefrontal cortex result in a failure to discriminate odors. Olfactory functions are not known to be associated with any of the other choices. Instead, the primary auditory receiving area is located in the auditory cortex; the posterior parietal lobule is concerned with such processes as the programming mechanisms associated with complex motor tasks; the cingulate gyrus has been associated with such functions as spatial learning and the modulation of autonomic and emotional processes; and the prefrontal gyrus contains the primary motor area.

**346. The answer is e.** *(Kandel, pp 510–521.)* Lateral inhibition within the retina is generated most effectively by the horizontal cells. A horizontal cell receives inputs from a given receptor cell and, when activated, inhibits adjacent receptor cells. It is possible for a given cone cell to differentially affect two neighboring bipolar cells and for an on-center bipolar cell to hyperpolarize an adjacent off-center ganglion cell. However, the primary flow of information through these neuronal elements is in the plane of orientation that most directly connects the receptor cell to the ganglion cell

through a bipolar cell. Therefore, the contribution of these elements to lateral inhibition is relatively minimal (if at all) in comparison to the effects generated by horizontal cells. The ganglion cell is not known to play any role in lateral inhibition.

**347. The answer is c.** *(Kandel, pp 523–543.)* Cells in the lateral geniculate nucleus respond very much like ganglion cells in the retina because of the point-to-point projection pathway from the retina to the lateral geniculate. Accordingly, lateral geniculate cells have small concentric receptive fields that are either on-center or off-center in which the cells respond best to small spots of light that are in the center of the receptive field. On the other hand, cells in the visual cortex display a much greater complexity in their responses to images in the visual field. Instead of responding to small spots of light, they respond to lines and borders in the different areas of the visual field. In particular, the simple cell responds as a function of the retinal position in which the line-stimulus is located as well as its orientation (e.g., whether it is in a vertical or horizontal position). As a result, when a bar of light is positioned in the appropriate part of the visual field with the appropriate orientation, the cells in area 17 will respond maximally. When either of these parameters is altered, the firing pattern of the cell will be reduced or totally inhibited. Complex cells lack clear excitatory and inhibitory zones (i.e., these neurons respond to bars of light in a given orientation but they are not position-specific). Hypercomplex cells are stimulated by bars of light of specific lengths or by specific shapes.

**348. The answer is e.** *(Kandel, pp 625–634.)* The olfactory cilia are extensions of the receptor cell, and it is this part of the cell that initially responds to an olfactory stimulus. The cilia contain protein membranes that bind with different odorants, which constitutes a necessary condition for excitation of the olfactory cell. Mitral and granule cells are situated in the olfactory bulb and, consequently, are not part of the receptor mechanism. Sustentacular cells are supporting cells and are not part of the receptor mechanism. Basal cells are the precursors for receptor cells and, thus, are also not directly part of the receptor mechanism.

**349. The answer is b.** *(Kandel, pp 626–636.)* A number of recent studies have indicated that different olfactory glomeruli respond to different kinds of olfactory stimuli. In a sense, this represents a type of organization of the

olfactory bulb that bears a functional similarity to the spatial organization that exists for other sensory systems. There is no evidence that such a spatial arrangement exists for other components of the olfactory system, nor is there any evidence that temporal summation plays any role in the process of olfactory discrimination.

**350. The answer is d.** *(Nolte, pp 316–323.)* The principal output pathways of the olfactory bulb arise from mitral cells and a related cell, called a *tufted cell.* The mitral cells project their axons out of the olfactory bulb to other regions of the forebrain associated with the transmission of olfactory information to the cerebral cortex. The major pathway subserving this is the lateral olfactory stria. Other cells that are mentioned in this question are either not present in the olfactory bulb (Golgi cells), or they have no known projections outside of the olfactory bulb. Receptor cells project only as far as the glomerulus. The granule cell has no axon. The periglomerular cell makes only local connections among neighboring glomeruli.

**351. The answer is b.** *(Kandel, p 633; Nolte, pp 316–323.)* Mitral cell axons enter the lateral olfactory stria and project caudally through this bundle to supply the medial amygdala and pyriform cortex. Olfactory projections to other nuclei, such as the hippocampal formation, prefrontal cortex, medial thalamus, and septal area, require at least one additional synaptic connection such as in the pyriform cortex, amygdala, or olfactory tubercle.

**352. The answer is a.** *(Afifi, p 489; Nolte, p 323.)* Uncinate fits are characterized by seizure activity involving portions of the anterior aspect of the temporal lobe. The structures most often implicated include the uncus, parahippocampal gyrus, the region of the amygdala and adjoining tissue, and the pyriform cortex. During the occurrence of uncinate fits, a person experiences olfactory hallucinations of a highly unpleasant nature.

**353. The answer is e.** *(Kandel, pp 636–644.)* A primary afferent fiber innervates many taste receptors. Recordings from the primary afferent fiber reveal that it responds to different modalities of taste stimuli, although it may preferentially respond to a single, given modality. This would suggest that taste discrimination and perception occur as a result of the comparison of the activation pattern of different groups of taste fibers. Different types of

taste receptors may be positioned in the same region of the tongue. Primary afferent taste fibers respond to more than one modality of taste stimuli. Taste fibers from the anterior two-thirds of the tongue are carried in the facial nerve; fibers from the posterior one-third of the tongue are carried in the glossopharyngeal nerve; and taste fibers from the epiglottis are carried in the vagus nerve. The cellular mechanism for transduction of taste stimuli depends upon the stimulus. For example, receptors for molecules associated with sweet and bitter tastes utilize second messengers, while those associated with sour- and salty-tasting molecules act directly upon the ion channels.

**354. The answer is c.** *(Nolte, pp 230–239, 295–304, 319–323, 547–560.)* The pathway for conscious proprioception from the body utilizes the ventral posterolateral nucleus as its thalamic relay. Conscious proprioception from the head utilizes the ventral posteromedial nucleus as its relay. The taste pathway utilizes the ventral posteromedial nucleus as well. The visual system utilizes the lateral geniculate nucleus, and the auditory system utilizes the medial geniculate nucleus. In contrast, the olfactory system can transmit olfactory information to the prefrontal cortex without engaging thalamic nuclei. Thus, olfactory information reaches the pyriform cortex and amygdala from the olfactory bulb and then is transmitted directly to the prefrontal cortex. However, it should be noted that olfactory information also can reach the prefrontal cortex by virtue of projections from the olfactory tubercle and pyriform cortex via the mediodorsal thalamic nucleus. Thus, the olfactory system may utilize a parallel processing mechanism in transmitting inputs to the prefrontal cortex.

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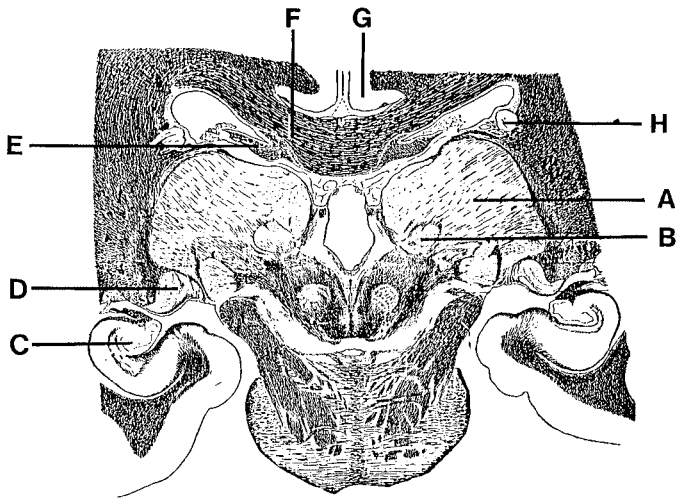
# Anatomy of the Forebrain

## *Questions*

**DIRECTIONS:** Each group of questions below consists of lettered options followed by a set of numbered items. For each numbered item, select the **one** lettered option with which it is **most** closely associated. Each lettered option may be used once, more than once, or not at all.

**Questions 355–359**




**355.** Neurons in this region project their axons to the inferior parietal lobule

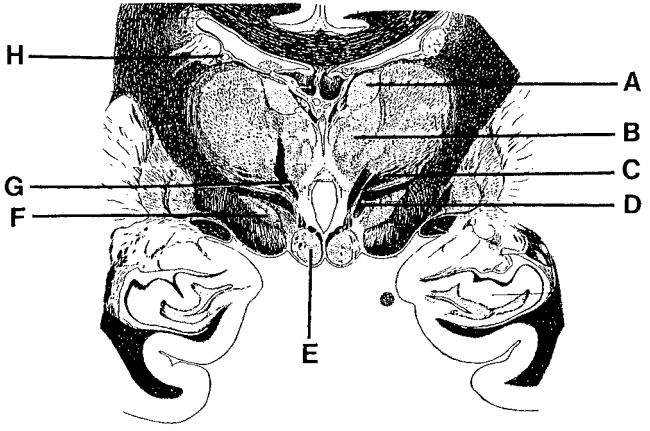
**356.** This fiber bundle arises from the hippocampal formation

**357.** A lesion of this structure produces short-term memory deficits

**358.** This is a specific relay nucleus

**359.** Neurons in this region innervate the striatum

### Questions 360–367




**360.** This structure has pallidal fibers that project to the ventrolateral (VL) and ventral anterior (VA) nuclei of the thalamus

**361.** This nucleus maintains reciprocal connections with the globus pallidus

**362.** This structure receives fibers from the mamillary bodies

**363.** These fibers project to the medial hypothalamus

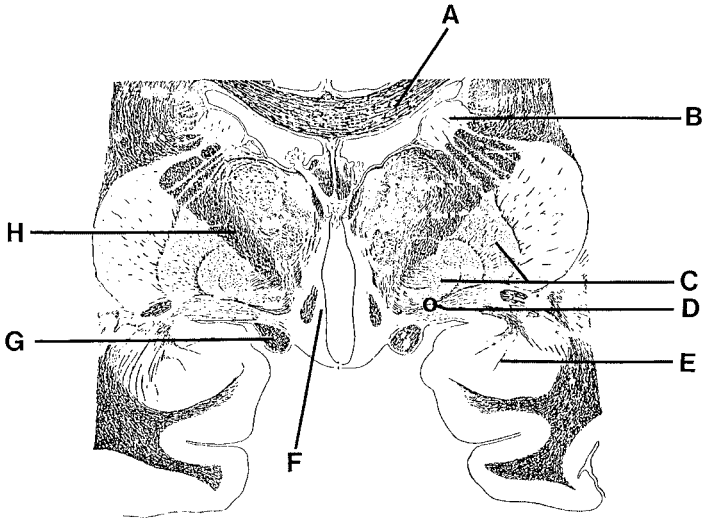
**364.** This structure contains fibers that arise from both the pallidum and cerebellum

**365.** This region receives inputs from the hippocampal formation and projects its axons to the anterior thalamic nucleus

**366.** These fibers arise from the mamillary bodies

**367.** This region has reciprocal connections with large parts of the frontal lobe

### Questions 368–374




**368.** These fibers arise from layers V and VI of the cerebral cortex

**369.** These fibers arise from layers II and III of the cerebral cortex

**370.** These fibers arise from the globus pallidus

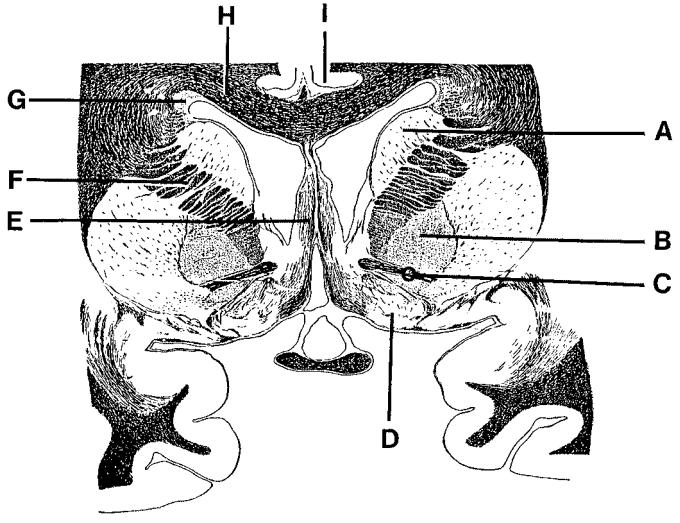
**371.** This region receives dopaminergic inputs from the substantia nigra

**372.** Cells in this region produce hormones that are released in the posterior pituitary

**373.** This structure powerfully modulates hypothalamic functions

**374.** A lesion of this structure will produce a hemianopsia

## Questions 375–379




**375.** This structure receives afferent fibers from the cerebral cortex and thalamus

**376.** This structure is the source of a major cholinergic projection to the neocortex

**377.** This structure receives inputs from the neostriatum

**378.** This structure transmits olfactory information from the anterior olfactory nucleus

**379.** This structure (which is considerably larger in nonhumans, such as the rat, cat, and monkey) is a component of the limbic system and receives a major afferent projection from the hippocampal formation

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

### Questions 380–384

Susan is a 32-year-old woman, who recently stopped taking her birth control pills in order to become pregnant. However, after several months, her menstrual period failed to resume. Prior to beginning the birth control pills several years before, she had been having normal cycles. She also noticed headaches, which had been increasing in severity over the past several months. Recently, she became aware of difficulty with her peripheral vision. Thinking that she might be pregnant, she sought the attention of her gynecologist. Her doctor ran a pregnancy test, which was negative. She told her that there may be another cause of the absence of her menstrual cycle, and she sent Susan's blood for levels of various hormones. When Susan returned to find out the results of the tests, her gynecologist told her that the level of the hormone prolactin was high. Susan remembered her headaches and visual symptoms, and informed her doctor, who promptly referred her to a neurologist. The neurologist listened to Susan's story, and examined her. She found only that Susan was unable to see fingers in the temporal fields (lateral half of each visual field) of both of her eyes. The remainder of her neurologic exam was normal. The neurologist told Susan that she would like to order a magnetic resonance imaging (MRI) test of her head, in order to find out why she had the headaches, visual problem, and high prolactin levels.

**380.** A tumor in which area could cause a high prolactin level?

a. Adenohypophysis
b. Neurohypophysis
c. Amygdala
d. Hippocampus
e. Adrenal gland

**381.** What type of neurologic visual loss can cause a loss of peripheral vision?

a. Central scotoma
b. Superior quadrantanopsia
c. Bitemporal hemianopsia
d. Homonymous hemianopsia
e. Papilledema

**382.** A lesion adjacent to which structure caused Susan's visual problem?

a. Optic nerve
b. Optic radiations
c. Retina
d. Optic chiasm
e. Lateral geniculate nucleus

**383.** Which hypothalamic nucleus regulates prolactin secretion?

a. Suprachiasmatic nucleus
b. Preoptic nucleus
c. Paraventricular nucleus
d. Supraoptic nucleus
e. Arcuate nucleus

**384.** Which neurotransmitter system regulates prolactin secretion?

a. Tuberoinfundibular dopaminergic system
b. Nigrostriatal dopaminergic system
c. Mesolimbic dopaminergic system
d. Mesocortical dopaminergic system
e. Mesostriatal dopaminergic system

### Questions 385–389

Norma is a 75-year-old woman who had a stroke several months ago, manifested by numbness on her right side, including her arm, face, trunk, and leg. The numbness had improved somewhat over time, but did not completely disappear. One day, she noticed that brushing her right arm against a door was very painful. Thinking that perhaps this was "in her mind," she tried touching the right arm with her left hand, and this, too, was painful. Fearful that she may be having another stroke, she went immediately to see her neurologist at her local hospital. Norma's neurologist examined her and

found that sensation for a pin, temperature, and vibration were diminished on the entire right side of her body. The degree of sensory loss was unchanged from an examination several months before. However, she had a large amount of discomfort with any type of stimulus, accompanied by some emotional disturbance. The discomfort was far out of proportion to the degree of the stimulus (e.g., a light touch to her right arm would engender a scream similar to that elicited by a knife). The remainder of her examination was normal. The neurologist told Norma that he didn't think that she had had a new stroke, but would order a head CT to be sure that there was no tumor or bleeding. In addition, he told her that if the head CT showed nothing new, she could begin a new medication that would help with the pain.

