

Pathophysiology: The Central Nervous System

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Faculty

Jane C. Norman, RN, MSN, CNE, PhD, received her undergraduate education at the University of Tennessee, Knoxville campus. There she completed a double major in Sociology and English. She completed an Associate of Science in Nursing at the University of Tennessee, Nashville campus and began her nursing career at Vanderbilt University Medical Center. Jane received her Masters in Medical-Surgical Nursing from Vanderbilt University. In 1978, she took her first faculty position and served as program director for an associate degree program. In 1982, she received her PhD in Higher Education Administration from Peabody College of Vanderbilt University. In 1988, Dr. Norman took a position at Tennessee State University. There she has achieved tenure and full professor status. She is a member of Sigma Theta Tau National Nursing Honors Society. In 2005, she began her current position as Director of the Masters of Science in Nursing Program.

Faculty Disclosure

Contributing faculty, Jane C. Norman, RN, MSN, CNE, PhD, has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

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Division Planner/Director Disclosure

The division planner and director have disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

Audience

This course is designed for nurses working in critical care and general and specialty medical-surgical units in which patients with multiple organ system problems are found.

Accreditations & Approvals



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Course Objective

As health care becomes more complex, it is essential that the theoretical concepts of the basis of illness (pathophysiology) be well understood. The purpose of this course is to reinforce the scientific rationales for the interventions nurses perform and the decisions nurses make as patients move through the ever-changing management of their central nervous system disorder.

Learning Objectives

Upon completion of this course, you should be able to:

1. Identify the key structures and functional inter-relationships in the central nervous system (CNS).
2. Discuss structures and mechanisms in place to protect the CNS.
3. Describe the components and functions of CNS circulation.

4. Discuss the pathophysiologic and environmental influences and effects on the CNS.
5. Outline the role of subjective data in completing a full nursing assessment of the CNS.
6. Describe objective data compiled during a nursing assessment of the CNS.
7. Identify diagnostic tests used in the identification and classification of CNS diseases.
8. Outline the nursing diagnoses, planning, and management of conditions related to CNS dysfunction.
9. Discuss clinical manifestations of congenital diseases of the CNS.
10. Review signs and symptoms of CNS disorders of multifactorial origin and related nursing actions.
11. Describe the common causes, appearances, and treatment of degenerative CNS disorders.
12. Analyze the presentation and nursing management of immunologic CNS disorders.
13. Evaluate pathologic causes and manifestations of infectious and inflammatory disorders of the CNS.
14. Discuss the pathophysiology and clinical manifestations of neoplastic and obstructive CNS disorders.
15. Outline the concepts and information the nurse should provide for the patient who has sustained a traumatic CNS injury.



Sections marked with this symbol include evidence-based practice recommendations. The level of evidence and/or strength of recommendation, as provided by the evidence-based source, are also included so you may determine the validity or relevance of the information. These sections may be used in conjunction with the course material for better application to your daily practice.

INTRODUCTION

The nervous system is the body's most organized and complex structural and functional system, and it profoundly affects both psychological and physiologic function. This course discusses the importance of the central nervous system (CNS) to human function and the major consequences of central neurologic disorders. The onset of neurologic problems may be sudden, as in traumatic spinal cord severance or ruptured aneurysm, or insidious, as in Parkinson disease or multiple sclerosis. Providing care to patients experiencing neurologic disorders is challenging and requires extensive knowledge of neurologic structures and function and neurologic disease processes.

Neurologic problems can be frightening and even devastating to the patient and family involved, especially if the process is irreversible. Many such problems produce varying degrees of physical and/or psychosocial dependency. Physical disabilities may limit self-care, and memory loss and confusion can occur. Subtle or gross changes in consciousness may develop, and patients may not be responsible for their behavior at times. A person's entire way of life may be altered. This course provides the information necessary to plan appropriate nursing care for individuals experiencing CNS problems in both acute and rehabilitative states. An overview of CNS anatomy, physiology, and pathophysiology is detailed, as are assessment, diagnostic tests, pathologic findings, planning, intervention, and evaluation of nursing care provided for these patients.

THE NERVOUS SYSTEM IN HEALTH AND ILLNESS: STRUCTURAL AND FUNCTIONAL INTER-RELATIONSHIPS

Every physical, mental, and emotional aspect of a person's existence is influenced by continually changing internal and external environments. To deal with these changes effectively, the nervous system perceives and interprets the changes and then quickly and continuously initiates, coordinates, and modulates body responses. This job is endless, because the body and the environment are never static. Internal changes include the process of aging, anabolic and catabolic activities, and psychological states; external changes include ambient temperatures, light, noise levels, and colonies of micro-organisms.

The nervous system not only plays a key role in the management of body functions; it also depends on other body systems. Indeed, the nervous system quickly malfunctions if its sources of nourishment, its waste management systems, or its protective mechanisms are lost or impeded.

The brain is structurally divided into components according to its embryologic development. These components, known as the forebrain (prosencephalon), midbrain (mesencephalon), and hindbrain (rhombencephalon) are further divided according to their location within the adult brain. Knowing these divisions is useful because they are often involved in CNS function or pathology [7; 10].

An understanding of structure and function and the interdependence of body systems is an essential part of the nurse's knowledge base. With this knowledge, nurses can effectively assess and plan care of the patient with CNS dysfunction.

NEURONS

Structure of the Neuron

Neurons are the primary components of the nervous system. Working alone or as units, neurons detect environmental changes and initiate body responses needed to maintain homeostasis. Each neuron is composed of a cell body, an axon, and a number of dendrites. Both axons and dendrites vary in size and shape. The axons, ranging in length from miniscule to over a meter, transmit messages throughout the central and peripheral nervous systems. Each cell has only one axon, but axonal branching is common and allows for broader dissemination of neuronal transmissions. Dendrites, the processes of neurons that conduct electrical impulses to the cell body, also have varying branching patterns. These characteristics allow for the efficient transmission and reception of impulses throughout the body [2; 3].

Many central axons are wrapped in insulating sheaths of a white, fatty substance called myelin. The myelin is encased in special cells lying end to end along the axons. Junctions, known as nodes of Ranvier, occur where these cells abut, allowing for more rapid transmission of electrical impulses. Axons often branch at these nodes [2; 3].

Two distinct types of cells cover axons of the nervous system. Those located in the CNS are known as oligodendrocytes. These cells and the neurons they protect cannot be replaced or repaired if damaged. Schwann cells, which surround peripheral nervous system myelinated and unmyelinated axons, form the neurilemma, the outer membrane that supports and protects peripheral nervous system axons and may facilitate the healing of damaged axons [2; 3].

Groups of neurons called nuclei provide routes for the transmission of complex afferent and efferent impulses. Fasciculi are bundles of neurons; groups of fasciculi, encased in a covering called epineurium, are referred to as nerves. Most nerves contain afferent (toward the CNS) and efferent (from the CNS) fibers [2; 3].

Structurally distinct neurons are responsible for receiving and sending specific messages to the brain about the body's internal and external environment. There are five major types of sensory receptors [2; 3]:

- Mechanoreceptors: Receive impulses related to pressure, touch, and mechanical deformation of the receptor
- Thermoreceptors: Respond to heat and cold
- Nociceptors: Receive messages about pain caused specifically by physical or chemical damage
- Electromagnetic receptors: Respond to light on the retina
- Chemoreceptors: Sense flavors, odors, oxygen levels, osmolality of body fluids, and the concentration of carbon dioxide

Each type of receptor is sensitive to the particular stimuli it is designed to receive and almost nonresponsive to other types of stimuli. For example, a nociceptor can be stimulated by electricity, heat, crushing, or other tissue damage that will be experienced as pain. The nociceptor will not, however, respond to light. Each sensory nerve terminates at specific points in the CNS, where the message is interpreted [2; 3].

CNS and peripheral nervous system neurons cannot function independently. Their nutritional and physical support and protection are provided by other cells commonly referred to as glial cells. Glial cells, unlike neurons, are able to undergo mitosis. Astrocytes, star-shaped cells with many projections, are the largest and most numerous glial cells. They provide structural support and nutrition to neurons and maintain a biochemical environment supportive of nerve impulse transmission and synaptic activity. If nervous tissue is destroyed, astrocytes multiply (a process called gliosis) to fill in the area or line a cavity. Microglia, considered the phagocytes of the CNS, are classified as part of the body's reticuloendothelial system. They remove dead tissue and foreign matter. Ependymal cells are involved in cerebrospinal

fluid (CSF) system function. They line the choroid plexuses of the ventricular system, the ventricles, and central canal of the spinal cord. Oligodendrocytes and Schwann cells, previously considered for their role in the encasement and protection of axons, are also classified as neuroglia [7; 10].

Neuronal Function

Functionally, neurons are recognized as being motor (efferent) neurons, sensory (afferent) neurons, or internuncial (transmitters of messages) neurons. Neuronal messages are transmitted through electrical impulses, with the necessary voltages created by positive and negative forces produced while ions line up inside and outside the cell's plasma membrane. When a nerve is in a resting state (known as a resting membrane potential), the electrical charge outside the wall is positive and the charge inside is negative [43; 44; 45].

The principal extracellular cation is sodium; the main intercellular cation is potassium. With stimulation of the cell, the charge is reversed, as sodium moves into the cell and potassium moves out. This reversal results in a flow of electric current. With sufficient stimulation, the reversal of polarity travels along the entire axon. This process, known as an action potential, requires only a few milliseconds. Quickly, electrical forces and ion concentration forces re-establish the resting membrane potential. If a stimulus is not sufficient to produce an action potential and another stimulus occurs before the membrane has completely stabilized, depolarization will be facilitated [43; 44; 45].

Information is transmitted from one neuron to another at synapses following the initiation of an action potential. In humans, chemical synapses initiate almost all action potentials. These synapses, located where axons and dendrites meet, employ various neurotransmitters, which are stored in and released from the axon terminal following an action potential. Action potentials are believed to

increase the permeability of the axon terminal to calcium, allowing it to move into the axon terminal to stimulate the release of neurotransmitters into the synaptic cleft. The neurotransmitter diffuses across the cleft and attaches to postsynaptic receptors [43; 44; 45].

The influence of neurotransmitters on the postsynaptic receptor depends on the combination of impulses received. This combination is derived from the total number and frequency of impulses received over a period of time from one or multiple synapses. Strong stimuli activate a greater number of neurons. Myelinated fibers speed the transmission of impulses. Inhibitory impulses also influence the postsynaptic receptors' response. Through these combinations, the nervous system "fine tunes" synaptic activity needed to manage the body's complex functions. Nerve impulses are binary (either "on" or "off"), so the CNS must discriminate among stimuli by interpreting variations in strength, frequency, and number of stimuli received [43; 44; 45].

Synapses between neuron effector junctions are similar to chemical synapses between two neurons. A review of the events that produce skeletal muscle contraction serves as a good example of how these synapses work. Following depolarization of the axon terminal and movement of calcium into the terminal, acetylcholine is released. It diffuses across the synaptic cleft at the neuromuscular junction to the plasma membrane of the muscle cell and attaches to the receptor sites, causing an increased permeability of the muscle fiber membrane to sodium and potassium ions. If the impulse, known as an endplate potential, is sufficient to depolarize the muscle-fiber membrane, a propagated action potential leads to contraction of the muscle fiber. At the cleft site, a small portion of acetylcholine diffuses away but most is quickly inactivated by the enzyme cholinesterase, located on the muscle-cell membrane, which prevents continued excitation of the muscle fibers [43; 44; 45].

There are many other neurotransmitters. About 30 are known or suspected to play a role in nerve-impulse transmission; some have multiple actions. For example, norepinephrine is involved in the maintenance of arousal and dreaming sleep and regulation of moods; dopamine has roles in the regulation of emotional responses and control of complex movements; and endorphins and enkephalin are believed to be involved in the perception and integration of pain and emotional experiences [43; 44; 45].

The Cranial Nerves

The functions of cranial nerves (CNs) vary; they may be motor, sensory, or mixed. Motor nerves are innervated with proprioceptive (sensory) branches. The parasympathetic branch of the autonomic nervous system provides a visceral component for some cranial nerves. The 12 pairs of cranial nerves, identified by roman numerals, are ordered by their position within the skull [47].

Cranial Nerve I (Olfactory Nerves)

CN I nerves, made up of sensory receptor cells within the epithelial lining of the nasal mucosa, are responsible for the perception of odors. Nerve impulses originating here are transmitted to the temporal lobes for interpretation [47].

Cranial Nerve II (Optic Nerves)

The optic nerves, which are actually nerve tracts, originate in the retina of the eye and enter the cranium via the optic foramina. Nerve impulses are transmitted to the occipital lobe, where vision is perceived. Optic nerve projections from the orbits meet at the optic chiasm. Here, each tract divides, the inner halves joining with fibers from the opposite orbit. From this point, each tract carries fibers from both eyes. Some fibers important for visual reflexes synapse in the midbrain, but most travel to the thalamus to synapse with neurons that form pathways called optic radiations. These fibers terminate in the visual cortex of the occipital lobe [47].

Cranial Nerve III (Oculomotor Nerves)

The oculomotor nerves emerge from the midbrain and enter the orbits through the superior orbital fissures. They are responsible for movement of four of the six extrinsic eye muscles and for opening the eyelid. Parasympathetic innervation supplies the ciliary muscle and the sphincter muscle of the iris to control visual accommodation and adjustment to light intensity [47].

Cranial Nerve IV (Trochlear Nerves)

The trochlear nerves arise from the dorsal side of the midbrain. CN IV is responsible for voluntary movement of the eyeball through its innervation of the superior oblique muscle [47].

Cranial Nerve V (Trigeminal Nerves)

Trigeminal nerve fiber locations are widespread. Both sensory and motor fibers exit from the pons, and some sensory nuclei are located in the medulla. Deep and superficial sensory fibers innervate the face and anterior portion of the head through the ophthalmic, maxillary, and mandibular branches. Sensory fibers for pain, light, touch, and proprioception (i.e., awareness of one's position in space) can be readily identified. The motor components of this nerve are responsible for mastication [47].

Cranial Nerve VI (Abducens Nerves)

CN VI nerves exit from the medulla just below the pons and enter the orbits with CN III and IV. They function to roll the eyes outward [47].

Cranial Nerve VII (Facial Nerves)

CN VII nerves project from the lower edges of the pons. They supply motor neurons for the facial and scalp muscles. Sensory fibers supply the taste buds to detect sweet, sour, and salt on the anterior two-thirds of the tongue. Parasympathetic fibers supply the lacrimal glands and the submandibular and sublingual salivary glands [47].

Cranial Nerve VIII (Vestibulocochlear Nerves)

There are two sensory divisions to the eighth cranial nerves—an auditory division and a vestibular division. Both originate at inner-ear receptors located in the petrous portion of the temporal bones. The two divisions, enclosed in a single sheath, pass to the brain stem just below the pons. Some of the vestibular fibers travel directly to the cerebellum. Auditory impulses are transmitted to the temporal lobes for interpretation [47].

Cranial Nerve IX (Glossopharyngeal Nerves)

The nuclei for CN IX, located in the medulla oblongata, innervate the tongue and pharynx. The motor component is important in swallowing. Sensory responsibilities include perception of bitter taste on the posterior one-third of the tongue; sensory awareness for the mucous membranes of the pharynx, tonsils, and middle ear cavity; carotid body receptor sensitivity to serum oxygen and carbon dioxide levels; and baroreceptor information regarding blood pressure. Parasympathetic neurons innervate the parotid gland [47].

Cranial Nerve X (Vagus Nerves)

The vagus nerve nuclei, also located in the medulla, carry motor impulses to and sensory impulses from the pharynx and larynx. Extensive parasympathetic nerve fibers innervate the pharynx, larynx, and trachea and extend into the thorax and abdomen. Thoracic and abdominal vagal branches influence the function of the esophagus, lungs, aorta, stomach, gallbladder, spleen, small intestine, kidneys, and upper two-thirds of the large intestine. Sensory fibers from the vagus nerve related to visceral functions generally operate at an unconscious level. An exception is nausea, which is perceived via the vagus nerve [47].

Cranial Nerve XI (Accessory Nerves)

CN XI is formed by two nerves. One projects from the medulla; the other, projecting from the fifth or sixth cervical segment of the spinal cord, is a spinal nerve. Fibers from the cranial portion join with the vagus nerve to supply muscles of the larynx and pharynx. Fibers from the spinal component innervate the trapezius and sternocleidomastoid muscles [47].

Cranial Nerve XII (Hypoglossal Nerves)

The hypoglossal nerves exit from the medulla oblongata and pass through the hypoglossal canals located beneath the tongue. These nerves are responsible for tongue movement [47].

THE BRAIN

The outer area of the cerebral cortex is composed of gray matter in complex folds (gyri) or convolutions separated by deep depressions (fissures) and shallow depressions (sulci). These folds make the surface area much greater. The patterning of gyri and sulci is similar in all individuals. The following well-marked fissures are distinguishable in all brains [7; 10]:

- The longitudinal fissure: Separates the right and left hemispheres of the brain
- The central sulcus (fissure of Rolando): Extends outward and downward over the hemisphere
- The lateral fissure (fissure of Sylvius): Begins on the underside of the brain and moves out and around the brain along its side

Each hemisphere of the cerebral cortex is divided into lobes. The frontal lobe is located anterior to the central sulcus and above the lateral fissure. The parietal lobe is positioned behind the central sulcus.

The temporal lobe is located below the frontal and parietal lobes (below the lateral fissure) and merges posteriorly with the occipital lobe. The occipital lobe extends from the parieto-occipital sulcus inferiorly around the base of the cerebrum. The insula (island of Reil or central lobe) lies within the lateral cerebral fissure. The cerebellum, which lies below the occipital lobe, is separated by the deep transverse fissure into which a dural fold, called the tentorium cerebelli, extends [7; 10].

Nerve fiber tracts establish connections between areas within the brain. The corpus callosum consists of fibers extending between the right and left hemispheres. The commissural tracts of the corpus callosum located deep in the longitudinal fissure extend from one convolution to a corresponding one in the opposite hemisphere. The internal capsule also allows networking between areas within the brain. The internal capsule is comprised of two distinct sections referred to as anterior and posterior limbs. Here, afferent and efferent fibers extend from an extensive, fanlike radiation of fibers in the cerebrum to link with the brain stem and spinal cord. Short association fibers extend from one convolution to another in the same hemisphere. Long association tracts interconnect cortical regions in different lobes of each hemisphere [7; 10].

The basal ganglia are coated with the white matter of the cerebral hemispheres. This area contains the caudate nucleus, putamen, globus pallidus, thalamus, subthalamus, substantia nigra, and the red nucleus. These structures have many and varied functions, including sensory and motor activities and transmission of afferent and efferent signals to appropriate parts of the nervous system. The hypothalamus, a small but extremely important area of the brain, is situated just below the thalamus. It receives input from all parts of the body, both by neuronal transmission and its blood supply. The hypothalamus, in turn, influences body functions via these same routes [7; 10].

The Cerebellum

The cerebellum lies below the tentorium cerebelli in the posterior inferior portion of the cranial vault. It is made up of two hemispheres connected in the center by a structure called the vermis. The superficial area of the cerebellum is composed of gray matter, which lies in even, horizontal folds forming fissures and sulci. White fiber tracts lying below the gray matter provide extensive afferent and efferent connections with the brain stem, cortex, thalamus, and basal ganglia. Cerebellar efferent signals travel to the brain stem, thalamus, and motor cortex [18].

