EPPP Fundamentals

Review for the Examination for Professional Practice in Psychology

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EPPP Fundamentals
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To Rob, Alex, and Jacob—for your unconditional love and support

—ASK

To my daughter, Kaitlyn, and wife, Lori; a perfect joy and loving partner

—BAM
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Preface

Most professions require some type of state or national examination before formal entrance into their respective fields can occur. For example, medicine requires completion of the U.S. Medical Licensing Exam. Prospective lawyers must pass the Bar. Accountants are required to take a test if they wish to become certified. And although it varies by state, to become a teacher, one must successfully navigate a certification exam. The profession of psychology is no different. Before one can practice psychology independently, the Examination for Professional Practice in Psychology (EPPP) must be passed.

The EPPP is a 225-question examination that focuses on eight content areas: biological bases of behavior; cognitive–affective bases of behavior; social and cultural bases of behavior; growth and life span development; assessment and diagnosis; treatment, intervention, prevention, and supervision; research methods and statistics; and ethical, legal, and professional issues. It is a broad-based test that assesses the individual’s depth and breadth of knowledge of psychology. The underlying assumption behind inclusion of the exam content is that the questions assess the knowledge base required to successfully function as a psychologist in professional practice (ASPPB, 2012). It is not all encompassing of what an effective psychologist should know, only a mere sampling. (You can find detailed information about the EPPP and the examination process at www.asppb.net)

The EPPP is arguably one of the most anxiety-provoking milestones associated with becoming a psychologist. Without successful completion, it will delay one’s entry into the profession leading to uncertainty, potential financial strain and, for some, thoughts of inadequacy and myriad negative emotions. Therefore, the anxiety is understandable. However, it is most often unwarranted and unnecessary. The truth is that the vast majority of individuals pass the test the first time they take it (Schaffer et al., 2012). And for those who don’t, most all will pass it in a subsequent attempt. For the reasons mentioned above, passing the examination the first time is highly desirable. And the best way to maximize your chances of passing the examination right out of the chute is to adequately prepare.

EPPP Fundamentals: Review for the Examination for Professional Practice in Psychology provides a comprehensive review of core exam content and includes over 300 sample questions. EPPP Fundamentals goes beyond merely “teaching the test” through rote memorization. Instead, it covers the eight content domains of the EPPP and their representative knowledge areas in a stepwise, narrative, and review format. Another unique aspect of EPPP Fundamentals is that it is an edited volume. Consequently, it includes contributions from psychologists associated with some of the top psychology training and internship programs in the United States. The lead contributors are professors, training directors, and
practitioners with expertise in the content areas of the chapters they authored. This combined approach helps users obtain the depth and breadth of knowledge required for passing the exam, and mirrors how doctoral level courses are commonly taught.

*EPPP Fundamentals* can be used in a variety of ways. We believe that the guide can serve as an instrumental text for supporting traditional systematic study methods or a stand-alone resource for those who are not able to invest in a formalized study program. Many students will find the textbook format of the guide useful as a primer at the beginning of the study process or as a review at the end. Questions found at the end of each chapter can serve as a gauge for successful review of the chapter material and the questions at the end of the text can serve as a review of the entire content of the book.

This book contains a considerable amount of information. If you have any questions or suggestions for updates or corrections, please email us at EPPPreviewupdates@yahoo.com.

Regardless of how you use *EPPP Fundamentals*, we wish you tremendous success with the exam and your future career as a psychologist. Passing the EPPP and becoming a professional psychologist will be one of the most memorable stages of your professional and personal life. We are honored to be a small part of that process.

**References**


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Biological processes are responsible, whether in isolation or concert with other processes, for all human and animal behavior. The biological system consists of highly complex, delicate, and integrated structures and mechanisms that are not fully understood by science. However, our knowledge in this area is growing at a rapid pace. Below we present the core components involved in the critical role biophysiological processes play in human and animal behavior with a focus on structures, functions, interventions, and methodologies.

Central Nervous System

It is important to understand that the brain forms only one part of what is also known as the central nervous system (CNS), which as a unit, comprises the brain and spinal cord. Elegant in design but complex in function, the brain and spinal cord make up the biological core of the human experience. To understand this system in terms of its functions and dysfunctions requires an understanding of the major structures and their role in the integration of our internal and external experiences.

Spinal Cord

The human spinal cord is a segmented cord linked with the organs and muscles of specific body regions. The spinal cord has four major divisions with 30 total segments, from the neck to the sacrum. The segments are identified according to their location in one of the three regions: cervical (C1–C8), thoracic (T1–T12), lumbar (L1–L5), and sacral (S1–S5). Fibers entering the dorsal portion of the spinal cord carry sensory information from the body to the brain. Descending fibers exiting the ventral portion of the spinal cord carry motor information to the muscles. When the spinal cord is damaged, a person loses the ability to feel and/or move...
the corresponding portion of his or her body at and below the site of the damage. For example, damage to the upper cervical regions of the spinal cord results in quadriplegia (inability to move the arms and legs), whereas damage to lower cervical regions results in paraplegia (inability to move the legs). Incomplete damage to the spinal cord may result in muscle weakness (paresis) as opposed to total immobility (paralysis). In addition to mediating voluntary movement, the spinal cord is also involved in involuntary movements such as reflexes (e.g., the withdrawal reflex from pain).

**Brain**

**Skull and Cranial Meninges**
The CNS, and the brain in particular, is guarded by several layers of protection. The most obvious is the skull, which is the bone structure forming the cranial vault. Just inside, and in various places, closely attached to the skull is the dura mater. This fibrous membrane also forms the falx cerebri, which extends down into the longitudinal fissure separating the two hemispheres of the brain. The arachnoid mater is a thinner and more delicate membrane separated from the dura by the subdural space through which passes a series of veins. Finally, the pia mater is the most delicate and highly vascular membrane, which closely follows the contours of the brain. The pia is separated from the dura by the subarachnoid space, which contains a network of arteries, veins, and connective tissue known as trabeculae.

**Ventricles**
Providing both protection and structural support, the internally located ventricular system comprises open chambers and channels filled with cerebral spinal fluid (CSF). This colorless fluid circulates through the two large lateral ventricles, located internally in each cerebral hemisphere, to the centrally located third ventricle, through the cerebral aqueduct and into the fourth ventricle in the dorsal brain stem. From there, the fluid flows in the subarachnoid space around the brain and spinal cord. The fluid is formed predominantly in the linings of the lateral ventricles known as the choroid plexus and then reabsorbed after its circulation. The CSF maintains the brain’s neutral buoyancy in the cranial vault and plays an important role in protection from infection and regulation of cerebral blood flow.

**Cerebrum**
Gross examination of the human brain reveals that the surface comprises convolutions of fissures (the inward folds) and gyri (the smoothly curved hills), both serving to increase the surface area of the cortex. Comprising six layers of cell bodies and interconnections, the cortex forms the outer and most visible layer of the brain, also known as the cortex or gray matter. Although functionally significant, the fissures and gyri also form the definitions and boundaries of the major structures of the telencephalon, or cerebrum, which includes the four major lobes (frontal, temporal, parietal, and occipital). Each lobe is represented bilaterally in the right and left hemispheres.

**Frontal Lobes**
Located anterior of the central sulcus, the frontal lobe is the largest of the four lobes, governs output, and is considered the seat of higher cortical and cognitive
functioning. Major anatomical subdivisions include the primary motor cortex, pre-motor cortex, orbitofrontal cortex, and prefrontal cortex. These regions are particularly devoted to attention, cognition, reasoning, problem solving, and voluntary movement.

Directly anterior to the central sulcus is the primary motor cortex. This gyrus, which runs laterally from superior to inferior, is crucial in the initiation of motor movements and isolated muscle groups are specifically represented along the surface of this gyrus. Moreover, the relative representation in this region corresponds directly to the requisite accuracy of motor control. For example, hands, fingers, lips, and tongue are heavily represented, whereas other regions such as the trunk and torso are not as heavily represented. Damage to this region will produce deficits in motor learning and more severe forms of lateralized damage will produce hemiparesis.

Directly anterior to the primary motor cortex is the premotor cortex, a region dedicated to the initiation and execution of limb movements in conjunction with input from other cortical regions. Mirror neurons located here have been associated with imitation and empathy and have been the focus of some autism studies (Schulte-Ruther, Markowitsch, Fink, & Piefke, 2007; Williams, Whiten, Suddendorf, & Perrett, 2001).

Prefrontal and orbitofrontal regions, located anterior to the primary motor cortex, are most often associated with higher-level cognitive functions also known as the executive functions, which includes reasoning, planning, and judgment. Dysfunction in this region has been associated with many disorders, including attention deficit hyperactivity disorder (ADHD) and schizophrenia. Inhibitory control is also most often associated with this region. The frontal lobe injury sustained by Phineas Gage in the mid-1800s, a railroad construction worker, is often considered an illustrative example of classic frontal lobe impairment.

For the majority of individuals, the inferior lateral region of the left frontal lobe is known as Broca's area. This area is particularly dedicated to the fluent production of oral and written speech, as well as grammar and comprehension of syntax. The dysfunction associated with a lesion here is most often recognized as Broca’s (or expressive) aphasia (an acquired disorder of language).

Temporal Lobes
Located inferior to the lateral sulcus, the temporal lobes are divided into the superior, middle, and inferior temporal gyri. Located in the superior temporal gyrus is the site of primary auditory processing, where conscious perception of sound takes place. This region, typically found in the infolded region of superior temporal gyrus, is also known as Heschel’s convolutions. Reception of stimuli in this region is considered “tonotopic,” which corresponds to individual frequencies detected at the level of the cochlea located in the inner ear. Stimuli arrive here by way of the vestibulocochlear nerves and the medial geniculate nuclei of the thalamus and undergo only partial “decussation,” the process by which incoming stimuli are transmitted to the contralateral hemisphere for processing. Because of partial decussation, or crossing of fibers, sound stimuli critical for auditory language comprehension will still arrive at the language-dominant hemisphere. Immediately adjacent and posterior to the primary auditory cortex is the auditory association cortex, where sound is further processed. In the language-dominant hemisphere, this region is known as Wernicke’s area, which is dedicated to the comprehension of language. Lesions in this region will disrupt not only the ability to comprehend
language but also the meaningful expression of language. This deficit is known as Wernicke’s (or receptive) aphasia.