**385.** What is the most likely location of the old stroke?

a. Right precentral gyrus
b. Left precentral gyrus
c. Right ventral thalamus
d. Left ventral thalamus
e. Left cerebral peduncle

**386.** Which two nuclei mediating sensation of the arms, face, legs, and trunk may have sustained damage from the original stroke?

a. Lateral and medial geniculate nuclei of the thalamus
b. Ventral posterior lateral and ventral posterior medial nuclei of the thalamus
c. Putamen and globus pallidus
d. Caudate and putamen
e. Anterior and lateral dorsal nuclei of the thalamus

**387.** Which pathway mediating pain is the afferent input into the infarcted area?

a. Fasciculus gracilis
b. Fasciculus cuneatus
c. Spinocerebellar tract
d. Spinothalamic tract
e. Corticospinal tract

**388.** Surgical stimulation of various regions of the central nervous system (CNS) has been shown to alleviate pain. What is the location of one of these areas producing analgesia?

a. Anterior nucleus of the thalamus
b. Caudate nucleus
c. Anterior horn of the spinal cord
d. Globus pallidus
e. Periaqueductal gray

**389.** Neurotransmitters implicated in pain modulation, which may be the targets of pain-alleviating drugs, include

a. Aspartate
b. Glutamate
c. Epinephrine
d. Dopamine and norepinephrine
e. Opiates and serotonin

# Anatomy of the Forebrain

## *Answers*

**355–359. The answers are 355-A, 356-E, 357-C, 358-D, 359-B.** *(Nolte, pp 375–387, 417–421, 538–561.)* This section is taken at the level of the posterior thalamus and, because of the oblique cut, also includes parts of the midbrain and pons. The fornix (E), situated just below the corpus callosum, arises from the hippocampal formation and supplies the septal area, anterior thalamic nucleus, and the mamillary bodies. The hippocampal formation (C) is associated with a number of different processes, including short-term memory. Thus, a lesion of this structure will likely produce deficits in short-term memory. The lateral geniculate nucleus (D), situated in the far VL aspect of the posterior thalamus, is a relay nucleus for the transmission of visual information to the cortex. The centromedian (CM) nucleus (B), identified by its encapsulated appearance, can be found in posterior levels of the thalamus, where it projects to the neostriatum. Also located at the level of the posterior aspect of the thalamus is the pulvinar nucleus (A). This large structure projects its axons to the inferior parietal lobule.

**360–367. The answers are 360-D, 361-F, 362-A, 363-H, 364-C, 365-E, 366-G, 367-B.** *(Nolte, pp 375–394, 451–467, 538–545, 548–555.)* This section is taken at the level of the mamillary bodies (at ventral levels) and includes parts of the anterior thalamus (at dorsal levels). The lenticular fasciculus (D), situated just below the thalamic fasciculus and immediately above the subthalamic nucleus at the level of this brain section, arises from the dorsomedial aspect of the medial pallidal segment and projects to the VL, VA, and CM nuclei of the thalamus. Note that the thalamic fasciculus (C) also projects to these nuclei. While many of the fibers contained in this bundle arise from the pallidum, others arise directly from the cerebellum. The subthalamic nucleus (F), which lies on the dorsal surface of the internal capsule, maintains reciprocal connections with the globus pallidus through a pathway called the *subthalamic fasciculus*. The anterior nucleus of the thalamus (A), which lies at the rostral end of the thalamus in a dorso-

medial position, receives a major input from the mamillary bodies via the mamillothalamic tract. The region immediately below the tail and body of the caudate nucleus is occupied by a major output pathway of the medial amygdala, the stria terminalis (H). It supplies the medial preoptic region, bed nucleus of the stria terminalis, and medial hypothalamus.

The thalamic fasciculus (C) can be seen in sections taken through the caudal half of the thalamus and is clearly visualized in a position dorsal to the subthalamic nucleus and lenticular fasciculus. It contains fibers that arise from both the dentate nucleus of the cerebellum and the medial pallidal segment. The mamillary bodies (E), situated at the base of the posterior aspect of the hypothalamus, are the origin of the mamillothalamic (G) tract, which innervates the anterior thalamic nucleus. A large nuclear mass situated in the medial aspect of the posterior two-thirds of the thalamus is the mediodorsal thalamic nucleus (B). This nucleus projects extensively to wide regions of the frontal lobe, including the prefrontal cortex. In turn, the prefrontal region of the cortex and adjoining regions of the frontal lobe project their axons back to the mediodorsal nucleus. Thus, there are reciprocal connections linking the mediodorsal nucleus and rostral portions of the frontal lobe.

**368–374. The answers are 368-H, 369-A, 370-D, 371-B, 372-F, 373-E, 374-G.** *(Nolte, pp 239–240, 261, 507–515, 517–523, 538–561.)* This section is taken from rostral levels of the diencephalon. Corticobulbar and corticospinal fibers contained within the internal capsule (H) arise from the deeper layers of cerebral cortex (i.e., layers V–VI), while those of the corpus callosum (A) arise from more superficial layers of the cortex (i.e., layers II–III) and project to the homotypic region of the contralateral cortex. Fibers of the ansa lenticularis (D) arise from the ventral aspect of the medial pallidal segment and can be visualized at more anterior levels of the pallidum. Its axons supply the VL, VA, and CM nuclei of the thalamus. The caudate nucleus (B) receives dopaminergic inputs from the substantia nigra.

Different cells of the paraventricular nucleus of the hypothalamus (F), situated in the dorsomedial region at anterior levels, synthesize oxytocin and vasopressin. These hormones are transported down their axons to the posterior pituitary. Different fiber groups of the amygdala (E) provide major inputs into the medial and lateral regions of the hypothalamus and thus constitute a significant modulator of hypothalamic functions. The

optic tract (G) arises from the retina. Each optic tract represents fibers associated with the visual fields of the opposite side. Therefore, a lesion of the optic tract will result in a homonymous hemianopsia.

**375–379. The answers are 375-A, 376-D, 377-B, 378-C, 379-E.** *(Nolte, pp 277–278, 380–388, 544–561.)* This section is taken at the level of the septum pellucidum, anterior commissure, and the substantia innominata. Fibers from the region of the basal nucleus of Meynert located in the substantia innominata (D) (at the base of the brain in the far rostral forebrain) send a cholinergic projection to wide areas of the neocortex. The globus pallidus (B) receives inputs from the neostriatum (i.e., caudate nucleus and putamen). The anterior commissure (C) can be clearly seen at the level of the forebrain just rostral to the level of the preoptic area. It transmits olfactory information from the anterior olfactory nucleus on one side of the brain to the olfactory bulb and anterior olfactory nucleus of the contralateral side. The septal area (E), seen at this level of the forebrain as a thin structure separated by the lateral ventricles on both sides, receives major inputs from the hippocampal formation and is a principal component of the limbic system. The caudate nucleus (A) and the putamen constitute the principal structures for receipt of afferent fibers. Primary sources of such input include the cerebral cortex and CM nucleus of the thalamus.

**380–384. The answers are 380-a, 381-c, 382-d, 383-e, 384-a.** *(Adams, pp 676–678; Afifi, pp 404–409; Kandel, pp 544, 978–980.)* The MRI of Susan's head revealed a pituitary microadenoma, a benign tumor arising from the anterior pituitary or adenohypophysis. This particular tumor consisted of cells that secrete the hormone prolactin, which is not only the stimulating factor for lactation, but inhibits menstruation when levels are high. It is common for this tumor's symptoms to be manifested during the child-bearing years. The visual problem is called *bitemporal hemianopsia.* Since the pituitary gland is in very close proximity to the optic chiasm, pituitary tumors often invade this structure. Since only the medial fibers (which perceive the temporal field of each eye) in each optic nerve cross, these are the fibers damaged by these tumors, and the patient will be unable to see either temporal visual field. Both the central scotoma (an island of visual loss surrounded by normal vision in one eye), which is usually seen with lesions of the retina or optic nerve, and the papilledema (blurring of the optic disc margin when viewed by fundoscopic examina-

tion due to increased intracranial pressure) would not be caused by damage to the optic chiasm. The optic chiasm can be compressed by pituitary tumors, causing bitemporal hemianopsia (see the answer for the previous question). The prolactin-releasing factor is found in the arcuate nucleus of the hypothalamus and activates the lactotropic cells of the anterior pituitary gland. Several different peptides, including dopamine, have the capacity to raise the level of prolactin in the blood. Specifically, the tuberoinfundibular dopaminergic system regulates prolactin secretion through direct projection to the pituitary. For this reason, a newer treatment for prolactin-secreting microadenomas is the drug bromocriptine, a dopamine agonist commonly used in the treatment of Parkinson's disease. By giving a dopamine agonist, serum prolactin increases, inhibiting production by the tumor cells, and eventually the tumor size shrinks. This has become either an alternative or first-line treatment prior to trying radiation or surgery.

**385–389. The answers are 385-d, 386-b, 387-d, 388-e, 389-e.** *(Kandel, pp 446–450, 454–460, 473–475, 480–487, 874–875.)* Norma's head CT showed an old stroke in her left ventral thalamus and no new lesions. A stroke involving the ventral posterolateral nucleus of the thalamus, especially several months after the stroke can produce an entity called the *Déjérine-Roussy syndrome,* or *thalamic pain syndrome*. Although there is sensory loss on the contralateral side, there is pain or discomfort out of proportion to the stimulus on the affected side of the body. Emotional disturbance aggravates the response. Some patients describe the sensation as knifelike or hot. As the deficit (numbness) resolves, the pain may lessen. This syndrome may also occur in lesions of the parietal white matter, and is thought to occur as a result of an imbalance of afferent sensory impulses. Sensation of the limbs and trunk are projected through the ventral posterior lateral nucleus of the thalamus to the somatosensory cortex. Sensory information from the face is carried through the trigeminal system to the ventral posteromedial nucleus, from which it is projected to the somatosensory cortex. The spinothalamic tract is the only sensory pathway listed that mediates pain. The periaqueductal gray is one area of many that produces analgesia when stimulated in both animals and in humans. It is an area with a high density of opiate receptors and opioidergic neurons, and is thought to represent a key area in gating pain. Many neurotransmitters have been implicated as pain modulators, including the opiates and

enkephalins, norepinephrine, serotonin, substance P, GABA, and acetylcholine. Most analgesic medications are designed to target a particular aspect of the pain pathway. In more recent years, the advent of a class of drugs called *tricyclic antidepressants* has added another dimension to medical pain treatment. The methylated forms of these medications are useful blockers of serotonin reuptake. Since serotonin is known to be a pain modulator, it is thought that blocking the reuptake of serotonin enhances its action and facilitates the action of intrinsic opiates to relieve pain. This is a common class of drugs used to treat chronic pain, since these medications are not addictive.

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# Motor Systems

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

**390.** A patient delays initiation of movement, displays an uneven trajectory in moving her hand from above her head to touch her nose, and is uneven in her attempts to demonstrate rapid alternation of pronating and supernating movements of the hand and forearm. She probably has a lesion in the

a. Hemispheres of the posterior cerebellar lobe
b. Flocculonodular lobe of the cerebellum
c. Vermal region of the anterior cerebellar lobe
d. Fastigial nucleus
e. Ventral spinocerebellar tract

**391.** Spasticity may result from a lesion of

a. Ventral horn cells
b. Corpus callosum
c. Postcentral gyrus
d. Internal capsule
e. Substantia nigra

**392.** In studying the functional relationships between the motor cortex and the spinal cord, which of the following effects of cortical stimulation on synaptic potentials would an investigator be likely to observe?

a. The largest potentials would be seen in spinal motor neurons that innervate proximal muscles
b. The largest potentials would be seen in spinal motor neurons that innervate distal muscles
c. The potentials seen in spinal motor neurons that innervate proximal and distal muscles would be approximately equivalent
d. The largest potentials would be seen in spinal sensory neurons that carry information from spindle afferents to the cerebellum
e. The largest potentials would be seen in spinal sensory neurons that carry information from proprioceptors to the thalamus

**393.** Which of the following statements correctly characterizes the properties of neurons in the motor cortex?

a. In the resting state, the membranes of motor cortex neurons are more permeable to sodium than to potassium ions
b. Motor cortex neurons receive information from the muscle to which they project or from a region of skin that is related to the function of that muscle
c. Motor cortex neurons have reciprocal connections with the red nucleus
d. Motor cortex neurons that excite alpha motor neurons generally have little effect upon gamma motor neurons that project to the same muscle group
e. Motor neurons of the cerebral cortex have reciprocal, monosynaptic connections with neurons in the cerebellar cortex

**394.** Paralysis of the right side of the lower face, right spastic paralysis of the limbs, deviation of the tongue to the right with no atrophy, and no loss of taste from any region of the tongue will likely result from a lesion of the

a. Internal capsule of the right side
b. Internal capsule of the left side
c. Right pontine tegmentum
d. Base of the medulla on the right side
e. Base of the medulla on the left side

**395.** An impairment in the ability to perform certain types of learned, complex movements (referred to as *apraxia*) usually results from a lesion of the

a. Precentral gyrus
b. Postcentral gyrus
c. Premotor cortex
d. Prefrontal cortex
e. Cingulate gyrus

**396.** The overwhelming majority of fibers that supply the basal ganglia terminate in the

a. Paleostriatum
b. Neostriatum
c. Subthalamic nucleus
d. Substantia nigra
e. Claustrum

**397.** Neurons in the neostriatum are

a. Inhibited by γ-aminobutyric acid (GABA) released at corticostriate terminals
b. Inhibited by GABA released at nigrostriatal terminals
c. Inhibited by substance P released at corticostriate terminals
d. Excited by acetylcholine (ACh) released from hypothalamic-caudate terminals
e. Excited by glutamate released at corticostriate terminals

**398.** The primary transmitter released from terminals of both neostriatal and paleostriatal neurons is

a. Glycine
b. Enkephalin
c. Dopamine
d. GABA
e. Glutamate

**399.** Since motor dysfunctions associated with disturbances of basal ganglia are expressed on the contralateral side of the body, one may conclude that the basal ganglia project

a. Fibers to the spinal cord that are crossed
b. Fibers to motor nuclei of the brainstem whose axons then project to the contralateral spinal cord
c. Fibers to structures that ultimately influence motor regions of the ipsilateral cerebral cortex
d. Axons to the cerebellum, whose outputs are known to modulate the contralateral side of the body
e. Fibers directly to the contralateral motor cortex

**400.** The major afferent input to the flocculonodular lobe is from the

a. Clarke's nucleus dorsalis of the spinal cord
b. Red nucleus
c. Vestibular nuclei
d. Cerebral cortex
e. Midbrain reticular formation

**401.** In Huntington's disease, there is a loss of

a. Dopamine in the neostriatum
b. Substance P in the substantia nigra
c. ACh and GABA in intrastriatal and cortical neurons
d. Serotonin in the neostriatum
e. Most of the pallidal neurons

**402.** Damage to the subthalamic nucleus will result in

a. Torsion dystonia
b. Tremor at rest
c. Hemiballism
d. Spastic paralysis
e. Tardive dyskinesia

**403.** Which of the following drugs ameliorate choreiform movements?

a. ACh blockers because there is an excess of this transmitter in the caudate nucleus
b. Dopamine blockers because there is too low a ratio of ACh to dopamine in the neostriatum
c. Serotonin blockers because there is too low a ratio of serotonin to ACh and dopamine in the neostriatum
d. Substance P antagonists because the ratio of substance P to ACh is too high in the neostriatum
e. Norepinephrine antagonists because the ratio of norepinephrine to ACh is too high in the subthalamic nucleus

**404.** Tardive dyskinesia is most likely the result of

a. A change in serotonin receptors that causes a hypersensitivity to serotonin
b. A change in ACh receptors that causes a hypersensitivity to ACh
c. A change in enkephalin receptors that causes a hypersensitivity to enkephalin
d. A change in dopamine receptors that causes a hypersensitivity to dopamine
e. A change in GABA receptors that causes a hypersensitivity to GABA

**405.** The neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) has recently been applied experimentally with considerable success as a model for

a. Huntington's disease
b. Hemiballism
c. Parkinson's disease
d. Tardive dyskinesia
e. Dystonia

**406.** The dorsal spinocerebellar tract, the ventral spinocerebellar tract, and the cuneocerebellar tract, in a general sense, show convergence in their projections to the cerebellum. The principal region within the cerebellum where these fibers converge is the

a. Anterior lobe
b. Posterior lobe
c. Flocculonodular lobe
d. Fastigial nucleus
e. Dentate nucleus

**407.** Information arising from the cerebral cortex is known to reach the cerebellum. The fibers carrying it are

a. Somatotopically distributed only to the anterior lobe
b. Somatotopically distributed only to the vermal region of the anterior and posterior lobes
c. Somatotopically distributed to the cerebellar hemispheres
d. Not somatotopically organized but do project to the hemispheres of the anterior and posterior lobes
e. Distributed mainly to the interposed and dentate nuclei

**408.** A cerebellar glomerulus includes

a. Mossy fiber terminals, Golgi axons, and axon terminals of granule cells
b. Climbing fiber terminals, Golgi axons, and granule cell dendrites
c. Mossy fiber terminals, Purkinje cell axons, and granule cell dendrites
d. Mossy fiber terminals, Golgi and granule cell dendrites, and Golgi cell axon terminals
e. Climbing fiber terminals, Golgi cell dendrites, Purkinje cell dendrites, and axon terminals of parallel fibers

## Questions 409–411

The cerebellum contains a number of important feedback relationships with different regions of the central nervous system (CNS). In each of the following circuits, one or more of the structures has been omitted. Indicate the structure(s) that must be added to complete that circuit.