Cerebellum Function

The cerebellum modulates and coordinates skeletal muscle activity and maintains body posture and muscle tone. It controls movement with both excitatory and inhibitor signals, which modulates fine movements in ways the cerebral cortex is incapable of carrying out. Each hemisphere influences the movement of the ipsilateral side of the body and modifies activity initiated elsewhere in the body. There is no conscious input [48].

Activities of the cerebellum derive from the multiple inputs from the CNS and peripheral nervous system. Afferent fibers travel to the cerebellum from the cerebral cortex by way of the corticocerebellar tracts and the pons. Peripheral afferent impulses from muscle spindles, Golgi tendon organs, skin, and joint receptors travel to the cerebellum via the ventral and dorsal spinocerebellar tracts. The reticular substance of the brain stem and vestibular tracts also provide the cerebellum with information [48].

Cerebellar efferent impulses are sent to the motor cortex via the thalamus. Additional efferent signals are transmitted to the basal ganglia, red nucleus, reticular formation of the brain stem, and vestibular nuclei. The connections with vestibular nuclei integrate changes in the direction of body movement and posture. The semicircular canals of the inner ear perceive these changes and transmit this information to the cerebellum via the vestibular nerve and the brain stem. Balance is maintained through modification of muscle tone. The cerebellum has no direct influence on lower motor neurons [48].

The cerebellum is also involved with predictively coordinating visual cues with bodily motion. For example, the cerebellum is the area of the brain responsible for processing how rapidly an object is approaching. The findings of an experiment on monkeys illustrate the value of this function. In this experiment, when the portion of the cerebellum involved in vision was removed, the monkey could not judge distance from a corridor wall and repeatedly charged into the wall [48].

Frontal Lobes

The frontal lobes of the brain are involved in mental, emotional, and physical functions. Anterior portions have a major role in the control of conscious and unconscious behaviors such as personality, social behavior, judgment, and complex intellectual activity. The central and posterior portions of the frontal lobes control motor function. The primary motor areas, located in precentral gyri, control voluntary movement via the pyramidal tracts. The premotor areas control and coordinate complex, learned movements such as typing, writing, scanning eye movements, conjugate deviation of the eyes, and movement of the head. These activities are affected via the extrapyramidal tracts. Pyramidal and extrapyramidal centers control movements of the opposite side of the body. The dominant frontal lobe also contains Broca's motor speech area [7; 10].

Parietal Lobes

The parietal lobes interpret sensory input. The postcentral convolutions, organized similarly to the major motor strip, receive conscious sensory input. Sensations perceived on one side of the body are interpreted by the contralateral parietal lobe. Somatic sensations perceived include pain, temperature, touch, pressure, and proprioception. The parietal lobes contain the somesthetic association areas, which lie in the superior portion of the lobes and extend to the medial surface of the hemisphere.

Many other connections within the parietal lobe allow for interpretation of sensory input such as stereognosis (i.e., perceiving and understanding an object by touch and relating the sensations to experience and knowledge). Awareness of body parts and establishment of body image also take place here. The angular gyrus, located in the parietal lobe of the dominate hemisphere, is responsible for interpretation of written language [7; 10].

Temporal Lobes

The temporal lobes receive input from three senses—hearing, taste, and smell—and have a role in memory processes. Association fibers, especially in the dominate lobe, allow the comparison of sensory input with past experiences. These fibers, particularly those of the dominant lobe, inter-relate somesthetic visual and auditory stimuli to give them meaning. Wernicke's area, also located on the dominate side, is involved in the hearing component of speech and in the formulation of language [7; 10].

Occipital Lobes

The occipital lobes contain the primary visual and visual association areas. The primary visual areas receive information and perceive color, while the visual association areas give visual input meaning and have a role in visual reflexes for fixing the eyes on a stationary or moving object. Injury to the medial surface on the dominate side can result in loss of the ability to recognize objects and know their function, although recognition of faces still is possible. A consequence of damage to the nondominant side may be the inability to recognize faces and differentiate various animals, such as horses and elephants [7; 10].

Insula

The insula, thought by some to be a fifth lobe of the brain, lies deep within the lateral fissure, where it is covered by portions of the frontal, temporal, and parietal lobes. It is believed to be involved in visceral activities related to intra-abdominal sensations and visceral motility. Little information is available regarding function [7; 10].

Limbic System

The limbic system consists of a group of structures, including the olfactory bulbs, septum pellucid, fornix, cingulate gyrus, parts of the basal ganglia (including the amygdaloid nucleus), hippocampus, uncus, mammillary bodies, and various thalamic and hypothalamic nuclei. This system's multiple interconnections with brain structures influence behavior and responses to stimuli. For example, the sensory system, cerebral cortex, and limbic system are involved in the stimulation of visceral and somatic effectors, which results in psychological expressions of behavior and emotions [7; 10].

The limbic system influences memory, drives, motivation, visceral functions, and interactions with the environment. Emotional expressions believed to evolve from this complex group of structures include rage, placidity, fear, and attack reactions. Animal studies have demonstrated that the limbic system contains centers of reward and punishment, with both serving as important imitators of behavior and affecting memory. The hippocampus is thought to be involved in the transfer of short-term memory into long-term memory, especially with events related to elements perceived in the environment [25; 26].

Amygdala

The amygdala is thought to have major responsibilities for the control of behavior in social and environmental circumstances. It is also believed to influence visceral responses to emotions and various movements related to posturing and eating [25; 26].

Thalamus

The thalamus, a large, ovoid gray mass, surrounds the third ventricle. Specific areas within the thalamus receive axons from the cord, brain stem, cerebellum, basal ganglia, and various parts of the cerebellum. These connections allow it to influence motor function and have a role in arousal, alerting mechanisms, and reflex movements [7; 10].

The thalamus influences the motor cortex through its connections with the pyramidal tract neurons. It is involved with the initiation of movement, control of muscle tone, and regulation of cortical reflexes through connections with the cerebellum, globus pallidus, and substantia nigra. The thalamus interprets and relays sensory impulses from all parts of the body, except the olfactory nerve. Recognition of crude sensations, such as pain, temperature, and touch, also take place here. Sensory impulses that the thalamus is unable to interpret are relayed to appropriate primary sensory and association nuclei in the cerebral cortex. The thalamus is even involved in emotional responses—interpreting sensations as pleasant or unpleasant [7; 10].

Hypothalamus

The hypothalamus, a small but important area of brain tissue situated just below the thalamus, plays a major role in the maintenance of many homeostatic functions. Numerous regulatory activities initiated here are affected through the pituitary gland and the autonomic nervous system. The pituitary gland, also known as the hypophysis, lies below the hypothalamus in the sella turcica. Hypothalamic nuclei influence pituitary gland function through neural and endocrine activity [45].

The hypothalamus receives input from all parts of the body. Autonomic nervous system activity is initiated in response to input received from areas within the thalamus, medulla oblongata, spinal cord, and limbic system. The influence of the hypothalamus in autonomic nervous system activity includes regulation of heart rate, blood pressure, and body temperature [45].

The limbic system, important in emotions and behavior, surrounds the hypothalamus and has connections with it. Hypothalamic connections with the thalamus, which interprets feelings of pleasantness and unpleasantness, and with the reticular activating system, which influences wakefulness, provide additional input to which the hypothalamus responds [45].

Many hypothalamic activities are initiated by changes in the perceived composition of its blood supply. For example, specific areas within the hypothalamus are sensitive to changes in water balance, glucose, and insulin levels. The hypothalamic response to an increase in osmotic pressure illustrates this sensitivity. With a loss of body fluid, the hypothalamus detects an increase in osmotic pressure. In response, it initiates the release of antidiuretic hormone by the posterior pituitary gland to concentrate the urine and stimulate the thirst center to increase the oral intake of fluid [45].

Other centers within the hypothalamus regulate appetite. Specific nuclei credited with the initiation of feeding behavior and satiety have been identified. These centers are reciprocal in their inhibition of one another. The hypothalamus also influences gastrointestinal function and sexual activity [45].

Speech Centers and Cerebral Hemisphere Specialization

About 95% of the population has speech centers in the left hemisphere of the cerebral cortex. In the remaining 5%, these centers are in the right hemisphere or (rarely) in both. There is a relation between the dominant hand and the hemisphere controlling speech; most right-handed persons' speech centers are in the left hemisphere, while left-handed person's speech centers tend to be in the right hemisphere. The term "cerebral dominance" refers to the hemisphere containing the speech centers. Two areas within the brain concerned with speech and language are Broca's motor speech area, located in the frontal lobe, and Wernicke's area, located in the superior posterior aspect of the temporal lobe [25; 26].

Research has demonstrated that both hemispheres of the brain have many types of specialization in addition to speech. The dominant hemisphere appears to excel in mathematical calculation and logical analysis of problems, where the other hemisphere appears better able to understand complex

visual patterns and spatial relations and to appreciate music. Development and effective use of these special skills require that the fibers connecting the hemisphere be intact [25; 26].

Brain Stem

The brain stem, which lies between the diencephalon and the spinal cord, has three sections: the midbrain (superior portion), the pons (center portion), and the medulla oblongata. The medulla oblongata joins the spinal cord at the foramen magnum, located at the base of the skull. The brain stem contains many fiber tracts that transmit messages to and from the brain. It also serves as a relay station between the cerebellum and brain. Ten of the twelve cranial nerves located here function much like peripheral nerves (spinal nerves). With the exception of CN IV (the trochlear nerve), they are unlike peripheral nerves in that they only innervate tissues ipsilaterally (i.e., on the same side of the body) [7; 10].

Brain Stem Function

Each of the structures of the brain stem has unique responsibilities, but they function as a unit to serve as a conduit for impulses passing to and from the cerebral cortex and the spinal column. The midbrain, the uppermost portion of the brain stem, contains afferent and efferent nerve tracts that travel to and from the cerebral hemispheres. It also houses the red nucleus, which serves as a relay station for coordination of impulses traveling between the cerebellum and cerebral hemispheres, and the corpora quadrigemina, which are involved in reflex responses to visual stimuli and the relay of auditory impulses [25; 26].

The pons sits between the midbrain and the medulla oblongata and anterior to the cerebellum. It contains nerve fiber tracts that provide communication between upper and lower levels of the CNS and the cerebellum. The lower third of the pons contains respiratory reflex centers influenced by the carbon dioxide levels of the blood and spinal fluid. The pons also influences vasomotor activity [25; 26].

The medulla oblongata forms the inferior portion of the brain stem. The pyramids for the motor tracts are located on its ventral surface. Sensory tracts ascend through the medulla to the thalamus. Major reflex centers in the medulla influence respiratory and cardiovascular function [25; 26].

THE SPINAL CORD

The spinal cord is continuous with the brain stem. It begins at the foramen magnum and descends through the vertebral canal to the level of the first or second lumbar vertebrae. Nerve roots, known collectively as the cauda equina, extend off the base of the spinal cord and travel for some distance before exiting at the appropriate intervertebral foramina [18].

The spinal cord contains neuronal cell bodies, ascending sensory tracts, and descending motor tracts. A cross-section of the cord shows a gray center shaped like an H surrounded by white fibers. The gray area is made up of neuronal cell bodies, internuncial neurons, neuroglial cells, and synapses. In the center of the gray matter lies the differential canal, each is continuous with the fourth ventricle. It may contain CSF but is often filled with cellular debris [18].

The white fiber area of the cord contains myelinated and unmyelinated fiber tracts, which transmit the many messages essential for maintenance of the body's complex functions. Both sensory and motor tracts are located on each side of the cord [18].

There are 31 pairs of spinal nerves, each numbered according to the level of the cord section from which it originates. There are 8 pairs of cervical nerves, 12 pairs of thoracic nerves, 5 pairs of lumbar nerves, 5 pairs of sacral nerves, and 1 pair of coccygeal nerves. The first pair of cervical nerves exits between the occipital bone and the first cervical vertebrae. Because there are eight cervical nerves and only seven cervical vertebrae, spinal lesions are identified according to the cord level rather than the vertebral level. Generally, each cord segment is named for the vertebral body below its exit point. Peripheral

nerve trunks extend from anterior and posterior roots, which unite in the intervertebral foramina. Upon emerging from the vertebral foramina, they form mixed nerves, which divide into anterior and posterior branches and extend into the periphery. Also present are white rami, which contain autonomic nervous system fibers. The posterior rami divide into smaller nerves connected to the muscles and skin of the exterior surface of the head, neck, and trunk. Anterior rami (except for the thoracic nerves) divide to supply fibers to the skeletal muscle, skin of the extremities, and the anterior and lateral surfaces. Subdivisions of the anterior rami form three complex networks or plexuses: the cervical plexus, graphical plexus, and lumbosacral plexus. Smaller nerves emerge from these plexuses and continue to subdivide to innervate distal regions of the extremities [18].

Spinal Cord Function

The spinal cord is a conduit for messages to and from the higher levels within the CNS and participates in reflex motor activities. Descending pathways within the cord carry motor instructions to the anterior horn (ventral roots) from the cerebral cortex, brain stem, and cerebellum. Impulses synapse in the anterior horn (motor gray area) just before leaving the cord. This synaptic activity involves upper motor neurons, which are located within the cord, and lower motor neurons, which extend beyond the cord. Ascending (dorsal) roots transmit sensory impulses from the skin and viscera to the cord and CNS. Synaptic activity necessary for transmission for signals occurs at various levels within the cord. Dermatome charts provide a "map" of the area of skin supplied by the dorsal root to each spinal nerve [48].

Specific sensory and motor tracts have been identified within the spinal cord. These tracts, located in the white matter, are anterior, lateral, and posterior funiculi. These funiculi, further divided into tracts called fasciculi, carry similar types of nerve impulses to specific destinations [48].

THE AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system comprises two efferent subsystems: the sympathetic and parasympathetic subsystems. Organs influenced by the autonomic nervous system are controlled by one of these two subsystems [18].

Sympathetic Nervous System

Sympathetic nervous system impulses are transmitted to the periphery by tracts of sympathetic fibers containing cell bodies and dendrites that extend with the intermediolateral gray horns of the spinal cord from thoracic spinal nerve 1 to lumbar spinal nerve 2. Because of its location, the sympathetic nervous system has been referred to as the thoracolumbar division. Axons leave the cord with the anterior roots of the thoracic and first four lumbar spinal nerves. After exiting, they quickly join the sympathetic trunk via white rami. The sympathetic trunks located on both sides of the cord extend from the second cervical vertebrae to the coccyx. The axons, upon entering the trunks, extend branches up and down the chain. The sympathetic nervous system preganglionic axons terminate on many post-synaptic ganglia present in organs [18].

Parasympathetic Nervous System

Cell bodies of the preganglionic neurons of the parasympathetic nervous system are located in two areas. The nuclei of CN III, VII, and X are located in the brain stem and the lateral gray columns of the sacral cord. In the sacral region, parasympathetic axons are present in spinal nerves. Because of these anatomic locations, the parasympathetic nervous system has been referred to as the craniosacral division [25; 26].

THE RETICULAR ACTIVATING SYSTEM

The reticular activating system is composed of a diffuse system of neurons extending through the medulla, pons, midbrain, diencephalon, and cortex. Afferent and efferent connections also exist between the cerebellum and spinal cord [7; 10].

The reticular activating system regulates spinal motor activity as well as voluntary and reflex muscle activity. Projections to the diencephalon and cortex effect and maintain arousal and alerting states. In addition to maintaining wakefulness, this system also participates in the regulation of sensory input from the periphery, regulation of respiration, and vasomotor activity [25; 26].

MOTOR FUNCTION

Effective skeletal muscle function involves many components of the CNS and peripheral nervous system. Muscle function requires the perception and interpretation of sensory stimuli and an intact motor system to initiate and carry out muscle contraction. Areas of the CNS involved in motor function include the premotor cortex, the primary motor area, pyramidal and extrapyramidal tracts, basal ganglia, thalamus, brain stem, spinal cord, and cerebellum [48].

The premotor cortex (associative cortex), positioned just anterior to the major motor strip, is involved in muscle activities that produce hand skills, voluntary eye movements, eyelid blinking, and vocalization. To appreciate the complexity of the functions carried out by the premotor cortex, consider the many coordinated activities needed to speak. Speaking requires groups of muscles in the tongue, larynx, pharynx, and chest to contract and relax in carefully programmed sequences. Function of the premotor area requires intact connections with the sensory association areas of the parietal lobe, temporal lobe, frontal lobe, occipital lobe, components of the basal ganglia, primary motor cortex, thalamus, brain stem, and spinal cord [48].

The primary motor area is believed responsible for the initiation of movement by individual groups of muscles, such as those involved in the movement of the fingers, toes, and mouth. The cross section of the precentral gyrus illustrates specific areas of the primary motor area identified as initiating willed movement by various muscles. A large amount of gray matter is allocated to muscle groups involved in complex movements of the hands and mouth [48].

Nerve cells of the major motor strip and their conducting fibers make up the pyramidal (corticospinal) motor system. Nerve fibers descend from the motor strip through the internal capsule, midbrain, and pons to the medulla oblongata, where the pyramidal fibers cross. After crossing, the fibers descend in the spinal cord to appropriate levels. Most pyramidal fibers descend via the lateral corticospinal tracts to ventral horns of gray matter in the cord. Some motor fiber travel via ventral corticospinal tracts [48].

Extrapyramidal motor tracts are most complex in their arrangement and synaptic activity in the cerebrum and brain stem. A functional (rather than anatomic) unit, extrapyramidal tracts are involved in maintaining balance and posture by facilitating some muscle movements and inhibiting others. Movements initiated in one hemisphere influence movements on the opposite side of the body. The basal ganglia are part of the extrapyramidal tract. In addition, the thalamus, subthalamus, substantial nigra, and red nucleus have roles in motor function. Multiple connections exist among all of these areas. A second pathway allows for feedback control of extrapyramidal motor activity [48].

The basal ganglia have three motor functions. A major responsibility of the basal ganglia as a whole is believed to be the inhibition of postural muscle tone. The caudate nucleus and putamen, collectively referred to as the striate body, are thought to initiate and regulate gross intention movements such as body posture and major arm movements. This regulation involves pyramidal and extrapyramidal pathways. The globus pallidus is believed to provide background muscle tone for intended movements initiated by the striate body or the cerebral cortex (e.g., the muscle contractions needed to support the arm and trunk while using a tennis racket) [48].

Final pathways for extrapyramidal signals into the cord are the reticulospinal tracts that lie in other ventral and lateral tracts of the cord. Also involved in transmission to a lesser degree are the rubrospinal, tectospinal, vestibulospinal, and possibly olivospinal tracts [48].

PROTECTION AND MAINTENANCE OF THE CNS

Understanding the complex capabilities of the nervous system allows for an appreciation of the system's means of protection and maintenance. Recognizing the safeguards of the bony cranial vault and vertebral column is easy. Less obvious but also valuable is the protection provided by the hair, skin, scalp, fascia, muscle, meninges, fluid cushioning, and complex vascular supply. The superficial structures help to limit injuries from external trauma, and the ventricular and vascular systems provide a reinforcement for optimal neuronal function [56].