### Parietal Lobes

The parietal lobes are located posterior to the central sulcus and include the site of primary somatosensory processing on the postcentral gyrus. Major neuroanatomical structures also include the inferior and superior parietal lobules. Within the parietal lobes are large regions of the heteromodal cortex, where different sensory modalities are integrated to construct a complete picture. The parietal lobes process visual information along dorsal and ventral pathways from the occipital lobes to help coordinate movements and behaviors with the environment. Damage to posterior regions of the parietal lobe can result in neglect syndromes such as hemispatial neglect, which is characterized by an inability to attend to features of the environment in the space contralateral to the lesion site.

As noted, primary somatosensory processing occurs on the postcentral gyrus where “somatotopic” detection of touch, pressure, pain, and temperature takes place. As on the primary motor cortex, regions of the sensory cortex proportionally represent body regions depending on their relative sensitivity, for example, there is heavy representation of the finger tips, face, and lips. Lateralized lesions here will result in hemisensory loss (loss of sensation on one side of the body).

### Occipital Lobes

Located posterior to the temporal and parietal lobes, the occipital lobes are geographically defined by the parieto-occipital sulcus visible on the medial surface of the hemisphere. Primarily dedicated to visual processing, primary visual processing is located in the region of the occipital pole, posterior to the calcarine sulcus. Primary visual processing is phototopic in nature, receiving its stimuli from the retina and optic nerve by way of the lateral geniculate nucleus of the thalamus.

Properties such as color and movement are processed at the primary visual, or striate, cortex. They are then sent for further processing and integration along the dorsal stream to parietal regions for processing of object location and along the ventral stream to temporal regions for object identification. Areas adjacent to primary processing regions are considered visual associations areas, which further process and integrate visual stimuli. Lesions in primary visual processing regions result in cortical blindness. Other lesions can result in disturbances in color perception and inability to detect orientation or movement.

### Subcortical Brain Regions

#### Hippocampus

The inferior temporal lobe curls in toward the midline and forms a region known as the hippocampus. As part of the limbic system, the hippocampus is critical for memory formation such as the transfer of memories to longer-term stores. The classic cases of a patient initially known as HM whose hippocampi were surgically removed to control seizures, and Clive Wearing, a British musician who contracted encephalitis, illustrate the debilitating memory impairments associated with bilateral hippocampal lesions.
Amygdala
Also part of the limbic system, the amygdala is located anterior of the hippocampus and is involved in processing olfactory stimuli. However, the amygdala is most often associated with processing emotions. Its connections to midbrain structures make the amygdala an essential component of the "fight-or-flight" response.

Thalamus
Located superior to and contiguous with the brain stem is the thalamus. This structure performs the critical relay functions between the cortex and the brain stem. Specific nuclei, or collections of nerve cells, form the specific transmission sites in the thalamus to and from specific cortical regions. Because of these very rich interconnections, the thalamus also performs important attention and perceptual functions.

Basal Ganglia
The basal ganglia is an important subcortical structure comprising a network of complex loops involved in motor output (i.e., descending motor pathways), emotions, cognition, and eye movements. The main components of the basal ganglia include the caudate nucleus, putamen, globus pallidus, subthalamic nucleus, and substantia nigra. The cerebral cortex provides most of the input to the basal ganglia, and the primary outputs of the basal ganglia are sent to the thalamus. Motor abnormalities due to basal ganglia do not involve paresis or paralysis, but rather the coordination and rhythm of movement. These syndromes are referred to as "extrapyramidal syndromes." For example, slow movements (i.e., bradykinesia) or excessive muscle rigidity result from basal ganglia dysfunction. Movement disorders such as Parkinson's disease (PD) and Huntington's disease result from abnormal activity in the basal ganglia.

Brain Stem
Comprising the medulla (also referred to as the medulla oblongata), pons, and midbrain, the brain stem forms the core of the brain. The midbrain and its component structures are surrounded by the cerebral hemispheres. Located caudally, or toward the tail, is the pons (or bridge), followed by the medulla, which is essentially contiguous with the spinal cord. Functionally, the brain stem, as a unit, is involved in the control and regulation of autonomic functions and maintaining the body's homeostasis, including breathing, heart rate, temperature regulation, and blood pressure. The reticular formation, including the reticular activating system, plays important roles in alertness, consciousness, and pain. It also plays important roles in regulating the respiratory and cardiovascular systems. Ascending sensory pathways from the spinal cord rise through dorsal regions, whereas descending, motor fibers pass through anterior regions of the brain stem.

Cerebellum
Attached to the posterior brain stem is the cerebellum. Rich in neurons, the cerebellum is structurally divided into the superior, middle, and inferior cerebellar peduncles. The middle cerebellar peduncle is the only structure visible on surface examination of the brain. The cerebellum comprises a gray matter cortex and subcortical white matter with rich interconnections to cortical regions of the other hemispheres of the brain. Functionally, the cerebellum is most often associated
with the regulation of movement, including automatic and rhythmic movements, coordination of the limbs, and postural control. Studies have also associated the cerebellum with cognitive functions such as learning and attention (Helmuth, Ivry, & Shimizu, 1997).

The cerebellum is particularly vulnerable in multiple sclerosis, which can result in disruption of ocular movements such as nystagmus (a rapid rhythmic eye movement which is particularly enhanced when the gaze is in the same direction of the lesion site). Lesions of the cerebellum can also produce motor incoordination and a characteristic wide-based stance and gait.

Neurons

The neuron is the building block of the nervous system. Neurons vary in size and shape and are highly specialized to a specific function. A typical neuron consists of a cell body (containing the nucleus), dendrites (short processes emerging from the cell body which receive inputs from other neurons), and axons (long processes that carry output away from the cell body). Most neurons in the human brain are “multipolar,” that is, they have multiple dendrites and axons. A myelin sheath, which is an insulating fatty layer, surrounds the axon and speeds up transmission. Axons can range in length from 1 mm to 1 m. The synapse is the space between two neurons in which chemical and/or electrical communication occurs. In most cases, the axon from one neuron communicates with the dendrites of another neuron.

Chemicals known as neurotransmitters are released “presynaptically” by the axon terminal of one neuron and bind to neurotransmitter receptors on the “postsynaptic” neuron, which may then cause postsynaptic excitation or inhibition. When postsynaptic excitation reaches a minimum threshold, that neuron then fires an action potential, causing that neuron to send the neural signal down its axon. The firing of a neuron is an “all-or-nothing” phenomenon, that is, the strength of neuronal firing does not vary in response to the strength of the input. In other words, a neuron either fires or it does not. After a neuron fires, there is a refractory period during which it is unable to fire again until it reestablishes an electrochemically based resting potential state. Different neurotransmitters have different effects on cells (excitatory or inhibitory), and the amount of a neurotransmitter that is available for binding to the postsynaptic neuron can be affected by various medications.

Neurotransmitters

Neurotransmitters are chemicals that transmit signals from one neuron to another and are classified according to their molecular size. Biogenic amines (e.g., acetylcholine [ACh] and serotonin), catecholamines (e.g., dopamine [DA], norepinephrine [NE], and epinephrine), and amino acids (e.g., gamma-aminobutyric acid [GABA] and glutamate) are smaller molecular messengers, whereas neuropeptides (e.g., vasopressin, oxytocin, and substance P) are larger molecules. Neurotransmitters fit into a receptor site like a lock and key, although a variety of neurotransmitters may fit into a single type of receptor (Zillmer & Spiers, 2001). The most significant neurotransmitters to psychopharmacology include NE, serotonin, DA, GABA, ACh, and glutamate (Wegman, 2012).
Norepinephrine or “noradrenalin” is a catecholamine and functions as a hormone and a neurotransmitter. It is formed in the brain stem at a site called the “locus coeruleus” and is found in the sympathetic nervous system and CNS. It regulates mood, memory, alertness, hormones, and the ability to feel pleasure. Elevated levels may lead to anxiety, whereas low levels may cause depression (Wegman, 2012). NE also underlies the “fight-or-flight” response and is released into the blood as a hormone by the adrenal gland in response to stress or arousal. It is primarily considered an excitatory neurotransmitter, but may result in inhibition in some areas.

Dopamine is also a catecholamine and can be both excitatory and inhibitory. The majority of DA neurons are in the substantia nigra. Dopamine pathways extend to the frontal lobes, basal ganglia, and hypothalamus. Overactivity of DA in the pathway to the frontal lobes has been implicated in schizophrenia, and the loss of DA-producing neurons in the basal ganglia pathway is the underlying cause of PD. Underactivity of DA has also been implicated in ADHD (Wegman, 2012). Dopamine plays a role in emotions, movement, endocrine functioning, as well as attention, sociability, motivation, desire, pleasure, and reward-driven learning.

Serotonin (5-HT) is a biogenic amine and is primarily inhibitory. It is widely distributed throughout the brain and originates in the raphe nuclei in the brain stem. Pathways extend to the limbic system, and serotonin levels are associated with the regulation of mood, anger, aggression, anxiety, appetite, learning, sleep, sexual functioning, level of consciousness, and pain. Low levels of serotonin are associated with depression, obsessive-compulsive disorder, and anxiety disorders (Wegman, 2012).

Acetylcholine is also a biogenic amine and plays a major role in the parasympathetic nervous system and autonomic nervous system (Zillmer & Spiers, 2001). It is the primary neurotransmitter at the neuromuscular junction (the synapse between neuron and muscle cells) and is involved in movement. Degeneration of ACh in the striatum of the brain is associated with a movement disorder called Huntington's disease. ACh also plays a major role in activating the brain through the reticular activating system and regulates alertness and attention. Another cholinergic system involving the hippocampus influences attention, learning, and memory (Zillmer & Spiers, 2003).

Gamma-aminobutyric acid is an amino acid and is the major inhibitory neurotransmitter of the CNS. It is widely distributed throughout the CNS but is most concentrated in the striatum, hypothalamus, spinal cord, and temporal lobes. GABA is associated with emotion, balance, and sleep patterns. Low levels of GABA are associated with high anxiety and agitation, and higher levels are associated with a reduction in anxiety (Wegman, 2012). GABA deficiencies are also implicated in epilepsy, and many antiepileptic drugs increase GABA activity.