**409.** Frontal lobe → deep pontine nuclei → cerebellar cortex → _____? _____ → _____?_____ → motor cortex (frontal lobe)

a. Fastigial nucleus → red nucleus
b. Interposed nuclei → red nucleus
c. Dentate nucleus → ventrolateral (VL) nucleus of the thalamus
d. Dentate nucleus → ventral anterior (VA) nucleus of the thalamus
e. Purkinje cell axons → reticular formation of pons

**410.** Red nucleus → inferior olivary nucleus → cerebellar cortex of the anterior and posterior lobes → _____?_____ → red nucleus

a. Fastigial nucleus
b. Interposed nuclei
c. Dentate nucleus
d. Purkinje cells of the cerebellar hemispheres
e. Vestibular nuclei

**411.** Spinal cord (via dorsal and ventral spinocerebellar tracts) → anterior lobe of the cerebellum → _____?_____ → reticular formation and vestibular nuclei → spinal cord

a. Fastigial nucleus
b. Globose nucleus
c. Emboliform nucleus
d. Dentate nucleus
e. Red nucleus

**412.** Based upon your knowledge of the anatomic and neurophysiologic relationships of the anterior lobe of the cerebellum, you would predict that electrical stimulation of the medial vermal aspect of the cerebellar cortex of the anterior lobe will

a. Produce movement of the arms
b. Produce spasticity
c. Cause tonic seizures to occur
d. Modulate extensor muscle tone
e. Have little effect upon muscle tone

**413.** A man presents with a wide-based, ataxic gait during his attempts at walking. He also is unsteady and sways when standing and displays a tendency to fall backward or to either side in a drunken manner. A lesion is most likely located in the

a. Hemispheres of the posterior cerebellar lobe
b. Anterior limb of the internal capsule
c. Dentate nucleus
d. Anterior lobe of the cerebellum
e. Flocculonodular lobe of the cerebellum

## Questions 414–418

Sam is a 62-year-old man, previously healthy, who was brought to a neurologist by his daughter because of increasing difficulty walking. His daughter noticed that for the past year, he had difficulty getting out of a chair and took a lot of time to begin to walk. When he did walk, he walked with a slow, shuffling gait. In addition, she had noticed some changes in his face, and that he had been drooling excessively. His signature on checks became progressively smaller from the beginning of his name to the end, and he had developed a new tremor. She brought him in to make sure this wasn't just "aging." The neurologist examined Sam and noticed immediately that Sam's facial expression was masklike, with few eyeblinks. When asked to write a sentence, the letters became progressively smaller toward the end of the sentence. His speech was soft and monotonous, and he had a slow, resting pill-rolling tremor in both of his hands. He had very little spontaneous movement, and his arms, legs, and trunk were stiff. When the neurologist tried to flex his arm, he felt many catches, similar to a cogwheel. There was no weakness, sensory problems, or abnormalities in his reflexes. When asked to walk, Sam took many tries to rise from his chair. When he finally stood up, his posture was stooped and flexed. His gait was slow, his feet shuffled when he walked, and his arms didn't swing with his steps. The neurologist told Sam's daughter that she was correct that this wasn't aging, and explained to her all of the details about a new medication that Sam needed to take.

**414.** Damage to which structure in particular causes Sam's problem with movement?

a. Substantia gelatinosa
b. Substantia nigra, pars reticularis
c. Substantia nigra, pars compacta
d. Caudate nucleus
e. Thalamus

**415.** What is the blood supply of the main structure damaged?

a. Lenticulostriate branches of the middle cerebral and anterior cerebral arteries
b. Perforating branches of the basilar and vertebral arteries
c. Anterior choroidal artery and anterior cerebral artery
d. Posteromedial branches of the posterior cerebral and posterior communicating arteries
e. Anterior cerebral and anterior communicating arteries

**416.** What neurotransmitter is deficient?

a. Norepinephrine
b. Glutamate
c. Dopamine
d. ACh
e. GABA

**417.** Which of the following is a precursor to the deficient neurotransmitter, and can be given as a medication to improve Sam's movement?

a. Tyrosine
b. Choline
c. Acetyl-CoA
d. Tryptamine
e. L-dopa

**418.** The antagonism of which enzyme by drugs will increase the amount of the deficient neurotransmitter?

a. Choline acetyltranferase
b. Monoamine oxidase
c. GABA transaminase
d. Acetylcholinesterase
e. Tyrosine hydroxylase

## Questions 419–424

John is a 57-year-old man who has always been a very heavy drinker, often consuming 2 pints of whiskey per day, for many years. Upon the urging of his wife, he decided to seek medical attention for help with problems with his gait, which has steadily worsened over the past several months. He noticed that he now needed to stand with his feet far apart in order to maintain his balance and that he waddled when he walked. The doctor who evaluated him tested his memory and speech carefully, as well as his cranial nerves, and was unable to find any deficits. There was no weakness, sensory loss, or abnormalities in his reflexes. When asked to touch the doctor's finger, then his nose, John missed his nose slightly, but rapidly corrected the movement on both sides. When asked to slide his right heel down his left shin, his heel slid sideways and clumsily across the bone until it reached his ankle. The response with the left heel was similar. When asked to walk, John walked with his feet very far apart. If he attempted to walk in a tandem fashion, with one heel in front of the other toe, he began to fall, and the doctor needed to catch him. The doctor ordered an magnetic resonance imaging (MRI) of John's head.

**419.** What term could one use for John's gait?

a. Stiff
b. Festinating
c. Ataxic
d. Spastic
e. Shuffling

**420.** A gait problem of this type could be caused by lesions in what (which) system(s)?

a. Cerebellar tracts only
b. Posterior columns only
c. Corticospinal tracts
d. Both the cerebellar and posterior column systems
e. Spinothalamic system

**421.** Where in the brain would a neurologist expect to visualize it on an MRI scan?

a. Red nucleus
b. Cerebellar vermis
c. Substantia nigra
d. Internal capsule
e. Basilar pons

**422.** The region of the affected area is associated with which functional division of the cerebellum?

a. Cerebrocerebellum
b. Spinocerebellum
c. Dentate nucleus
d. Superior cerebellar peduncle
e. Brachium pontis

**423.** To which deep nucleus does the damaged region project?

a. Globose
b. Dentate
c. Fastigial
d. Vestibular
e. Emboliform

**424.** Which cell type most likely sustained the most damage from John's alcohol consumption?

a. Schwann cell
b. Pyramidal cell
c. Stellate cell
d. Anterior horn cell
e. Purkinje cell

### Questions 425–429

Louise is an 86-year-old woman who has had difficulty with high blood pressure, high cholesterol, diabetes, strokes, and blood clots in her legs for many years. One day, her grandson arrived at her apartment in a senior citizen center for his weekly visit, only to find her lying unconscious on the floor. He immediately called an ambulance to bring her to the nearest emergency room. The paramedics in the ambulance gave Louise some

medications, including glucose, but she did not awaken. She was brought to the nearest emergency room, where a physician was called to evaluate her. She was breathing on her own and had a pulse, but could not be aroused to any stimulus. Her arms and legs were stiff, and would not move in response to a painful stimulus. Her eyes moved in response to moving her head. Finally, in response to a very loud shout and pinch on the arm, she briefly opened her eyes; however, she immediately shut them again. Further attempts to arouse Louise were unsuccessful. She was taken for a CT scan of her head, and then brought to an intensive care unit.

**425.** An acute stroke in which portion of the CNS could cause this picture?

a. Right frontal lobe
b. Left frontal lobe
c. Right temporal lobe
d. Pons and midbrain
e. Right occipital lobe

**426.** What is the cause of the stiffness in Louise's arms and legs?

a. Infarction of the corticospinal tracts bilaterally in the pons
b. Damage to the basal ganglia
c. Infarction of the precentral gyrus
d. Infarction of the internal capsules bilaterally
e. Thalamic infarction

**427.** Infarction of which artery may cause this picture?

a. Anterior cerebral artery
b. Middle cerebral artery
c. Anterior choroidal artery
d. Basilar artery
e. Lenticulostriate branches of the middle cerebral artery

**428.** If the stroke occurred in the brainstem, which region is most likely affected?

a. Facial nerve nucleus
b. Trochlear nerve nucleus
c. Reticular formation
d. Trigeminal system
e. Medial longitudinal fasciculus

**429.** What are the main monoaminergic systems of the region infarcted?

a. Dopamine
b. Norepinephrine
c. Serotonin
d. GABA
e. Norepinephrine and serotonin

# Motor Systems

## *Answers*

**390. The answer is a.** *(Nolte, pp 485–492.)* The classic appearance of a patient with a lesion of the cerebellar hemispheres is one in which voluntary and skilled movements are affected. They are uncoordinated and there are errors in the range, force, and direction of movement. The relationships between the cerebellum and the motor regions of the cerebral cortex have been disrupted. Lesions of other regions such as the flocculonodular lobe, vermal region of the anterior cerebellar cortex, or fastigial nucleus produce different symptoms (disturbances of balance, muscle tone, or nystagmus). Although pure lesions limited to the ventral spinocerebellar tract have not been reported, it is likely that such a lesion could not account for the symptoms indicated in this question. Information carried by this tract concerns activity of Golgi tendon organs of muscles of the lower limbs.

**391. The answer is d.** *(Nolte, pp 269–271, 439–448.)* An upper motor neuron (UMN) paralysis occurs following a lesion of the internal capsule. Such a lesion disrupts not only fibers destined for the spinal cord, but others that project to parts of the reticular formation and activate inhibitory reticulospinal mechanisms. Loss of such inhibitory input to spinal cord motor neurons then leads to increased levels of excitation of these neurons. The behavioral manifestation of this process is spasticity. Lesions of ventral horn cells produce a flaccid paralysis. Lesions of the postcentral gyrus primarily produce sensory loss, not spasticity. Since the corpus callosum is concerned with interhemispheric transfer of information, a lesion of this bundle will not produce spasticity. A lesion of the substantia nigra will result in Parkinson's disease, which is associated with tremors at rest and rigidity, but not spasticity.

**392. The answer is b.** *(Kandel, pp 764–777.)* The largest synaptic potentials produced by cortical stimulation would most likely be seen in spinal motor neurons that innervate distal muscles. One of the primary functions of the corticospinal tract is to control the distal muscles of the hands and fingers. Penfield and others constructed a homuncular map from stimulation studies of the cortex. Such studies reveal that the region of the cortex

that is associated with the hands and fingers is considerably larger than those regions that are associated with the proximal musculature. Accordingly, stimulation of the hand region of the cortex would activate more fibers than other cortical regions. It is likely that more ventral horn neurons (located in a lateral position) innervate distal musculature than neurons (located in a medial position) innervate proximal musculature. Since the size of the synaptic potential is a function of both the number of fibers that provide a converging input into a given region and the number of cells that discharge in response to that converging input, it is reasonable to conclude that the largest potentials would be observed following stimulation of the cortical regions associated with the distal musculature. Since neurons situated in the motor cortex project their axons to motor horn cells and interneurons but not to sensory neurons of the dorsal horn (although the component of the corticospinal tract that arises from the parietal lobe does project to the dorsal horn), stimulation of the motor cortex could only produce weak potentials at best among sensory neurons in the dorsal horn.

**393. The answer is b.** *(Kandel, pp 150–164, 764–777.)* Motor cortex neurons receive information from the muscle to which they project or from a region of skin related to the function of that muscle. The anatomic pathway includes dorsal column–medial lemniscal fibers that terminate in the ventral posterolateral (VPL) nucleus of the thalamus. Fibers from the VPL nucleus then project to the postcentral gyrus. Fibers from a given region of the primary sensory cortex project to the region of the primary motor cortex whose projection target in the spinal cord involves the same muscle group (or body part) from which the sensory stimulus originated. All other choices are incorrect. The properties of membrane potentials of neurons in the motor cortex follow the same principles as those found elsewhere in the nervous system; namely, in the resting state, the cell membrane is more permeable to potassium than to sodium. The red nucleus does not have an ascending projection (and, therefore, cannot be reciprocally connected with the motor cortex). Its fibers project, instead, to the spinal cord and lower brainstem. In general, corticospinal fibers that activate alpha motor neurons that innervate a given muscle group will also synapse with gamma motor neurons associated with that same muscle group. Coactivation of both alpha and gamma motor neurons is an important principle because it enables muscle spindles to react to changes in the length of the muscle even during the process of movement of the limb associated with that mus-

cle. The projection to the cerebellum from the motor cortex is disynaptic. Projections from the cerebellum to the motor cortex synapse in the dentate nucleus and the ventrolateral nucleus of the thalamus.

**394. The answer is b.** *(Kandel, pp 1306–1309.)* This constellation of deficits, including paralysis of the lower right face, paralysis of the right lower limbs, and right deviation of the tongue, requires a lesion located in the left internal capsule. Since the motor fibers from the cortex that supply all three of these regions (i.e., limbs, lower face, and tongue) are all crossed, a lesion of the internal capsule will produce each of these deficits. Also, recall that the tongue will deviate to the side of the lesion when the lesion affects the lower motor neuron (LMN) (i.e., cranial nerve XII) directly. When it affects the UMN (i.e., fibers in the internal capsule), inputs into the contralateral nucleus of cranial nerve XII are affected. Thus, the tongue in this instance will deviate to the side opposite the lesion. A lesion of the pontine tegmentum will not affect descending corticospinal or corticomedullary fibers since these fibers are contained in the basilar part of the pons. A lesion of the medulla would be too caudal to affect cortical fibers that terminate on cells of the facial nucleus whose axons innervate muscles of the lower face.

**395. The answer is c.** *(Kandel, pp 654–672, 770–777.)* The premotor areas play an important role in the programming or sequencing of responses that compose complex learned movements. They receive significant inputs for this process from the posterior parietal lobule and, in turn, signal appropriate neurons in the brainstem and spinal cord (both flexors and extensors). Lesions of the postcentral gyrus produce a somatosensory loss. Lesions of the precentral gyrus produce paralysis. Neither lesions of the prefrontal cortex nor those of the cingulate gyrus have been reported to produce apraxia.

**396. The answer is b.** *(Kandel, pp 853–864.)* The neostriatum (i.e., caudate nucleus and putamen) constitutes the principal, if not exclusive, receiving area for afferent fibers to the basal ganglia. The subthalamic nucleus and the substantia nigra share reciprocal connections with the paleostriatum (i.e., globus pallidus) and the neostriatum, respectively. However, these areas receive few, if any, fibers from the cerebral cortex or the centromedian nucleus of the thalamus, which are the major afferent

sources to the basal ganglia. Functions of the claustrum are not well understood, but it is believed to be more closely associated with the neocortex than with the basal ganglia.

**397. The answer is e.** *(Kandel, pp 853–864.)* The cerebral cortex is a principal source of afferent fibers to the neostriatum and utilizes glutamate as its transmitter, which is excitatory to caudate neurons. Thus, neither GABA nor substance P are transmitters from the cortex to the neostriatum; nor is GABA a transmitter released from the nigrostriatal terminals. Projections from the hypothalamus to the caudate nucleus have never been demonstrated and, presumably, do not exist.

**398. The answer is d.** *(Kandel, pp 853–864.)* The major transmitter released at terminals of neostriatal and paleostriatal fibers is GABA. Thus, the output of the basal ganglia is mainly inhibitory. This suggests that thalamic influences upon the cortex are generated through the process of disinhibition, whereby neurons of the basal ganglia are inhibited. The presence of glycine in striatal neurons has yet to be demonstrated. Enkephalins are released from terminals of neostriatal-pallidal fibers but not from other efferent neurons of the striatum. Dopamine is released from the brainstem and some adjoining hypothalamic neurons but certainly not from striatal neurons. The neostriatum receives cortical inputs that utilize glutamate, but the release of GABA from terminals of striatal efferent fibers has not been demonstrated.

**399. The answer is c.** *(Kandel, pp 853–864.)* The basic principle governing how the basal ganglia control motor activity is that they do so by modulating neurons of the motor cortex and premotor areas (of the ipsilateral side) via synaptic connections in the VL and VA nuclei of the thalamus. One can see from the circuits:

globus pallidus → ventrolateral nucleus → area 4 of cortex (medial segment) (VL)
globus pallidus → ventral anterior nucleus → area 6 of cortex (medial segment) (VA)

that damage to the basal ganglia on one side of the brain will affect cortical neurons on the same side. This will result in dyskinesia expressed on the contralateral side of the body because the corticospinal tract is crossed. The

other possibilities listed in the question are not viable. Projections of the basal ganglia to the brainstem nuclei are minimal. The basal ganglia do not project fibers down to the spinal cord nor do they project to the cerebellum.