The meninges in the cranial vault and vertebral column protect the CNS from physical harm and support the CSF system and circulation. The dura mater, the outermost layer of meninges, forms a double layer over the brain tissue. Its outer layer is an inner periosteal lining for the skull and vertebral canal. In the cranium, the inner layer of dura, for the most part fused with the outer layer, helps to secure the brain to the cranial vault. To provide extra support and protection, the inner layer of dura separates in areas, dipping down between the longitudinal fissures, between the cerebellar hemispheres, and passing over the pituitary glands nestled in the sella turcica. In other areas, dura layers separate to form venous sinuses that collect and carry venous blood away from the brain. Arachnoid processes (villi) project into the dural sinuses [56].

In the spinal cord, the inner layer of dura is continuous with the spinal dura mater. The spinal dura extends to the second sacral vertebrae, where it joins with the external filum terminal and attaches to the back of the first segment of the coccyx. The middle layer of the meninges, the arachnoid, is a thin, fibrous membrane that adheres closely to the inner surface of the dura, allowing only a narrow space between the two. The inner layer of the meninges, the pia mater, adheres so closely to the brain that it follows the contour of the fissures and sulci.

The space between the pia mater and arachnoid is bridged with weblike strands of arachnoid called trabeculae. A rich network of pia blood vessels extends into the brain. The area between the arachnoid and pia mater is called the subarachnoid space. Located here are arteries, veins, arachnoid trabeculae, and CSF. Within the spinal cord, fibrous bridges join the pia mater with the arachnoid and dura mater. These bridges, known as denticulate ligaments, help to stabilize the cord within the spinal canal [56].

THE CEREBROSPINAL FLUID SYSTEM

The CSF protects the brain and spinal cord by supporting the tissues, acting as a shock absorber, and serving as a medium for the transfer of elements from the bloodstream to nervous system tissues. CSF flows through the elaborate ventricular system in the brain and through the subarachnoid space surrounding the brain and spinal cord. Two large ventricles are positioned in each cerebral hemisphere. Their central portions extend in the parietal lobes. The anterior horns extend into the temporal lobes, and the posterior horns project into the occipital lobes. A small third ventricle lies below and communicates with each lateral ventricle via a small channel known as the foramen of Monro. The thalamus forms the lateral walls of the third ventricle. The third ventricle is connected via the cerebral aqueduct to the fourth ventricle, which lies below. The pons and medulla are positioned below the fourth ventricle. The cerebellum lies above [56].

CSF flows from the ventricular system to the arachnoid space of the brain and spinal cord by way of the lateral aperture (foramina of Luschka) and the medial aperture (foramen of Magendie). CSF is constantly being produced by capillary tufts called choroid plexuses, located in the ventricles. Arachnoid villi, projecting into the dural sinuses, provide routes for the reabsorption of CSF into the venous circulation [56].

THE BLOOD-BRAIN BARRIER

The blood-brain barrier theory stems from observations that only water, oxygen, carbon dioxide, and alcohol can readily enter or leave the capillaries of the CNS. Large molecules penetrate slowly through special systems or not at all. This protective barrier is believed to prevent sudden, extreme fluctuations in the composition of CNS tissue fluid while allowing nutrients to pass. The blood-brain barrier is thought to be formed within the capillaries by a continuous layer of endothelial cells connected by tight junctions, with a basement membrane surrounding the endothelium. Astrocytes that lie in close apposition are not considered part of this barrier [25; 26].

The blood-brain barrier protects most of the brain and cord tissue. Exceptions include the pineal body and the posterior lobe of the hypophysis, which are believed to be nourished by vessels with fenestrated endothelia that provide specific sites for the transfer of proteins and solutes irrespective of molecular size and lipid solubility. Tight junctions at the intracellular clefts of the choroid epithelium serve as the blood-CSF barrier in the vascular choroid plexuses of the CSF system [25; 26].

CENTRAL NERVOUS SYSTEM CIRCULATION

The viability and functioning of the CNS depends on a rich and continuous blood supply. The brain utilizes approximately 20% of the body's oxygen supply and requires about 400 kcal of glucose per day. The average cerebral blood flow is about 750 mL per minute [56].

The external carotid arteries supply the scalp and parts of the head and neck. Secondary branches of the external carotids (the middle meningeal arteries) supply blood to the meninges of the brain. The right and left internal carotid arteries, after passing through the carotid canals, branch into the anterior cerebral arteries and middle cerebral arteries at the level of the optic chiasm. The right and left anterior

cerebral arteries are connected by the small anterior communicating artery to form the anterior portion of the circle of Willis. Anterior cerebral arteries perfuse the caudate and putamen nuclei of the basal ganglia, the corpus callosum, and portions of the internal capsule and frontal and adrenal lobes. The middle cerebral arteries are the major supplier of blood to the precentral and postcentral gyri and feed portions of the temporal, parietal, and frontal lobes [56].

The vertebral arteries, whose source is the subclavian artery, travel to the brain via the foramina of the cervical vertebrae and the foramen magnum. Posterior communicating arteries extending back from the internal carotid arteries complete the anastomosis with the posterior cerebral arteries to form the circle of Willis. This anastomosis, intended to maintain circulation to the brain tissue if one of the vessels closes, is not always functional. Other vessels providing collateral circulation are vessels at the base of the brain; small pial anastomotic branches on the surface; external carotids to the eyes; and anterior, middle, and posterior cerebral anastomoses on the surface of the brain [56].

Passage of venous blood from surface and deep brain tissue takes place via thick veins lacking valves. The blood flows into the dural sinuses and then drains into the internal jugular veins. The superior sagittal sinus serves as a major route for the removal of the constantly forming CSF. The cavernous sinus drains blood from the eye, orbit, and face, while the transverse sinus lies close to the ear [56].

REGULATION OF CEREBRAL BLOOD FLOW

Control of blood flow in the CNS is essential for viability and optimal function. The body has several built-in mechanisms to maintain effective circulation.

Three major factors that have a direct and potent effect on cerebral blood flow are elevations in concentrations of carbon dioxide, hydrogen ions, and oxygen. The increase in hydrogen ion concentration causes vasodilation of the cerebral vessels. Vasomotor reflex response that affects the body's general vascular perfusion also affects the perfusion of blood in the CNS. Vasomotor centers in the pons and medulla maintain vascular tone through impulses transmitted via the spinal cord to all blood vessels in the body. The reticular areas of the brain stem and hypothalamus have both excitatory and inhibitory effects on vasomotor activity. The hypothalamus also influences vasoconstriction activity through excitatory or inhibitory action on the vasomotor centers. It also helps to regulate total body water (and therefore blood pressure) by increasing or decreasing the release of antidiuretic hormone [56].

A severe drop in blood pressure to 50 mm Hg or less will result in ischemia in the vasomotor center. The resulting local increase in concentration of carbon dioxide causes a profound stimulation of the sympathetic nervous system, which initiates the constriction of blood vessels—some to the point of occlusion. This response, meant to shunt blood to the CNS, is known as the ischemic response [56].

Cushing phenomenon occurs when an increase in pressure in the CSF system equals the pressure in the cerebral vascular bed, hampering the flow of blood to the brain. At this point, the CNS ischemic response is initiated to raise the CNS blood pressure above the CSF pressure and facilitate blood flow to the brain [56].

SPINAL CORD CIRCULATION

As noted, multiple arteries feed the spinal cord. The vessels join, forming a complex network that supplies the vertebrae, periosteum, and dura. Branches supply the ventral and dorsal roots and penetrate deeply into the cord. Anterior and posterior spinal arteries extending the length of the cord originate from the carotid and vertebral arteries [56].

There are no valves in this venous network. As a result, blood flow varies depending on pressure. With elevation of intra-abdominal pressure, venous blood from the pelvic plexus passes into the vertebral venous channels. If the jugular vein is occluded, blood from the skull can drain via the vertebral channels. This venous plexus is believed to provide potential routes for metastasis of neoplasms [56].

PATHOPHYSIOLOGIC INFLUENCES AND EFFECTS

The CNS's complex structure and diverse functions predispose it to a multitude of pathologies, each capable of causing varying types and degrees of dysfunction. The protective structures and mechanisms are obviously not fail-proof. In fact, these structures often produce or contribute to nervous system trauma. The rigid skull and vertebral column allow little room for neuronal swelling, tumor growth, or circulatory congestion. Trauma may also occur when external forces drive the nervous tissue against the inside of the bony structures. Fractures and bony degenerative processes can perforate or crush neurons or supportive tissues [72].

The elaborate CSF system, designed to support and cushion the CNS, is subject to localized or generalized buildup if reabsorption is impeded. This extensive fluid system also provides routes for the spread of infection. Foramina of the skull and vertebral column, serving as avenues for the passage of blood vessels and nerves traveling to and from the periphery, also provide routes for micro-organisms into the CNS [72].

Meningeal tissues also present hazards as well as protection. With increases in intracranial pressure (ICP), herniation of brain tissue through the tentorial notch formed by the tough dura can seriously injure the brain stem and surrounding tissues. The dura mater, pia mater, and arachnoid fissures also provide extensive, uninterrupted routes for the spread of infection [72].

The CNS's circulatory bed, so important in providing nourishment, provides routes for migrating micro-organisms and metastases from other parts of the body. Additional risks include an increase in ICP from hemorrhage and/or abnormal vascular structures, such as arteriovenous malformations or reflex vasodilation in response to hypoxia [72].

The neurons, nerves, and nerve tracts, with their long communication chains and multiple connections required for effective afferent and efferent activity, form the most complex part of the nervous system. Because they are present throughout the body, these structures are vulnerable to many pathologic elements, including alterations in the biochemical environment, structural damage from disease, degeneration of the myelin sheath, trauma, and neurotransmitter abnormality [72].

Various pathologic changes can damage any or many of the components of the nervous system. Diagnosis of the cause or area affected is often difficult because of the inter-relationships of structures and their dependence on nerve fibers and neurotransmitters to carry signals to organs outside the CNS. Each component of a system and its innervated organs may be the cause of a particular symptom or deficit [72].

Nervous system damage often produces complex physical and mental disabilities. A stroke, for example, may produce spasticity rather than flaccid limbs. This spasticity can result in severe positioning problems and contractures. Personality changes can occur from physical deficits produced by the stroke or from psychological stressors stemming from the disability [72].

INFLAMMATION AND INFECTIOUS PROCESSES

Inflammatory processes affecting nervous tissue alter the metabolism and thus the tissue's nutritional immune processes. Causes of CNS inflammation include trauma, lumbar punctures, and infectious processes, such as meningitis.

Inflammatory processes are known to attack the spinal cord's gray matter. Inflammation can occur in response to acute infections or can be part of a primary infectious process, such as poliomyelitis. These inflammatory processes can cause necrosis, emboli, or thrombotic complications. Sensory and motor deficits may result [72]. In herpes zoster (shingles), an acute unilateral and segmental inflammation of the dorsal root ganglia produces localized vesicular lesions, confined to one dermatome. Severe pain is experienced in the peripheral areas innervated by the inflamed ganglia [72].

TRAUMA, TUMORS, AND COMPRESSION

Trauma to the nervous system may result from external forces or elements in the nervous system. The skull and vertebral bodies can make it difficult to discover, locate, and assess trauma or physical changes. Symptoms indicating progressing damage may be subtle or lacking until the condition is too advanced to treat effectively [72].

At times, the system's response to the trauma causes more damage than the insult itself. This secondary damage occurs when edema, bleeding, and/or increased ICP destroy nervous tissue by compression or restriction of circulation. Edema results from trauma associated with contusions and lacerations, injury to capillary walls, or hematomas or tumors that obstruct venous blood outflow. Obstruction causes the blood to back up and fluid to move out of the capillaries. Expanding tumors cause an increase in ICP and edema, but they may also cause bleeding by damaging vessels. All of these elements carry the hazard of herniation through the tentorial notch or foramen magnum [72].

Spinal injuries may cause many of the same problems as cranial injuries, but the complexity of the vertebral column's structure and the concentration

of neural tracts at all levels of the cord present special concerns. Injuries to the spinal cord are more common in areas of greater mobility, such as at the lower cervical spine (C4 through C7 and T1) and at the lumbar juncture (T12, L1, and L2). Vertebral injuries can cause compression of nerve roots by bone, ligaments, extruded disk material, hematomas, or disruption or overstretching of the neural tissue. Edema caused by trauma can compromise cord function [72].

DEGENERATIVE PROCESSES

Many nervous system diseases can be categorized as degenerative. Their causes vary, as do their severity and influence on lifestyle. For example, progressive muscular atrophy follows degeneration of lower motor neurons in the spinal cord that inhibits impulses that trigger muscle contraction. The upper motor neuron degeneration associated with amyotrophic lateral sclerosis (ALS) leads to impaired speech, chewing, swallowing, and breathing [72].

HEREDITARY AND CONGENITAL DISEASES

Hereditary diseases result from conferred genetic errors that affect development, maturation, and/or aging. These illnesses present varying degrees of risk to offspring both in the threat of developing the disease and carrying it on to another generation. For example, Huntington chorea is transmitted as an autosomal dominant trait, with each child born of a parent with this trait having a 50% chance of developing the disease [72].

Congenital defects causing CNS abnormalities or malfunctions may occur alone or in combination. Causes include a hereditary tendency, intrinsic factors (e.g., inadequate circulation for the embryo), and in-utero exposures to infectious diseases (e.g., rubella) [72].

RELATED SYSTEM INFLUENCES AND EFFECTS

As discussed, the CNS orchestrates activities to maintain body functions and homeostasis. Each major system has a role in maintaining homeostatic activity through the regulation of the body's biochemical environment. Alteration in system functions by disease or trauma can result in abnormal neuronal activity or tissue destruction within the CNS. For example, effective pumping by the heart is essential to nourish the CNS and remove waste. Along with the respiratory system, the cardiovascular system provides the oxygen necessary for function. Failure of these systems quickly results in neuronal death [2; 3].

The kidneys serve several functions to support the nervous system. They participate in the maintenance of blood pressure and in fluid, electrolyte, and acid-base balance. They also contribute to ensuring an adequate oxygen supply through the manufacture of erythropoietin, the hormone that stimulates the production of oxygen-carrying red blood cells [2; 3].

Gastrointestinal absorption maintains an adequate nutritional level of food elements, vitamins, and minerals. The liver also helps by ensuring an effective level of blood glucose and other nutrients and provides detoxification of drugs and other foreign substances [2; 3].

The endocrine system, which has multiple roles in the regulation of body function, influences metabolism, heart rate, water balance, and mental function. Pathology in this system can cause a variety of neurologic abnormalities or deficits [2; 3].

PSYCHOSOCIAL/LIFESTYLE INFLUENCES AND EFFECTS

Bodily changes with aging are the result of alterations in effector tissues, receptor systems, and impairment of the body's homeostatic regulatory system. There is also a gradual decline in activities requiring rapid sensory and motor coordination. Isometric muscle strength usually peaks at about 18 years of age and is maintained through the fifth decade, after which there is a gradual decline related to a decrease in number of muscle fibers and muscle atrophy [4; 27].

Although some changes in muscle mass occur in all aging persons, epidemiologic studies indicate that physical exercise contributes to longevity by decreasing the incidence of heart disease. Exercise may reverse or delay age-related changes in synaptic function and nerve-conduction velocity. Physical training in the elderly has been found to improve heart rate, cardiac output, blood pressure, and joint mobility and to decrease stiffness, although it does not improve pulmonary function [4; 27].

Intellectual performance, as measured by vocabulary and information comprehension, peaks between 20 and 30 years of age and is maintained through life or until the mid-70s, in the absence of disease. As with physical activity, individuals who continue to be active mentally can perform better than those who do not. However, the speed of central processing for mental functioning is impaired with age [4; 27].

The loss of vibratory perception in the lower extremities usually begins at about 50 years of age. Touch becomes significantly diminished due to skin changes and a decrease in the number of sensory receptors. This fact may be particularly significant to nurses during neurologic assessment. Corneal sensitivity, an accurate measurement of sensory perception, shows a decrease with age. Visual, auditory, gustatory, and olfactory senses are also diminished [4; 27].

Cortical size and blood flow decrease over time. Brain weight peaks in the early 20s and then slowly declines. Along with the weight loss, the cortical area is reduced, with a broadening of sulci and a flattening of gyri. Cerebral blood flow in healthy adults is about 50–60 mL per minute per 100 g of tissue. (The base requirement for normal cortical function is about 40 mL per minute per 100 g tissue.) Between 30 and 70 years of age, the rate of cerebral blood flow decreases by approximately 20%. Alterations in blood flow from atherosclerosis, structural changes, and heart disease can easily decrease the oxygen supply, compromising neuronal function [4; 27].

Changes in autonomic nervous system function in the elderly can be seen in the deterioration of pupillary, cardiovascular, thermal, and secretory functions. It is not clear whether these changes are the result of peripheral or CNS changes [4; 27].

The elderly face the threat of altered homeostasis due to health problems unrelated to neurologic pathology. If an imbalance occurs in any other system, the nervous system can be affected. Even psychological reactions to stress can alter neurologic function [4; 27].

THE NURSING PROCESS FOR PATIENTS WITH CNS DYSFUNCTION

The pervasive influence of the CNS on mental and physical functions often complicates the analysis of neurologic symptoms. Identification of nervous system pathology can be difficult, because symptoms are often far removed from the source. For example, a cerebrovascular accident (CVA) can result in weakness in a lower extremity. Furthermore, similar symptoms and signs among some diseases of the CNS can confound diagnosis; for example, increased ICP can be a symptom of subarachnoid hemorrhage, stroke, or hydrocephalus [30; 40].

SUBJECTIVE DATA

Collection of subjective data from patients with CNS diseases can be difficult, because the disease often compromises the patient's ability to provide reliable information. In some instances, the patient will be unresponsive, unconscious, or unreliable as a historian. In these cases, family members, friends, or persons who were present when the problem arose should be consulted [30; 40].

The fear or apprehension that often accompanies diagnosis of neurologic disease can also limit patient disclosure. A review of the patient's long-term and recent health history is essential, because, as discussed, neurologic problems can result from diseases affecting other systems (e.g., peripheral neuropathy resulting from diabetes). Neurologic problems may also be misdiagnosed as psychiatric diseases. A variety of psychiatric and other health problems should be part of differential diagnosis, and comorbidities should be considered in planning treatment or care. For example, diet, pharmacotherapy, and intravenous therapy are more complex if a patient with neurologic dysfunction also has diabetes, cirrhosis, or renal disease [30; 40].

Head and Neck

Patient symptoms related to the head and neck should be carefully reviewed during the history of the present illness. Headaches are common in a variety of health problems, including stress, tumors, meningitis, or one of the many diseases causing increased ICP. Ear infections can spread into the brain via adjacent blood vessels and the mastoid bone of the skull. Infections of the scalp, paranasal sinuses, and the nasopharynx also present the risk of meningitis or encephalitis because of their proximity to venous sinuses, blood vessels, and foramina [30; 40].

Reported hearing loss may be the result of a conduction problem or of damage to CN VIII or cortical tissue. The patient should be asked about tinnitus (i.e., ringing or buzzing in the ears). Dizziness and vertigo are significant symptoms of tumors or degenerative changes in the vestibular branch of CN VIII, the brain stem, or the cerebellum [30; 40].