Glutamate is also an amino acid and is the brain's primary excitatory neurotransmitter. It is widely distributed throughout the CNS. It is a basic building block of proteins and plays an important role in learning and memory (Wegman, 2012). Excessive glutamate causes excitotoxicity (cell death due to excessive stimulation and excitation), and is implicated in cell death following traumatic brain injury and stroke.

**Psychopharmacology**

Clinical psychologists may or may not have prescription privileges depending on whether they have specialized training and whether this training is recognized by
the state in which they practice. It is imperative for even nonprescribing psychologists to possess a general understanding of psychotropic agents and whether they may be indicated. Often the psychologist will act as the intermediary between the patient and a prescribing clinician. It is necessary to be able to recognize when an evaluation for psychotropic medication (or for termination of medication) may be beneficial to the patient. Each patient is unique and will have different requirements based on age, sensitivities to medications, and other characteristics.

**Pharmacokinetics and Pharmacodynamics**

Psychotropic medications cross the blood–brain barrier and cause physiological and biochemical changes. The mechanism of action for these medications is complex. To put it simply, these medications alter the activity of neurotransmitter communication between neurons by doing one or more of the following: disrupting the action of the neurotransmitter at the synapse (thus blocking the action of the neurotransmitter), inhibiting the enzymes that break down neurotransmitters in the synaptic cleft (thus boosting the overall transmission of that neurotransmitter), changing the sensitivity of postsynaptic neurons to neurotransmitters, or increasing the amount of neurotransmitter produced and available at the synapse.

Psychoactive drugs are able to cause downstream biochemical and physiological changes by binding to receptor sites on neurons and either boosting the action of a particular neurotransmitter system or blocking the action of a neurotransmitter system. An agonist is a chemical that binds to a receptor site and mimics the activity of a neurotransmitter, thus causing the same downstream effects as that neurotransmitter and boosting the overall system. A partial agonist also binds to a receptor site and mimics the activity of a neurotransmitter, but cannot produce 100% of the effect of a full agonist, even at very high doses. An inverse agonist binds to the same receptor site as an agonist but has the opposite effect of full agonists by causing a reduction in the overall efficacy of a neurotransmitter system. An antagonist also blocks or reverses the effect of agonists or inverse agonists, but when an agonist is not present, they have no effect of their own (Stringer, 2011).

Pharmacodynamics describes the biochemical and physiological effects of drugs on the body. Pharmacokinetics describes how the body handles the drug through absorption, distribution, metabolism, and elimination. Absorption is the process through which drugs reach the bloodstream. This process occurs mainly in the small intestine and results in the drug’s onset and degree of action. A poorly absorbed drug may not reach the minimal effective concentration required in the blood for clinical efficacy. The blood stream transporting a drug to its site of action serves as the distribution. The speed of distribution varies depending on how the drug is administered. For example, drugs taken orally must first travel through the digestive system, whereas drugs injected intramuscularly have a faster response. When a drug enters the bloodstream, metabolism begins. The body recognizes the drug as a foreign substance and attempts to eliminate it via chemical transformation. Metabolism occurs primarily in the liver. People metabolize psychotropic agents differently, and the sensitivities and preferences of each individual patient must be considered to get optimum risk–benefit ratios. For example, children and the elderly may metabolize drugs differently than young adults, and therefore dosage or scheduling adjustments may be required to achieve the best risk–benefit
ratios. Once a drug is in circulation, elimination is a function of renal and hepatic processes. The elimination half-life of the drug is the time it takes for drug concentration to decrease by half due to excretion and metabolic change. In the steady state, the rate of elimination is equal to the rate of administration of the drug.

A therapeutic window is defined by the range of a drug dose that can result in desired clinical efficacy without resulting in unsafe side effects. For example, if a drug has a narrow therapeutic window, then there is only a small range of dosages that can result in the desired benefit before it becomes unsafe. A therapeutic index is the ratio of the amount of drug that causes the desired benefit to the amount of the drug that produces dangerous side effects. It is more desirable for a drug to have a high therapeutic index, as it is a measure of drug safety.

Psychoactive drugs can be classified according to the clinical disorders that they treat (e.g., anxiolytics, antidepressants, antipsychotics, stimulants, and pain medications). All psychoactive drugs have a unique mechanism of action, even those that treat similar clinical disorders, and all have notable side effects and possible drug interactions that must be considered. The following section summarizes the primary psychoactive drug classifications, their mechanism of action, side effects, and drug interactions.

**Anxiolytics**

Anxiolytics refer to the psychotropic medications that may be used to treat anxiety disorders and can be classified into benzodiazepines and nonbenzodiazepines. Examples of anxiolytics that are benzodiazepines are alprazolam (Xanax), clonazepam (Klonopin), diazepam (Valium), and lorazepam (Ativan). Buspirone (Buspar) is an example of a nonbenzodiazepine anxiolytic. Some benzodiazepines are used as sleep agents and others may treat seizure disorders and alcohol withdrawal (Pies, 2005). Anxiety disorders that may be treated with benzodiazepines include generalized anxiety disorder, panic disorder, phobic disorders, adjustment disorder with anxiety, anxiety disorder due to a general medical condition, and substance-induced anxiety disorder (Pies, 2005; Wegman, 2012).

Benzodiazepines act through the CNS and cause muscle relaxation as well as sedative, anxiolytic, and anticonvulsant effects. They enhance the action of GABA (which is an inhibitory neurotransmitter) and block the rapid release of stress hormones associated with anxiety and panic. These medications are rapidly and completely absorbed after oral administration and distributed throughout the body. Some are short acting and some are long acting.

The most significant side effects of benzodiazepines include drowsiness, confusion or feelings of detachment, dizziness, imbalance, and high potential for dependence. When discontinued, they must be tapered slowly to prevent withdrawal symptoms. In regard to drug interactions, benzodiazepines increase the effects of alcohol and other CNS depressants. They should be used cautiously in patients with liver disease and avoided in patients with a history of substance abuse (Stahl, 2011).

Since the 1990s benzodiazepines have been increasingly replaced by selective serotonin reuptake inhibitors (SSRIs) and other antidepressants as clinicians’ first choice for the treatment of anxiety disorders due to their increased safety, lower side effect profile, and decreased likelihood of dependence (Asho & Sheehan, 2004).

Barbiturates are medications that were formerly used for sedation and to induce sleep, but have now been essentially replaced by benzodiazepines. The side effects of barbiturates are extreme, including tolerance, physical dependency,
and very severe withdrawal symptoms. They also enhance the function of GABA in the CNS (Stringer, 2011).

**Antidepressants**

Antidepressants are a diverse group of medications that have different mechanisms of action. The primary classifications of antidepressants include monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants (TCAs), SSRIs, and newer agents such as norepinephrine–dopamine reuptake inhibitors (NDRIs) and serotonin–norepinephrine reuptake inhibitors (SNRIs). In general, antidepressants do not cause dependence, tolerance, or addiction. These medications are used to treat disorders such as unipolar major depression, dysthymic disorder, adjustment disorders, and mood disorder due to general medical condition. These medications are also used in the treatment of many anxiety disorders (Pies, 2005), ADHD, and eating disorders (Stein, Lerer, & Stahl, 2012). The use of antidepressants is contraindicated in patients with bipolar disorder, as they may induce mania.

The medications listed above target a class of neurotransmitters called the monoamines, which include NE, serotonin, and DA. The “monoamine hypothesis” of depression dates back to the 1960s and postulates that depression is caused by abnormal functioning of these neurotransmitters. Based on this hypothesis, antidepressant medications are thought to increase the availability of these neurotransmitters at the synaptic level. However, a simple deficiency of monoamines at the synaptic level is no longer thought to explain the mechanisms of action of these medications in full (Patterson, McCahill, & Edwards, 2010), and antidepressants likely affect many biological systems in addition to neurotransmitter uptake (Mycek, Harvey, & Champe, 1997).

**Tricyclic antidepressants** These drugs are categorized on the basis of their chemical three-ring structure (Pies, 2005). Examples of TCAs include amitriptyline (Elavil), nortriptyline (Pamelor and Aventyl), imipramine (Tofranil), and desipramine (Norpramin). Absorption of the tricyclic drugs occurs in the small intestine, and peak levels occur within 2 to 8 hours following ingestion (Golan, Tashjian, Armstrong, & Armstrong, 2008). Tricyclics block the reuptake of serotonin and NE (thus increasing the activity of these neurotransmitter systems by making them more available for binding to postsynaptic neurons); however, the precise mechanism of action of the TCAs is unknown (Stringer, 2011).

Unfortunately, this class of medications has side effects that make them unattractive. Side effects of TCAs fall into three categories: cardiac/autonomic, anticholinergic, and neurobehavioral. Orthostatic hypotension (a drop in standing blood pressure) is one of the most common reasons for discontinuation of this medication (Pies, 2005).

**Monoamine oxidase inhibitors** Monoamine oxidase inhibitors (MAOIs) are rarely used today because of serious drug–drug and drug–food interactions. They block the reuptake of monoamine neurotransmitters (serotonin, NE, and DA) by blocking their respective monoamine transporters, thus increasing the levels of these neurotransmitters in the synaptic cleft (Keltner & Folks, 2005). Examples of MAOIs include phenelzine (Nardil) and tranylcypromine (Parnate). The most dangerous side effect of MAOIs is hypertensive crisis, which can occur when an MAOI is taken with tyramine (Stahl, 2011).

**Selective serotonin reuptake inhibitors** These medications, which block the reuptake of serotonin by selective binding, are especially effective for the treatment
of depression with agitation and/or comorbid anxiety. The term “selective” is used because they have weaker affinity for blocking the action of other monoamines. Examples of SSRIs include fluoxetine (Prozac), paroxetine (Paxil), fluvoxamine (Luvox), setraline (Zoloft), citalopram (Celexa), and escitalopram (Lexapro). SSRIs are less likely to cause anticholinergic and cardiac/autonomic side effects than the TCAs; however, side effects do include gastrointestinal side effects, headache, sexual dysfunction, insomnia, psychomotor agitation, and occasional extrapyramidal reactions.