**400. The answer is c.** *(Kandel, pp 833–846.)* The principal source of afferent fibers to the flocculonodular lobe is the vestibular complex, in particular, the inferior and medial vestibular nuclei. For this reason, this lobe of the cerebellum is sometimes referred to as the *vestibulocerebellum.* The red nucleus and cerebral cortex project topographically (via relays in the inferior olivary nucleus and deep pontine nuclei, respectively) to the anterior and posterior lobes. Pathways arising from the spinal cord, such as the spinocerebellar tract, project to the anterior lobe. Other fibers arising from the spinal cord enter the cerebellum through a relay in the inferior olivary nucleus. Such fibers terminate in both anterior and posterior lobes.

**401. The answer is c.** *(Kandel, pp 864–866.)* In Huntington's disease, the essential neurochemical change is in the basal ganglia, where there is a significant reduction in the two transmitters ACh and GABA. In particular, there are reduced levels of choline acetyltransferase, glutamic acid decarboxylase, and GABA.

**402. The answer is c.** *(Kandel, pp 864–866.)* A lesion of the subthalamic nucleus results in hemiballism, a form of dyskinesia in which the patient displays severe involuntary movements. It is believed to occur as a result of an imbalance in the output signals of the basal ganglia. There is a change in the relationship between efferent pathways associated with the two pallidal segments (i.e., a direct pathway from the medial pallidal segment to the VL and VA nuclei of the thalamus versus an indirect pathway, involving connections between the lateral pallidal segment, subthalamic nucleus, and substantia nigra). Thus, in hemiballism the indirect pathway is disrupted, resulting in a change in the output signals of the pallidum to the thalamus.

**403. The answer is b.** *(Kandel, pp 861–866.)* Choreiform movements have generally been associated with damage to the neostriatum (the cortex and the globus pallidus have occasionally been implicated). Normally, there is a balance in what seems to be opposing effects of ACh, dopamine, and GABA in the neostriatum. In this disorder, the levels of ACh and GABA

are significantly reduced. This creates an imbalance in which dopamine levels now become (relatively) too high. Accordingly, effective pharmacologic treatment involves the use of dopamine receptor blockers.

**404. The answer is d.** *(Kandel, p 1201; Gilroy, pp 172–174.)* Tardive dyskinesia, a disorder involving involuntary movements of the mouth, face, and tongue, is caused by long-term treatment with antipsychotic drugs that block or decrease dopaminergic synaptic transmission. Such treatment eventually produces a hypersensitivity in dopamine receptors to dopamine. An imbalance is created between dopamine, GABA, and cholinergic systems within the striatum and this is believed to be responsible for the disorder.

**405. The answer is c.** *(Kandel, pp 862–864.)* MPTP was discovered by accident when drug abusers who were using a synthetic heroin derivative developed signs of Parkinson's disease. It was discovered that their drug included the contaminant MPTP. As a consequence, MPTP has been applied systemically in a number of experimental animals, resulting in significant decreases in dopamine content of the brain due to the loss of dopaminergic neurons in the substantia nigra. These animals also developed symptoms similar to those seen in Parkinson's patients. For these reasons, this drug is currently being used for research purposes in order to develop a better understanding of this disease and to establish possible drug therapies for its treatment and eventual cure.

**406. The answer is a.** *(Nolte, pp 473–485.)* One of the most important features of the anterior lobe of the cerebellum is that it receives major inputs from structures that mediate information concerning muscle spindle and Golgi tendon organ activity (sometimes referred to as *unconscious proprioception*). The pathways that mediate unconscious proprioception include the dorsal and ventral spinocerebellar tracts and the cuneocerebellar tract. Accordingly, the cerebellar anterior lobe is sometimes referred to as the *spinocerebellum*. The fastigial and dentate nuclei receive their principal inputs from the cerebellar cortex, and their axons project out of the cerebellum. The posterior lobe receives few, if any, inputs from pathways that mediate unconscious proprioception information.

**407. The answer is c.** *(Kandel, pp 833–846; Nolte, pp 480–484.)* A unique feature of the connections between cerebral cortex and the cerebellum is

the somatotopically organized projection from the cerebral cortex largely to the cerebellar hemispheres (some fibers terminate in the vermis). The somatotopic maps are arranged in both anterior and posterior lobes in a manner that has the distal musculature functionally represented in the lateral aspect of the hemispheres, while the proximal musculature is represented toward or in the vermal region. Because of this somatotopic arrangement, the lateral hemispheres are concerned with functions associated with detailed movements of the limbs, while more medial regions are concerned with regulation of the proximal musculature (e.g., postural mechanisms).

**408. The answer is d.** *(Kandel, pp 835–837.)* The cerebellar glomerulus consists of mossy fiber terminals, Golgi dendrites, axon terminals of Golgi cells, and granule cell dendrites. The flow of information in the glomerulus is as follows: (1) Information reaches the cerebellar cortex through mossy fibers. (2) Axon terminals of mossy fibers terminate upon dendrites of either granule or Golgi cells. (3) Collaterals of parallel fibers (axons of granule cells) may contact dendrites of Golgi cells, whose axons then feed back onto the granule cells. (4) Mossy fiber terminals synapse with Golgi cell dendrites, whose axons then make synaptic contact with the granule cell (feed-forward mechanism). The axons of the granule cells run parallel to the cortex and perpendicular to the orientation of the Purkinje cell dendrites with which they synapse. The circuitry for feedback and feed-forward mechanisms is as follows:

Feedback mechanism:
mossy fiber axon terminal → granule cell dendrites → granule cell axon → Golgi cell → (inhibits) granule cell

Feed forward mechanism:
mossy fiber axon terminal → Golgi cell dendrites → Golgi cell axon → (inhibits) granule cell

**409–411. The answers are 409-c, 410-b, 411-a.** *(Kandel, pp 833–846.)* For each of these questions, the central point relates to the projection targets of the relevant deep cerebellar nuclei and their relationship to their afferent sources. In question 409, note that the inputs from the frontal lobe eventually reach the cerebellar cortex (which involves the cerebellar hemispheres of the anterior and posterior lobes to a large extent).

Because many of these cerebellar afferents terminate in the lateral aspect of the hemisphere, the return (or feedback) pathway will initially involve Purkinje cell axons that synapse with cells in the dentate nucleus. Fibers of the dentate nucleus then supply the VL nucleus of the thalamus, which, in turn, supplies the primary motor cortex. Question 410 concerns the feedback pathways associated with the red nucleus. Inputs to the cerebellum from the red nucleus utilize the inferior olivary nucleus as a relay. These inputs supply the anterior and posterior cerebellar lobes in a topographic manner. Since many of these fibers are distributed to an intermediolateral position within the cerebellar cortex, Purkinje cells from this area supply the interposed (i.e., globose and emboliform) nuclei. The interposed nuclei, in turn, supply the red nucleus via the superior cerebellar peduncle. In question 411, the issue concerns the relationship of the cerebellum to those spinal cord mechanisms relating to descending fibers of the vestibulospinal and reticulospinal systems. Recall that many of the spinocerebellar fibers are distributed to the medial vermal region of the anterior lobe. Thus, the return flow of information to the spinal cord with respect to the regulation of muscle tension will involve the vestibulospinal and reticulospinal systems. To achieve this objective, the Purkinje cells of the medial (vermal and paravermal) regions of cerebellar cortex project to the fastigial nucleus. The fastigial nucleus, in turn, projects to both the reticular formation and vestibular nuclei, which then complete the feedback circuit by sending their axons down to the spinal cord.

**412. The answer is d.** *(Kandel, pp 833–846.)* This experiment was actually carried out many years ago by several investigators. They observed that stimulation of the medial vermal region of the cerebellar cortex could either inhibit or facilitate extensor muscle tone, depending on the precise site of stimulation. It is most likely that stimulation directly affected local populations of Purkinje cells, which then inhibited other local populations of neurons within the fastigial nucleus. Since the fastigial nucleus (as well as the other deep cerebellar nuclei) has excitatory effects upon its target neurons, inhibition or excitation of the fastigial nucleus following local stimulation of the cerebellar cortex would result in either decreased or increased activation of the vestibulospinal system. Thus, such a mechanism could account for the changes of muscle tone that are seen after stimulation of the anterior vermal region of cortex.

**413. The answer is e.** *(Nolte, pp 488–492.)* Since the flocculonodular lobe receives and integrates inputs from the vestibular system, it is understandable why lesions that disrupt this integrating mechanism for vestibular inputs would result in difficulties in maintaining balance. Indeed, this is a classic feature of lesions of the flocculonodular lobe but is not associated with lesions in the hemispheres of the posterior lobe, anterior limb of the internal capsule, or the dentate nucleus, which are functionally linked to the frontal lobe. Lesions of the anterior lobe also do not affect mechanisms of balance.

**414–418. The answers are 414-c, 415-d, 416-c, 417-e, 418-b.** *(Adams, p 1074; Kandel, pp 861–866, 1306–1309.)* Sam has Parkinson's disease, a degenerative condition caused by progressive loss of dopaminergic cells in the substantia nigra, pars compacta. This is an area that controls the speed and spontaneity of movement, so damage to this area can produce deficits that include: a slow shuffling gait with a tendency to move progressively faster (festinating gait); problems with maintaining size in handwriting, with a tendency to write with small letters (micrographia); masklike facial expression with a paucity of eyeblinks; and difficulty getting out of a chair. Other problems include a soft, monotonous voice; muscle rigidity (lead-pipe rigidity); a tremor at rest that is "pill-rolling"; and a combination of a tremor and rigidity, especially in the arms, which, when flexion is attempted, elicits a "cogwheeling" property. Failure to swallow with a normal frequency makes drooling a problem. Dementia (senility) is also a problem with Parkinson's patients, especially later in the course of the disease. The blood supply to the substantia nigra arises from the posterior circulation, specifically the posteromedial branches of the posterior cerebral artery and branches of the posterior communicating artery. The lenticulostriate branches of the middle cerebral artery supply other portions of the basal ganglia, such as the striatum and the globus pallidus. The anterior choroidal artery also supplies some of the telencephalic nuclei of the basal ganglia. The majority of cells that are lost in this disease are dopaminergic cells in the substantia nigra, pars compacta. Only the pars compacta region of the substantia nigra contains dopaminergic neurons. Medications are currently available to lessen the symptoms of Parkinson's disease. Some of these medications contain various concentrations of L-dopa, an immediate precursor to dopamine. Dopamine itself doesn't cross the blood-brain barrier, so it cannot be directly replaced. Medications that

antagonize the breakdown of catecholamines by monoamine oxidase can increase the amount of dopamine available for the remaining cells in the substantia nigra.

**419–424. The answers are 419-c, 420-d, 421-b, 422-b, 423-e, 424-e.** *(Kandel, pp 833–849, 879–885; Adams, pp 89, 118, 1156–1158.)* An ataxic gait is an unsteady gait. Gaits due to motor weakness or spasticity tend to involve circling of the weak leg (circumduction); festinating or shuffling gaits, which are often due to parkinsonism or disease of the basal ganglia, involve a stooped posture with shuffling of the feet and very small steps. An ataxic gait may result from motor incoordination due to cerebellar disease, or from lack of proprioception in the lower extremities due to disease in the posterior column system (gait becomes unsteady when a patient is unable to detect the location of his or her feet). Degeneration of both systems may occur due to alcoholism, although in this case, we are told that John does not have any sensory deficits when this modality is tested in isolation. This is an example of alcoholic cerebellar degeneration. It is caused by degeneration (probably through nutritional deficiency) of neurons in the cerebellar cortex, particularly of the Purkinje cells, and is usually restricted to anterior and superior parts of the vermis, as well as anterior portions of the anterior lobes. For this reason, most of the deficits in this syndrome involve midline structures such as the trunk, which are represented most in the vermis. Trunk instability usually causes problems with gait. In addition, because the cerebellar homunculus represents the legs in the anterior portion of the anterior lobe, the legs are affected more than the arms. Loss of volume within the vermis of the cerebellum is readily visualized, especially on an MRI of the brain, because this technique allows good visualization of the posterior fossa. If these changes are visualized, then the condition is most likely chronic (as also indicated by the history) and most likely irreversible. However, it is important to make sure that the patient is well nourished, takes vitamins, and stops drinking in order to prevent other neurologic problems from occurring. Damage to other brain regions listed do not cause such damage. The spinocerebellum receives sensory inputs from the spinal cord and is instrumental in controlling posture and movement. It includes the vermis and the intermediate hemisphere. The cerebrocerebellum consists of the lateral hemispheres and is instrumental in the planning of movement. The dentate nucleus comprises the cell bodies that form the superior cerebellar peduncle. The brachium pontis corre-

sponds to the middle cerebellar peduncle. The spinocerebellar cortical (Purkinje) cells project to the fastigial and interposed nuclei. Purkinje cells are found in the cerebellar cortex. None of the other choices are cells that are found in the cerebellum.

**425–429. The answers are 425-d, 426-a, 427-d, 428-c, 429-e.** *(Adams, pp 347, 350; Kandel, pp 887–888, 898–902, 1307–1311.)* The CT scan of Louise's brain revealed a large, acute stroke of her upper pons and midbrain. Strokes of these areas often result from occlusion of the basilar artery and can produce coma, or a variant of hypersomnia called *akinetic mutism* or *coma vigil*. An EEG of a patient like this shows a pattern associated with slow-wave sleep, but eye movements are preserved. It is likely that the corticospinal tracts within the pons were damaged during this very large stroke, causing the increased tone from lack of inhibition, as well as the lack of movement in Louise's arms and legs. Infarctions of perforators of the basilar artery, supplying the reticular formation of the pons may cause coma. These perforators also supply the corticospinal tracts, causing the increased tone and weakness of Louise's legs, so a large stroke may involve both functions. Coma occurs because there is damage to the brainstem tegmentum, which is a major component of the ascending reticular activating system. Although it is not known exactly which area is precisely responsible for consciousness, lesions of this region, as well as projections from the medial regions of the midbrain reticular formation can produce coma. The two main monoaminergic systems of the reticular formation are the noradrenergic and serotonergic systems, originating in the locus ceruleus and raphe nuclei, respectively. The mesolimbic, mesostriatal, and mesocortical dopaminergic systems are located within the ventrorostral aspect of the brainstem, but not within the reticular formation.

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# Higher Functions

## *Questions*

**DIRECTIONS:** Each item below contains a question or incomplete statement followed by suggested responses. Select the **one best** response to each question.

### Questions 430–431

A 67-year-old man suffers an infarct of the geniculothalamic branch of the posterior cerebral artery. In particular, there is involvement of nuclei of the posterior thalamus.

**430.** The most likely effects of such an infarct include

a. Emotional volatility in response to an innocuous statement
b. Short-term memory loss that occurs about 1 week following the infarct
c. Long-term memory loss that occurs about 1 month following the infarct
d. Severe pain triggered by cutaneous stimuli applied to the patient
e. Spastic paralysis of the contralateral limbs

**431.** The neurons affected by this infarction project primarily to the

a. Hypothalamus and midbrain
b. Parietal and occipital cortices
c. Precentral and postcentral gyri
d. Basal ganglia and premotor cortex
e. Prefrontal cortex and medial aspect of the frontal lobe

### Questions 432–434

A 52-year-old woman has an infarct involving a branch of the posterior communicating artery, causing damage to the ventral anterior (VA), ventrolateral (VL), dorsomedial, and anterior thalamic nuclei.