Uncontrolled head movements may be indicative of Parkinson disease, other extrapyramidal disease processes, or multiple sclerosis. A partial loss of motor function of the face can be the result of Bell palsy, CVA, or pathology affecting the nuclei of the brain stem. Loss of smell from insult to the olfactory bulbs or tracts can be related to shearing trauma, orbital fractures, or tumors; it may also be an early sign of Parkinson disease. Diseases causing visual changes include multiple sclerosis, myasthenia gravis, stroke, tumors, and trauma [30; 40].

Bowel and Bladder

Bowel and bladder function is controlled by various components of the autonomic nervous system. For patients with known or suspected CNS deficits, it is important to thoroughly review symptoms such as constipation, urinary retention, and fecal and urinary incontinence. Symptoms of ataxia (i.e., lack of muscle coordination) should raise concern about degeneration of the posterior tracts of the spinal cord or cerebellar dysfunction. Spasticity of muscles occurs with CVA and multiple sclerosis. In contrast, flaccidity (i.e., decreased or absent muscle tone) can result from isolation of muscles from neuronal impulses. Fasciculations—fine, rapid, twitching movements originating in small groups of muscle fibers—are often present in patients with ALS [30; 40].

Motor Function

Changes in motor function are often unique to a disease process. Inquiry into changes in motor function should include thorough questioning about localized or generalized weakness. Asking specifically about difficulty arising from or turning in bed; flopping of ankles during walking; difficulty in lifting legs to go up and down steps or lifting objects; or problems keeping eyes open will help patients explain their symptoms better [30; 40].

As noted, ataxia may signal degeneration of the posterior tracts of the spinal cord or cerebellar dysfunction. Plasticity of muscles may occur with CVA or multiple sclerosis, which releases muscles from upper motor neuron control. In contrast, flaccidity can result from isolation of muscles from neuronal impulses [30; 40]. Twitching of the trapezius muscle may occur with lesions in the nucleus of CN XI [30; 40].

Reports of other abnormal movements include spasm of a muscle or muscle groups (myoclonus), as in Creutzfeldt-Jakob disease, dyskinesias, athetosis (i.e., slow, twisting, snakelike movements in the upper extremities), and dystonia (i.e., intense, irregular torsion and muscle spasms). Movement disorders can result from antipsychotic medications, such as chlorpromazine and haloperidol, or from Huntington chorea [30; 40].

Sensory Function

Diseases of the spinal cord's sensory tracts can alter or even prevent transmission of stimuli to the brain for interpretation. Symptoms of this pathology include numbness, tingling, pain, increased or decreased sensitivity to touch, and alteration in perception of cold and heat. Sharp, lightning-like root pain occurs with lesions of the dorsal roots of the spinal nerves. This pain may result from ruptured disks, cord tumors, fractures, inflammatory diseases of the vertebrae, or meningitis [30; 40].

OBJECTIVE DATA

A thorough neurologic assessment of patients with probable CNS disease is important initially and often throughout the nurse-patient interaction. Comprehensive baseline data assist in effective evaluation of the patient's changing status. In addition to the formal assessment, frequent contact with the patient during treatment and activities of daily living allows for closer observation [30; 40].

Physical Assessment

The patient's mental status is an important component of the neurologic assessment, providing valuable insights into the cause of disease and its effect on the body. Determination of level of consciousness is often used to evaluate improvement or decline of health status in patients with traumatic brain injury or tumors [30; 40; 41].

Assessment of level of consciousness begins with noting the degree of alertness. Is the patient awake and alert? If sleeping, is the patient aroused by verbal stimuli or tactile stimulation, such as touch or gentle shaking, or does the patient respond only to noxious stimuli? After arousal, is alertness maintained after the stimulus is removed? Does the patient appear drowsy, restless, irritable, or combative? The patient should be questioned regarding time, place, person, and self. If the patient is becoming disoriented, awareness of time will usually be lost first, then place, then person, and last, self. Inappropriate responses to such questioning may not necessarily indicate mental dysfunction. The patient may fail to respond appropriately because of a language barrier, impaired hearing or vision, or some degree of expressive or receptive dysphasia. Patients who have undergone trauma may be confused about time or place because of a period of unconsciousness or the rapidity of events. The patient transferred from another hospital or even from another unit may have trouble keeping up with the changes. This is especially true of the elderly [11; 41].

The patient should also be asked to follow simple commands, such as "squeeze my hand," "hold up your arm," or "wiggle your toes." The response permits assessment of motor and mental function. If the patient does not respond to verbal or tactile stimulation, noxious or painful stimulus may be required. As a general rule, the least invasive/painful but effective stimulus should be used. Even in unconscious patients, noxious stimuli may cause a precipitous rise in blood pressure [11; 41].

Charting should include the stimulus used as well as a description of the patient's response to it. Responses are casually classified as appropriate, inappropriate, or absent. In an appropriate response, the patient localizes the unpleasant stimulus and attempts to withdraw from it or push it away. An inappropriate response involves random or purposeless movements and decerebrate or decorticate posturing. In extreme situations, no response can be elicited and the patient remains flaccid. Because subtle changes in level of consciousness can be significant, precise documentation and clear communication are essential. Avoid words like "confused," "stuporous," and "comatose." Instead, describe the patient's behavior, what is said, and what can or cannot be done. At change of shift, it may be especially beneficial for the nurse coming on duty and the nurse reporting off duty to do an assessment together. In this way, the nurse about to assume responsibility for the patient has a clearer picture of the patient's condition and subtle/minor but significant changes are less likely to be missed or misinterpreted [11; 41].

The Glasgow Coma Scale is an objective measure of level of consciousness that can also be somewhat predictive of recovery. Eye, motor, and verbal responses are measured. Coma is defined as a score of 7 or less. With a score of 3 or 4, there is an 85% chance of dying or remaining vegetative. A score greater than 11 is associated with an 85% chance of moderate disability or good recovery. Response to pain is also significant in determining the prognosis [11; 41].

Station and Gait

Observation of the patient's station (i.e., manner of standing) and gait can provide useful insight. A decortication response is indicative of cortical damage, and unusual gait, stance, or settling posture can result from motor or sensory deficits. With decorticate posturing, the patient demonstrates hyperflexion of the upper extremities and hyperextension of the lower extremities. With decerebrate posturing, both the upper and lower extremities are hyperextended. This response indicates brain-stem injury [11; 41]. Postures can also be influenced by mental and physical problems.

Meningeal Irritation

Assessment for nuchal rigidity, Kernig sign, and Brudzinski sign aid in diagnosing meningeal irritation associated with meningitis or subarachnoid hemorrhage. In nuchal rigidity, neck flexion is limited by involuntary muscle spasm and the patient is unable to place the chin on the chest [41].

To test for Kernig and Brudzinski signs, the patient is in the supine position. For Kernig sign, one hip is flexed to 90° with the knee also flexed to 90°. If meningeal irritation is present, the patient in this position will be unable to extend the knee past 90° without pain. To test for Brudzinski sign, the patient is supine with the legs extended. When the examiner flexes the patient's neck, the hips and knees will spontaneously flex to avoid the accompanying pain if meningeal irritability is present [41].

DIAGNOSTIC STUDIES

As discussed, diagnosis is often complicated because the CNS is contained within the skull and vertebral canal. For this reason, most diagnostic studies for neurologic disease are invasive. Careful patient preparation is necessary for all diagnostic studies. The invasiveness of neurologic tests and the consequences of misdiagnosis increase the importance of patient teaching and nursing care before and after studies are conducted [5; 6; 8].

Most laboratory studies specific for assessment of neurologic function are based on the contents of the CSF or its pressure within the CNS. Other laboratory studies, such as analysis of blood samples, are used to determine whether malfunction of other body systems is compromising CNS function. For example, serum iron and hemoglobin tests can help determine if anemia is reducing the brain's oxygen supply and altering CNS function [5; 6; 8].

X-Rays

X-rays of the skull are used as a basic, noninvasive screening for patients with suspected trauma or neoplasm. They can also reveal pathologic changes, such as pituitary gland tumors, and detect calcified abnormalities or abnormal position of the calcified pineal gland [5; 6; 8].

Spinal radiography may reveal changes in spinal bones resulting from fractures, tumors, or infections. It may also show the bony ridges and spur formations characteristic of osteoarthritis and help to identify congenital defects [5; 6; 8].

Nursing Implications

Before x-rays are taken, the purpose of the procedure and the systems involved should be explained to the patient. Tangled or braided hair is combed, and pins and wigs are removed. Ocular prostheses can produce confusing shadows in a radiograph, so their presence should be noted [17].

Electroencephalography

Electroencephalography (EEG) is essentially a non-invasive test that records a portion of the brain's electrical activity. The EEG is valued for its ability to reveal abnormal brain-wave patterns that help in diagnosing seizure disorders, brain tumors, abscesses, and psychiatric disorders. Diagnosis is made by evaluating patterns and characteristics of brain waves recorded, along with the patient's clinical state. An absence of brain waves generally establishes brain death; however, acute drug intoxication or severe hypothermia can also cause a flat EEG [5; 6; 8].

To conduct an EEG, the patient is placed in a bed or on a lounge chair in a quiet, secluded area. Surface electrodes, or occasionally needle electrodes, are positioned on or in the scalp. The patient is instructed to close his or her eyes, relax, and rest quietly. Various stimuli are introduced to determine whether seizure activity can be produced. The patient may be asked to hyperventilate for three minutes, to watch a flashing light, to endure a sensory stimulus (e.g., a minor electrical shock), to observe a black-and-white checkerboard image, and/or to listen to sounds through earphones. The patient is observed carefully and protected if seizure activity occurs. If a sleep EEG is requested, the patient will be instructed to stay awake the night before the test. Medication may be administered to promote sleep [5; 6; 8].

Nursing Implications

Patients scheduled for EEG should be instructed to eat meals as usual, with the exception of coffee, tea, cocoa, and cola, which may be withheld because of their stimulant effects. Medications that may influence the test are often withheld, including anticonvulsants, tranquilizers, barbiturates, or sedatives. Hair should be clean and free from oils, sprays, or pins [17].

An explanation of the procedure and its purpose, the function of the electrodes, and the length of the test will help the patient relax. The test usually takes 40 to 60 minutes, with pretest and post-test care taking another hour. Following test completion, patients may require assistance removing electrode paste from their hair. During this time, it is important to observe for seizures and recovery from sedation, if given during the test. Vital signs and neurologic signs are also assessed, as appropriate [17]. The physician's order for resumption of medications should be reviewed with the patient.

Computed Tomography

Computed tomography (CT) is used to diagnose intracranial locations of hemorrhage and spinal cord lesions. Scans are also used to monitor the effects of surgery, radiotherapy, or chemotherapy and to reveal vascular displacement, hematomas, cerebral atrophy, infarction, edema, and hydrocephalus. An iodinated contrast dye may be administered to make large blood vessels visible or to define lesions. Administered intravenously, the dye increases the blood density and delineates intracranial masses [5; 6; 8].

Nursing Implications

Before a CT is done, the purpose of the procedure should be explained to the patient. If no contrast medium is used, restriction of food and drink is not necessary. If dye is used, the patient should fast for four to six hours to prevent emesis if nausea occurs. Before administering a contrast dye, any allergies to shellfish, iodine, or contrast media should be identified, as dye may be contraindicated in these patients. Skin testing to determine allergy may be indicated [17].

Patients benefit from a knowledge of the details of the procedure, including special positioning, noise emitted by the machine, and length of test. Some machines require the patient to be strapped to the table, which moves into a gantry during the test. The test takes 15 to 30 minutes if no dye is used; with dye, the time is doubled. Patients receiving the dye should know that it is normal to feel flushed and warm and that sometimes a headache, salty taste, or nausea occurs [17].

Positron Emission Tomography

Positron emission tomography (PET) is a noninvasive nuclear-imaging technique available in large medical centers. It is used to study oxygen uptake, blood flow, and glucose utilization in patients with cerebrovascular disease, seizure disorders, cardiovascular disease, and some degenerative disorders. With PET, viable tissue can be discriminated from nonviable tissue and the amount of nutritional blood flow to an area can be identified. PET is often combined with CT [5; 6; 8].

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is possible because hydrogen nuclei in abnormal tissues behave differently in a magnetic field. A computer can manipulate these differences into a detailed picture of the organ under study. This noninvasive study does not require contrast media or exposure to radiation. The patient lies supine in a large magnet. Loud clacking or knocking noises are normally emitted during this time. Bone tissue is not visualized with MRI, but soft tissue close to bone is easily viewed, making MRI useful for assessing problems of the skull and spine [5; 6; 8]. Patients should be instructed to remove all jewelry and to avoid makeup or nail polish.

Radionuclide Scan of the Brain

A radionuclide scan detects intracranial masses, vascular lesions, and areas of ischemia, infarction, and hemorrhage. A radionuclide, usually administered intravenously, accumulates in affected areas if the blood-brain barrier has been compromised. Oral or intra-arterial administration can also be used.

Some scans also include oscilloscope scanning of the carotid and cerebral blood flow. After the radioactive isotope has circulated for at least one hour, a scanner records the accumulation of isotopes. Another scan can be done three to four hours after injection [5; 6; 8].

Nursing Implications

Allergies to the isotope should be assessed prior to a radionuclide scan. No dietary or fluid restrictions are required. Potassium perchlorate may be given to block uptake of the isotope by the thyroid, choroid plexus, and salivary glands. Patient education should include the steps of the procedure, the time requirement, and the fact that the injection will be the only discomfort. All jewelry and metal objects should be removed. To prevent patient apprehension, explain that the radioisotope is harmless to self and others and is quickly excreted from the body. This test is often combined with CT and angiography to help confirm the diagnosis [17].

Cerebral Angiography

Cerebral angiography (also referred to as intra-arterial digital subtraction angiography) is used to diagnose intracranial lesions. With this approach, a radiopaque contrast medium is injected into blood vessels of the head and neck to allow visualization of intracranial and extracranial vessels on x-ray. Cerebral angiography can reveal aneurysms, arteriovenous malformations, and displacement of vessels by masses, edema, or herniation. The test is also used during surgery to check the position and integrity of aneurysm clips [5; 6; 8].

Contrast material used with cerebral angiography can be injected into a variety of sites. The most common are the carotid, brachial, and femoral arteries. Catheters are often used to facilitate access. Injection is done under local or general anesthesia in the operating room or in a special procedures area where resuscitation equipment is available. This test is contraindicated in patients with renal, hepatic, thyroid, or clotting disorders as well as in those who are hypersensitive to iodine or contrast materials [5; 6; 8].

Nursing Implications

Patient education includes a review of the procedure's purpose. The risks of the test should be explained, including CVA, thrombus, allergic reactions, seizures, pulmonary emboli, and visual disturbances. Informed consent forms should be signed. To reduce apprehension, patients should be walked through the procedure and informed that a supine position, with the head secured to prevent movement, will be required throughout the test, which lasts approximately two hours. Periodic assessment of heart function and blood pressure is routine [17].

Careful explanation of sensations expected when the dye is injected is essential to reduce fear; injections of contrast medium into the blood vessels of the head can be painful. Sensations vary from warmth to severe burning behind the eyes and in the jaw, teeth, tongue, and lips. Even fillings in the teeth can feel warm. The sensation of heat lasts four to six seconds after the dye is injected. More than one injection may be necessary [17].

Nursing care also includes collection of baseline data and preparation of the patient. Vital signs and neurologic status should be recorded and pulses distal to the puncture site marked to facilitate assessment after the procedure. If the carotid site is used, documenting neck measurements allows for comparison after the test. Hairpins, nets, and dentures should be removed. The patient should also void before the test begins [17].

Preprocedure medications may include anti-anxiety agents (to help the patient relax) and atropine sulfate to protect against a reflex response (hypotension, syncope, and bradycardia) by the carotid artery. Patients should be well-hydrated to promote clearance of the dye by the kidneys but otherwise should fast for six to eight hours before the test. The injection site(s) should be shaved and prepared with an antiseptic solution. Local anesthetic is usually given before insertion of the catheter or needle [17].

Immediately following the removal of the needle or catheter from the artery, pressure should be applied to the puncture site for 15 minutes to prevent hemorrhage and development of a hematoma. Vital signs and neurologic checks are conducted and recorded every hour for 4 hours, then every 4 hours for 24 hours. Intake and output should also be noted [17].

The puncture site, surrounding areas, and distal extremities should be carefully monitored. The puncture site should be assessed for redness, swelling, and superficial-to-deep hematoma. A pressure dressing and ice bag may decrease the risk of bleeding and discomfort. When the carotid artery is used, the patient should be monitored for respiratory distress and swallowing difficulty, which may indicate excessive edema or an expanding hematoma. For puncture of the femoral or brachial site, pulses in the distal limb are monitored for 12 hours and the limb is maintained in an extended position and observed for normal temperature, color, and sensation. Blood pressure is not monitored in the involved arm. A physician should be alerted immediately of any untoward effects. The patient should rest quietly in bed for two to four hours after the procedure, with food and medications given as tolerated [17].

Electromyography and Nerve Conduction Studies

Electromyography (EMG) records electrical activity in muscle at rest and during contraction. Findings allow differentiation of muscle disease from lower motor neuron dysfunction. Recorded electrical patterns can be diagnostic of various diseases, including myositis, dystrophy, and myasthenia gravis. EMG can be used to assess function in the spinal cord, nerve root, nerve plexus, peripheral nerves, and/or myoneural junction, and the test can detect and measure regeneration of nerve and muscle before clinical signs appear. This information can be used to predict recovery [5; 6; 8].

A nerve conduction test is often administered along with an EMG. This test measures the strength and speed of conduction in the sensory and motor fibers of peripheral nerves. Motor conduction studies are valued for assessing nerve damage when minor symptoms of motor weakness or atrophy exist. Sensory fiber conduction rates are especially useful for diagnosing neuropathies in patients with diabetes, alcoholism, metabolic or nutritional disorders, and trauma. Sensory nerve fiber conduction is assessed with a single electrical stimulus. The action potential is recorded by an electrode placed on the skin where the nerve is close to the surface. The recorded conduction time is compared with established norms for healthy nerves [5; 6; 8].

Nursing Implications

Fluid or food intake is not restricted prior to EMG or nerve conduction study. However, in some cases cigarettes, coffee, tea, cola, or medications may be restricted before the test. A written consent is obtained [17].

Patients should be educated about the time EMG takes (one hour or more), steps of the procedure, the need to insert a needle into the muscle, and the changing of needle position, which may cause discomfort. Patients will need to cooperate in flexing and releasing muscles during the test [17].

Residual pain after the test is treated with warm compresses and prescribed analgesics. If medications were withheld for the test, the physician will indicate when they should be resumed [17].

Lumbar Puncture

Lumbar puncture, also referred to as a spinal tap, is an invasive procedure used to obtain samples of CSF, to measure CSF pressure, and therapeutically to reduce pressure in conditions such as subarachnoid hemorrhage. Lumbar puncture is also done to instill antibiotics, steroids, and/or dye or oxygen for diagnostic studies and to evaluate CSF flow [5; 6; 8].

Lumbar puncture can be done at the bedside or in the diagnostic lab. The procedure is done with the patient positioned to one side (typically the left) with head and knees flexed toward the abdomen. The patient is assisted in maintaining this position, which separates the vertebrae, allowing the needle to enter the subarachnoid space at the level L3-to-L4 or L4-to-L5. Aseptic technique is required [5; 6; 8]. In some patients, particularly those who are overweight, sitting and leaning forward may provide better access.