Norepinephrine–dopamine reuptake inhibitors These antidepressants are relatively newer antidepressants, and they work by blocking the reuptake of NE and DA. An example of an NDRI is bupropion (Wellbutrin).

Serotonin–norepinephrine reuptake inhibitors These medications block the reuptake of serotonin and NE. An example of an SNRI is venlafaxine (Effexor).

Over-the-counter products St. John’s wort, S-adenosyl methionine (SAMe), 5-HTP, Omega-3 fatty acids, and folic acid have all been shown to have some efficacy in treating depression. Omega-3 fatty acids and folic acid are typically used in conjunction with antidepressants (Preston & Johnson, 2012). However, it is important to note that these alternative remedies can also have negative side effects and adverse drug interactions. For example, St. John’s wort can reduce the effectiveness of oral contraceptives and Omega-3 fatty acids can increase the risk of bruising and bleeding especially when combined with blood thinners (Wegman, 2012).

Antipsychotics Antipsychotics are primarily used to treat schizophrenia, schizotypal disorder, schizoaffective disorder, brief psychotic disorder, bipolar disorder, and agitation (Pies, 2005). Several neurochemical abnormalities are associated with schizophrenia but the DA system is the most studied (Patterson et al., 2010). All traditional (or first-generation) antipsychotic medications block DA receptors, whereas atypical (or second generation) also block serotonin receptors (Patterson et al., 2010).

Conventional antipsychotics ("typical" or "first generation") First developed in the 1950s, all of the drugs in this group seem to have equal efficacy but differ in potency and side effects. Examples of conventional antipsychotics include haloperidol (Haldol), thioridazine (Mellaril), molindine (Moban), thiothixene (Navane), fluphenazine (Prolixin), trifluoperazine (Stelazine), and chlorpromazine (Thorazine).

Conventional antipsychotics may cause extrapyramidal symptoms (EPSs), including parkinsonism, acute dystonia, akathisia, and tardive dyskinesia. The first three EPSs are early drug reactions. The fourth, tardive dyskinesia, results from long-term use. Parkinsonism includes bradykinesia (slowed movements), tremor, and rigidity. Acute dystonia includes muscle spasms in the tongue, face, neck, and back. Akathisia is characterized by restless movements and symptoms of anxiety and agitation. Tardive dyskinesia is characterized by abnormal involuntary, stereotyped movements of the face, tongue, trunk, and extremities. Unfortunately, this syndrome may be irreversible, even when antipsychotic medications are discontinued (Lehne, 2013).

Another potential side effect is neuroleptic malignant syndrome (NMS), a rare but life-threatening reaction characterized by catatonia, stupor, fever, and autonomic instability. Additional side effects of the antipsychotics may include
orthostatic hypotension, sexual dysfunction, and sedation, as well as anticholinergic effects such as dry mouth, constipation, and difficulty with urination (Lehne, 2013).

**Atypical antipsychotics (second generation)** Atypical antipsychotics became available in the 1990s. In addition to blocking DA receptors in the CNS, the atypicals also block serotonin receptors (Patterson et al., 2010). Initially, the atypical antipsychotic medications were thought to be more effective than the typical or first-generation antipsychotics; however the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE), a National Institutes of Health-(NIH) funded, nationwide clinical trial, revealed that the typical or first-generation antipsychotics may indeed be just as effective as some of the newer atypical antipsychotic medications (Lieberman et al., 2005). The newer atypical drugs may produce milder extrapyramidal symptoms than the typical antipsychotics; however, they may also cause dangerous metabolic effects such as weight gain, diabetes, and dislipidemia (Lehne, 2013).

Atypical antipsychotics include olanzapine (Zyprexa), quetiapine (Seroquel), ziprasidone (Geodon), aripiprazole (Abilify), paliperidone (Invega), iloperidone (Fanapt), asenapine (Saphris), clozapine (Clozaril), and risperidone (Risperdal). Clozapine is actually one of the more effective atypical antipsychotics although it is also the most dangerous. Fatal agranulocytosis (dangerously low white blood cell count causing decreased ability to fight infection) is a potential side effect. Therefore, testing of white blood counts is done on a regular basis, which improves outcome. Risperdal (risperidone) is a first-line medication for new onset schizophrenia and is also well accepted for treatment of agitation and aggression in dementia and in bipolar disorders. Risperdal is also Food and Drug Administration–(FDA) approved for minimizing self-harm in autism and disruptive behavior disorders in children and adolescents (Stringer, 2011).

**Mood Stabilizers**

Lithium was the first mood-stabilizing medication approved by the FDA for the treatment of acute mania and hypomania. It has well-documented efficacy in preventing relapse in bipolar disorder. Lithium’s mechanism of action is complex and simply a theory. It is suspected to involve NE and serotonin (Wegman, 2012). Unfortunately, lithium has a slow onset of action and a narrow therapeutic index (i.e., the therapeutic dose is close to toxic).

Common side effects of lithium include nausea, diarrhea, vomiting, thirst, excessive urination, weight gain, and hand tremor. A reversible increase in white blood cell count frequently occurs with lithium use. Chronic use side effects include hypothyroidism, goiter, and rarely, kidney damage. Toxicity may result in lethargy, ataxia, slurred speech, shock, delirium, coma, or even death (Wegman, 2012). Drug interactions with diuretics can increase plasma lithium concentration, and those with nonsteroidal anti-inflammatory agents can increase serum lithium levels (Stahl, 2011).

Antipsychotics and anticonvulsant medications may also be used as mood stabilizers and are considered first-line treatments for bipolar disorder (Wegman, 2012). For example, the atypical antipsychotics Zyprexa and Abilify are both FDA approved for acute and maintenance treatment of bipolar mania. Symbyax is FDA approved for treatment of depression associated with bipolar disorder (Wegman, 2012). Examples of anticonvulsants used as mood stabilizers include divalproex (Depakote), lamitrogine (Lamictal), carbamezepine (Tegretol), and topiramate (Topamax). Anticonvulsants work by enhancing the actions of GABA, the brain’s
major inhibitory neurotransmitter (Wegman, 2012). One example of a serious side effect of an anticonvulsant medication (Lamictal) is Stevens–Johnson syndrome, a potentially fatal skin rash.

**Opiates (Narcotic Analgesics)**

Opiates refer to natural or synthetic compounds obtained from the juice of the opium poppy that are used as drugs. Natural opiates include opium, morphine, and codeine. Semisynthetic derivatives of opiates include morphine, heroin, Percodan (oxycodone hydrochloride and aspirin) and Dilaudid (hydromorphone hydrochloride). Drugs with opiumlike mechanism of action are called opioids. The brain produces its own version of opiates called endogenous opiates (Mycek et al., 1997), and there are naturally occurring binding sites in the brain called opiate receptors.

Opiates are used to relieve intense pain and the anxiety that goes along with it. They also induce sleep. Some opiates are prescribed for severe diarrhea or coughs (Stringer, 2011). Opiates are often manufactured in combination with nonopiate analgesics, such as aspirin and acetaminophen (e.g., Percodan or oxycodone hydrochloride and aspirin). The two work well in combination because these different classes of drugs affect pain pathways via different mechanisms of action (Stringer, 2011).

Long-term opiate use changes the way nerve cells work in the brain, which can lead to withdrawal symptoms when they are suddenly discontinued. These withdrawal symptoms may include diarrhea, vomiting, chills, fever, tearing and runny nose, tremor, abdominal cramps, and pain (Stringer, 2011). Opiates may be abused for their euphoric effects. Regarding drug interaction, the depressant actions of morphine are enhanced by MAOIs and TCAs (Mycek et al., 1997).

**Psychostimulants**

Psychostimulants increase prefrontal cortex levels of NE and DA (Pies, 2005) and are primarily used to treat ADHD. Some examples of psychostimulants include amphetamine (Adderall), methylphenidate (Concerta), and modafinil (Provigil), which is also used for sleep disorders such as narcolepsy. Some antidepressants can be used in treating ADHD because they also enhance the actions of NE and DA in the prefrontal cortex; however, SSRIs are considered a poor choice for treating ADHD due to their effects on serotonin (Wegman, 2012).

Side effects of the psychostimulants may include insomnia, headache, tics exacerbation, nervousness, irritability, overstimulation, tremor and dizziness, weight loss, abdominal pain or nausea, possibly slow normal growth in children, and blurred vision (Stahl, 2011). There are numerous potential drug interactions. For example, they should not be used with MAOIs as they may cause hypertensive crisis (Stahl, 2011).

**Combined Treatments: Psychopharmacology and Beyond**

It is important to note that the psychoactive medications described above often complement other nonmedication approaches to treatment for many psychiatric and neurological illnesses. For example, in the treatment of mild depression, cognitive behavioral therapy (CBT), antidepressants, and their combination have been shown to result in equal benefit (Otto, Smits, & Reese, 2005). For the treatment of severe depression, antidepressants used in combination with CBT have been
shown to be better than either CBT or medication alone (Keller et al., 2000). Cognitive behavioral therapy for insomnia is shown to be equally effective and have longer lasting effects than psychotropic medication when active treatment is discontinued (Perlis, 2011)

Psychoactive medications are almost always used in the treatment of psychotic disorders. Unfortunately, schizophrenia and other forms of psychoses continue to be very difficult to treat. Psychotherapy, family therapy, skills training, psych-education, and vocational training often complement medication management (Patterson et al., 2010). Antipsychotics and anticonvulsant medications are also commonly used as first-line treatments for bipolar disorder, although they are commonly used in combination with psychotherapy (Wegman, 2012). Psychostimulants, which are primarily used to treat ADHD, are commonly used as part of a treatment plan involving multiple therapy modalities, including behavioral modification, parent and social skills training, and school-based interventions. Finally, environmental, social, and psychological interventions are crucial when managing patients with dementia.

Neuroimaging

The types of brain imaging techniques that can be used to visualize neuroanatomy and assess for neurological disorders are usually divided into “structural” and “functional” imaging techniques.

Structural Imaging

Computerized Tomography

Computerized tomography (CT) uses x-rays to look at slices of the brain, providing information on the density of brain tissue. There are two primary features that distinguish CT scans from traditional x-rays. First, rather than taking one view, the x-ray beam in CT is rotated around the patient to take many different views from different angles. Then, x-ray data are reconstructed by a computer to obtain detailed images of soft tissues, liquid, air, and bone.