**432.** The most likely clinical manifestations of this infarct include

a. Hemiparesis and neuropsychological impairment
b. Loss of sleep and apnea
c. Loss of appetite and thermoregulation
d. Total blindness of the contralateral eye
e. Marked endocrine dysfunction

**433.** The VA nucleus receives inputs primarily from structures associated with

a. Somatosensory functions
b. Motor functions
c. Autonomic functions
d. Auditory and taste functions
e. The regulation of sleep

**434.** The primary outputs of the VA nucleus include

a. Prefrontal and premotor cortices
b. Precentral and postcentral gyri
c. Posterior parietal lobe
d. Middle temporal gyrus
e. Wernicke's area

### Questions 435–437

A patient has an infarct involving the medial branches of the basilar root of the posterior cerebral artery. The primary region affected includes nuclei of the medial thalamus. One likely effect of this infarct is

a. Grand mal epilepsy
b. Severe acute depression and hyperphagia
c. Drowsiness and abnormalities in memory and attention
d. Marked somatosensory loss, including pain and temperature
e. Upper motor neuron (UMN) paralysis

**436.** The probable basis for the effects of the infarct is the loss of processing of information from

a. Hypothalamus
b. Parietal cortex
c. Reticular formation
d. Basal ganglion
e. Hippocampal formation

**437.** The major output of the mediodorsal thalamic nucleus is the

a. Precentral gyrus
b. Postcentral gyrus
c. Prefrontal cortex
d. Posterior parietal lobe
e. Temporal lobe

**438.** The superior temporal gyrus receives primary inputs from

a. Centromedian thalamic nucleus
b. Medial geniculate thalamic nucleus
c. Lateral geniculate thalamic nucleus
d. Dorsomedial thalamic nucleus
e. Anterior thalamic nucleus
f. VA thalamic nucleus

**439.** Of the following thalamic nuclei, which nucleus contains neurons that have properties of both specific and nonspecific thalamus?

a. Centromedian thalamic nucleus
b. Medial geniculate thalamic nucleus
c. Lateral geniculate thalamic nucleus
d. Dorsomedial thalamic nucleus
e. Anterior thalamic nucleus
f. VA thalamic nucleus

**440.** Which one of the following thalamic nuclei makes local connections with other thalamic nuclei and, additionally, projects to the basal ganglia?

a. Centromedian thalamic nucleus
b. Medial geniculate thalamic nucleus
c. Lateral geniculate thalamic nucleus
d. Dorsomedial thalamic nucleus
e. Anterior thalamic nucleus
f. Pulvinar

**441.** Rapid eye movement (REM) sleep is characterized by

a. Slow-wave EEGs
b. Sleep spindles
c. Low-voltage EEGs
d. High-voltage biphasic waves
e. An increase in most skeletal muscle tone

**442.** Nuclei that have been shown to regulate REM sleep are located in the

a. Spinal cord–medulla border
b. Rostral aspect of the medulla
c. Rostral aspect of the pons
d. Tectal aspect of the rostral midbrain
e. Hypothalamic-thalamic border

**443.** During relaxed periods of wakefulness, a normal individual's EEG will display

a. α rhythms (8 to 13 Hz)
b. β rhythms (13 to 30 Hz)
c. θ rhythms (4 to 7 Hz)
d. δ rhythms (0.5 to 4 Hz)
e. Spike and wave activity

**444.** A patient is confused and displays localized jerks in his right hand, which progress to jerks of the entire arm with a brief loss of consciousness. This disorder can best be characterized as a

a. Generalized seizure
b. Absence seizure
c. Simple partial seizure
d. Complex partial seizure
e. Petit mal seizure

**445.** The display shown by the patient described in question 444 is then followed by his falling to the ground with a further loss of consciousness in which all of his extremities are extended and are rigid, and jerks of these limbs are displayed as well. This disorder would best be characterized as a

a. Generalized seizure
b. Absence seizure
c. Simple partial seizure
d. Complex partial seizure
e. Petit mal seizure

**446.** Vasopressin is released from the posterior pituitary. However, it is synthesized in the

a. Mamillary bodies
b. Lateral hypothalamus
c. Supraoptic hypothalamic nucleus
d. Ventromedial hypothalamic nucleus
e. Posterior hypothalamus

**447.** Which of the following statements concerning temperature regulation is correct?

a. Stimulation of the posterior hypothalamus results in panting, dilation of blood vessels, and suppression of shivering
b. Neurons in the anterior hypothalamus respond to local warming of hypothalamic tissue but not to warming of the skin
c. Stimulation of the anterior hypothalamus may produce constriction of blood vessels and shivering
d. Neurons in the preoptic region and septal area act in concert to intensify increases in body temperature generated by pyrogens
e. Temperature regulation requires the integration of skeletomuscular, endocrine, and autonomic responses

**448.** The supraoptic nucleus is most closely associated with

a. Feeding behavior
b. Temperature regulation
c. Sexual behavior
d. Short-term memory functions
e. Water balance

**449.** Lesions of the lateral hypothalamus will likely produce

a. Feeding behaviors
b. Drinking behaviors
c. Sexual behaviors
d. Aphagia
e. Hypertension

**450.** A number of investigations have provided strong evidence that the suprachiasmatic nucleus plays an important role in

a. Water intake
b. Food intake
c. Hypertension
d. Circadian rhythms
e. Short-term memory

**451.** The Klüver-Bucy syndrome is typically associated with lesions of the

a. Septal area
b. Amygdala
c. Cingulate gyrus
d. Medial hypothalamus
e. Lateral hypothalamus

**452.** The central nucleus of the amygdala

a. Projects its axons to the medial hypothalamus via the stria terminalis
b. Is a major receiving area for information concerning tertiary auditory and visual signals
c. Has high concentrations of enkephalins, somatostatin, and dopamine
d. Is a primary location of norepinephrine-containing cell bodies in the forebrain
e. Projects axons that directly inhibit spinal motor neurons

**453.** Neurochemical and related theories of schizophrenia postulate that schizophrenia is

a. Basically caused by environmental factors rather than genetic ones
b. Linked to increases in brain dopamine levels
c. Linked to increases in brain serotonin levels
d. Linked to decreases in brain endorphin levels
e. Linked to decreases in brain neuropeptide levels

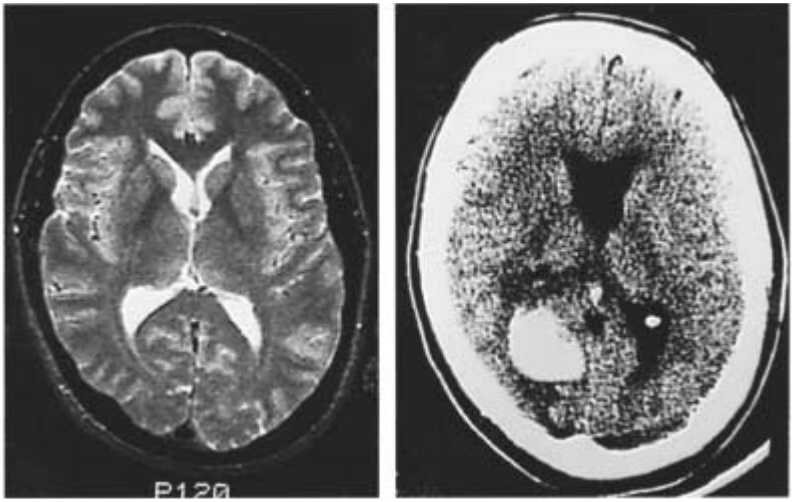
**454.** While the hippocampal formation has few if any direct (monosynaptic) connections with the lateral hypothalamus, it is known to modulate functions associated with the hypothalamus. The underlying anatomic substrate for such effects is mediated via a synaptic relay in the

a. Cingulate gyrus
b. Habenular nucleus
c. Mediodorsal thalamic nucleus
d. Septal area
e. Bed nucleus of the stria terminalis

**455.** Which of the following constitutes the Papez circuit?

a. Hippocampal formation → mamillary bodies → anterior thalamic nucleus → prefrontal cortex → hippocampal formation
b. Hippocampal formation → septal area → hypothalamus → hippocampal formation
c. Hippocampal formation → mamillary bodies → anterior thalamic nucleus → cingulate gyrus → hippocampal formation
d. Amygdala → hippocampal formation → mamillary bodies → amygdala
e. Prefrontal cortex → hippocampal formation → septal area → medial hypothalamus → prefrontal cortex

## Questions 456–457



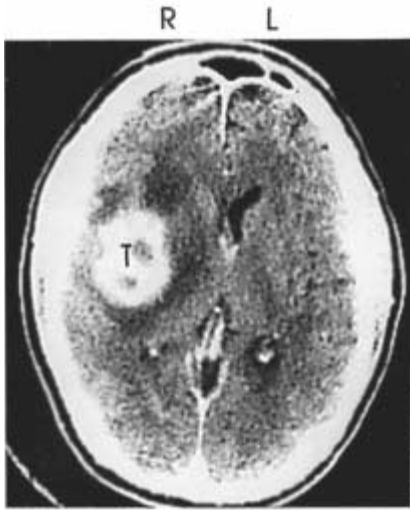

**456.** The T2-weighted MRI scan on the left side of the figure above is of a normal patient. In the CT scan on the right side, the patient had sustained a right cerebral hemorrhage, indicated by the large white area. It is likely that the cerebrovascular accident produced

a. Right homonymous hemianopsia
b. Left homonymous hemianopsia
c. Loss of intellectual and emotional processes
d. Aphasia
e. Hemiparesis of the right side of the body

**457.** The blood vessel(s) affected in the figure above would most likely be the

a. Anterior cerebral artery
b. Middle cerebral artery
c. Posterior cerebral artery
d. Superior cerebellar artery
e. Striate arteries

### Questions 458–459



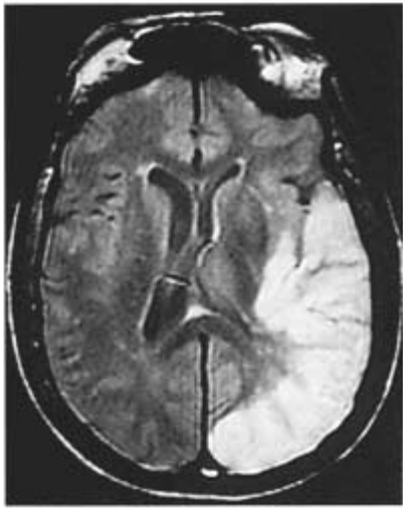

**458.** The CT scan above reveals that the patient has a glioma (T) on the right side of the brain. It is likely that the patient has sustained

a. A UMN paralysis of the left side
b. Dyskinesia
c. Intention tremor
d. Upper left quadrantanopia
e. Upper right quadrantanopia

**459.** The tumor in the scan above has most likely damaged the

a. Lentiform nucleus only
b. Internal capsule only
c. Thalamus only
d. Lentiform nucleus and internal capsule
e. Lentiform nucleus, internal capsule, and thalamus

### Questions 460–461



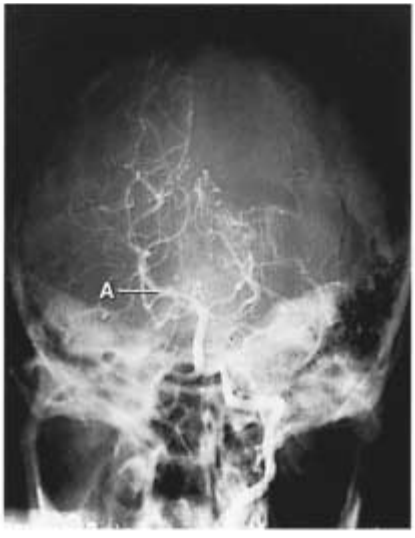
**460.** The patient whose CT scan is shown in the figure above sustained an occlusion of a major artery on the left side of the brain. The most prominent deficits will most likely include

a. A right homonymous hemianopsia only
b. Aphasia only
c. A right homonymous hemianopsia coupled with aphasia
d. Marked intellectual deficits
e. Marked intellectual deficits coupled with hemiballism

**461.** The blood vessel occluded in the figure above is the

a. Anterior cerebral artery
b. Middle cerebral artery
c. Posterior cerebral artery
d. Posterior choroidal artery
e. Superior cerebellar artery

## Questions 462–463



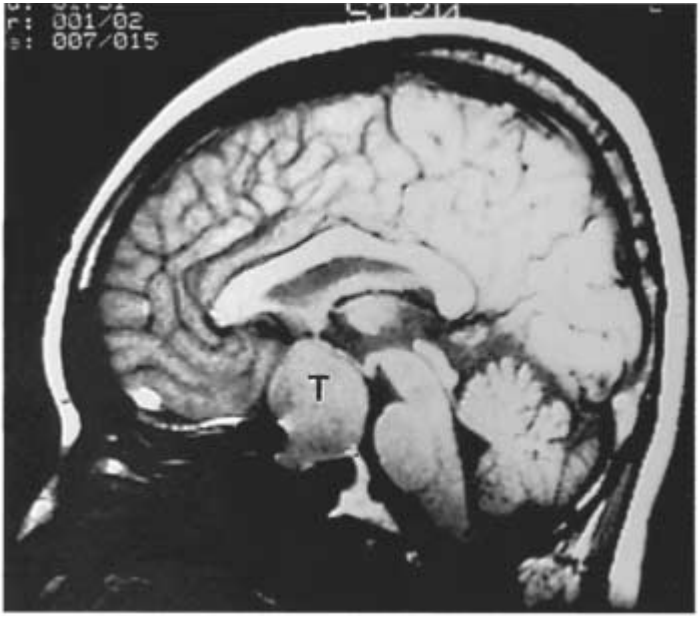

**462.** The vertebral angiogram in the figure above reveals the effects of a severe motorcycle accident upon a 21-year-old woman. As a result of the accident, she most likely suffers from

a. A UMN paralysis of the right side of the body
b. A right homonymous hemianopsia
c. A left upper quadrantanopia
d. Aphasia
e. Dyskinesia

**463.** The artery occluded on the left side and labeled in the figure above on the normal side as A is

a. Vertebral
b. Basilar
c. Middle cerebral
d. Anterior cerebral
e. Posterior cerebral

**464.** The MRI scan in the figure below reveals a large chromophobe adenoma (T) of the pituitary that impinges on the adjoining brain tissue. This tumor caused a




a. Binasal hemianopsia
b. Bitemporal hemianopsia
c. Loss of the accommodation reflex
d. Loss of the pupillary light reflex
e. Loss of conjugate gaze

## Questions 465–470

June is a 65-year-old woman who was previously healthy. One day, while taking a walk in the park, she noticed her right fingers twitching, then her right hand, then her arm and shoulder, followed by a march of twitches down her leg. She did not remember any more than this, because she lost consciousness. An onlooker saw her drop to the ground and deviate her neck backward, while making a high-pitched noise. Then, both of her arms and legs began to jerk for approximately 1 to 2 minutes, then stopped abruptly. She had lost control of her bladder during this event. When the onlooker attempted to speak to June to ask her if she was okay, she was unresponsive. The onlooker called an ambulance, which brought June to the nearest hospital. A doctor met June at the emergency room entrance, and asked her what had happened. By this time, June was slightly drowsy, but able to answer questions appropriately. Her speech was fluent and grammatically correct. She knew the month, but not the day of the week, or where she was. She moved the left side of her body better than her right, but had too much difficulty following commands for an effective motor examination. The remainder of her examination was normal. The doctor ordered a CT of June's head, and drew some blood.

**465.** From which area of the brain did June's seizure begin?

a. Left precentral gyrus
b. Right precentral gyrus
c. Right temporal lobe
d. Left temporal lobe
e. Thalamus

**466.** What could account for June's loss of consciousness following the seizure?

a. Involvement of the reticular activating system
b. Head trauma
c. Bilateral postictal suppression
d. Thalamic involvement
e. Brain hemorrhage from the seizure

**467.** The "march" of twitching that June experienced can be explained by

a. Proximity of the body part to the spinal cord
b. Proximity of the body part to the cerebral cortex
c. Somatotopic representation within the brainstem
d. Somatotopic representation within the basal ganglia
e. Somatotopic representation within the precentral gyrus

**468.** Which cell type is the predominant cause of the seizure?

a. Basket cell
b. Purkinje cell
c. Stellate cell
d. Schwann cell
e. Pyramidal cell

**469.** A burst of what type of potentials may initiate an epileptic seizure?

a. Inhibitory postsynaptic potentials (IPSPs)
b. Membrane potentials
c. Resting potentials
d. Excitatory postsynaptic potentials (EPSPs)
e. Nernst potential

**470.** A chemical mechanism that could underlie seizure generation includes

a. $Na^+$ channel blockade
b. γ-aminobutyric acid (GABA) inhibition
c. Glutamate inhibition
d. Aspartate inhibition
e. Substance P inhibition

### Questions 471–475

Helen is a 76-year-old woman who has had high blood pressure and diabetes for more than 10 years. One day, as she was reaching for a jar of flour to make an apple pie, her right side suddenly gave out, and she collapsed. While trying to get up from the floor, she noticed that she was unable to move her right arm or leg. Helen attempted to cry for help because she was unable to reach the telephone; however, her speech was slurred and rather unintelligible. She lay on the floor and waited for help to arrive. Helen's son began to worry about his usually prompt mother when

she didn't arrive with her apple pie. After several attempts at telephoning her apartment without an answer, he drove to her apartment and found her lying on the floor. She attempted to tell him what had happened, but her speech was too slurred to comprehend, so assuming that his mother had had a stroke, her son called an ambulance to bring her to the nearest emergency room. A neurology resident was called to see Helen in the emergency room because the physicians there, too, felt that she had had a stroke. The resident noted that Helen followed commands very well, and, although her speech was very slurred, it was fluent and grammatically correct. The lower two-thirds of her face drooped on the right, but when asked to raise her eyebrows, her forehead appeared symmetric. Her tongue pointed to the right side when she was asked to protrude it. Her right arm and leg were severely, but equally, weak; her left side had normal strength. She felt a pin and a vibrating tuning fork equally on both sides.