Contraindications for lumbar puncture include skin lesions in the lumbar area, epidural infection or abscess, and lumbar deformity near the puncture site. Lumbar puncture is also contraindicated with increased ICP because of the heightened risk of brain compression or herniation through the tentorial hiatus when the spinal fluid pressure is lowered. In some circumstances, such as when meningitis is suspected, the need to establish a diagnosis outweighs the dangers of the procedure [5; 6; 8].

Possible complications of lumbar puncture include headache, transitory low back pain and root irritation, and meningitis or abscess. Post-lumbar puncture headache is believed to result from the loss of CSF at the puncture site, which lowers the spinal fluid pressure and places tension on the intracranial structures [5; 6; 8].

Nursing Implications

The purpose of the test and steps of the procedure should be thoroughly explained to the patient, emphasizing the importance of lying still in the flexed position during the lumbar puncture. Patients should be advised of the brief episodes of pain when the anesthetic and spinal needle are inserted. A discussion of postprocedure activity is also helpful [17].

After a lumbar puncture, most physicians require the patient to remain recumbent, but turning should be encouraged. Forcing fluids will help to promote

replacement of withdrawn spinal fluid. Headaches, experienced by many, can be treated with hydration, caffeine, and/or prescribed analgesics. Patients should be carefully monitored for signs of meningitis and drainage or discharge at the puncture site. Abnormal findings, such as the presence or absence of glucose and abnormal spinal fluid color, should be reported immediately [17].

Suboccipital Puncture

Suboccipital (cisternal) puncture is an alternative procedure for obtaining CSF when lumbar puncture is contraindicated. For this test, the patient's head should be flexed forward so the chin touches the chest. A short, beveled needle is inserted into the subarachnoid space of the cisternal magna. Suboccipital punctures are hazardous because of the proximity to the brain stem and the risk for subarachnoid hemorrhage [5; 6; 8]. Fluoroscopic guidance can help avoid these complications.

Nursing Implications

Preprocedure care for suboccipital puncture is the same as for lumbar puncture. Bed rest is usually maintained for several hours after the procedure. Respiratory and cardiac function is observed frequently because of the puncture's proximity to medullary centers controlling these functions. Headaches are uncommon [17].

Myelography

In a myelography, fluoroscopy and radiography are combined to study the subarachnoid space, spinal cord, and vertebral bodies. The test reveals spinal cord tumors, herniated or ruptured intervertebral disks, and nerve root injury. In this study, a lumbar puncture is used to replace a small amount of CSF with a radiopaque dye or gas. The patient is positioned on a movable table that tilts to various positions to allow the flow of dye through the subarachnoid space. Abnormalities in flow provide the diagnostic information [5; 6; 8].

Nursing Implications

Prior to myelography, it is important to assess the patient for allergies to iodine, shellfish, or radiographic dye, and to obtain a written consent for the patient. Preprocedure fasting for four to eight hours is necessary, and baseline vital signs and neurologic status should be recorded. An enema may be ordered to reduce x-ray shadows. A sedative (to relax the patient) and atropine (to reduce secretions) may be ordered [17].

Patient education should cover information about the procedure, including the length; the purpose of the lumbar puncture (or suboccipital puncture, if lumbar deformity or skin infection is present); the usual response to the dye, which may include flushing, a warm sensation, a salty taste, headache, nausea, and vomiting; the positioning and strapping to the table; and the need to tilt the table during the study and to remove the dye [17].

After the procedure, the patient's head should be positioned to keep the dye from entering the cranium. Vital signs and neurologic status, including nuchal rigidity, nausea, vomiting, and reports of back pain and spasms, should be monitored for at least 24 hours or as indicated by policy. Headache may be treated by positioning and analgesics. Patients should be encouraged to consume plenty of fluids while their intake, output, and ability to void are monitored [17].

CSF Analysis

Because CSF is in contact with the components of the CNS, it can be valuable in the diagnosis and evaluation of CNS disease progression or healing process. CSF is colorless and consists of water and traces of protein, glucose, sodium, chloride, and potassium. The average volume in adults ranges from 100–150 mL. CSF pressure in the supine patient ranges from 7–20 cm/H₂O. Fluid that is dark in color or even pink-tinged indicates hemorrhage or a cerebral bleed [5; 6; 8].

The collection and handling of CSF should be carefully controlled to ensure proper analysis. The fluid is collected in sterile tubes and delivered to the laboratory immediately. Some diagnostic evaluations require processing within one hour [5; 6; 8].

Certain analyses of CSF require special considerations. For example, the evaluation of the glucose level requires that a blood glucose level be obtained not more than three hours before the puncture. Because serum glucose levels are reflected in the CSF, an abnormal CSF level may be a reflection of the serum level, rather than a neurologic disease process. Similarly, CSF chloride levels can be affected by serum levels. Because of this influence, CSF chloride levels will not be valid if the patient has received IV therapy with electrolyte solutions prior to or during the lumbar puncture [5; 6; 8].

NURSING DIAGNOSES, PLANNING, AND IMPLEMENTATION

Careful gathering and analysis of patient information, physical findings, and diagnostic data are needed for all patients with suspected CNS dysfunction. Patients who have CNS conditions often require long periods of treatment and time to adapt to the changes in their lives brought on by the disease. These adaptations may involve physical and mental changes, including the reorganization of self-image and the adjustment of expectations of life. Specific nursing interventions depend on when care is sought for the disease process and how the individual responds physically and mentally to the illness [41].

INEFFECTIVE AIRWAY CLEARANCE

Patency of the respiratory tract depends on a person's ability to maintain proper positioning, effective functioning of the muscles of respiration, and healthy respiratory tract mucosa. Neurologic diseases often cause dulled consciousness, confusion, and

decreased motor function, all of which can affect respiration. Patients may not be able to assume positions independently that keep the tongue from obstructing the airway. Disease processes that affect cranial nerve function (e.g., myasthenia gravis, ALS) or level of consciousness can make it difficult to swallow or cough to clear the airway of sputum, foreign objects, or vomitus [30; 40].

Ingestion of adequate fluids can be a problem for patients with neurologic problems as well. Those with cognitive impairment may forget to drink, and patients with communication problems may be unable to request fluids. Physical impairments may make drinking difficult or impossible. These situations can result in dehydration and drying and crusting of the mucosal tissue in the oropharyngeal area and respiratory tree, with related damage to mucous cells and cilia. This damage compromises an individual's normal phagocytic action and ability to expel organisms, increasing the risk of respiratory tract infections [30; 40].

Promoting Airway Clearance

For the physically impaired or obtunded patient, careful positioning with pillows and special devices promotes drainage of secretions, maintains a patent airway, and reduces the risk of aspiration. This includes careful positioning during meals, supplemental oral intake, nasogastric feedings, and oral care. A suction machine should be at hand if choking is a risk or secretions are unmanageable. Active or passive range-of-motion exercises and frequent position changes help to promote mobilization of secretions [41].

Steps to ensure adequate hydration support healthy respiratory function both by promoting the elimination of secretions and the destruction of organisms. Careful, ongoing assessment of the oral pharyngeal mucosa, breath sounds, and activity to manage secretions is necessary because the patient's condition may change quickly and the change may not be obvious [41].

ALTERATION IN BOWEL ELIMINATION

Constipation

Many CNS diseases can cause constipation. Neurologic alterations can result in decreased fluid intake, decreased physical activity, impaired communication, inability to monitor bowel patterns, inability to initiate changes in diet to correct constipation, limitations in using toilet facilities, and increased dependence resulting in a lack of privacy [30; 40].

Diseases such as stroke, myasthenia gravis, and ALS may result in paralysis of the lips, tongue, mouth, pharynx, or larynx (bulbar paralysis). The resultant difficulty in swallowing frequently reduces fluid intake, making stools hard and difficult to pass [30; 40].

Immobility from motor neuron damage or decreased level of consciousness limits abdominal muscle contraction and bowel activity slows. Positioning constraints, as with spasticity or contractures, can prevent a patient from assuming positions that facilitate defecation. For example, being confined to bed for treatment of a lumbar disk problem often causes constipation brought about by both immobility and positioning constraints [30; 40].

Loss of toilet privacy frequently accompanies the physical disabilities of weakness, paralysis, and immobility. Embarrassment over exposure during toilet activities and related helplessness can hamper normal defecation [30; 40].

Special nursing techniques for the patient with limited motor function who needs to increase fluid intake to keep stools soft include special positioning and the use of special cups, straws, and other adaptive devices. Recognizing the patient's physical and mental deficits allows for effective planning and assistance. If the patient's communication is limited, family or friends may be able to suggest favored fluids and foods that will increase fluid intake and bulk to relieve or prevent constipation [41].

Incontinence

Bowel incontinence may accompany a wide variety of disease processes. Sphincter control can be lost because of cortical, spinal cord, or peripheral nerve damage; recovery of control may or may not be possible. Following brain trauma, patients may have temporary or chronic loss of bowel continence. Aphasia predisposes some patients to incontinence because they cannot express their need to defecate. Tube feedings and medications may cause diarrhea, which is more difficult to control [30; 40].

INEFFECTIVE BREATHING PATTERN

CNS diseases can interfere with the brain stem's regulation of respiratory function. Related changes in body function—decreased level of consciousness, immobility, obstruction of airway, and aspiration—can result in decreased ventilation, decreased gas exchange, and hypoxia. Hypoxia presents two threats: anoxia in vital neurons and dilation of cerebral vessels leading to an increase in ICP. Signs and symptoms include increased lethargy, rising blood pressure, and depressed respirations [30; 40]

Pathologic conditions producing an increase in ICP can lead to herniation, damaging respiratory centers in the brain stem. This trauma and other pathologies involving the brain stem, such as stroke or tumor, alter the rate, depth, and rhythm of respirations. Diseases, injuries, or infections affecting phrenic innervation of the diaphragm can result in loss of stimulus for breathing [30; 40].

Hyperventilation can result from problems such as encephalitis, drug overdose, and hypoxia. It can also be initiated by psychological stress or pain, both common in patients with CNS diseases [30; 40].

ALTERATION IN CARDIAC OUTPUT

Impairment of cardiac output can follow injury to the brain stem's vasomotor center, which influences cardiac function via the autonomic nervous system. The brain stem's vasomotor center also controls blood pressure. With sharp elevation in blood pres-

sure initiated to perfuse a severely edematous brain, the vasomotor center initiates a reflex slowing of the heart, reduces contractility, and produces vasodilation. Cardiac output can also be compromised by anoxia stemming from alterations in respiratory function [30; 40].

Decreases in vasomotor tone with various neurologic problems can affect cardiac function due to the decrease in blood returning to the heart. In addition, spinal cord injuries producing spinal shock severely decrease vasomotor tone. Orthostatic hypotension after long periods of bed rest is also the result of a loss of vasomotor tone [30; 40].

IMPAIRED COMMUNICATION

A variety of conditions affecting the CNS, including stroke, gunshot wounds, and carbon monoxide poisoning, can damage speech centers located in the dominant hemisphere. If Broca's area or the nerve fibers connecting Wernicke's area and Broca's area are damaged, speech may be severely limited, but understanding of one's own and others' speech will be intact. If Wernicke's area is damaged, speech may be fluent but nonsensical [30; 40].

Speech can also be impaired if cranial nerves responsible for movement of the lips, tongue, oral pharynx, and larynx are injured. Damage to spinal nerves that control respirations can also affect verbal communication [30; 40].

Destruction or swelling of neuronal tissue in the parietal, occipital, and/or temporal lobes impairs perception and interpretation of stimuli including touch, sight, and hearing. As such, aspects of awareness needed for communication can be altered [30; 40].

Limitations in motor function will impair nonverbal forms of communication, including writing, gesturing, facial expressions, and various postures that add expression to oral communication. Impaired nonverbal communication can influence how information is received, because gestures, facial expressions, and posturing enhance oral communication [30; 40].

IMPAIRED PHYSICAL MOBILITY

Hemiplegia from stroke, tumor, or spinal cord injury, or motor deficits from peripheral nerve injury may be obvious or subtle. Subtle limitations, such as decreases in range of motion, lesser involvement in activities of daily living, or unsteady gait, are often first identified by the nurse, especially in patients with progressive diseases. In extrapyramidal tract diseases, the impairment in mobility may be difficult to detect. Nevertheless, careful analysis may reveal clumsiness and limitations in movement resulting from tremors and rigidity [30; 40].

Mobility may also be compromised by cerebellar dysfunction, alterations in sensory perception, changes in mentation, and changes in mood and energy level. Cerebellar dysfunction can produce ataxia, uncoordinated movements, and limitations in depth perception. Damage to the parietal, temporal, or occipital lobes or to the sensory tract impairs an individual's ability to perceive and interact with the environment to varying degrees. Limitations from sensory deficits can often be difficult to determine and measure; careful observation and diagnostic testing are necessary [30; 40].

Activity levels frequently increase or decrease with alterations in mental function. Both states are potentially dangerous. Patients who are restless and confused may also have immobility if restraints are used to ensure safety. Body image changes that accompany neurologic diseases frequently lead to depression, fatigue, decreased participation in daily activities, and fewer interactions with the environment [30; 40].

ALTERATION IN NUTRITION

The physical, psychological, and social factors accompanying neurologic diseases can have a profound effect on an individual's nutritional status. Common physical problems that may limit intake include lack of exercise, inability or awkwardness in self-feeding, social isolation, difficulty chewing and swallowing, fear of choking, and limited energy for eating [29; 30].

For the individual at home, physical and mental deficits can make buying food and preparing meals challenging. A limited income (because of illness-related unemployment and healthcare costs) can also influence the amount and type of food available. As a result, the quality and quantity of food consumed may be reduced [29; 30].

Sight, smell, taste, and touch—all important to an interest in eating—are altered by many CNS diseases. In addition, mental changes from injury to the cerebral cortex or the psychological response to disease can diminish interest in eating [29; 30].

SELF-CARE DEFICIT

CNS diseases predispose patients to many types and degrees of self-care deficits. Planning effective care depends on determining how the following factors influence the treatments and activities planned for or by the patient [30; 40]:

- Cause of the illness
- Degree of influence on self-care
- Prognosis and expected outcomes
- The patient's personality and response to deficits
- The family's interest and ability to support the patient mentally and physically

ALTERATION IN SENSORY PERCEPTION

CNS dysfunction can change one's perception of the environment and spatial relations. The parietal, occipital, and temporal lobes all have major roles in the interpretation of messages received from peripheral nerves. The interdependence of these areas within the cerebral cortex and in the cerebellum, subcortical areas, brain stem, and peripheral nerves influences the patient's physical and mental interaction with the environment. Perceptual changes and deficits predispose the patient to injury, depression, confusion, fear, and isolation [30; 40].

SEXUAL DYSFUNCTION

Sexual dysfunction is relatively common in patients with CNS conditions and can result from spinal cord trauma, peripheral nerve trauma, or damage to the nerves necessary for sexual activities. Pharmacotherapy may also produce sexual dysfunction. Mental changes stemming from brain damage may result in inappropriate sexual behaviors [30; 40].

ALTERATION IN URINARY ELIMINATION

Urinary retention or incontinence can result from diseases affecting the cerebral cortex, spinal cord, and/or peripheral nervous system. Patients with CNS dysfunction often experience both a diminished awareness of bladder fullness and a decreased ability to empty the bladder. Alterations in consciousness from trauma, electrolyte imbalance, anoxia, and disease processes can produce temporary or permanent urinary incontinence [30; 40].

CONGENITAL DISORDERS AFFECTING THE CENTRAL NERVOUS SYSTEM

A congenital disorder of the CNS can be defined as a developmental defect. Although the causes of maldevelopments are often unknown, the majority are considered to result from the hereditary transmission of a chromosomal abnormality or are secondary to embryonic damage. The developing CNS in the fetus is particularly vulnerable to radiation effects, anoxia, metabolic diseases, and infections in the mother [14; 15].

CEREBRAL PALSY

Cerebral palsy is not a disease entity per se but a variety of neuromotor disorders resulting from cerebral hypoxia or damage to the nervous system in utero, at birth, or in early life; most cases are the result of damage that occurs before or during birth. Cerebral

palsy occurs most often in infants born prematurely or after a difficult labor, at the rate of about 2 cases per 1,000 live births [42; 46]. Causal factors can be divided into four groups:

- Genetic defects associated with chromosomal abnormalities
- Prenatal factors, including:
 - Maternal infections (e.g., rubella, cytomegalovirus, toxoplasmosis)
 - Irradiation
 - Harmful drug intake
 - Malnutrition
 - Toxemia
 - Gestational diabetes
 - Rhesus (Rh) and ABO blood incompatibilities
- Prenatal factors causing anoxia of the brain, such as:
 - Difficult breech and midforceps deliveries
 - Improper anesthesia during labor and delivery
 - Premature birth
 - Low birth weight
- Postnatal factors causing injury to the neonate's brain, including:
 - Cerebral vascular lesions
 - Infections
 - Trauma
 - Malnutrition
 - Prolonged convulsive seizures

Developmental disorders are more frequent in infants of mothers younger than 20 or older than 35 years of age. Data from CT scanning confirm that perinatal and/or postnatal cerebral vascular bleeding is the major cause [42; 46].

Clinical Manifestations

The symptoms and signs of cerebral palsy are variable, ranging from mild muscle incoordination to severe spasticity. Intellectual performance is often hampered by seizures and speech, visual, hearing, and motor impairment. As a result, individuals with cerebral palsy often develop serious emotional and social problems. In addition, at least half have intellectual disability as a primary aspect of the disorder. Patients with cerebral palsy often live into adulthood and develop other health problems [42; 46].

Therapeutic and Nursing Measures

The treatment of cerebral palsy is dependent on the extent of damage and the patient's needs. Although the initial damage in the brain cannot be reversed, earlier and aggressive treatments may help to improve function and adjustments for the young nervous system and musculoskeletal system [1]. Common treatment approaches include physical therapy and rehabilitation, braces and other orthotic devices, assistive technology, pharmacotherapy, and surgery.

MUSCULAR DYSTROPHY

Muscular dystrophy is a hereditary, degenerative neuromuscular disorder characterized by chronic, progressive wasting and weakness of voluntary muscles. Far more common in men than women, the disease affects both children and young adults. The nine different types of muscular dystrophy (i.e., myotonic, Duchenne, Becker, Emery-Dreifuss, limb-girdle, facioscapulohumeral, congenital, oculopharyngeal, and distal) vary in the age of onset, rate of symptom progression, and clinical manifestations. All types exhibit degenerative changes in the muscle fibers [42; 46].

Clinical Manifestations

Muscular dystrophy is characterized by a progressive weakness and atrophy of all voluntary muscles, with eventual contracture and confinement to a wheelchair. Failure of cardiac or respiratory musculature usually leads to death in the second or third decades of life [42; 46].

Therapeutic Measures

There is no cure for the muscular dystrophies. Accurate diagnosis is essential, however, to rule out similar muscle diseases for which effective treatments are available. Patients with muscular dystrophy are managed with supportive interventions and assistive devices. Physical therapy may enable patients to gain optimal use of affected muscles, and muscle stretching helps prevent contractures. Tendon-lengthening surgeries have varying degrees of success [42; 46].