The appearance of brain tissue on a CT scan depends on the tissue density. Very dense tissue, such as bone, appears white. Less dense tissue, such as air, appears black. The term hyperdense refers to brighter areas and hypodense refers to darker areas. Areas of intermediate density are referred to as isodense. Brain tissue that is rich in cells has a different density than areas rich in axons. White matter is slightly darker than gray matter due to its high myelin content. Cerebral spinal fluid is denser than air and is usually dark gray in color. In some instances, intravenous contrast material containing iodine is injected into the patient prior to obtaining the CT scan for better visualization of certain tissues. This contrast material is denser than brain tissue and will therefore appear hyperdense (white) in areas of increased vascularity or breakdown of the blood–brain barrier. CT images are often obtained with and without contrast for comparison. An enhancing lesion refers to areas that are absorbing this contrast material and may be indicative of brain neoplasms, abscess, infarct (area of dead tissue resulting from obstructed blood flow), demyelinating disease, resolving hematoma, or vascular malformation.
Clinically, CT is often used in the emergency room to detect acute hemorrhage or skull fracture following trauma. Fresh intracranial hemorrhage coagulates almost immediately and shows up as hyperdense (white) areas. Acute cerebral infarcts often cannot be seen with CT, although areas of abnormality resulting from cell death after a cerebral infarct are later visible. CT scans are also useful in the detection of neoplasms, tumor, mass effect, or ventricular enlargement, for example, in the context of hydrocephalus.

**Magnetic Resonance Imaging**

Magnetic resonance imaging (MRI) was developed in the 1980s and uses powerful magnetic fields that cause protons to align themselves in response to the magnetic field's line of force. Unlike CT scans, MRI scans are not described in terms of density, rather they are described in terms of intensity, or brightness, of the signal. The term “hyperintense” refers to a brighter area, and the term “hypointense” refers to a darker area.

Clinically, MRI provides high-contrast, high-resolution imaging with good anatomical detail. MRI is the preferred method for detecting small lesions such as plaques found in patients with multiple sclerosis, subtle tumor, or chronic hemorrhage. CT is not as sensitive in detection of white matter or neurodegenerative disorders. However, MRI costs more and takes longer. CT is preferred in urgent assessment of head trauma with suspected intracranial hemorrhage, and it is better at visualizing bony structures (e.g., skull fracture). CT is also preferred for patients who have metallic implanted devices, such as a pacemaker. MRI is preferred in nonurgent situations in which a higher-resolution imaging method is required for better anatomical detail (Blumenfeld, 2010).

**Neuroangiography**

Neuroangiography is used to visualize lesions of blood vessels through the use of radiographs and injection of contrast material into the vasculature. It is the gold standard for evaluating vascular diseases in head, neck, and spine, such as atherosclerotic plaques and other vessel narrowings, aneurysms, and arteriovenous malformation (AVM). Angiography is often invasive and requires general anesthesia.

**Wada Test**

The Wada test is an example of neuroangiography that is helpful in localizing language function and aiding in presurgical planning, particularly in patients with epilepsy who are undergoing brain resection. For this procedure, amobarbital is selectively infused into each carotid artery while the patient is awake, essentially “putting to sleep” the contralateral hemisphere, so that various cognitive functions (e.g., memory and language) can be assessed in that hemisphere.

**Functional Neuroimaging**

**Electroencephalography**

Electroencephalography (EEG) is considered the original method for measuring brain activity. To measure brain activity using an EEG, a small metal disk that records the electrical activity of the neurons in the underlying brain area is
attached to the scalp. These small electrical impulses are amplified and displayed on paper using a chart recorder called an electroencephalograph. EEG is useful in detecting widespread abnormality in brain function in a variety of contexts (e.g., sleep, anesthesia, coma, traumatic brain injury, and epilepsy), but its sensitivity and spatial resolution for detecting brain lesions are poor.

**Positron Emission Tomography**

Positron emission tomography (PET) uses small amounts of injected radioactive material to measure regional cerebral blood flow via glucose metabolism or oxygen consumption. The idea is that areas of the brain that are more active will use more glucose (and hence become radioactive) than less active areas. However, the brain is always active, so the brain's normal background activity is usually measured first to establish a baseline, which is then “subtracted” from the activity measured during the test. PET scans are useful for mapping the distribution of neurotransmitters and identifying brain dysfunction due to stroke, epilepsy, tumor, dementia, and other brain-impairing conditions.

**Functional Magnetic Resonance Imaging**

Functional magnetic resonance imaging (fMRI) was developed directly from MRI and can detect functionally induced changes from blood oxygenation. The basic idea is that oxygen distribution varies with brain activity and the amount of oxygen in the blood changes the magnetic properties of the blood without having to inject any radioactive materials, such as with PET or CT. Like MRI, fMRI has excellent resolution and provides a detailed structural map, while also providing functional information. fMRI can be used to measure the brain's real-time response to motor activities or neuropsychological tests.

**Disorders**

**Aphasia**

Aphasia refers to an acquired disorder of language (as opposed to a developmental language disorder) and can affect expressive speech, receptive speech, reading (alexia), and/or writing (agraphia). Aphasia syndromes can be subdivided into three major classifications: fluent aphasia (in which speech is fluent but there are difficulties with comprehension and/or repetition of words or phrases spoken by others), nonfluent aphasia (in which expressive speech is notable for poor articulation or poor grammar, but comprehension is relatively preserved), and pure aphasia, in which select aspects of language are affected, such as reading or writing.

Under the category of fluent aphasia is Wernicke's aphasia (aka sensory aphasia or receptive aphasia), in which the primary deficit is the inability to understand language. Speech is usually fluent (with normal rate and articulation) but the content of the speech is often nonsensical and meaningless, often containing neologisms (nonwords) or incorrect combinations of words (“word salad”). People with Wernicke's aphasia often have poor insight into their deficit and may expect others to understand what they are saying. The ability to repeat what others say is also impaired. The lesion typically associated with Wernicke's aphasia is in the left temporal lobe.
Transcortical sensory aphasia is similar to Wernicke’s aphasia in that it is also a fluent aphasia in which comprehension is poor, but the individual can repeat what others say (unlike Wernicke’s). The lesion is usually in the border zones between the parietal and temporal lobes.

Broca’s aphasia (also known as motor or expressive aphasia) is a nonfluent aphasia in which the person speaks in a slow, halting manner, with poor grammar and limited prosody. Only keywords are used, and use of verbs or connecting words is limited. Damage is usually in the left frontal lobe around Brodmann areas 44 and 45, also known as “Broca’s area.” Writing is usually slow and effortful. Repetition is also impaired. Auditory comprehension and reading comprehension are relatively preserved.

Transcortical motor aphasia is similar to Broca’s aphasia (and is sometimes referred to as “little Broca’s”) in that it is also a nonfluent aphasia, but the person is able to repeat what others say (unlike Broca’s aphasia). Damage usually occurs in the left frontal areas surrounding Broca’s area, leaving Broca’s area and its connections to Wernicke’s area intact.

Conduction aphasia is a specific disorder in which people can speak normally (therefore it is considered a fluent aphasia), name objects, and understand speech, but the sole deficit is in the repetition of what others say. Conduction is considered a “disconnection syndrome” in which the expressive speech center of the brain and the receptive speech center are disconnected. Damage is thought to affect the arcuate fasciculus, which is the large white matter tract connecting Broca’s area and Wernicke’s area.

Anomic aphasia consists of a focal deficit in naming objects, although the person can adequately produce meaningful speech, comprehend speech, and repeat speech. The angular gyrus is thought to be affected in this type of aphasia, although some degree of anomia, or problems with word finding, is present in most types of aphasias and is not consistently localized to a particular brain region.

In global aphasia, all aspects of language are impaired, including expressive speech, comprehension, repetition, reading, and writing.

**Alexia**

Alexia is the acquired inability to read (as opposed to dyslexia, which refers to a developmental disorder of reading starting in childhood). Pure alexia refers to impairments with reading, whereas the ability to write is relatively preserved. The pathology is usually a stroke in the posterior region of the left hemisphere, affecting the posterior region of the corpus callosum, disconnecting the visual centers of the brain from the language centers of the brain.

**Agraphia**

Agraphia refers to an acquired disorder of writing (as opposed to a developmentally based writing disorder beginning in childhood). Agraphia may affect a variety of components of writing, including spelling, grammar, letter formation, or visuospatial errors (e.g., poor spacing or orientation of letters). Different types of agraphia are usually classified based on accompanying symptoms such as alexia, apraxia, or visuospatial disorders. The site and extent of damage can range from parietal lobe, frontal lobe, corpus callosum, and subcortical structures.
**Apraxia**

Apraxia is an acquired disorder of skilled, purposeful movement that is not due to a primary motor or sensory impairment such as paresis or paralysis. For example, a person may not be able to demonstrate how to brush his or her hair or wave goodbye on command. There are many types of apraxia. In some cases, the action may be carried out accurately but in a clumsy manner, and in other forms of apraxia, the person may commit errors such as performing sequenced actions in the wrong order (such as sealing an envelope before placing the letter inside). The lesion site may vary depending on the type of apraxia but is usually in the left hemisphere.

**Dementia**

Dementia is an umbrella term that refers to a decline in two or more areas of cognitive functioning resulting in significant impairments in activities of daily living. The term *dementia* does not imply a specific cause and could be due to progressive, static, or reversible etiologies (National Institute of Neurological Disorders and Stroke (NINDS)—National Institutes of Health, n.d.). Although cognitive functions do decline with age, dementia is not a normal part of the aging process. Dementia is also distinct from delirium, which is an acute and potentially reversible form of cognitive decline. The term dementia has recently been replaced by the term “neurocognitive disorder” in the *DSM-5* (American Psychiatric Association, 2013).

Alzheimer’s disease (AD) is the most common cause of dementia in those aged 65 and older. Approximately 10% of people over the age of 65 are living with AD in the United States, and nearly half of those older than 85 have the disease (www.ninds.nih.gov/disorders/dementias/dementia.htm). A neurocognitive disorder due to AD is defined by the *DSM-5* as a decline in memory and at least one other cognitive domain and a progressive, steady decline in cognition, and no evidence of mixed etiology. The onset must be insidious with gradual progression over time. These features should not be better accounted for by an Axis I disorder, medical disorder, or delirium.