**471.** Where in the central nervous system (CNS) did Helen's stroke occur?

a. Left precentral gyrus
b. Right precentral gyrus
c. Left basilar pons or left internal capsule
d. Right putamen or globus pallidus
e. Left thalamus

**472.** A computerized tomography (CT) scan revealed a new infarct in the left internal capsule. Which artery was occluded, causing the stroke?

a. Lenticulostriate branches of the middle cerebral artery
b. Posterior cerebral artery
c. Anterior cerebral artery
d. Vertebral artery
e. Posterior choroidal artery

**473.** Damage to which two tracts caused Helen to be weak on her right side?

a. Spinothalamic and corticospinal tracts
b. Spinothalamic and corticobulbar tracts
c. Corticospinal and corticobulbar tracts
d. Corticospinal and spinocerebellar tracts
e. Corticospinal and rubrospinal tracts

**474.** Helen's forehead is spared from the weakness because

a. The forehead is innervated by different fibers originating in the postcentral gyrus
b. There are two cranial nerves innervating the forehead
c. The forehead is represented bilaterally at the cortical level
d. The forehead is stronger than the rest of the face
e. Thalamic regions receiving inputs from the forehead contain few inhibitory neurons

**475.** How can Helen's speech deficit be classified?

a. Wernicke's aphasia
b. Broca's aphasia
c. Anomia
d. Dysarthria
e. Conduction aphasia

## Questions 476–480

Lindsey is a 12-year-old girl who has never had medical problems. One day, while in the kitchen with her mother, she told her mother that she felt very frightened all of a sudden and had a funny feeling in her stomach. Immediately after this, she turned her head to the right, stared persistently, and began to chew. Her mother called her name several times, but Lindsey, who was usually a very obedient child, did not answer. After approximately 1 minute of staring, Lindsey slowly turned her head back to her mother. Apparently confused, she asked her mother where she was. Over the next 10 to 15 minutes, she became less and less confused, and by the time she was in the car being driven to the pediatrician by her mother, she felt like she was back to normal. The pediatrician listened to Lindsey's mother's story when they arrived. He examined Lindsey and could find no abnormalities on general physical examination or on neurologic examination. The pediatrician told her mother that he would refer Lindsey to a pediatric neurologist for further evaluation, as well as further evaluation for the need for medication.

**476.** What type of problem did Lindsey most likely have?

a. Attention deficit disorder (ADD)
b. Temporary psychosis
c. Conversion disorder
d. Epilepsy
e. Schizophrenia

**477.** From which area of the brain is this problem most likely emanating?

a. Medulla
b. Occipital lobe
c. Temporal lobe
d. Thalamus
e. Midbrain

**478.** If the amygdala is involved with this problem, which two major efferent pathways from this structure may be affected?

a. Corticospinal tract and stria terminalis
b. Mamillothalamic tract and stria terminalis
c. Medial forebrain bundle and stria terminalis
d. Ventral amygdalofugal pathway and stria terminalis
e. Corticospinal tract and mamillothalamic tract

**479.** If the hippocampal formation is involved in this problem, which structures may be damaged?

a. Hippocampus, dentate gyrus, and subiculum
b. Hippocampus, amygdala, and subiculum
c. Hippocampus, fornix, and amygdala
d. Hippocampus, fornix, and habenulae
e. Hippocampus, dentate gyrus, and fornix

**480.** If Lindsey develops this problem with a high frequency, what ongoing problem may she eventually develop?

a. Hemiparesis
b. Diminished memory function
c. Diminished sensation
d. Improved attention
e. Dyslexia

### Questions 481–485

Jane is a 75-year-old woman who has taken medication for high blood pressure and high cholesterol for the past 10 years. One morning, upon awakening, she attempted to get up from her bed, only to find that she had difficulty walking, but didn't know why. When she tried to walk, her left leg collapsed beneath her. Jane couldn't understand why she was having so much difficulty waking, because she felt fine. Thinking that perhaps some-

thing was wrong, she edged her way across the floor to her telephone and promptly called for an ambulance. Jane hadn't noticed until now that her speech was slightly slurred. She was taken to the nearest emergency room for an evaluation. Upon arriving in the emergency room, the staff noted that her face drooped on the left and that she persistently looked to her right side, and called a neurologist to see Jane. The neurologist tested Jane's language functions by asking her to name objects, repeat sentences, and write sentences, and thought that all of these tests were normal. Her speech was mildly slurred, and she had a right gaze preference. She would not cross the midline with her eyes when asked to look to the left, but instead, immediately returned her eyes to their right-sided gaze. When asked to raise her left hand, she raised her right hand. The neurologist asked Jane if her left hand belonged to her and she replied "no, it's yours." When asked to fill in the numbers of a clock, Jane put numbers 1 through 12 on the right side of the clock. When asked to bisect a line, she placed the perpendicular line on the right side. She did not blink to hand waving in the temporal visual field of her left eye, and the nasal visual field of her right eye. Other cranial nerves were normal, except for a left facial droop that spared the forehead. Her left arm and leg were markedly weak, and the muscle tone was flaccid (floppy). All reflexes were depressed on the left side and normal on the right. The neurologist thought that all sensory modalities were depressed on the left side. The neurologist ordered a CT scan of Jane's head, and admitted her to the hospital for further workup and treatment.

**481.** What kind of neurologic deficits does Jane have?

a. Left hemiparesis, hemineglect, left homonymous hemianopsia, left hemisensory loss
b. Left hemiparesis, right superior quadrantanopsia
c. Left hemiparesis, left hemisensory loss, hemineglect, left superior quadrantanopsia
d. Left hemisensory loss, hemineglect, bitemporal hemianopsia
e. Left hemisensory loss, hemineglect, left superior quadrantanopsia

**482.** Where in the nervous system has the damage occurred?

a. Left temporal and parietal lobes
b. Right frontal and temporal lobe
c. Right frontal and parietal lobes
d. Left frontal and parietal lobes
e. Left occipital lobe

**483.** If this damage was caused by a stroke, which artery became occluded?

a. Right anterior cerebral artery
b. Left anterior cerebral artery
c. Right posterior cerebral artery
d. Right middle cerebral artery
e. Left middle cerebral artery

**484.** Damage to which fibers caused Jane's inability to blink in response to the hand waving in her left temporal visual field?

a. Left facial nerve
b. Right oculomotor nerve
c. Left optic nerve
d. Optic chiasm
e. Right optic radiations

**485.** Damage to which specific area caused Jane's inability to notice the left side of her body?

a. Left anterior frontal cortex
b. Right anterior frontal cortex
c. Right posterior frontal cortex
d. Right posterior parietal cortex
e. Right anterior parietal cortex

### Questions 486–490

Morris is a 79-year-old man who was brought to the emergency room (ER) because his family was worried that he suddenly was not using his right arm and leg, and seemed to have a simultaneous behavior change. He was unable to write a reminder note to himself, even with his left hand, and he put his shoes on the wrong feet. A neurologist was called to the ER to examine the patient. A loud bruit (pronounced as *bru-ē;* a rumbling sound) was heard with a stethoscope over the left carotid artery in his neck. When asked to show the neurologist his left hand, he pointed to his right hand, since it could not move. The neurologist asked him to add numbers, and he was unable to do this, despite having spent his life as a bookkeeper. Morris was unable to name the fingers on either hand, and he could not form any semblance of a letter, using his left hand. His eyes did not blink when the neurologist waved his hands close to Morris' eyes in the left temporal and right nasal visual fields. The right lower two-thirds of his face

drooped. There was some asymmetry of his reflexes between the right and left sides, and there was a positive Babinski response of his right toe.

**486.** Where in the CNS is the damage?

a. Right frontal and parietal lobes
b. Left frontal and parietal lobes
c. Right frontal lobe
d. Left frontal lobe
e. Right temporal lobe

**487.** Assuming that Morris had a stroke, which artery has become occluded?

a. Left anterior cerebral
b. Right anterior cerebral
c. Right middle cerebral
d. Left middle cerebral
e. Left posterior cerebral

**488.** Damage to which area of the brain caused Morris' inability to move his right side?

a. Right precentral gyrus
b. Left precentral gyrus
c. Right angular gyrus
d. Left angular gyrus
e. Left supramarginal gyrus

**489.** Damage to which region caused Morris' inability to tell right from left and inability to write, even with his nondominant hand?

a. Left parietal
b. Left frontal
c. Right frontal
d. Left temporal
e. Right temporal

**490.** Damage to which structure caused the visual defect?

a. Right optic nerve
b. Left optic nerve
c. Optic chiasm
d. Right optic radiations
e. Left optic radiations

## Questions 491–494

Bob is a 75-year-old male college graduate who was brought to a neurologist by his family because he was having problems with his gait and suffered from urinary incontinence for the past 6 months, and recently, began to have problems with his short-term memory and paying his bills. The gait problem mainly manifested itself as difficulty in climbing stairs and frequent falls. Bob had no past medical history other than a subarachnoid hemorrhage resulting from a ruptured cerebral aneurysm many years earlier. When the neurologist examined Bob, she found that he could not remember three objects 5 minutes after they were shown to him, even when he was prompted. He was unable to figure out how many quarters were in $1.75, and spelled the word *world* incorrectly. A grasp reflex (squeezing the examiner's hand as a reflex reaction to stroking of the palm) was present. Although his motor strength was full in all of his extremities, when asked to walk, he took many steps in the same place without moving forward, then started to fall. His cranial nerve, sensory, and cerebellar examinations were normal.

**491.** Bob has a grasp reflex and dementia. A lesion in which region can cause this deficit?

a. Occipital lobe
b. Frontal lobe
c. Medulla
d. Thalamus
e. Pons

**492.** You are asked to evaluate Bob with the neurologist. The nurse in the office asks if you would like to order a CT scan, and you request one. The CT scan shows that all the ventricles are dilated, especially the frontal horns of the lateral ventricles, without any evidence of obstruction by a tumor. What would be a possible mechanism underlying the enlargement of the ventricles?

a. Decreased cerebrospinal fluid (CSF) absorption
b. Low blood pressure
c. Decreased central nervous system (CNS) blood flow
d. Decreased intracranial pressure
e. High blood pressure

**493.** If there is diminished CSF absorption, where does the blockage occur?

a. Pyramidal cells
b. Renshaw cells
c. Arachnoid villi
d. Purkinje cells
e. Sagittal sinus

**494.** Where would the greatest damage be done by the expanding ventricles?

a. Thalamus
b. Brainstem
c. Pituitary gland
d. Parietal cortex
e. Deep frontal white matter (corona radiata)

### Questions 495–499

Joe is a 75-year-old man who is right-handed and was told in the past by his internist that he had an irregular heartbeat. Unfortunately, Joe decided that he didn't wish to learn anything further about this condition, so he didn't return to this physician, and it remained untreated. One morning, he awoke to find that his face drooped on the right side, and that he couldn't move his right arm or right leg. When he tried to call an ambulance for help, he had a great deal of difficulty communicating with the operator because his speech was slurred, nonfluent, and missing some pronouns. The call was traced by the police, and an ambulance arrived at his house and brought him to an emergency room. A neurologist was called to see Joe in the emergency room. When he listened to Joe's heart, he detected an irregular heartbeat. It was very difficult to understand Joe's speech, because it was halting, with a tendency to repeat the same phrases over and over. He had a great deal of difficulty repeating specific sentences given to him by the neurologist, but he was able to follow simple commands, such as: "Touch your right ear with your left hand." His mouth drooped on the right when he attempted to smile, but his forehead remained symmetric when he wrinkled it. He couldn't move his right arm at all, but was able to wiggle his right leg a little bit.

**495.** What kind of language problem does Joe have?

a. Dysarthria
b. Wernicke's aphasia
c. Broca's aphasia
d. Alexia
e. Pure word deafness

**496.** Which area of the brain is damaged?

a. Internal capsule and thalamus
b. Right occipital lobe
c. Pontine reticular formation
d. Corpus callosum
e. Left precentral gyrus and Broca's area

**497.** Which artery was blocked when the event occurred?

a. Anterior cerebral artery
b. Posterior cerebral artery
c. Anterior inferior cerebellar artery
d. Middle cerebral artery
e. Basilar artery

**498.** Which term best describes Joe's facial weakness?

a. Peripheral nerve VII
b. Central nerve VII
c. Nerve XII
d. Nerve V
e. Oculomotor nerve weakness

**499.** With which hand does Joe most likely write?

a. Right
b. Left
c. Ambidextrous (both)
d. Cannot be determined

**500.** A 68-year-old man goes to a sleep clinic after he has repeated episodes of loud snoring during sleep coupled with sudden periods of restlessness and cessation of breathing. After extensive analysis, the physicians concluded that the patient's problem was not a result of obstructive sleep. Instead, it was judged that this condition reflected central sleep apnea due to loss of chemoreceptor sensitivity of the neuronal control mechanisms governing respiration. The most likely site within the CNS that is most closely associated with these effects is the

a. Dorsal horn of the thoracic spinal cord
b. Reticular formation of the medulla
c. Midbrain periaqueductal gray
d. Hippocampal formation
e. Border of occipital and parietal lobes

# Higher Functions

## *Answers*

**430–431. The answers are 430-d, 431-b.** *(Afifi, pp 235–252, 266–271; Simon et al., p 203.)* The infarct caused damage to posterior thalamic nuclei. When these structures are damaged, a disorder referred to as *thalamic pain* can ensue. In this condition, light cutaneous stimulation is sufficient to produce severe pain. The projections from nuclei situated in this region project principally to the parietal and occipital lobes and play a role in the regulation of pain (although the precise mechanisms remain unknown). The other processes offered as alternate choices have not been shown to be related to functions of the posterior thalamus.

**432–434. The answers are 432-a, 433-b, 434-a.** *(Afifi, pp 235–252, 266–271.)* Damage to the VA, VL, dorsomedial, and anterior thalamic nuclei would most likely result in motor impairment such as a hemiparesis (because of the connections of these nuclei with the motor and premotor cortices). Damage to the dorsomedial nucleus could also be linked with neuropsychological impairment, because of its connections with the prefrontal cortex and adjoining regions of the frontal lobe. The other processes mentioned in question 432 have not been shown to be related to these groups of nuclei. As noted earlier, the VA nucleus is associated with motor functions, not only in its projections to motor regions of the cerebral cortex—the premotor and prefrontal cortices—but also in the inputs that it receives from structures associated with motor functions such as the globus pallidus and substantia nigra.

**435–437. The answers are 435-c, 436-c, 437-c.** *(Afifi, pp 235–252, 266–271.)* An infarct that affects the medial thalamus, which includes the dorsomedial nucleus, and midline thalamic and intralaminar nuclei, can result in abnormalities in memory, attention, and drowsiness. The other choices offered for question 435 have not been shown to be related to functions associated with medial thalamic structures. A key input into the medial thalamus is the reticular formation. In this manner, the medial and intralaminar thalamus represent a relay from reticular formation to the cerebral cortex. Since a major function of the reticular function is to regu-

late states of sleep and wakefulness, these thalamic nuclei thus contribute to these states. When these nuclei are damaged, this mechanism is affected, resulting in drowsiness. Because of the connections of the medial thalamus with much of the frontal lobe, including the prefrontal cortex, damage to the medial thalamus would also affect the functions of these cortical regions, which involve memory and other cognitive processes.

**438. The answer is b.** *(Afifi, p 250.)* The superior temporal gyrus is the primary auditory receiving area in the cerebral cortex. Accordingly, the primary afferent source to this region arises from the medial geniculate nucleus, which constitutes a specific thalamic relay for processing of auditory information.

**439. The answer is f.** *(Afifi, pp 242–245, 249–251; Kandel, pp 341–345.)* The VA nucleus has properties of both the specific and nonspecific thalamus. By this, it is meant that, on the one hand, the VA nucleus receives motor inputs from the basal ganglia, including the substantia nigra, and projects its axons to an important motor region of the brain—the premotor cortex. In this context, the VA nucleus functions as a relay nucleus for the transmission of information associated with motor functions. On the other hand, the VA nucleus also receives various inputs from other thalamic nuclei (such as the centromedian nucleus) and projects its axons in a widespread manner to other parts of the frontal lobe, including the prefrontal cortex. In this context, the VA nucleus can modulate a wide variety of neurons in the cerebral cortex, both directly and indirectly, which is characteristic of nonspecific thalamic nuclei.

**440. The answer is a.** *(Afifi, pp 242–245, 249–251; Kandel, pp 341–345.)* The centromedian nucleus is a classical nonspecific thalamic nucleus. It can modulate cortical activity by making local connections with specific thalamic nuclei, and therefore modify the specific thalamic inputs to different regions of the cerebral cortex. In addition, the centromedian nucleus also projects to the putamen. This projection is sometimes referred to as the *thalamostriatal projection.* Since the centromedian nucleus receives considerable inputs from the cerebral cortex, this connection to the putamen provides a basis by which the cerebral cortex can influence the basal ganglia in addition to its direct projections to the neostriatum.

**441. The answer is c.** *(Kandel, pp 937–940.)* REM sleep is characterized by a low-voltage EEG pattern typical of an alert person. For this reason, REM sleep is sometimes referred to as *paradoxical sleep.* Other EEG patterns, such as slow waves, high-voltage EEGs, and sleep spindles, occur at other stages of sleep. In addition, REM sleep is characterized by a general loss of skeletal muscle tone with the exception of the eye muscles, which govern the REMs.