Specific Nursing Measures

Patients with muscular dystrophy should remain physically active as long as possible. Physical therapy regimens are essential. Stretching and resistive exercises preserve joint range of motion, prevent or minimize contractures, decrease atrophy, and promote mobility. Exercises should be done at least twice every day for the outpatient or four times per day for inpatients. Each joint should be put through its normal arc of motion, as tolerated, while avoiding moving joints beyond the point of resistance. Exercises should be halted whenever the patient has pain [42].

Patients with muscle spasticity experience an increased tonus in a weak muscle. The objective is to promote muscle relaxation and prevent complications, such as contractures, muscle atrophy, pressure injuries, and urinary tract infections. Patients and their families should be taught to avoid stimuli that can increase spasticity, including fatigue, maintaining one position for too long, and cold temperature [42].

Braces may be used to stabilize the lower limbs and trunk, but they should be light enough for weakened muscles to support. Other potentially helpful assistive devices include bed trapezes, handrails, raised toilet seats, and wheelchairs [42].

Management of alteration in nutritional intake is another priority. The physical inactivity of patients with muscular dystrophy may contribute to unnecessary weight gain. Patients can plan meal schedules that permit smaller, more frequent meals. Evaluation of weight patterns and energy requirements can help [46]. The goal is to promote independence as long as possible.

HUNTINGTON CHOREA

Huntington chorea, also known as Huntington disease, is a rare hereditary disease of the CNS. The disorder is progressive, degenerative, and fatal. It is characterized by severe choreiform movements and mental deterioration. Although the disease was first recognized as a medical entity more than 100 years ago, public awareness has lagged. As noted, Huntington chorea is inherited through the autosomal dominant gene with full penetrance. Therefore, 50% of the children of individuals with the disease eventually inherit it [42; 46].

Clinical Manifestations

Huntington chorea usually develops insidiously and runs its course over 15 to 20 years. Onset is typically in middle life (35 to 40 years of age). The earlier the age of onset, the more rapid the deterioration [42].

The choreiform movements are the most striking characteristic of Huntington chorea. They begin slowly, usually first in the face and upper extremities. Facial grimacing and jerking limb movements occur. Over time, movements become frequent, erratic, and violent [42].

As the disease progresses, communication becomes poor, with increasing dysarthria and unintelligible speech. Early behavioral changes include periods of irritability, labile mood swings, and impulsiveness. Periods of apathy, elation, depression, and aggression can be expected. Progressive memory impairment, inattention to personal hygiene, and cognitive impairment accompany the personality changes [46].

Seizures also occur in the end stages of classic Huntington chorea. Death is usually from cardiac or respiratory failure, extreme systemic exhaustion, or suicide [46].

Therapeutic Measures

Treatment of Huntington chorea is difficult. There are no known methods of arresting the disease, and drug regimens have been generally unsuccessful. Exercise, physical therapy, nutritional support, and speech and language therapy can be helpful.

Specific Nursing Measures

The chronic, disabling nature of Huntington chorea requires interventions that are preventive, protective, and supportive. These patients are at high risk for injury, and as the disease progresses, a safe physical environment should be maintained, with precautions similar to those for seizures. As mental and cognitive capacity deteriorate, patients will rely more on caregivers to carry out activities of daily living—daily hygiene, nutrition, and elimination. Genetic counseling is crucial to screen families whose children may later develop the disease, to counsel these families regarding the risks, and to support other family members carrying the gene [42; 46].

NEUROFIBROMATOSIS

Neurofibromatosis is a hereditary disorder characterized by a variety of congenital abnormalities. The skin, CNS, bones, and endocrine glands are most commonly affected. Usually, some form of benign tumor is the typical finding [14; 15].

Neurofibromatosis is one of the most common hereditary disorders. The incidence is approximately one case per 3,000 live births [14; 15]. In the United States, there are an estimated 100,000 cases. The disease is slightly more common in males. Each child of an affected parent has a 50% chance of inheriting the gene and developing neurofibromatosis [14; 15].

Clinical Manifestations

In the peripheral form of neurofibromatosis, multiple cutaneous and subcutaneous nodules occur. Cutaneous tumors are palpated in the dermis as discrete soft or firm papules varying in size from millimeters to centimeters. If pressed, these soft nodules feel like a seedless grape, which aids in distinguishing lesions of neurofibromatosis from other tumors. Subcutaneous tumors are usually multiple, assuming two forms: discrete or plexiform neuromas. Discrete tumors are firm nodules that attach to the peripheral portion of a nerve. These nodules may cause neurologic or paresthetic pain to pressure and rarely cause weakness, atrophy, or sensory loss in the distribution of the affected nerve. The number

of nodules varies from a few to thousands, and the size varies from pea-sized to orange-sized. Plexiform neuromas are an overgrowth of subcutaneous tissue and can reach enormous sizes. The face, scalp, chest, and neck are typically affected with growths that feel like a “bag of worms” when palpated. The hypertrophy is highly disfiguring and often accompanied by underlying bone abnormalities. These tumors, if large enough, can cause increased ICP and brain stem compression [19].

Therapeutic Measures

Neurofibromatosis has no cure. The most promising approach is surgery for removal of symptomatic lesions. In cases of multiple CNS lesions, the decision to have surgery depends on the severity of symptoms, risk for survival, and the quality of life. Some have advocated aggressive plastic surgical treatment for cosmetic reasons or for removing lesions that might degenerate into sarcomas. Radiotherapy is not justified because of unsatisfactory results and the risks associated with radiation exposure [20].

Specific Nursing Measures

Patients with neurofibromatosis should be helped to recognize that there are many variants of the disease and that no two individuals have the same course and prognosis. Patient education should include the current extent of the illness and possible treatment strategies, with emphasis on the patient’s right to make decisions regarding treatment choices.

One challenge is helping the patient recognize and cope with the uncertainty of the disease course and prognosis. Lesions can change from asymptomatic to symptomatic at any time, and new lesions can develop spontaneously without warning. Some patients may experience symptom-free periods [42].

ARTERIOVENOUS MALFORMATIONS OF THE BRAIN

An arteriovenous malformation is characterized by the direct shunting of arterial blood into veins. Two-thirds of all patients with intracranial arteriovenous malformations experience symptoms before 30 years of age; most cases are congenital. About 1 in 20 patients with arteriovenous malformation have aneurysms, but only 1 in 75 patients with intracranial aneurysm has an associated arteriovenous malformation [9; 13].

Clinical Manifestations

Clinical manifestations of arteriovenous malformations include seizures, hemorrhage, headaches, motor and sensory deficits, organic mental impairments, visual dysfunction, and syncopal episodes. The most common are seizure activity and hemorrhage [9; 13].

Severe intracerebral bleeds can occur from these malformations. In these cases, presenting symptoms and signs include vomiting, intractable headache, and loss of consciousness. Less severe arteriovenous malformations can cause aphasias and hemiparesis. An estimated 10% to 15% of patients experience sudden, severe paralysis after seizure. An equal percentage develops a progressive rather than sudden hemiparesis. Sizable bleeds in the brain stem can lead to coma and death [9; 13].

Therapeutic Measures

Complete excision of the arteriovenous malformation is the treatment of choice for most patients and eliminates the possibility of rebleeding. Few patients are in the high-risk or inoperable category. Microneurosurgical techniques permit the removal of most lesions, regardless of size and depth. Those with subarachnoid hemorrhage require a recovery period before surgery to permit careful evaluation of neurodiagnostic studies. Angiography is essential to visualize the circulation of the malformation [13].

Specific Nursing Measures

Assessment of initial symptoms and signs (usually related to either seizures or hemorrhage) is one of the most important aspects of care for the patient with intracranial arteriovenous malformation. Although a subarachnoid hemorrhage with an arteriovenous malformation is generally less severe than a ruptured intracranial aneurysm, many of the symptoms will be the same. Intracerebral hemorrhage can occur [39].

Transient episodes of syncope and dizziness increase the patient's risk for injuries from falls. Patients should be instructed to assume a safe position and call for assistance at the initial onset of any of these symptoms. Any episodes should be carefully documented [39].

CNS DISORDERS OF MULTIFACTORIAL ORIGIN

CNS disorders of multifactorial origin can be associated with a combination of lifestyle factors, trauma, environmental toxins, and inherited defects.

STROKE

The two primary types of stroke are ischemic and hemorrhagic. In the United States, approximately 87% of all strokes are ischemic; 10% are hemorrhagic [76]. An ischemic stroke occurs when any artery that supplies the brain with oxygen becomes stenosed or occluded, resulting in infarction. In the case of hemorrhagic stroke, bleeding occurs below the arachnoid, the location of the brain's blood supply, allowing blood to directly contact and damage brain tissue. In addition, transient ischemic attacks (TIAs) are often a precursor to ischemic stroke.

Hemorrhagic Stroke

Hemorrhagic strokes are categorized by the location of the hemorrhage, either intracerebral or subarachnoid, with the former being more than twice as common—because of this, the term hemorrhagic stroke often refers to intracerebral hemorrhage (ICH) [76; 77; 78]. ICHs are characterized by bleeding directly into the brain parenchyma [78; 79]. Intraventricular hemorrhage describes bleeding that extends into the ventricles [79; 80]. Nontraumatic ICH is categorized as primary (unrelated to congenital or acquired lesions), secondary (caused by a congenital or acquired condition), or spontaneous (unrelated to trauma or surgery) [79].

The signs and symptoms of ICH include headache, vomiting, seizures, depressed consciousness, meningeal irritation, and blood-tainted CSF. The onset of symptoms may occur within seconds to minutes after the start of an ICH. Individuals with this type of stroke often feel more ill than those with an ischemic stroke.

ICH is the least treatable type of stroke [81]. Functional independence is regained within six months in approximately 20% of survivors [82]. The morbidity and mortality depend on the volume and location of the hematoma. The one-year mortality rate varies according to anatomic location, with the highest mortality rate (65%) associated with ICH in the brain stem; the rate is 57% for lobar hemorrhage, 51% for deep hemorrhage, and 42% for cerebellar hemorrhage [83]. Overall, 46% of patients with ICH survive one year and 29% survive five years [84].

As many as 80% of primary ICHs occur after small vessels are compromised by chronic hypertension [85]. Hypertension is associated with ICH originating in the periventricular deep white matter, deep subcortical structures, pons, and cerebellum [86]. In individuals older than 70 years of age, cerebral amyloid angiopathy, a condition that leads to amyloid protein infiltration into the cortical arterioles, is responsible for approximately 20% of ICHs [87].

Other causes of ICH include anticoagulant and antiplatelet use, drug use (e.g., cocaine, phenylpropanolamine), and other bleeding diathesis [81; 88]. Fewer than 15% of all cases of ICH are secondary to congenital vascular abnormalities and malignant brain lesions [79].

Subarachnoid hemorrhages occur less frequently than ICHs. The hallmark of subarachnoid hemorrhage is the immediate onset of a severe headache with signs of meningeal irritation [89]. Individuals may describe this headache as their “worst ever.” Nausea, vomiting, neck pain, and photophobia are also classic symptoms, although they are not always present [89]. Neurologic deficits may be acute or may manifest hours to days after the onset of bleeding.

Nontraumatic subarachnoid hemorrhages are subcategorized as aneurysmal or non-aneurysmal [90]. Aneurysmal subarachnoid hemorrhage is associated with higher rates of morbidity and mortality than non-aneurysmal hemorrhage. Among patients who live 3 months after the event, the risk of death is 8.7% within 5 years and 17.9% within 10 years [91]. In contrast, non-aneurysmal subarachnoid hemorrhages are associated with better outcomes and are less likely to cause death [92].

Most nontraumatic subarachnoid hemorrhages involve rupture of an intracranial aneurysm or cerebral arteriovenous malformation. Congenital arteriovenous anomalies are more likely to cause stroke in adolescents and young adults [93]. The incidence of perimesencephalic subarachnoid hemorrhage, a non-aneurysmal type, is increasing. Although the cause remains unknown, increased use of antithrombotic medications may be a factor [94; 95].

Therapeutic Measures

The primary focus of medical care after initial aneurysm rupture is to prevent rebleeding. Measures should be taken to manage blood and intracranial pressure. If necessary, a ventriculostomy tube may be placed to help alleviate remove blood and/or cerebrospinal fluid. [19; 20].

Communicating hydrocephalus, the third most common complication of subarachnoid hemorrhage, can occur with the bleed or weeks later as a result of a malabsorption or blockage of CSF. Hydrocephalus should be suspected if any of these signs appear:

- Mental status changes
- A decrease in level of consciousness
- Dementias
- Flat affect
- Urinary incontinence
- Disturbances in gait

Hydrocephalus is confirmed by a CT scan that shows an enlargement of the ventricles due to blocked absorption of fluid from the arachnoid space [19].

Surgical repair that includes clipping of the aneurysm neck is the best treatment of a ruptured intracranial aneurysm for patients who are neurologically stable. Early repair (24 to 28 hours after rupture) is sometimes advocated, particularly for those who are asymptomatic. Those who demonstrate more extensive signs of meningeal irritation or neurologic deficit may be at greater risk if repair is attempted during the first week following rupture. One complication of clipping the aneurysm is the potential for the client to develop the syndrome of inappropriate secretion of antidiuretic hormone. Clinical signs include confusion and increased specific gravity of urine [20].

Specific Nursing Measures

Unfortunately, about 40% to 50% of clients with subarachnoid hemorrhage due to ruptured intracranial aneurysms die from catastrophic bleeds before receiving medical attention [39]. For those who do have medical care, mortality and morbidity rates can be greatly reduced with careful nursing and medical management. Nursing care is aimed at preventing rebleeding, the most life-threatening complication, and other possible complications, including cerebral edema and hydrocephalus [39].

A patient's cardiovascular status may change because of hypothalamic dysfunction; a common cardiac change in a patient with a subarachnoid hemorrhage is sinus bradycardia with ST-segment or T-wave changes. These alterations should be differentiated from those of a myocardial infarction. Electrocardiogram pattern and vital signs should be monitored with the neurologic assessment [39].

Ischemic Stroke

Within minutes of the onset of ischemic stroke, the core of an infarct can begin to form at the least-perfused site. This site is encircled by an area partially altered metabolically and ionically by cytotoxic edema [96]. This area, the ischemic penumbra, is structurally intact and generally salvageable if reperfusion is achieved promptly. Because cerebral function deficits develop rapidly (within minutes to hours) as an ischemic stroke progresses, these brain attacks are a medical emergency. Although irreversible damage occurs, most individuals with stroke have recoverable penumbral tissue for at least three hours following the onset of symptoms [97].

The physical signs, symptoms, and sequelae of ischemic stroke are usually unilateral because of the circulatory anatomy of the brain. In general, ischemic strokes are categorized according to etiology: thrombotic and embolic [98].

Therapeutic Measures

Thrombolytic therapy with recombinant tissue plasminogen activator (rt-PA) is the only treatment approved by the U.S. Food and Drug Administration (FDA) for ischemic stroke [97]. Anticoagulant and antiplatelet agents are also used, but their appropriateness is a source of debate and ongoing research. Intra-arterial rt-PA may be beneficial for select patients; however, the drug is not FDA approved for this use [99]. Mechanical thrombectomy is a consideration as both a primary reperfusion strategy and in conjunction with pharmacologic fibrinolysis [97].



EVIDENCE-BASED
PRACTICE
RECOMMENDATION

With a goal to improve functional outcomes, the American College of Emergency Physicians recommends that intravenous tPA should be offered and may be given to selected patients with acute ischemic stroke within three hours

after symptom onset at institutions where systems are in place to safely administer the medication. The increased risk of symptomatic intracerebral hemorrhage should be considered when deciding whether to administer tPA.

(https://www.acep.org/globalassets/new-pdfs/clinical-policies/tpa-for-stroke_new.pdf. Last accessed November 18, 2021.)

Strength of Recommendation: B (Recommendation based on moderate clinical certainty)

Although emergent angioplasty and stenting are high-risk procedures, progressing strokes, which occur when patients' moderate neurologic deficits deteriorate significantly within 72 hours after onset, are associated with very poor outcomes and high mortality rates [100]. Therefore, some case studies suggest that emergency angioplasty followed by immediate or delayed stenting is appropriate for patients with a progressing stroke caused by carotid artery occlusion or stenosis, respectively [101; 102].

In the setting of acute ischemic stroke, justification for emergent (within the first 24 hours) or early revascularization with carotid endarterectomy is based on reports of increased risk of recurrent stroke in patients undergoing medical therapy while awaiting revascularization. However, the risk associated with emergency carotid endarterectomy is believed to be high, for several reasons, particularly in patients with an unstable neurologic status [103]. For some patients, however, the benefit of carotid endarterectomy may outweigh the risk.

Specific Nursing Measures

For individuals who have had a stroke and are medically stable, rehabilitation assessment, prevention of medical complications, and secondary prevention become the focal points [97; 104; 105]. To begin, the rehabilitation team's systematic evaluation of the patient addresses various issues, including the need for rehabilitation services; the risk of complications; physical functioning, cognition, and communication; and psychosocial conditions [105]. Next, the team works with the patient and family to implement a rehabilitation plan that includes a detailed exercise program and general as well as tailored strategies for secondary prevention [104]. Throughout, the team should strive to foster a climate of familial support [105].

Medical complications related to illness, being bed-ridden, or lack of proper care/attention can prolong hospitalization, impede rehabilitation, increase disability, or result in death. Living in an inappropriate post-stroke environment also substantially increases a patient's risk for complications. Complications may develop in as many as 85% of hospitalized patients who have had a stroke [106]. Thus, medical examinations before and during a patient's rehabilitation program should assess the most common risks of complications: skin breakdown, deep vein thrombosis, swallowing dysfunction, bowel and bladder incontinence, falls, and pain [105].

Transient Ischemic Attacks

TIA's are sometimes referred to as "ministrokes" because, like ischemic strokes, they are caused by inadequate cerebral blood flow. TIA's are also called warning strokes, as they often precede an ischemic stroke [107]. The proposed definition states that a TIA is "a brief episode of neurologic dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction" [108].

This definition was designed to reflect the ischemic pathogenesis of TIA, promote its early management, and support the use of diagnostic imaging techniques to ensure that the patient does not have infarction [108]. The American Heart Association/American Stroke Association guideline defines TIA as, "a transient episode of neurologic dysfunction caused by focal brain, spinal-cord, or retinal ischemia, without acute infarction" [104; 109].

The risk of ischemic stroke is dangerously high in the period following a TIA. Research indicates that one-half of subsequent strokes occur within the first 48 hours, and a meta-analysis showed that approximately 5% of patients who have a TIA will have an ischemic stroke within seven days of that event [107; 110].

Although the most common focal neurologic signs of TIA are sudden-onset unilateral weakness and numbness or tingling in a limb, a TIA can cause any of the following symptoms [111; 112]:

- Numbness of the face, hand, or leg, with or without weakness
- Paralysis
- Slurred speech
- Dizziness
- Double vision
- Hemianopia
- Transient monocular blindness
- Imbalance
- Aphasia
- Confusion
- Head pain

By the time of evaluation, however, most patients appear asymptomatic because TIA's usually resolve within five minutes [113]. A clinician should highly suspect a TIA if the patient says, "I don't know why I'm here. Whatever it was, it is all better now" [114].