AD is considered a “cortical dementia” because it primarily results in neuronal loss and atrophy of the cerebral cortex, namely, the medial temporal areas, including the amygdala, hippocampal formation, and entorhinal cortex. In later stages of the disease, the following brain areas may also be affected: basal temporal cortex, parietal–occipital cortex, posterior cingulate gyrus, and frontal lobes. The primary motor, somatosensory, visual, and auditory cortices are relatively spared. The primary pathological changes in AD are beta-amyloid plaques (insoluble protein cores) and neurofibrillary tangles (intracellular protein tangles) which can be found throughout the cortex, although primarily in the limbic cortex region (e.g., the hippocampus), which is involved in memory. Neurotransmitter changes are also present, with primary dysfunction in the cholinergic neurons, which are involved in learning and memory.

Current medications used to treat AD, such as cholinesterase inhibitors, prevent the breakdown of ACh. These medications include galantamine, rivastigmine (Exelon), and donepezil (Aricept). Weight gain, sedation, and rarely, seizures, are some of the side effects. These drugs should not be combined with other cholinesterase inhibitors (Stahl, 2011).
Another medication known as memantine (Namenda) works by regulating glutamate, which, in excess, can lead to cell death. These medications typically slow the progression of AD, rather than restore previously lost cognitive functions. Side effects include dizziness, headache, and constipation (Stahl, 2011).

The diagnosis of AD is based on clinical presentation, obtained through a detailed clinical history and evaluation of cognitive abilities (McKhann et al., 2011). Sophisticated imaging and biomarker techniques, such as PET and CSF assays, are also being developed to identify the pathological hallmarks of the disease, which develop years before the clinical signs of memory loss appear (Sperling et al., 2011). Not everyone who has these pathological changes in their brain will go on to develop AD.

In the early stage of the disease (1–3 years), mild impairments may be seen in memory, particularly new learning and retention of new memories over time, with remote memory being relatively spared. Other cognitive areas affected include visuospatial functioning (e.g., topographic disorientation and difficulty with construction) and language (e.g., word finding and naming). Increased frustration and irritability may also be present. In the intermediate stage (2–10 years), increased impairments in memory, visuospatial skills, and language are present, with the emergence of apraxia, acalculia, aphasia, or agnosia. In the later stages (8–12 years), intellectual functions may be severely impaired, verbal output may be minimal, and the patient may develop problems with his or her gait and motor control.

The greatest risk factor for developing AD is age. Most cases of AD are sporadic, although several risk genes have been implicated. The risk gene with the strongest influence is called apolipoprotein E-e4 (APOE-e4). Scientists estimate that APOE-e4 may be a factor in 20% to 25% of Alzheimer's cases. There is also a rare form of “familial” AD, which is caused by an autosomal dominant gene and often onsets before age 60.

Pick's disease is a rare form of cortical dementia that is caused by degeneration of the frontal and temporal lobes of the brain. Pick's disease is one specific cause of a heterogeneous group of dementias referred to as frontotemporal dementia (FTD). Pathologically, Pick's disease is distinguishable on autopsy by characteristic Pick inclusion bodies usually found in cortical and hippocampal neurons in the frontal and anterior temporal lobes (as opposed to the amyloid plagues and neurofibrillary tangles, which are the hallmark of AD; Heilman & Valenstein, 2003). Dementia due to Pick's disease, as well as other types of FTDs, are characterized by personality changes such as behavioral disinhibition, which often occur early in the course of the disease, as well as executive dysfunction and language abnormalities. Memory problems are also present, but tend to become more obvious later in the disease (as opposed to AD where memory loss is typically the primary presenting problem). Onset is typically younger than that of AD, occurring between ages 50 and 60. There is no treatment for Pick's disease.

Cerebrovascular disease is the second leading cause of acquired dementia following AD and is caused by multiple infarcts, or strokes, in either large vessels or smaller vessels which penetrate deeper in the brain. Dementia due to cerebrovascular disease tends to begin earlier than AD and is more common in men than women. Alternative terminology includes “multi-infarct dementia,” “vascular dementia,” or “vascular cognitive impairment.” The onset is typically abrupt with a “stepwise” or fluctuating course. Risk factors include hypertension, abnormal lipid levels, smoking, diabetes, obesity, cardiovascular disease,
or previous stroke or transient ischemic attacks. Cerebrovascular disease may co-exist with other causes of dementia, including AD (O’Brien et al., 2003). The types of deficits present in vascular dementia are variable and depend on the nature, type, and extent of the cerebrovascular lesions. Focal deficits may be present, as well as gait disturbance or psychomotor retardation. Depression or mood changes are also common. Cognitive deficits common in vascular dementia include psychomotor processing speed, complex attention, and executive functioning. Diagnostic criteria are similar to that of AD but differ in the onset and course of the disease, and focal neurological signs (e.g., gait abnormalities or weakness of an extremity) or evidence of cerebrovascular disease on neuroimaging is required (American Psychiatric Association, 2013). Treatments are often preventive, focusing on the underlying risk factors (e.g., smoking cessation, exercise, and dietary modifications) as well as aspirin, anticoagulants, or antihypertensive medications.

Parkinson’s disease (PD) is a progressive neurodegenerative condition that is characterized clinically by tremor, rigidity, bradykinesia (slowed movement), and postural instability. PD is considered a movement disorder and is caused by the degeneration of the substantia nigra, which is a nucleus in the basal ganglia, and the loss of DA, which is produced by this nucleus. The basal ganglia is a subcortical structure involved in regulating voluntary movement. Lewy bodies are often present in the substantia nigra on autopsy.

Dementia occurs in 20% to 60% of patients (American Psychiatric Association, 2013). Parkinson’s dementia is considered a “subcortical” dementia and may be characterized by deficits in executive functioning, learning and recall aspects of memory, slowed psychomotor speed, and bradyphrenia (slowed thinking). There are typically no cortical disturbances such as aphasia or apraxia. Depression is relatively common and affects approximately 30% of patients with PD.

The major motoric symptoms of PD can be broken down into “positive” and “negative” symptoms. The positive symptoms (actions that are not seen in “normals”) include a resting tremor that often has a “pill rolling” quality, muscular rigidity, or increased muscle tone, and involuntary movements, or akathisia. The negative symptoms (the inability to engage in behaviors that “normals” can do) include difficulty with positioning, difficulty standing from a sitting position, shuffling gait, bradykinesia or slowed movement, and blankness in facial expression (e.g., masked facies).

Treatment includes medications that boost the DA system in the brain, such as levodopa (L-DOPA), a precursor to DA. These medications may become less effective over time as the disease progresses, and there is less and less DA available. Dopamine agonists such as L-DOPA primarily help with the motor symptoms of the disease, but the cognitive symptoms are not improved. Neurosurgery such as deep brain stimulation (DBS) uses a surgically implanted device called a neurostimulator to deliver electrical stimulation to block the abnormal electrical signals within the basal ganglia. This type of treatment treats the motor symptoms of the disease and is used with patients whose symptoms are not adequately controlled with medication (NINDS, n.d.).

Huntington’s disease or Huntington’s chorea is also a movement disorder and is caused by a degenerative loss of neurons in the basal ganglia, particularly the caudate nucleus. Neurotransmitters such as GABA and NE, which normally inhibit the DA pathways, die during the course of the disease, thus creating a hyperactive DA system.
It is an autosomal dominant genetic disorder affecting approximately 5/100,000. The defect causes a part of DNA, called a cytosine, adenine, guanine repeat, to occur many more times than normal.

Offspring have a 50% chance of developing this disorder. The disorder typically appears in the third or fourth decade of life. Dementia almost always occurs and is characterized by a decline in memory retrieval and executive functioning, with more severe deficits in memory and global intellectual functioning later in the disease. Behavioral disturbances occur in up to 50% of cases and are often the initial feature of the disease. These behavioral changes may include depression, personality changes, anxiety, irritability, restlessness, or psychosis (NINDS, n.d.). The abnormal movements associated with this disease include “choreiform movements” (frequent, brisk jerks of the pelvis, trunk, and limbs), athetosis (slow uncontrolled movements), and unusual posturing. These motor symptoms often present months to a year after the disease onsets. Subtle changes in personality, memory, and coordination are often the first symptoms of the disease. There is no treatment for Huntington’s disease. Genetic counseling plays an important role for those with a family history of Huntington’s disease.

Dementia due to HIV disease is primarily a subcortical dementia caused by direct pathophysiological changes in the brain due to HIV. Alternative terminology includes AIDS dementia complex (ADC) or HIV/AIDS encephalopathy. Neuropathological findings include diffuse, multifocal destruction of white matter and subcortical structures, resulting in cognitive, behavioral, and motor symptoms. Cognitive symptoms include forgetfulness, slowness, concentration problems, and problem-solving difficulties. Behavioral manifestations include apathy and social withdrawal as the primary features, although some individuals may experience visual hallucinations, delusions, or delirium. Motor symptoms include tremors, balance problems, impaired repetitive movements, ataxia, and hypertonia.

CD4 counts are an important biomarker of HIV disease, and dementia due to HIV disease is more likely to occur as CD4+ count levels fall below 200 cells per microliter. With the advent of highly active antiretroviral therapy (HAART), the frequency of dementia due to HIV disease has declined from 30% to 60% of people infected with HIV to less than 20%.

**Pseudodementia**

The term “pseudodementia” has been used in the past to describe a dementia-type presentation in a variety of psychiatric illnesses, although depression tends to be the most common cause. This type of dementia may occur in a subset of patients with mood disorders and these features may resemble other neurological etiologies of dementia. Individuals with depression may report various cognitive problems in their daily lives, including slowed processing speed, memory problems, and attention problems. The onset of these cognitive symptoms can sometimes be linked to a precise date of onset (perhaps associated with onset of a life stressor or emotional upset), and the course tends to progress more rapidly than in dementia. Subjective cognitive complaints in depression are typically greater in severity than the actual impairment on testing. Conversely, patients with AD may underestimate their impairments due to the poor insight that is often a hallmark of the later stages of the disease.