**442. The answer is c.** *(Kandel, pp 940–943.)* Animal research studies have indicated that the region of the rostral pons bordering on the caudal midbrain contains special sets of cholinergic neurons that are maximally active during REM sleep (and during wakefulness, as well). These neurons are located in the reticular formation. One group of neurons has been identified as the nucleus reticularis pontis oralis. Other neurons include the region of the pedunculopontine nucleus. It appears that these cholinergic neurons depolarize GABAergic neurons, which prevent rhythmic firing of reticular formation neurons. This latter effect allows for the asynchronous firing of thalamocortical neurons that take place during periods of wakefulness and REM sleep.

**443. The answer is a.** *(Kandel, pp 916–917.)* During states of quiet wakefulness or drowsiness, the EEG pattern becomes slower (8 to 13 Hz; average amplitude of 50 V) than what is seen during an alert state. This pattern is called an $\alpha$ *wave.*

**444. The answer is b.** *(Kandel, pp 910–918; Simon, pp 47, 258.)* This person displays a complex partial seizure, which is characterized by a confusional state with brief losses of consciousness. It is called a partial seizure because the seizure involves a localized region, reflected by jerks of the muscles of a specific part of the body. The focus of this seizure is typically in the temporal lobe, such as the amygdala, hippocampal formation, or adjoining cortical regions. A simple partial seizure does not involve loss of consciousness. Absence seizures are nonconvulsive seizures and are also called *petit mal seizures.* Generalized seizures typically involve all of the limbs. The patient falls to the ground and loses consciousness.

**445. The answer is a.** *(Kandel, pp 910–918; Simon, pp 47, 258.)* The seizure described in this patient has progressed from a complex partial

seizure to a generalized seizure. As indicated previously, this type of seizure involves all of the limbs. The patient falls to the ground and typically loses consciousness. As stated in the answer to question 444, the other choices involve seizures that are characterized differently than what was described in the progression of this case.

**446. The answer is c.** *(Kandel, p 979.)* Certain magnocellular neurons of the hypothalamus synthesize the hormones vasopressin and oxytocin. These include the paraventricular and supraoptic nuclei. The cell bodies of the magnocellular neurons that produce vasopressin are found mostly within the supraoptic nucleus. Vasopressin is important because it makes the membranes of the convoluted tubules and collecting ducts of the kidneys more permeable to water. This results in water conservation.

**447. The answer is e.** *(Kandel, pp 1000–1002.)* The process of temperature regulation requires the integration of autonomic, skeletomuscular, and endocrine responses. For example, dilation of blood vessels of the skin (an autonomic response) facilitates heat loss while constriction of these vessels helps to conserve heat. Panting and shivering (skeletomuscular responses) aid in the processes of heat loss and conservation (heat generation), respectively. Finally, when an organism is exposed to cold for long periods of time, there is an increase in thyroxine release from the anterior pituitary gland, which helps to increase body temperature by increasing metabolism. The classic interpretation of the role of the hypothalamus in temperature regulation has been that the anterior hypothalamus constitutes a heat loss center, while the posterior hypothalamus is a heat conservation center. Although such a generalization is somewhat oversimplified, the general phenomenon has been demonstrated. For example, stimulation of the anterior hypothalamus has been shown to dilate blood vessels and inhibit shivering, and lesions of this region produce hypothermia. Stimulation of the posterior hypothalamus produces heat conservation by constricting blood vessels and causing shivering. Neurons in this region respond to both local warming of hypothalamic tissue as well as to warming of the skin. Neurons in both the septal and preoptic regions constitute antipyretic areas, in that they respond to increases in fever by limiting the magnitude of the fever. Activation of these antipyretic regions is thought to occur through a mechanism utilizing the peptide vasopressin. The precise mechanism by which these regions become activated remains unknown.

**448. The answer is e.** *(Kandel, p 979.)* The supraoptic nucleus, like the paraventricular nucleus, contains magnocellular neurons that synthesize vasopressin and oxytocin and transport these hormones down their axons to the posterior pituitary. For this reason, the supraoptic nucleus plays a significant role in the regulation of water balance. There is no evidence to support the notion that the supraoptic nucleus has a role in feeding behavior, temperature regulation, sexual behavior, or short-term memory functions.

**449. The answer is d.** *(Kandel, pp 1002–1003.)* Lesions of the lateral hypothalamus are likely to produce aphagia. Feeding behavior is elicited by stimulation of the lateral hypothalamus. Neurons in this region respond to the sight or taste of food. Since drinking is also associated with lateral hypothalamic functions, a lesion of this structure would also disrupt this behavior. Lesions of the lateral hypothalamus do not produce either hypertension or sexual behaviors. The neurons regulating these functions are elsewhere within the hypothalamus.

**450. The answer is d.** *(Kandel, pp 45, 322, 937, 946.)* Recent studies have demonstrated that the suprachiasmatic nucleus controls the biologic clock of internal circadian rhythms. During the light phase of the light-dark cycle, metabolic activity (measured by $^{14}$C-2-deoxyglucose autoradiography) within the suprachiasmatic nucleus is significantly increased. In contrast, during the dark phase, there is very little metabolic activity.

**451. The answer is b.** *(Kandel, p 988.)* In this syndrome, produced experimentally in monkeys and also seen in cats, there is an extreme change in the personality of the animal. Its responses to emotion-laden stimuli are much reduced. It appears very tame. Aggressive tendencies are not evident. It also manifests oral tendencies and displays hypersexuality. This syndrome is the result of lesions of the temporal lobe in which parts of the amygdala are involved. Lesions of other regions such as the hypothalamus, cingulate cortex, or septal area do not produce the Klüver-Bucy syndrome.

**452. The answer is c.** *(Cooper, pp 275–276, 295; Nolte, pp 556–561.)* One of the most interesting discoveries concerning the amygdala made in recent years is that the central nucleus contains high concentrations of a number

of peptides. These include enkephalins and somatostatin, in particular. This region also receives large numbers of dopaminergic axon terminals. The central nucleus does not project its axons to the medial hypothalamus. It does not receive auditory or visual signals, nor does it project to the spinal cord, where it could inhibit spinal motor neurons. It receives norepinephrine-containing fibers from the brainstem rather than being a source of this neurotransmitter.

**453. The answer is b.** *(Kandel, pp 1200–1204.)* There have been a variety of neurochemical and related theories of schizophrenia that have evolved over the past 3 decades. Unfortunately, each of these theories has had its limitations. Nevertheless, one of the more popular theories has been that schizophrenia is linked to increased levels of brain dopamine. The hypothesis suggests that schizophrenia results from overstimulation of the brain by the dopaminergic system. Support for this view comes from the observation that antipsychotic agents are known to block dopamine receptors. Cotwin behavioral and developmental studies have shown that, while environmental factors are certainly important in the ontogeny of schizophrenia, genetic factors are also quite significant in the development of this disease. Other researchers have suggested that schizophrenia may bear some relationship to decreased levels of serotonin in the brain as evidenced by the hallucinogenic effects of LSD, which binds to serotonin receptors. Other investigations have shown that opioid peptide blockade by naloxone is effective in reducing hallucinations, which suggests that increased levels of endorphins may be linked to this disorder. Investigations involving neuropeptides have indicated that neuropeptides such as cholecystokinin (CCK) is colocalized with dopamine in brain neurons. In this fashion, CCK may function as a neuromodulator for dopamine, in which case increased levels of CCK may be linked with schizophrenia in the same fashion as are increased levels of dopamine.

**454. The answer is d.** *(Nolte, pp 548–560.)* A major target of efferent fibers from the hippocampal formation is the septal area. Fibers located in the precommissural fornix supply the septal area in an extensive and topographical manner. In turn, the septal area projects significant numbers of fibers to the lateral (and medial) regions of the hypothalamus. In this manner, the septal area serves as a relay for the transmission of signals from the hippocampal formation to the hypothalamus. The hippocampal formation

does not project to the habenular nuclei, mediodorsal nucleus, or the bed nucleus of the stria terminalis. Moreover, the cingulate gyrus does not project directly to the hypothalamus.

**455. The answer is c.** *(Kandel, pp 987–988; Nolte, p 554.)* For many years, it was believed that a neural circuit composed of the hippocampal formation → mamillary bodies → anterior thalamic nucleus → cingulate gyrus → hippocampal formation played a major role in the regulation of emotional behavior. More recent studies by a number of investigators have revealed that neither the mamillary bodies nor the anterior thalamic nucleus appears to contribute to the regulation of emotional behavior. Instead, it is believed that this circuit may subserve functions more closely related to short-term memory.

**456. The answer is b.** *(Kandel, pp 544, 1306–1309; Nolte, pp 418–424.)* The cerebrovascular accident produced damage of the right primary visual cortex. Therefore, this would result in a homonymous hemianopsia of the left visual fields. Since the damage was confined to the occipital lobe, there would be little effect upon other processes such as speech, motor functions, or intellectual activities.

**457. The answer is c.** *(Kandel, pp 544, 1306–1309; Nolte, pp 120–133.)* The occipital lobe is supplied by the posterior cerebral artery. The calcarine cortex (primary visual cortex) is supplied by a branch of this artery, the calcarine artery. The anterior cerebral artery supplies the medial aspect of the frontal lobe and the anterior-medial aspect of the parietal lobe. The middle cerebral artery supplies the lateral aspect of the frontal and parietal lobes. The superior cerebellar artery supplies the dorsolateral aspect of a portion of the pons and the cerebellum. The striate arteries arise from the anterior and middle cerebral arteries and supply portions of the internal capsule and neostriatum.

**458. The answer is a.** *(Kandel, pp 854–857; Nolte, pp 390–393, 451–467.)* The tumor is situated in the lentiform nucleus and internal capsule. Therefore, corticospinal fibers will be affected, causing a UMN paralysis of the left side. Dyskinesia would not be seen because any effects normally seen in association with damage to the basal ganglia would be masked by the

effects of the damage to the internal capsule. Since the cerebellum was not involved, there would be no intention tremor. Neither would there be any visual deficits from this glioma since optic nerve fibers are not involved. The following schematic diagram indicates the approximate extent of the tumor. Labeled are the caudate nucleus (C), the globus pallidus (GP), the internal capsule (IC), the putamen (P), and the tumor (T).

**459. The answer is d.** *(Kandel, pp 854–857; Nolte, pp 390–393, 451–467.)* The tumor clearly involves the lentiform nucleus of the basal ganglia and has expanded to include the internal capsule as well. At the stage when the CT scan was taken, the tumor had not involved the thalamus.

**460. The answer is c.** *(Nolte, pp 418–423, 515–529.)* The arterial occlusion involves both the temporal and occipital regions of cortex. Therefore, it would affect Wernicke's area as well as primary visual areas of the occipital lobe. The patient would most likely present with receptive aphasia as well as a right homonymous hemianopsia. The lesion would not likely produce marked intellectual deficits since the prefrontal cortex was spared; nor would it produce hemiballism since there was no damage to the subthalamic nucleus.

**461. The answer is b.** *(Kandel, pp 1305–1309; Nolte, pp 120–128.)* Although the tissue affected involves parietal, temporal, and occipital lobes, the primary artery affected is the middle cerebral artery. The unusual feature of this occlusion is that it appears that the middle cerebral artery extends more caudally than usual. Nevertheless, the middle cerebral artery is the only one of the choices presented that could account for the damage to the temporal and parietal cortices. The anterior cerebral artery supplies the medial aspects of the frontal and parietal lobes; the posterior cerebral artery supplies the occipital cortex (visual areas); the posterior choroidal artery mainly supplies part of the tectum, the medial and superior aspects of the thalamus, and the choroid plexus of the third ventricle. The superior cerebellar artery supplies the dorsolateral aspect of a portion of the pons and the cerebellum.

**462. The answer is b.** *(Kandel, pp 1305–1309; Nolte, pp 120–128, 421–425.)* An arterial occlusion compromised the blood supply to the

occipital lobe on the left side of the brain. Therefore, it would result in a right homonymous hemianopsia with no motor deficits (since no motor regions of the brain are affected).

**463. The answer is e.** *(Kandel, pp 1303–1311; Nolte, pp 117–128, 131–135.)* This vertebral angiogram is an anterior view of the back of the brain. It reveals an occlusion of the left posterior cerebral artery (A). It should be noted that the posterior cerebral arteries are formed from the bifurcation of the basilar artery. Follow the basilar artery caudally (see the bottom of the photograph) to the position where it is connected to the vertebral arteries.

**464. The answer is b.** *(Kandel, p 544; Nolte, pp 422–424.)* This large pituitary tumor is seen to compress the optic chiasm. Damage to the chiasm affects the crossing fibers of the nasal retina, which convey information from the temporal visual fields. This results in a bitemporal hemianopsia. Since some parts of the optic nerves are spared, pupillary reflexes are preserved. The neuroanatomic substrates for conjugate gaze (i.e., frontal eye fields; pontine gaze center; medial longitudinal fasciculus; and nuclei of cranial nerves III, IV, and VI) are unaffected by the tumor; the mechanism of conjugate gaze remains intact.

**465. The answer is a.** *(Kandel, pp 388, 759, 920.)* June had a seizure, which began focally on the left motor strip (the left precentral gyrus), moved up the motor strip, then secondarily generalized, or spread throughout the cortex. The phenomenon whereby there is twitching of an extremity that spreads to other areas on that extremity or other areas of the body is called a *Jacksonian march.* This phenomenon is named for Hughlings Jackson, a neurosurgeon who was instrumental in mapping out the cerebral cortex and describing the somatotopic organization of the cortex of the prefrontal gyrus called a *homunculus* (meaning *little man*). Observing patients with a Jacksonian march helped him to identify areas represented at each location of the motor strip.

**466. The answer is c.** *(Kandel, pp 919–927.)* Very often, there is inhibition following a seizure, which accounts for drowsiness or a postictal state after the seizure has finished. Sometimes, epileptic discharges spread to other areas of the cortex, recruiting contiguous areas of the cortex through

callosal, commissural, and sometimes thalamic circuits to eventually involve a large area of the cortex, causing the movements of the entire body. This occurs with a generalized seizure. If the cortices of both hemispheres become involved, there may be impairment or loss of consciousness. The cells (often pyramidal cells) in the cortex can generate a seizure through high-frequency, synchronous discharges in large groups. If the seizure begins focally, as this one did, there may be a *Todd's paralysis,* as June had, where there is transient paralysis of the involved motor area during the postictal period.

**467. The answer is e.** *(Kandel, pp 759, 919–927.)* There is somatotopic organization of the motor strip, and cortical neurons are included among the most likely to generate seizures, making this area the most likely to cause such a pattern.

**468. The answer is e.** *(Kandel, pp 919–927.)* The pyramidal cell is a cell in the cortex that uses glutamate, an excitatory neurotransmitter, whereas most other types of cortical neurons use GABA, an inhibitory neurotransmitter. The spike, one identifying feature of an epileptic seizure seen on an EEG recorded on the scalp, is initiated by a depolarization shift, which is thought to be generated by EPSPs.

**469. The answer is d.** *(Kandel, pp 925–927.)* Excitatory postsynaptic potentials are considered to be an initiating cellular event for a seizure. To become a seizure, however, the cellular discharges require enhancement and synchronization.

**470. The answer is b.** *(Kandel, pp 922–925.)* Since seizure generation requires excitation, or a loss of inhibition, the only correct choice is the inhibition of GABA, an inhibitory neurotransmitter. All the other choices cause inhibition only. Many new anticonvulsant medications are currently being designed to either enhance GABA activity, or inhibit the excitatory neurotransmitter, glutamate.

**471. The answer is c.** *(Kandel, pp 758–765.)* A CT scan of Helen's head was done in the emergency room, which showed a new infarct or stroke in the genu and anterior portion of the posterior limb of the left internal capsule. This is the region of the internal capsule through which most of the

fibers of the corticospinal and corticobulbar tracts pass in a somatotopically organized fashion before entering the brainstem. Because most of these fibers pass through a very small region, a small infarct can cause deficits in a wide distribution of areas. In this case, Helen has weakness in her face and tongue, causing her slurred speech, in addition to weakness of her arm and leg. In addition, since somatosensory fibers destined for the postcentral gyrus occupy a position in the internal capsule caudal to the corticospinal tract fibers, these fibers are spared and Helen has no sensory deficits. The only other area in the CNS that can cause a pure motor hemiparesis is the basilar pons, an area through which corticospinal and corticobulbar fibers also run. The vascular supply of this region consists of perforators from the basilar artery, which are small and subject to atherosclerotic disease.

**472. The answer is a.** *(Kandel, pp 1303–1307.)* The internal capsule is supplied primarily by the lenticulostriate branches of the middle cerebral artery. In addition, portions of the posterior limb of the internal capsule are supplied by the anterior choroidal artery, a branch of the internal carotid artery. Both the lateral striate branches and the anterior choroidal artery are small branches of larger arteries, and are more susceptible to damage (atherosclerosis) from high blood pressure and diabetes than the larger vessels.

**473. The answer is c.** *(Kandel, pp 757–763.)* The corticospinal and corticobulbar tracts contain motor fibers originating in the precentral gyrus, mediating voluntary motor function of the face, arms, legs, and trunk. They pass through the internal capsule to the crus cerebri in the midbrain. The spinothalamic tract is a sensory tract, and could not cause the observed deficits. The rubrospinal tract only affects the spinal cord.