TIAs are caused by conditions similar to those leading to ischemic stroke [104]. Among the common causes are atherosclerosis of large vessels, cardioembolism, and atrial fibrillation. Uncommon causes include hypercoagulable states, arterial dissection, sympathomimetic drugs (e.g., cocaine), and arteritis (caused by noninfectious necrotizing vasculitis, drugs, irradiation, or local trauma) [115].

Therapeutic Measures

The use of certain antiplatelet therapies rather than oral anticoagulation for noncardioembolic ischemic strokes and TIAs has been shown to reduce the overall risk of recurrent stroke and decrease the incidence of fatal recurrent strokes [104]. Clopidogrel is appropriate for patients who are allergic to aspirin or for patients in whom dipyridamole-associated headaches occur.

Aspirin (50–325 mg per day) monotherapy, the combination of aspirin 25 mg and extended-release dipyridamole 200 mg twice daily, or clopidogrel 75 mg monotherapy are all acceptable options for initial therapy [104]. The selection of an antiplatelet agent should be individualized on the basis of patient risk factor profiles, cost, tolerance, and other clinical characteristics.

Because TIA is associated with a significantly increased risk for stroke, secondary prevention strategies mirror those for ischemic stroke [104].

SEIZURE DISORDERS

Seizures are common neurologic disorders that occur across the entire spectrum of age, gender, race, and socioeconomic background. Rather than a diagnosis focused on location (e.g., “symptomatic temporal lobe epilepsy”), diagnosis should reflect important diagnostic features, such as seizure type, lesion type, localization (e.g., “epilepsy with focal seizures secondary to cortical dysplasia in the temporal lobe”), and possibly other factors, including age at onset, EEG patterns, or other features.

There are many possible etiologies that may lead to the development of seizures or the specific diagnosis of epilepsy. Epileptic seizures have three basic underlying causes: genetic, structural/metabolic, and unknown [65]. Some cases of epilepsy are of a genetic origin, but other forms of epilepsy are caused by structural or metabolic defects, which themselves may or may not have a genetic origin [65; 66]. Other cases of epilepsy do not have any identifiable cause. Similar to structural/metabolic defects, the unidentified causes may have a heritable component.

Some patients with epilepsy experience one type of seizure; others experience several different seizure events. Different syndromes account for the varying patient histories, etiology of seizures, seizure type, clinical presentation, EEG readings, and neuroimaging findings. Common epileptic syndromes include febrile epilepsy, childhood absence epilepsy, juvenile myoclonic epilepsy, primary idiopathic generalized epilepsy, and localization-related epilepsy with complex partial seizures [67]. In total, more than 50 epileptic syndromes have been identified, each with specific associated diagnostic criteria.

Clinical Manifestations

The four generally recognized phases of a seizure are the prodrome (or preictal), ictal, interictal, and postictal stages. Not all patients experience, nor do all seizures include, every phase. The prodromal phase can last several days preceding a seizure. The prodrome is generally characterized as malaise or emotional changes [68]. An aura occurs immediately prior to a seizure, usually lasting a few seconds. Patients often describe an aura as a warning. An aura may be autonomic or it may involve the auditory, olfactory, sensory, or visual senses. The description of an aura can vary and may include weakness, an epigastric sensation, a sense of fear, visual hallucinations, aphasia, headache, feelings of being hot or cold, or sensing unpleasant odors [68; 116]. If a patient experiences auras, the auras are usually fairly consistent in that individual. However, auras may vary in the same patient, and the use of antiepileptic drugs may alter or obscure the aura.

The ictal phase is the duration of the actual seizure activity. The patient experiences a paroxysmal, uncontrolled, abnormal, and excessive discharge of electrical activity in the brain. There are also corresponding EEG changes [116]. The clinical manifestations will coincide with the type of seizure activity that the patient is experiencing.

The interictal phase is the period between seizures. Many people with epilepsy suffer from emotional changes during this phase, including fear, anxiety, and depression [117]. These disturbances can be more troublesome to patients than the seizures themselves.

The postictal period is the interval after the seizure episode. The patient may experience some change in consciousness or behavior. Some patients experience Todd paralysis, a numbness or weakness of an affected extremity or the side of the face. After a tonic-clonic seizure, the postictal phenomena may be more severe. The patient may experience amnesia, confusion, fatigue, and/or coma [116]. Often, neuronal discharges remain abnormal and the EEG may indicate some slowing.

Seizure types are divided broadly into three groups: generalized seizures, focal seizures, and unknown (including epileptic spasms) [65]. Seizures that do not fit any category remain unclassified until further assessment reveals the seizure type; however, this is not a separate classification.

Generalized Seizures

Generalized seizures begin and spread rapidly in bilaterally distributed networks and are followed by a period of postictal phase of continued altered consciousness [65; 118]. They are thought to originate from structures deep within the brain, radiating outward to the cortical surface. The networks may include cortical and subcortical structures, but the entire cortex is not necessarily affected. When a generalized seizure begins, there is synchronous involvement of the entire brain with diffuse EEG abnormalities. Approximately 20% to 25% of seizures are classified as generalized at onset [116].

Generalized seizures may result in a loss of consciousness, convulsions, falls, or muscle spasms. Some generalized seizures may encompass all of these events, while others may involve only one symptom.

Focal Seizures

Focal seizures (sometimes referred to as “partial seizures”) are the more common classification and originate in a circumscribed area or areas of the brain (i.e., a localized brain disturbance). This type of seizure occurs in 75% to 80% of patients with epilepsy [117].

A focal (“partial”) motor seizure occurs from a focus in the region of the brain’s motor cortex. Motor activity occurs in the corresponding part of the body innervated by the motor neurons that are affected. The hands and fingers have a large cortical representation; consequently, seizures are frequently noticed in these areas. The duration of these seizures is usually one to two minutes, although the patient may require additional time to completely recover after the event [119]. The patient will present with twitching/jerking movements in an extremity, the face, the eyes, or another area of the body. The patient remains fully conscious and aware of the seizure but has no control of the event [120]. These seizures usually remain localized, but the involuntary movement may spread centrally and involve an entire limb, one side of the body, or the entire body [117; 121].

Therapeutic Measures

Although prevention of epilepsy is the ultimate goal, this is not always possible. However, there is a variety of treatment options. Comprehensive treatment is an important aspect of care for all patients with epilepsy. Managing the disorder improves the quality of life, and the consequences of not treating may be great. Seizures that go untreated or are poorly controlled have an increased risk of becoming more severe or more difficult to manage. Treatment recommendations tend toward more aggressive management and earlier surgical evaluation for patients with epilepsy.

The utilization of antiepileptic drugs is the mainstay of therapeutic options. The effectiveness of anti-epileptic drugs relies on the ability to classify the seizure type by its clinical presentation, history, and diagnostic test findings. It is important to know the correct seizure type, as some antiepileptic drugs will be more effective or will exacerbate certain seizures or seizure syndromes.

Although surgical techniques have improved markedly over the past few years, epilepsy surgery is rarely considered a first-line treatment and is usually considered only after years of medication treatment. A surgical approach may be deliberated sooner if the patient's ability to function is hampered by frequent or severe seizures and a specific epileptogenic focus, such as mesial temporal sclerosis, is identified.

The vagus nerve stimulator, first approved by the FDA in 1997, was the first successful medical device for patients with uncontrolled focal seizures [68]. The stimulator is an implanted device, similar to a cardiac pacemaker, that is connected to the vagus nerve in the neck and stimulates the nerve with electrical impulses. The device is programmed to send electrical discharges at specific intervals automatically and periodically throughout the day [68]. The device is adjusted according to each patient's individual requirements and tolerance.

Specific Nursing Measures

Epilepsy is a chronic disorder and often requires long-term management. The patient and family should be encouraged to obtain information about epilepsy through self-education. Local epilepsy organizations often provide written materials and information via other media. Frequently, the patient's family or other significant persons require as much education as the patient, because they will be observing the patient during the actual events. These significant persons should be educated to care for the patient during and after a seizure.

Patient education is crucial to obtain medication adherence and provide optimum care. The patient should receive instruction on the type, dose, and potential side effects of each medication [122]. The patient should also understand that the medication is to be taken every day, on time, and as prescribed. Although brand and generic drugs are comprised of the same active compounds, their absorption may vary and patients should be cautioned not to interchange the medications. To avoid undesirable drug interactions, all professionals writing prescriptions should be aware of all medications a patient is taking. The patient should be informed that many medications interact with other CNS depressants, including alcohol. Patients should bring medication bottles with them at each visit.



According to the American Academy of Neurology and the American Epilepsy Society, patients should be advised that their risk for antiepileptic drug adverse events ranges from 7% to 31% and that these adverse events are predominantly mild and reversible.

(<https://www.aan.com/Guidelines/home/GetGuidelineContent/688>. Last accessed November 18, 2021.)

Strength of Recommendation/Level of Evidence: B
(Probably useful based on at least randomized controlled study or two consistent cohort studies.)

Patients with seizure disorders should wear identification wristbands or necklaces. Injuries are the primary complication of seizures. Patients with epilepsy often cause harm to themselves by biting their tongues or falling down and hitting a piece of furniture. The patient should be safeguarded from injuries and falls, and injury prevention is a key aspect of patient education and the maintenance of quality of life. The patient and family should be instructed on constructive methods of safety planning without being overburdened with unnecessary restraints and concern. If possible, the environment should be altered during a seizure rather than restraining the patient.

DYSTONIA

Dystonia is an abnormality of involuntary movement (dyskinesia). The abnormality can involve a single, focal muscle group or can be a diffuse neurologic syndrome in which multiple muscle groups are involved [28].

Clinical Manifestations

Onset of dystonia usually occurs before 15 years of age and is commonly associated with more severe clinical signs. Later onset is rare and more benign. The disease is observed as a slow, sustained, involuntary twisting of affected muscle of the trunk, limbs, neck, and face. Dystonia is not usually present during sleep. During waking periods, dystonic movements can be continuous. Dystonic movements tend to intensify with stress or fatigue and can be alleviated by relaxation or sleep [28].

Therapeutic and Nursing Measures

A primary treatment choice for patients with intractable dystonia has been surgery. More commonly, patients with less severe disease are managed by pharmacotherapy. Anticholinergic drugs have been beneficial in reversing acute symptoms of dystonia induced by antipsychotic drugs [28]. The effect of dystonia on the muscles may be reduced by stretching exercises [38].

HEADACHE

The pathophysiology of headaches is multifactorial and complex. When lesions of the brain cause headache (e.g., mass, fluid, hemorrhage), they do so by involving pain-sensitive structures inside the skull, such as arteries at the base of the brain, the dura area blood vessels, and certain cranial nerves that carry pain fibers. In addition, the external structures of the head are all pain-sensitive and give rise to a variety of headaches.

Headaches may be categorized as either primary or secondary. Primary headaches are pure headache syndromes, meaning they are self-originating and not triggered or produced by other disorders.

Primary headaches include migraine, cluster, and tension-type headaches [123]. Secondary headaches, as the name indicates, occur secondary to another cause (i.e., they are a symptom of other diseases). These may include vascular, traumatic, neoplastic, infectious, pressure, and metabolic disorders [123]. Secondary headaches account for only 10% of headaches.

Clinical Manifestations

The various types of headache present with differing symptoms and/or signs. A migraine is a type of neurovascular headache that is initiated by a neurologic event that causes vasodilation, which in turn is interpreted by the brain as pain. Adults with migraine headache describe episodic attacks with pain of moderate-to-severe intensity that are often throbbing in quality, unilateral in position, and aggravated by physical exertion. Migraines are usually associated with nausea, vomiting, and sensitivity to light and sound. Nausea is the most common characteristic. The duration of these attacks, when not treated, may last anywhere from 4 to 72 hours [124].

Cluster headaches are one of several types of trigeminal autonomic cephalalgias, primary headaches characterized by unilateral pain in the trigeminal nerve region. As defined by the International Headache Society, cluster headaches are attacks of severe unilateral pain in the orbit and/or the surrounding region of the eye that last 15 to 180 minutes [125]. The term “cluster” refers to the headache attacks occurring in daily episodes. The patient is headache-free in between the clusters.



EVIDENCE-BASED
PRACTICE
RECOMMENDATION

According to the American College of Radiology, the imaging mode of choice for patients who present with sudden onset of severe headache (“worst headache of one’s life” or “thunderclap headache”) is CT without contrast.

(<https://acsearch.acr.org/docs/69482/Narrative>. Last accessed November 18, 2021.)

Level of Evidence: Expert Opinion/Consensus Statement

Tension headaches are among the most common headache type seen in practice today. Episodic tension headaches are usually associated with a stressful event. They are of moderate intensity, typically are self-limited, and usually respond well to over-the-counter headache treatments. They are usually described as soreness, tightness, or a band-like pressure around the entire head. They are often accompanied by stiffness in the neck and shoulders [126]. Chronic tension headaches generally have the same pain characteristics as episodic tension headaches, including phonophobia or photophobia. They occur more frequently, with an incidence of at least 15 headaches per month on average for greater than three months, or greater than 180 days in a given year. The headache may last hours, or it may be continuous [125].

Other primary headaches include primary exercise headache, hypnic headache, and headache associated with sexual activity. A secondary headache may be diagnosed when another disorder known to cause headache has been demonstrated; headache occurs in close temporal relation to the other disorder and/or there is other evidence of a causal relationship; and headache is greatly reduced or resolves within three months (this may be shorter for some disorders) after successful treatment or spontaneous remission of the causative disorder. It is helpful to think of secondary headaches in terms of etiologic categories, such as vascular, traumatic, neoplastic, infectious, pressure, metabolic/toxic ingestions, and medication overuse.

Therapeutic Measures

Treatment of headache focuses on pain relief and prevention of recurrence, in the case of primary headaches, or on amelioration of the underlying cause, in the case of secondary headaches. Non-pharmacologic measures tend to be more effective for tension headaches than for migraines or clusters. These measures include the use of hot or cold packs, ultrasound, electrical stimulation, improvement of posture, trigger point injections, regular exercise, and consistent sleep schedules.

Pharmacotherapy with analgesics (e.g., acetaminophen) and migraine-specific agents (e.g., triptans) is often effective. In general, narcotics should be avoided.

TRIGEMINAL NEURALGIA

Trigeminal neuralgia is the most common neurologic disorder to affect the CN V and is also the most frequent of the neuralgias. Paroxysms of recurrent, excruciating, sharp, stabbing pain of short duration along one or more branches of the trigeminal nerve characterize this disorder [69].

Clinical Manifestations

An episode of trigeminal neuralgia is often described as paroxysms of excruciating pain, with a lightning-like stab that burns. The onset is usually abrupt and related to a precipitating event that irritates a “trigger” point. Movements such as speaking, brushing the teeth, washing the face, shaving, laughing, and movements in the maxillary and mandibular divisions of CN V can precipitate an attack [69].

Therapeutic Measures

Standard analgesics are ineffective to address trigeminal neuralgia pain. Morphine provides some relief but is generally contraindicated because of its addictive properties and overdose risk. Phenytoin (Dilantin) may be given intravenously to prevent an acute attack, although long-term use of oral phenytoin is not effective in treating paroxysms of trigeminal neuralgia [64]. Carbamazepine is the usual drug of choice.

Specific Nursing Measures

Preventing attacks of trigeminal neuralgia is the priority of nursing care. Patients should be kept in an environment that avoids exposure to drafts or excessive heat or cold. Patients often lose weight because they fear that chewing movements will precipitate an attack; food choices that avoid excessive chewing can be encouraged. Fearing an episode may also result in patients avoiding washing their faces, performing oral care, or shaving. For patients with loss of corneal sensation, special eye care is necessary to avoid complications [39].

DEGENERATIVE CNS DISORDERS

Degenerative CNS disorders are those with an unknown etiology that have an insidious onset and involve atrophy of neurons and nerve fibers. It is not uncommon for degenerative diseases to occur after a long period of normal nervous system functioning [69].

ALZHEIMER DISEASE

Alzheimer disease was first identified and named in 1906 by Dr. Alois Alzheimer, a German neuropathologist [127]. Symptoms seen in individuals with Alzheimer disease are partially the result of damage to the hippocampus and the cerebral cortex, reflected in memory loss, impaired cognition, and atypical behaviors.

Alzheimer disease is characterized by insidious, severe, and progressive cognitive impairment that is irreversible and eventually fatal. Alzheimer disease accounts for roughly 60% to 80% of all dementias in the United States [128]. It proceeds relentlessly, gradually destroying all cognitive functions.

There are two types of Alzheimer disease: familial and sporadic. Familial Alzheimer disease follows an autosomal dominant inheritance pattern, while sporadic Alzheimer disease has no known inheritance factor. Familial Alzheimer disease can be further classified as early-onset, when it occurs in individuals younger than 60 years of age, or late-onset, when it affects individuals older than 60 years of age [129].

Clinical Manifestations

The onset of Alzheimer disease is slow and insidious; impaired memory is usually the initial symptom, followed later by deficits in other cognitive domains. Symptoms may be present for several months before the family realizes the severity of the problem. In some situations, a spouse may shelter and cover for the patient so even children and friends are unaware. In other cases, it is the death of the healthy spouse that causes other family members to recognize the changes that have occurred in the living partner.

After the diagnosis of Alzheimer disease, most patients will survive for 4 to 6 years; however, this number can vary from 3 to 20 years [130].

The early stages are especially challenging for patients with Alzheimer disease, as they realize that they are slipping away and are unable to do anything about it; each stage brings with it additional mental, emotional, or physical losses. Inevitably, nearly all patients develop amnesia (memory impairment), aphasia (language impairment), agnosia (inability to identify common objects), apraxia (inability to use objects, despite knowing their function), and visuospatial deficit and may exhibit apathy, depression, or psychosis. Afflicted individuals will become dependent on caregivers for meeting even the most basic physical needs. The model of the progressive cognitive and functional decline in Alzheimer disease as “childhood development in reverse” (i.e., from the functional capacity of a child to that of an infant) is one that is easy for nonmedical family members and caregivers to understand [131].

The disease progresses continuously, and it is useful to remember that staging of Alzheimer disease is an artificial construct meant to assist in diagnosis and management. Presentation of the disease is widely varied in patients, with symptoms and deficits affecting every individual differently or not at all.

Therapeutic Measures

There are no treatments that can cure or reverse the effects of Alzheimer disease. However, Alzheimer disease is not a condition for which nothing can be done. Patients and families can be helped with interventions designed to diminish the manifestations of the disease. The disease and its progression are evaluated by the behaviors exhibited by the individual. Care planning is directed toward the management of the identified behaviors. Although there are many common features, each person is unique and requires distinctive approaches based on an assessment that identifies the specific problems of each individual.

In the preclinical stage, the goal of management for susceptible patients is to prevent and/or delay the onset of the disease. Maintaining a healthy diet and lifestyle, with goals including reduction of oxidative stress and blood pressure and improving circulation, may help in preventing dementia or slowing the rate of disease progression [132]. Dietary, exercise, and pharmacologic treatment guidelines for lowering the risk of obesity, diabetes, cardiovascular disease, and particularly hypertension should be followed, as comorbidities complicate Alzheimer disease treatment and exacerbate the disease process. As noted, there is some evidence that certain nutrients, especially omega-3 fatty acids, can reduce the risk of dementia [133]. Engagement in cognitive activities is also highly recommended.