Several presenting features can distinguish “organic” dementia from dementia due to depression. First, cortical signs such as aphasia, apraxia, and agnosia are
typically absent in depression. Second, depressed patients may exhibit psychomotor slowing and inconsistent effort or attention during neuropsychological testing, rather than primary problems with retentive memory or visuospatial functioning. Cognitive impairments occurring during the acute stages of depression are typically reversible with treatment for the depressive symptoms. However, it is important to note that depression may also co-occur in the early stages of dementia, and cognitive symptoms in this context would be less likely to improve with antidepressant treatments.

**Mild Cognitive Impairment**

The term *mild cognitive impairment* (MCI) has been coined to capture the transitional time period between normal aging and dementia. MCI is defined as the state in which at least a single cognitive domain, usually memory, is impaired to a greater extent anticipated for someone’s age, although the patient does not meet criteria for dementia and does not exhibit significant changes in their everyday, functional abilities (Peterson, 1995). These individuals are at an increased risk for developing dementia in subsequent years. Since the conception of MCI, four clinical subtypes of MCI have been defined: amnestic MCI-single domain, amnestic MCI-multiple domains, nonamnestic MCI-single domain, and nonamnestic MCI-multiple domain (Busse, Hensel, Gühne, Angermeyer, & Riedel-Heller, 2006).

The course of MCI can last for up to 5 years (Peterson, 1997). When followed longitudinally, individuals with MCI have a significantly increased rate of developing dementia, with conversion rates ranging from 8% to 15% per year, compared to a rate of 1% to 2% per year for the “normal” aging population (Devanand et al., 2008; Peterson et al., 1997). Individuals with amnestic MCI have the highest risk of progressing to AD (Busse et al., 2006).

**Delirium**

Delirium, or acute confusional state, is defined as a disturbance in consciousness accompanied by a change in cognition that cannot be better accounted for by a dementia process. Delirium differs from dementia in that its onset is typically abrupt (developing over the course of hours or days), the course is often fluctuating, and it is oftentimes reversible. A delirium state can be caused by a general medical condition (e.g., infection or metabolic disturbance), substance intoxication, or withdrawal, medication, toxin exposure, or a combination of factors. Delirium is common in inpatient hospital settings, affecting up to 30% of medically hospitalized patients. It is more common in the elderly.

The hallmark feature of delirium is impairment in the ability to focus or sustain attention. A patient with delirium may have difficulty focusing on a conversation or may be easily distracted. In addition to this attention disturbance, the patient may demonstrate a change in memory, orientation, language, or perception.

**Concussion**

Concussion, which is a form of mild traumatic brain injury, is the result of a direct or indirect trauma to the head. Although a loss of consciousness may occur, it is
not necessary for diagnosis. Although many diagnostic frameworks have been developed, all require an identified trauma to the head, as well as at least some alteration of consciousness, posttraumatic amnesia (or amnesia for the event), or some focal neurological deficit.

Most of the literature suggests the presence of concussion-related symptoms for a few hours to several days after the injury. However, a more enduring syndrome of symptoms can occur and is known as postconcussion syndrome (PCS). For most, this syndrome will also resolve usually within the first 3 months. However, there is a smaller group of individuals who can remain symptomatic for over 3 months and, sometimes, up to several years following the injury. Post-concussion syndrome can be associated with a triad of somatic, cognitive, and behavioral symptoms. Somatic symptoms can include disordered sleep, fatigue, headaches, sensitivity to light and/or noise, vertigo or dizziness, and/or nausea. Personality/emotional changes can include anxiety, depressed affect, irritability, and/or apathy. Residual cognitive disturbances can include impaired attention and concentration, diminished short-term memory, slowed learning, decreased processing speed, lack of initiation, and poor planning, organization, and problem solving.

**Seizure Disorders**

A seizure is an episode of abnormal electrical firing of neurons resulting in abnormal behavior or experience of the individual (NINDS, 2004; Zillmer, Spiers, & Culbertson, 2008). The abnormal neuronal firing that occurs during a seizure may result in strange sensations, emotions, behavior, or sometimes convulsions or loss of consciousness. Epilepsy is a condition in which an individual experiences two or more unprovoked seizures. An unprovoked seizure means there is no identifiable cause or trigger. Having a seizure is not the same as being diagnosed with epilepsy. For example, someone may experience an isolated seizure without going on to develop epilepsy. Some children experience febrile seizures, in which a seizure occurs in the context of a high fever. However, most children with febrile seizures do not go on to develop epilepsy. Approximately 1% of the U.S. population has experienced an unprovoked seizure or has been diagnosed with epilepsy (NINDS, 2004). There are many causes of epilepsy. Anything that disrupts the normal pattern of neuronal activity may cause a seizure, for example, brain damage, abnormal development, illness, infection, toxins, drugs, or trauma. In about half of the cases of epilepsy, the exact cause is idiopathic, or unknown (NINDS, 2004).

There are several classifications of seizures. The first main classification is generalized versus partial or focal. In a generalized seizure, both sides of the brain are affected, resulting in loss of consciousness (or altered consciousness), falls, or muscle spasms. There are several types of generalized seizures. In a tonic–clonic generalized seizure, formally known as a grand mal seizure, the individual typically loses consciousness and exhibits stiffening of the body and repetitive jerking of the arms and/or legs. In an absence seizure, formally known as a petit-mal seizure, the person may appear to be staring into space.

Focal seizures, also called partial seizures, affect only one part of the brain. In a simple partial seizure, the individual does not lose consciousness. A person with a simple partial seizure may experience sudden and unexplained joy or anger or may hear, smell, or see things that are not real. In a complex partial seizure, the person experiences an alteration or loss of consciousness. A person having a complex partial seizure may display repetitive movements or behaviors, such as blinks,
twitches, mouth movements, or more complicated actions. Temporal lobe epilepsy is the most common type of recurring focal seizures and may be associated with memory problems due to involvement of the hippocampus. Some people with partial or focal seizures experience an aura, or unusual sensation that warns a seizure is about to happen.

Not all seizures are distinctly partial or generalized. Some seizures may begin as a partial seizure and then spread to the entire brain. In addition, some people may appear to have a seizure, but there is no evidence of seizure activity in their brain. These events are referred to as nonepileptic seizures, formally referred to as pseudoseizures. The cause of these nonepileptic seizures may be psychogenic in origin, and sometimes people with epilepsy also have psychogenic seizures. It can be difficult to distinguish between epileptic and nonepileptic seizures, and careful evaluation and monitoring are required.

Epilepsy is usually diagnosed with EEG monitoring, brain scans, blood tests, neurological or behavioral tests, and a thorough check of medical history. Epilepsy is usually treated with antiseizure medication, although not all individuals with epilepsy respond well to these medications. When medications are not effective in controlling seizures, surgery to remove the affected brain tissue may be considered, depending on the nature and type of seizures.

References


Review Questions

1. All of the following may occur following damage to the basal ganglia, EXCEPT:
   A. Bradykinesia
   B. Rigidity
   C. Paralysis
   D. Tremor

2. The primary function of the myelin sheath is to:
   A. Increase the strength of the nerve impulse
   B. Determine whether the postsynaptic neuronal response is excitation or inhibition
   C. Determine whether the postsynaptic nerve will fire an action potential
   D. Increase the speed of neuronal firing

3. An acute intracranial hemorrhage will appear ___________ on a CT scan, which is often referred to as a ___________.
   A. White/hyperdensity
   B. White/hypodensity
   C. Black/hyperdensity
   D. Black/hypodensity

4. Your patient is a 7-year-old child. His mother reports that he frequently exhibits staring spells during which time he is unresponsive. These spells last several seconds, and he subsequently appears lethargic. The first diagnostic tool you should recommend is:
   A. A WADA test
   B. The Glasgow Coma Scale
   C. An EEG
   D. A neuroangiogram

5. The person who spontaneously utters the following phrase, “window… break… ball” but who cannot repeat the phrase, “The ball broke the glass window” most likely has which of the following disorders of speech:
   A. Wernicke’s aphasia
   B. Conduction aphasia
   C. Transcortical motor aphasia
   D. Broca’s aphasia

6. The primary site of brain deterioration in Alzheimer's dementia is usually the:
   A. Medial temporal lobe
   B. Medial parietal lobe
   C. Frontal–temporal lobes
   D. Temporal–parietal lobes
7. Which of the following answers correctly pairs the stage of Alzheimer’s disease with the corresponding symptom presentation?
   A. Stage 1: Aphasia, apraxia, and/or acalculia
      Stage 2: Agnosia
      Stage 3: Declines in memory, visuospatial functioning, and language
   B. Stage 1: Declines in memory, visuospatial function, and language
      Stage 2: Apraxia, aphasia, and/or acalculia
      Stage 3: Impaired intellectual functioning and minimal verbal output
   C. Stage 1: Declines in memory and intellectual functioning
      Stage 2: Emergence of apraxia, aphasia, and/or acalculia
      Stage 3: Agnosia
   D. Stage 1: Impaired intellectual functioning, memory problems, and apraxia
      Stage 2: Minimal verbal output
      Stage 3: Declines in visuospatial functioning and language

8. Personality changes and executive dysfunction are primary features of which following disease?
   A. Parkinson’s disease
   B. Alzheimer’s disease
   C. Huntington’s disease
   D. Pick’s disease

9. The primary brain region affected in Huntington’s disease is the:
   A. Caudate nucleus
   B. Putamen
   C. Substantia nigra
   D. Globus pallidus

10. Which of the following statements is NOT true?
    A. Delirium is marked by an abrupt onset and fluctuating course.
    B. The hallmark feature of a delirium state is impaired attention.
    C. Delirium is relatively uncommon among patients hospitalized for non-neurological conditions.
    D. Delirium can be caused by prescription medications, toxin exposure, or metabolic disturbance.