**474. The answer is c.** *(Afifi, p 349.)* Helen's forehead is unaffected by the lesion because the forehead is bilaterally represented on the cortex, so the right side retains innervation despite a lesion in the left internal capsule. Motor fibers from each side pass into the internal capsule ipsilaterally, so a lesion in the internal capsule will not affect the forehead. This type of finding is called a *central seventh nerve lesion,* because it represents a lesion in the CNS superior to the level of the seventh nerve nucleus, where the fibers from both sides of the forehead coalesce.

**475. The answer is d.** *(Adams, p 1383.)* Dysarthria is slurred speech, occurring from lesions affecting innervation of the tongue, lips, and palate. We are given evidence that her tongue is weak in that her tongue points to the right. The interruption of fibers traveling to the hypoglossal nerve from the left side eventually innervates the right genioglossus muscle, which pulls the tongue to the left. Dysarthria is a motor phenomenon, unlike aphasia, which is a disruption of language. Language is primarily generated in the cerebral cortex; therefore, because the lesion spares the cortex, there were no signs of aphasia.

**476. The answer is d.** *(Adams, pp 321–322.)* This is an example of a complex partial seizure, most likely originating in the temporal lobe. A seizure is a paroxysmal derangment of the CNS due to rhythmic, synchronous discharges from cerebral neurons, causing changes in consciousness, sensation, and/or behavior. Complex partial seizures often start with a warning, or "aura." Since limbic structures are often involved, the seizure can include emotions, feelings of deja vu or jamais vu, or gastrointestinal sensations. Because olfactory pathways end in the temporal lobe, patients may experience smells as well. The seizure, itself, involves impairment of consciousness of some form, often manifested as staring, in addition to various stereotyped, automatic behaviors called *automatisms.* The latter may be manifested as chewing, repetitive swallowing, hand gestures, or vocalizations. These usually occur during the seizure, but may occur after it. After the seizure ends (the seizures usually last 1 to 2 minutes), the patient is often in a confused or postictal state for several minutes, or even up to several hours. Occasionally, a patient may manifest aggressive behavior while in the postictal state. Unless a structural lesion, such as a tumor, is present, the physical examination is usually normal. Verification of the diagnosis of epilepsy is done with the help of an EEG, which records potential differences of summed cortical action potentials over the scalp of a patient. Often, an epileptic spike, or sharp wave, is seen over the area from which the seizures arise. Epilepsy patients usually also have a CT scan or MRI to make certain that there is no structural lesion causing the seizures.

**477. The answer is c.** *(Adams, pp 321–322.)* Seizures similar to this one often begin with abnormal neuronal discharges in temporal lobe structures, which include the amygdala or hippocampus. These structures tend

to have a lower threshold for this type of activity than other structures in the brain.

**478. The answer is d.** *(Afifi, pp 435–438.)* The major descending pathways from the amygdala are the stria terminalis and the ventral amygdalofugal pathway. The medial forebrain bundle is a major pathway of the lateral hypothalamus. The mamillothalamic and corticospinal tracts do not involve the amygdala.

**479. The answer is a.** *(Afifi, pp 425–443.)* The hippocampal formation includes the hippocampus, the dentate gyrus, and the subiculum. All of the other structures listed are within the limbic system, but do not lie within the hippocampal formation.

**480. The answer is b.** *(Kandel, pp 988–992, 1228–1237.)* Since memory is a function that is mediated by the limbic system, a structure most likely involved in the generation of these seizures, it is possible that Lindsey will have memory problems in the future if she has frequent seizures. Early studies of patients who have undergone resection of portions of one or both temporal lobes have demonstrated the presence of memory deficits.

**481. The answer is a.** *(Kandel, pp 1306–1309; Adams, pp 456–457.)* Jane is not only unable to move her left side (hemiparesis), but ignores its existence (anosagnosia or the syndrome of hemineglect, see below). Even though she neglects her left side, the blink reflex should still be intact if she only neglects the side. Therefore, a visual field deficit, called a *homonymous hemianopsia,* is present on the left side, in which the left temporal and right nasal fields are damaged. There may also be some degree of primary sensory loss, which can be difficult to evaluate when a patient neglects the same side.

**482. The answer is c.** *(Adams, pp 443–446, 456–457.)* Jane's deficits result from lesions of the posterior frontal cortex, as well as from some contribution of corticospinal tract fibers to the parietal lobe and deeper motor cortical structures. In addition, the neglect and hemisensory loss result from damage to the parietal cortex. The homonymous hemianopsia results from damage to the deep portion of the parietal lobe where the optic radiations pass to the superior and inferior banks of the visual cortex, causing the visual field defect.

**483. The answer is d.** *(Kandel, pp 1303–1307.)* The posterior frontal lobe, as well as the parietal lobe, are supplied by the middle cerebral artery. Areas supplied by this artery, such as primary and supplementary motor areas, and the primary and secondary somatosensory cortices may be affected. As a result, the patient may have left-sided weakness and UMN facial weakness that spares the forehead, and hemisensory loss.

**484. The answer is e.** *(Kandel, pp 544–545, 1303–1307.)* If the lesion is deep enough, the patient may have a visual field cut, called a *homonymous hemianopsia,* where fibers traveling from the optic chiasm to the occipital cortex within the optic radiations are interrupted, and the patient doesn't see the left temporal and the right nasal visual field. It is common for patients with neglect not to notice the areas of blindness because they ignore the left side. Patients with this problem are usually advised not to drive a car.

**485. The answer is d.** *(Kandel, pp 1303–1307; Adams, pp 456–457.)* Jane's problem is an example of the syndrome of hemineglect, which arises from a lesion of the posterior parietal lobe. This area is essential for spatial organization. If this area, usually on the nondominant (right) side, is no longer functioning, the patient will live in a world that consists solely of a right side. Patients with the syndrome of hemineglect will look only to the right side (if the lesion is on the right), and when asked to look to the left, often will not cross the midline with their eyes. Especially when the lesion is acute, these patients will not acknowledge any person or objects on their left side, and it is not unusual for a patient to complain of losing her glasses when they are on a table on the left side. Since these patients see only the right side of everything, they will put all of the numbers of a clock on the right side of the clock, and will bisect a line on its right side. In addition, they will only comb the right side of their hair, dress the right side of their bodies, and shave the right side of their faces. When confronted with a left-sided entity, such as a left arm, they will often ignore the question, or may even go as far as claiming it belongs to someone else. In resolving lesions where the patient now has sensation and acknowledgment on the left side, she may still display extinction to double simultaneous stimuli where, if both sides are touched simultaneously, the patient feels the touch only on the right side and "extinguishes" the stimulus on the left. However, it is important to remember that neglect can resemble weakness because the patient won't move the left side.

**486. The answer is b.** *(Adams, pp 443–445, 453–459.)* This case is an example of a lesion of the left (usually dominant) parietal lobe, most often in the angular gyrus, with some involvement of the precentral gyrus in the posterior frontal lobe. There is contralateral UMN weakness (with a positive Babinski sign), as well as several cortical sensory defects—specifically, right-left confusion, agraphia (inability to write, independent of motor weakness), acalculia (the inability to calculate), and finger agnosia (the inability to designate the fingers). The latter four elements are sometimes referred to as the *Gerstmann syndrome* by neurologists, and all represent spatial discriminatory functions of the parietal lobe (often the dominant parietal lobe, which is usually the left). The parietal lobe also subserves other visual-spatial functions such as construction of complex drawings. There are other locations within the CNS where UMN weakness can occur; however, the combination with parietal lobe signs can only occur in this location. If the damage was slightly more extensive, it may have involved Broca's area, causing aphasia.

**487. The answer is d.** *(Kandel, pp 1303–1309.)* The artery serving this region (both posterior frontal and parietal lobes) is the right middle cerebral artery, which originates at Willis's circle. Because it continues in a nearly straight line from the internal carotid artery, it is a common route for small emboli formed from blood clots in the internal carotid artery. The bruit noted over the right common carotid artery in this patient is most likely a result of a thrombus (clot) that occludes part of the lumen of the artery. These emboli can occlude the middle cerebral artery because it is considerably smaller than the internal carotid artery. Since the middle cerebral artery has many branches through which an embolus may travel, but the territory of this stroke is large, it is likely that the embolus lodged in a more proximal location in this case.

**488. The answer is b.** *(Kandel, pp 1303–1309.)* Morris' leg weakness includes a positive Babinski sign, which is a UMN sign. Although this type of weakness may occur in several locations in the CNS, the combination with the cortical parietal signs can only occur in the left precentral gyrus if there is to be one lesion.

**489. The answer is a.** *(Adams, pp 443–445, 453–459.)* These deficits are visual-spatial in nature, and are characteristic of damage to the dominant parietal lobe.

**490. The answer is e.** *(Kandel, pp 1303–1309.)* The visual defect that Morris experiences is a homonymous hemianopsia, resulting from damage to the optic radiations traveling from the lateral geniculate nucleus to the visual cortex in the occipital lobe. These split so that inferior images are carried through the parietal lobe and superior images through the temporal lobes, but in large infarcts, the defect is more likely to involve more fibers of this tract. Since the optic radiations carry representations of the ipsilateral temporal field and the contralateral nasal field (only the nasal field fibers cross), this defect is noted clinically as the inability to detect objects in the regions described. Often, the patient will only notice bumping into objects on the side ipsilateral to the stroke, since turning of the eyes can compensate for the nasal field defect.

**491. The answer is b.** *(Rowland, pp 277–293.)* This case is an example of a condition called *normal-pressure hydrocephalus.* This may be caused by various nonprogressive meningeal and ependymal diseases, such as chronic meningitis and subarachnoid hemorrhages, which can initially block CSF absorption. Initially, the CSF pressure is high, which results in the enlargement of the ventricles. The CSF pressure becomes normal because the CSF absorption begins again. However, the enlarged ventricles, despite normal CSF pressure, cause hydrostatic impairment to the central white matter surrounding the ventricles. Maximal ventricular expansion is usually located in the frontal lobes with preservation of the cortical gray matter and other subcortical structures. As a result, patients with this condition have diminished frontal lobe functions, namely, gait problems without any weakness, as well as urinary incontinence and dementia. Frontal lobe dysfunction can also cause the reappearance of primitive reflexes, which disappear shortly after birth, such as the grasp reflex. Late in the course of normal-pressure hydrocephalus, the patient may develop *frontal lobe incontinence,* where he or she becomes indifferent to the incontinence, much like a very small child. Headaches are rare in this particular type of hydrocephalus. Normal-pressure hydrocephalus is usually diagnosed with a thorough neurological examination, in addition to a head CT, which shows enlarged ventricles and, occasionally, interstitial fluid within the white matter adjacent to the lateral ventricles. [Measurement of CSF pressures with a lumbar puncture and radionuclide cisternography (a procedure where a radionuclide is injected intrathecally, and its distribution is observed over a period of 24 hours) is also helpful.] Occasionally, shunting

procedures, which allow the CSF to drain into the peritoneal cavity or the blood, are helpful if performed early in the course of this condition.

**492. The answer is c.** *(Rowland, pp 277–293.)* The major mechanism underlying hydrocephalus is decreased absorption of CSF. In the case of normal-pressure hydrocephalus, the problem is described in the answer for question 491. Another cause of decreased absorption is obstruction of CSF flow by a tumor. Low blood pressure does not cause enlarged ventricles. High blood pressure only causes hydrocephalus as a result of hypertensive crisis, but not chronically. Decreased blood flow in the brain can actually be used as a temporizing measure to acutely decrease intracranial pressure in emergencies, in order to make room for expanding tissue through the mechanism of decreasing $P_{CO_2}$ in the brain with a ventilator.

**493. The answer is c.** *(Rowland, pp 277–293.)* The major location for reabsorption of CSF are the arachnoid villi within the ventricular system. In the case of this particular patient, there is a history of a subarachnoid hemorrhage, which may have caused obstruction within this area.

**494. The answer is e.** *(Rowland, pp 277–293.)* The frontal horns of the lateral ventricles are the area of greatest expansion; thus, the expansion would affect the adjoining white matter of the frontal lobe. The other areas listed are subcortical and gray matter areas, which are located further from the expanding frontal horns, and are less affected. The pituitary gland is quite distant from the frontal horns, as well.

**495. The answer is c.** *(Gilroy, pp 6–9; Rowland, pp 7–10.)* The language problem is an example of Broca's aphasia, a deficit seen with lesions of Broca's area and manifested by defects in the motor aspect of speech, leaving the patient's speech halting and nonfluent. People with Broca's aphasia tend to repeat certain phrases, as well as leave out pronouns. Since the language centers are usually located on the dominant side of the brain (the left side for a right-handed person), this lesion must be on the left side of Joe's brain. Wernicke's aphasia is a problem with the sensory aspect of speech, where the patient can speak fluently, but the speech sounds like gibberish. The area of disruption in this type of aphasia is usually in Wernicke's area, a region of the posterior superior temporal lobe. Dysarthria is slurred speech, but makes grammatical sense. Alexia is the inability to read. Pure-

word deafness is a type of sensory aphasia where language, reading, and writing are only mildly disturbed, but auditory comprehension of words is very abnormal. This arises from lesions of the posterior temporal lobe.

**496. The answer is e.** *(Gilroy, pp 225–230; Rowland, pp 7–10.)* Joe's condition is an example of a left inferior frontal lobe cortical stroke, including the region of Broca's area and the left precentral gyrus. The weakness on his right side confirms this, since the left side of the brain controls the right side of the body. The right leg is most likely less involved than the arm because the leg area of the precentral gyrus extends onto the medial aspect of the frontal lobe, an area served by a different artery than that serving the arm and face areas. The internal capsule contains motor fibers traveling to the cortex, but usually does not involve language. The thalamus contains many sensory, motor, and association areas, but only rarely causes language problems. Functions of the pontine reticular formation do not include language. The corpus callosum is a white matter structure that connects the hemispheres. Lesions of the posterior aspect may cause language problems, such as alexia without agraphia (the ability to write, but not to read), but would not cause both an aphasia as well as weakness.

**497. The answer is d.** *(Gilroy, pp 225–230, 235–239; Rowland, pp 7–10, 61–62.)* The middle cerebral artery subserves the precentral gyrus, the area which has been damaged. The damage can be more widespread, depending upon which portion of the vessel becomes occluded. The anterior cerebral artery supplies the orbitofrontal cortex, deep limbic structures, as well as the cingulate gyrus. The posterior cerebral artery supplies the thalamus, portions of the temporal lobes, and portions of the midbrain. The anterior inferior cerebellar artery supplies the lateral inferior pons and portions of the cerebellum. Perforating branches of the basilar artery supply medial portions of the brainstem. The irregular heartbeat observed in this case is an example of aerial fibrillation, a heart rhythm that is often recognized by being "irregularly irregular". This rhythm can cause strokes by throwing small blood clots or emboli from the heart to the cerebral blood vessels and occluding them.

**498. The answer is b.** *(Rowland, pp 7–11, 440–442.)* Joe's forehead doesn't droop like the rest of his face because this region receives innervation from both sides of the cerebral cortex, giving this area a backup in case

of damage. This can only occur when the lesion is above the level of nerve VII, where both sides no longer contribute to the innervation of the face. This type of weakness is called a *central nerve VII lesion,* because it occurs within the CNS. A *peripheral nerve VII lesion* is a lesion within the nerve VII nucleus, or distal. This type of lesion always involves the forehead in addition to the rest of the face. Nerve XII innervates the tongue, nerve V innervates sensation of the face, in addition to the muscles of mastication, but not the muscles of facial expression. The oculomotor nerve innervates four of the muscles that move the eyes.

**499. The answer is a.** *(Gilroy, pp 6–9; Rowland, pp 7–10.)* Joe is probably right-handed, which implies left-sided cerebral dominance. Since language is usually on the dominant side, and Joe's has an aphasia, his dominant cerebral hemisphere has been damaged. People who are left-handed may also have dominance of either side, or may have mixed dominance. True ambidexterity is rare.

**500. The answer is b.** *(Gilroy, p 337; Kandel, pp 951–953.)* Sleep apnea can occur for several reasons. One common basis is an obstruction of the airways (called *obstructive sleep apnea*). In this case, as indicated in the statement of the question, the physicians ruled out this possibility. Another possible cause involves central sleep apnea. This is due to disruption of the mechanism involving chemoreceptors in the carotid body that monitors carbon dioxide and oxygen levels in the blood. Axons in the carotid body project via the glossopharyngeal nerve (IX) to the reticular formation of the medulla. Therefore, disturbances involving the carotid body could result in central sleep apnea. Here, inappropriate signals are sent to the medullary reticular formation, which, in part, projects caudally to ventral horn sites in the spinal cord, governing such muscles as those that regulate the diaphragm and, therefore, disrupt the normal breathing process.

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