11. A seizure that is due to abnormal electrical activity in the left temporal lobe and is characterized by an alteration in consciousness and repetitive movements such as lip smacking or undoing a button would most likely be referred to as what type of seizure?
    A. Absence seizure
    B. Generalized tonic–clonic seizure
    C. Complex partial seizure
    D. Simple partial seizure

12. Which of the following is NOT a protective covering of the brain?
    A. Arachnoid
    B. Sphenoid
    C. Dura
    D. Pia
    E. C and B
13. Which of the following are regions of primary sensory processing?
A. Occipital lobe
B. Thalamus
C. Temporal lobe
D. A and B
E. A and C

14. Which of the following structure is NOT considered part of the basal ganglia?
A. Globus pallidus
B. Pineal
C. Subthalamic nucleus
D. Putamen

15. An impulse traveling away from the cell body of the neuron travels along the __________.
A. Axon
B. Dendrite
C. Soma
D. Terminal button

16. The process by which incoming stimuli cross over and are transmitted to the contralateral hemisphere is known as:
A. Myelination
B. Differentiation
C. Propagation
D. Decussation

17. The lateral geniculate nucleus of the thalamus __________.
A. Maintains balance and coordination
B. Relays visual information to the occipital lobe
C. Processes auditory information
D. Secretes cerebral spinal fluid

18. The telencephalon, or cerebrum, includes which of the following structures?
A. Frontal Lobes
B. Occipital lobes
C. Pons
D. A, B, and C
E. A and B

19. The role of mirror neurons has been associated with __________.
A. Empathy
B. Primary visual processing
C. Hand–eye coordination
D. Imitation
E. A and D
20. As part of the limbic system, the hippocampus is most often associated with
   __________.
   A. Memory formation and transfer to longer-term storage
   B. The regulation of emotional responses to the environment
   C. Relaying of sensory information to primary processing regions
   D. Hunger

21. Multiple sensory modalities are integrated in cortical regions known as
     __________.
     A. The parietal lobes
     B. The midbrain
     C. Heteromodal cortex
     D. The falx cerebri

22. Typically located in the language-dominant hemisphere, __________ is a secondary processing region dedicated to the comprehension of language.
    A. The parastriate region
    B. Broca’s area
    C. The orbitofrontal cortex
    D. Wernicke’s area

23. __________ has/have increasingly replaced __________ for the first-line treatment of chronic anxiety.
    A. Barbiturates, SSRIs
    B. Benzodiazepines, SSRIs
    C. SSRIs, benzodiazepines
    D. Alcohol, barbiturates

24. Cardiac/autonomic, severe anticholinergic, and neurobehavioral are types of side effects of __________.
    A. TCAs
    B. Anticonvulsants
    C. SSRIs
    D. Anxiolytics

25. A drug known for having a narrow therapeutic index is __________.
    A. Lithium
    B. Ambien
    C. Cymbalta
    D. Concerta

26. Fatal agranulocytosis is a side effect of __________.
    A. Clozapine (Clozaril)
    B. Alprazolam (Xanax)
    C. Dextroamphetamine (Adderall)
    D. Buspirone (BuSpar)
27. SAMe is ___________.
   A. An alternative remedy for depression
   B. An antipsychotic
   C. A stimulant
   D. A cognitive enhancing drug

28. SSRIs are used to treat ___________.
   A. Anxiety
   B. Depression
   C. Anxiety and depression
   D. Psychosis

29. Barbiturates are ___________.
   A. Essentially replaced by other psychotropic medications, including benzodiazepines
   B. Sleep medication
   C. Antidepressants
   D. Cognitive enhancing drugs

30. Pharmacokinetics refers to ___________.
   A. How the body handles a drug, including absorption, distribution, and elimination and metabolism
   B. A drug's mechanism of action
   C. Amino acids, biogenic amines, and peptides
   D. How a drug brings about unwanted side effects
Answers to Review Questions

1. C. Paralysis
   Motor abnormalities due to basal ganglia dysfunction do not involve paresis or paralysis, but rather refer to abnormal coordination and rhythm of movement. These syndromes are referred to as extrapyramidal syndromes.

2. D. Increase the speed of neuronal firing
   The myelin sheath increases the speed of neuronal firing down the axon. Whether a postsynaptic nerve is excited or inhibited depends on the neurotransmitters binding to that neuron. The relative threshold of postsynaptic excitation determines whether an action potential will fire. The action potential is an all-or-nothing phenomenon, and the strength of neuronal firing does not vary in response to the strength of the input.

3. A. White/hyperdensity
   The appearance of brain tissue on a CT scan depends on the tissue density. Very dense tissue, such as bone, appears white. Less dense tissue, such as air, appears black. The term hyperdense refers to brighter areas and hypodense refers to darker areas. Fresh intracranial hemorrhage coagulates almost immediately and shows up as a hyperdense (white) area.

4. C. An EEG
   This child could be having seizures, and an EEG is the primary diagnostic tool used to diagnose epilepsy. The Glasgow Coma Scale is used to determine the severity of traumatic brain injury. The WADA test helps to determine relative hemispheric capabilities underlying language and memory and is often used in patients with epilepsy prior to undergoing brain resection. A neuroangiogram is used to detect lesions of blood vessels.

5. D. Broca’s aphasia
   Broca’s aphasia is considered a nonfluent aphasia in which the person speaks in a slow, halting manner, with poor grammar and limited prosody. Only keywords are used, and use of verbs or connecting words is limited. Damage is usually in the left frontal lobe around Brodmann areas 44 and 45, also known as Broca’s area. Writing is usually slow and effortful. Repetition is impaired. Auditory comprehension and reading comprehension are relatively preserved.

6. A. Medial temporal lobe
   The primary site of brain involvement in AD is the medial temporal lobes, including the amygdala, hippocampal formation, and entorhinal cortex. In later stages of the disease, the basal temporal cortex, parietal–occipital cortex, posterior cingulate gyrus, and frontal lobes are also affected. The primary motor, somatosensory, visual, and auditory cortices are relatively spared.
7. **B. Stage 1: Declines in memory, visuospatial function, and language**  
   **Stage 2: Apraxia, aphasia, and/or acalculia**  
   **Stage 3: Impaired intellectual functioning and minimal verbal output**  
   In the early stage of the disease (1–3 years), mild impairments may be seen in memory, particularly new learning and retention of new memories over time, with remote memory being relatively spared. Other cognitive areas affected include visuospatial functioning (e.g., topographic disorientation and difficulty with construction) and language (e.g., word finding and naming). In the intermediate stage (2–10 years), increased impairments in memory, visuospatial skills, and language are present, with the emergence of apraxia, acalculia, aphasia, or agnosia. In the later stages (8–12 years), intellectual functions may be severely impaired and verbal output may be minimal.

8. **D. Pick's disease**  
   Pick's disease is one specific cause of a heterogenous group of dementias referred to as FTD. Dementia due to Pick's disease, is characterized by personality changes such as behavioral disinhibition, which often occur early in the course of the disease, as well as executive dysfunction and language abnormalities. Memory problems are also present, but tend to become more obvious later in the disease (as opposed to AD where memory loss is typically the primary presenting problem). Onset is typically earlier than that of AD, occurring between ages 50 and 60.

9. **A. Caudate nucleus**  
   Huntington's disease is an autosomal dominant genetic movement disorder that is caused by a degenerative loss of neurons in the basal ganglia, particularly the caudate nucleus. The abnormal movements associated with this disease include choreiform movements (frequent, brisk jerks of the pelvis, trunk, and limbs), athetosis (slow uncontrolled movements), and unusual posturing.

10. **C. Delirium is relatively uncommon among patients hospitalized for non-neurological conditions.**  
    Delirium can be caused by a variety of general medical conditions and occurs and affects up to 30% of medically hospitalized patients.

11. **C. Complex partial seizure**  
    Temporal lobe epilepsy is the most common type of complex partial seizure, in which the seizure activity is localized to the temporal lobe, and the person experiences an alteration of or loss of consciousness during the seizure.

12. **B. Sphenoid**  
    The sphenoid is a sinus. The dura, arachnoid, and pia are the three layers of the cranial meninges that form the cranial meninges and protective coverings of the brain.
13. E. A and C

The thalamus is typically considered a relay for pathways between cortex and lower brain regions. On the other hand, the occipital lobe is the site of primary visual processing and the temporal lobe is the site of primary auditory processing.

14. B. Pineal

The pineal is a small gland between the hemispheres. However, the globus pallidus, subthalamic nucleus, and putamen are all recognized as structures of the basal ganglia.

15. A. Axon

Only the axon carries the nerve impulse away from the cell. The impulse ends at the terminal buttons, which are located at the ends of the axons. The soma is the cell body and the dendrite carries impulses toward the cell body.

16. D. Decussation

Decussation refers to the structural crossing of fibers, which accounts for contralateral control and perception.

17. B. Relays visual information to the occipital lobe

The sensory pathways travel through modality-specific nuclei located in the thalamus. The lateral geniculate nuclei are specific to vision.

18. E. A and B

The pons is a midbrain structure. From these choices, only the frontal and occipital lobes are part of the telencephalon, which comprises the four lobes.

19. E. A and D

Research has suggested that mirror neurons located in premotor cortex are associated with empathy and imitation.

20. A. Memory formation and transfer to longer-term storage

Damage to the hippocampus, located in the inferior temporal lobe, is associated with severe impairments in memory because of its important role in the transfer of information to long-term storage.

21. C. Heteromodal cortex

Only the heteromodal cortex processes and integrates multiple sensory stimuli.

22. D. Wernicke’s area

Wernicke’s area represents a higher level of processing but is still modality specific. Therefore, it is considered a secondary processing region.

23. C. SSRIs, benzodiazepines

Because benzodiazepines have the potential to be addictive, SSRIs are now the first-line treatment for chronic anxiety.
24. A. TCAs
   *These are the serious side effects of the TCAs.*

25. A. Lithium
   *Lithium can be dangerous as the therapeutic dose is near fatal.*

26. A. Clozapine
   *Fatal agranulocytosis is a risky side effect of clozapine (Clozaril).*

27. A. An alternative remedy for depression
   *SAMe is an over-the-counter remedy for depression.*

28. C. Anxiety and depression
   *SSRIs are used to treat anxiety and depression.*

29. A. Essentially replaced by other psychotropic medications, including benzodiazepines
   *Barbiturates are now rarely used as psychotropic medication.*

30. A. How the body handles a drug, including absorption, distribution, and elimination and metabolism
   *Pharmacokinetics is the study of the mechanisms of absorption, distribution, metabolism, and excretion of a drug in the body.*