Management of diagnosed Alzheimer disease consists of pharmacologic and nonpharmacologic therapies. Some pharmacologic agents have shown modest benefits in alleviating problems with cognition and behavior in research settings, though these benefits are often not realized in clinical use [133; 134]. These agents include several cholinesterase inhibitors (ChEIs) and memantine, an N-methyl-D-aspartate receptor antagonist [135; 136]. The most common adverse effects of ChEIs are nausea, vomiting, and diarrhea, with the most serious being cardiac arrhythmia and other cardiovascular and neurologic effects [133]. Memantine produces fewer adverse effects, and the dropout rate is similar to placebo. Other medications, such as antipsychotic agents and antidepressants, are occasionally necessary, but these agents can cause many unacceptable side effects [135]. In 2021, the FDA approved aducanumab, an anti-amyloid antibody intravenous infusion intended for patients with mild cognitive impairment and the presence of beta-amyloid plaques [136]. Preliminary studies have been conflicting, and the FDA's decision to grant accelerated approval to aducanumab was controversial, with many experts calling for additional research to prove efficacy and questioning whether the approval gives false hope to patients and families.

Medications for Alzheimer disease may provide temporary improvement in cognition for a subset of patients; however, at present there is no pharmacologic agent or other treatment modality capable of substantially altering the progression of disease. Thus, nonpharmacologic interventions, including social, environmental, and behavioral measures, are the most important elements of a management strategy for patients with Alzheimer disease [137].

Specific Nursing Measures

Patients and family members should be encouraged to make long-term plans after a diagnosis of Alzheimer disease. When the diagnosis is made early in the course of the disease, the patient can and should fully participate. Decisions can be given some thought if they are made before a crisis occurs. The patient and family must be aware of the need for advance planning as a mechanism for protecting the individual's self-determination.

Unmet needs can cause the patient with Alzheimer disease to become agitated and anxious. Patients may be unaware of the source of discomfort or be unable to respond to the cues resulting from unmet needs. A routine should be established that will avoid problems resulting from thirst, hunger, lack of sleep and rest, inadequate exercise, and irregular elimination patterns.

For the patient with Alzheimer disease, palliative care is centered on the alleviation of agitation and anxiety, the prevention of catastrophic reactions, and the management of delusions and hallucinations. Comfort may be extended in a number of ways. Members of the interdisciplinary team work together to develop interventions that will facilitate the individual patient's physical and emotional comfort.



The Hartford Institute for Geriatric Nursing asserts that healthcare professionals should maximize the functional capacity of patients with dementia by maintaining mobility and encouraging independence as long as possible; providing graded assistance as needed with activities of daily living; providing scheduled toileting and prompted voiding to reduce urinary incontinence; encouraging an exercise routine that expends energy and promotes fatigue at bedtime; and establishing bedtime routine and rituals.

(<https://www.guidelinecentral.com/summaries/recognition-and-management-of-dementia-in-evidence-based-geriatric-nursing-protocols-for-best-practice>. Last accessed November 18, 2021.)

Level of Evidence: Expert Opinion/Consensus Statement

PARKINSON DISEASE

Parkinson disease is a neurodegenerative disorder that affects approximately 1% of those older than 60 years of age [138; 139]. This disorder is prominently characterized by the motor symptoms of resting tremor, rigidity, and bradykinesia. The non-motor features are increasingly identified and include sensory, autonomic, and neuropsychiatric symptoms that appear before motor dysfunction is evident. The onset is insidious and often asymmetrical. Numerous, potentially disabling non-motor symptoms are often present, and diagnosis is made clinically.

Clinical Manifestations

Parkinson disease is typically diagnosed following the onset of motor features that prompts the patient to seek medical attention. Pre-motor prodromal disease can manifest in non-motor features, such as depression, fatigue, rapid eye movement sleep behavior disorder, anosmia, and constipation, that reflect disease involvement in autonomic, enteric, or somatomotor systems. Visuospatial and cognitive dysfunction, especially mild cognitive impairment with dominant executive dysfunction manifested in

diminished multitasking, planning, retrieval, concentration, and attention performance, are increasingly recognized as prevalent in earlier stages [140]. As mentioned, appearance, severity, and progression of pre-motor and motor features corresponds to the nervous system and brain areas afflicted by pathologic infiltration [141; 142; 143; 144].

Motor symptoms can also appear long before diagnosis. In Parkinson disease, the cardinal motor features of bradykinesia, resting tremor, rigidity, and postural/gait impairment reflect parkinsonism [145]. A mnemonic for the core motor features is TRAP [146]:

- Tremor at rest
- Rigidity
- Akinesia (i.e., bradykinesia and hypokinesia)
- Postural instability

It is important to note that postural instability, while a cardinal motor feature, is seldom present at diagnosis, as it usually appears later in the disease course [147].

Therapeutic Measures

Since curative or disease-modifying agents are not yet available, Parkinson disease is treated symptomatically. Treatment strategies for Parkinson disease are influenced by disease stage, most problematic symptoms, and patient age, and clinical decision-making should balance possible efficacy with potential side-effect risk for each treatment option.

Initial treatment of early Parkinson disease generally involves monotherapy, and motor control problems can be improved in many patients. Treatment of later Parkinson disease becomes more complicated, with disease progression and prolonged dopaminergic drug administration. Requirements for dopamine replacement therapy become increasingly demanding as motor signs worsen. Patients initially well controlled using dopamine agonists require initiation of levodopa and, over time, increasing amounts given in higher doses with more frequent intervals.

Patients initiated on levodopa will require the addition of dopamine agonists and/or other adjuncts that improve response to levodopa [148].

Surgical treatment is considered in patients with advanced Parkinson disease when optimized medical treatment has failed to control motor symptoms. Deep brain stimulation can reduce symptoms of motor fluctuations, dyskinesia, and tremor, but symptoms unresponsive to levodopa in advanced Parkinson disease (e.g., cognitive impairment, gait instability, mood disorders, speech impairment, autonomic dysfunction) are unlikely to improve and may worsen with deep brain stimulation. Guidelines recommend that deep brain stimulation should only be performed in experienced centers [139].

A variety of nonpharmacologic, adjunctive interventions have been evaluated for management of Parkinson disease. These include exercise programs and occupational, physical, and speech therapies. While clinical study design and control group issues have confounded the quality of evidence, clinical experience suggests that these approaches have value. The American Academy of Family Physicians recommends physical therapy, speech therapy, and occupational therapy be offered to patients with Parkinson disease as part of an overall strategy for improving or maintaining function [139].



The American Occupational Therapy Association recommends engagement of patients with Parkinson disease in exercise and physical activity, specifically multisession, repetitive physical exercise (diachronic) to improve motor and sensory-perceptual performance skills.

(<https://www.guidelinecentral.com/summaries/occupational-therapy-practice-guidelines-for-adults-with-neurodegenerative-diseases>. Last accessed November 18, 2021.)

Level of Evidence: A or B (There is strong or moderate evidence that occupational therapy practitioners should routinely provide the intervention to eligible clients.)

AMYOTROPHIC LATERAL SCLEROSIS

ALS is a progressive degenerative CNS disease with a relentless, fatal course. The three major aspects of the disease are progressive muscle atrophy, progressive bulbar palsy, and upper motor neuron deficit [23; 24; 57].

The course of ALS varies from one to four years. Men are more frequently affected than women at a ratio of about 2:1. Disease onset typically occurs between 40 and 70 years of age, most frequently in the fifth and sixth decades. The cause of ALS is unknown, but researchers are investigating viral, metabolic, infectious, toxic, immunologic, and specific life events as possible causes [23; 24; 57].

Clinical Manifestations

An EMG of patients with ALS demonstrates muscle wasting, atrophy, fasciculations, and fibrillation. Nerve biopsy is normal; muscle biopsy may demonstrate degenerative fibers interspersed with normal ones. Laboratory tests find normal CSF [23; 24; 57].

Initial symptoms of ALS include skeletal muscle weakness and atrophy that progresses from distal to proximal and unilateral to bilateral involvement in the upper extremities. Damage to CN IX and CN X leads to dysphagia and aphonia. Swallowing problems affect eating, drinking, and swallowing of saliva. Vagus nerve involvement can also cause dyspnea and bradycardia. Damage to CN XII causes difficulty with tongue movements during swallowing and speech [23; 24; 57].

Primary lateral sclerosis results in upper motor neuron signs, including spastic paresis of extremities, positive Babinski's reflex, and hyperactive deep tendon reflexes. Progressive muscle spasm may cause pain because of intact afferent nerve fibers. Most patients remain mentally clear. With total paralysis and lower cranial nerve involvement, a "locked-in" syndrome occurs. Patients are fully conscious of the environment and themselves, but experience a paralyzed body—all movement and ability to verbalize are absent. Some patients may only be able to communicate by eyelid movement and blinking.

The immediate cause of death is often respiratory muscle weakness and bulbar palsy causing respiratory failure [23; 24; 57].

Therapeutic Measures

There are no effective treatments for ALS, and it will progress regardless of therapy. Supportive symptomatic therapy is recommended to improve and, in some cases, extend life. Diazepam, baclofen (Lioresal), and dantrolene (Dantrium) may be used to control spasticity. Neostigmine (Prostigmin) is used temporarily to manage bulbar weakness. Analgesics are indicated to control pain. An oral glutamate inhibitor, riluzole, is approved to treat ALS and can increase pre-tracheostomy survival by two to three months [156].

Efforts should be made to prevent infections, especially of the respiratory tract. In many cases, tracheostomy and mechanical ventilation become mandatory to maintain respiratory function. Regardless of treatment, the prognosis is poor and survival is brief for most patients [23; 24; 57].

Specific Nursing Measures

A common problem for patients with ALS is ineffective communication patterns related to muscular weakness. As such, patients should be supported with alternative methods of communication. Alteration in nutritional status is possible due to swallowing and chewing impairments. This can be addressed with small, frequent feedings to prevent fatigue and choking; a soft diet high in calories, protein, and carbohydrates; and an adequate daily fluid intake. When feeding patients with ALS, the head of the bed should be elevated, with oropharyngeal suction equipment nearby and adequate time allowed for meals. Tube feedings may be necessary for some patients [39].

Alterations in mobility accompany increased motor paresis. To keep patients independent as long as possible, active and passive range-of-motion exercises and physical and occupational therapy are helpful. Assistive devices may be necessary for daily activities. Patients should remain out of bed and up in a chair as long as their condition permits [39].

Management of ineffective breathing patterns related to muscle weakness or aspiration often becomes a nursing priority. Aspiration may occur from dysphagia and excessive salivation. Preventive measures include monitoring the patient during eating, suctioning if necessary, assessing respiratory patterns around meal times, and administering anticholinergic drugs as ordered. Good oral hygiene should be provided [39].

When respiratory involvement progresses, the maintenance of a patent airway and adequate ventilation also become nursing priorities. Respiratory function should be assessed every one to two hours, including observation of rate, depth, and rhythm of respirations and auscultation of breath sounds. Frequent coughing and deep breathing is encouraged. Monitoring of tidal volume, vital capacity, and arterial blood gasses becomes crucial.

Chest physical therapy, including postural drainage, may be required in addition to nasotracheal and oropharyngeal suctioning every hour and as necessary. Intubation and ventilator equipment should be kept on standby. The most difficult nursing task often becomes helping the patient and family members to accept the seriousness of the condition [39].

SPINAL STENOSIS

Spinal stenosis can occur in older persons with a history of osteoarthritis of the spine or chronic disk degeneration, or it can be a congenital problem. The cervical and lumbar spine are most affected [70].

Cervical Stenosis

Patients with cervical stenosis experience spinal cord compression and gradually progress to spastic quadraparesis or paraparesis, often accompanied by fasciculations and atrophy in the upper extremities. Myelography often demonstrates a complete block of the spinal canal when the patient's head is in extension. When the total bony canal is stenotic, posterior decompression laminectomy is indicated [70].

Lumbar Stenosis

Unlike cervical stenosis, lumbar stenosis is manifested mainly by pain. There is no cord damage or spasticity, although there may be considerable numbness and weakness of the lower extremities. More often, the syndrome is manifested by spinal claudication. The patient has rapidly developing severe pain on walking any distance. Unlike vascular claudication, the patient with spinal claudication obtains relief by bending forward, which widens the anterior-posterior diameter of the spinal canal. Diagnosis of lumbar stenosis is confirmed by spinal x-ray, CT scan, and myelography [70].

Patients with lumbar stenosis usually do well following laminectomy. Despite considerable incisional pain, they tend to ambulate on the first post-operative day and are back to their normal activities by the end of one month [70].

IMMUNOLOGIC CNS DISORDERS

MYASTHENIA GRAVIS

Myasthenia gravis is a chronic neuromuscular disorder that affects voluntary (striated) muscles and is characterized by fluctuating muscle weakness that becomes worse with use and shows some improvements with rest. Respiratory infection and emotional stress may briefly exacerbate symptoms. The highest mortality rate is seen in the first year of the disease [23; 24].

Myasthenia gravis is relatively common, with incidence estimated at 1 in 10,000 [23; 24]. Peak incidence is between the second and third decades; onset is rare in the first decade of life or after 70 years of age. Before 40 years of age, women are affected approximately two to three times as often as men. In later life, the incidence is about equal [23; 24].

This disorder usually persists for life, although there may be periods of spontaneous improvement for weeks or months followed by worsening. This disease is not hereditary, but 15% of infants born to mothers with myasthenia gravis have a transient case of the disease. With treatment, infants recover fully in two to three months [23; 24].

Despite numerous theories and a great deal of research, the cause of myasthenia gravis remains unknown. There is general agreement that the defect occurs at the neuromuscular junction, and myasthenia gravis is now considered an autoimmune disease. In the healthy individual, there are approximately 38 million acetylcholine receptors at each neuromuscular junction. In the patient with myasthenia gravis, acetylcholine receptors are reduced by about 20% [23; 24].

The thymus gland is also considered to be involved in myasthenia gravis. Active from birth until puberty, the gland is thought to initiate the body's immune response and to cease functioning after puberty. In about 85% of patients with myasthenia gravis, however, the thymus gland is abnormal and remains active. An autoimmune reaction is probably triggered by the thymus gland [23; 24].

Clinical Manifestations

The most characteristic finding in myasthenia gravis is an increasing weakness of certain voluntary muscles with activity and some improvement with rest. The muscles of the eyes are often most affected. Unilateral or bilateral ptosis and diplopia are common [24].

Other muscles affected are those of facial expression, chewing, swallowing, and speech. When chewing, patients with myasthenia gravis often become fatigued and must rest. Patients may experience problems with management of saliva, choking, and nasal regurgitation. Speech defects include a weak voice that fades during conversation and diminishes to a whisper. Speech becomes nasal, monotonous, and dysarthric [24].

The shoulder and neck muscles are also often affected. The head tends to fall forward, and patients may have difficulty holding their arms above the head; reaching for an object and fixing the hair are difficult. The muscles for fine hand movement can be affected, resulting in difficulty writing, serving, and moving the hands to the mouth. The most life-threatening situation occurs when the intercostal and/or diaphragm muscle are affected. An early sign of respiratory involvement is breathlessness. Respiratory weakness can develop rapidly [24].

Patients with myasthenia gravis may develop a “cholinergic crisis” as a result of taking too high a dose of their cholinesterase inhibitor medication. Signs of cholinergic crisis are dilated pupils, nausea, and tachycardia. A myasthenic crisis is life-threatening due to the increasing weakness of the respiratory muscles caused by a sudden withdrawal of cholinergic medications. In myasthenia gravis, EMG shows that the amplitude of the evoked muscle’s action potential decreases rapidly. This reaction can be diminished or prevented with a single 2-mg IV dose of edrophonium [24; 31].

Therapeutic Measures

Drug therapy is the first line of treatment of myasthenia gravis. The drugs indicated—pyridostigmine bromide (Mestinon), neostigmine bromide (Prostigmin), and occasionally ambenonium chloride (Mytelase)—act by inhibiting anticholinesterase, preventing the rapid destruction of the neurotransmitter acetylcholine. Although this effect does not change the basic abnormality, it increases the

amount of acetylcholine available, partially compensating for symptoms of a defective neuromuscular transmission. Drug dosage should be individualized to provide the greatest symptom relief and the fewest side effects [24].

Corticosteroids may be prescribed for patients who do not respond well to anticholinesterase drugs. The recommended approach is prednisone 100 mg every other day for 10 days. Patients may be hospitalized for close observation during the initial treatment period on full-dose therapy. Symptoms intensify after 7 to 10 days of treatment but improve after treatment ends. This treatment is given with anticholinesterase therapy [24].

In 2017, the FDA approved the use of the monoclonal antibody eculizumab for the treatment of generalized, refractory myasthenia gravis in anti-acetylcholine receptor antibody-positive adults [157]. Muscle strength and quality of life seem to be improved in patients receiving initial treatments, and the positive outcomes are sustained for at least one year with continuous (twice-monthly) therapy [158].

The surgical removal of the thymus gland (thymectomy) is indicated for patients with hyperplastic thymuses. This procedure is most effective for young women in the first two years of diagnosis and least effective for older men. Even the removal of the thymus gland without an associated tumor produces a high rate of improvement or remission of symptoms in patients with early onset [24].

Specific Nursing Measures

Myasthenia gravis has largely become a treatable condition. Nursing priorities will depend on whether the patient is stable or in crisis. For patients in crisis, ineffective breathing patterns can develop and the patient experiences an abrupt exacerbation of motor weakness, usually from undermedication resulting from an unresponsive neuromuscular junction [39]. In these cases, the nurse’s responsibility is to restore and maintain adequate respiration [39].

Breathing patterns are assessed by taking serial vital capacity measurements. Testing can be done at the patient's bedside with a respirometer attached to a mouthpiece, face mask, or tracheostomy or endotracheal tube. The patient should be in an upright position to promote maximum chest expansion, and emergency respiratory equipment should be available [39].

Another important function to evaluate is the patient's ability to swallow. This may be done by gently placing a hand over the anterior neck and instructing the patient to attempt "dry" swallowing. This maneuver is preferable to giving the patient liquids that might induce choking or aspiration. If the swallowing reflex is intact, the gag reflex should also be evaluated [39].

For patients who develop respiratory failure, endotracheal intubation may be necessary, including a planned tracheostomy. A volume-cycled ventilator should be used during periods of acute respiratory insufficiency. During the acute phase, all anticholinesterase medication should be discontinued to allow a "rest period" for the neuromuscular junction. After the patient's condition stabilizes, small doses may be restarted.

During this period, the patient is generally fed by nasogastric tube. To prevent aspiration, check for proper tube placement before each feeding, verify the amount and type of stomach aspiration, and ensure the patient is in the upright position with an inflated tracheal or endotracheal cuff. Aspiration can lead to major complications for patients with bulbar paralysis [39].

Intensive chest physiotherapy should be carried out during crises to prevent respiratory complications. In addition, a bedside physical therapy program should be instituted to prevent muscle atrophy or contractures. Adequate hydration and anti-embolism support stockings should be used to prevent thrombophlebitis [39].

Patients with myasthenia gravis often experience long-term muscle weakness that worsens with activity and fatigue. Anticholinesterase drugs should be taken at the same time every day, usually 30 to 45 minutes before meals. Patients' strength and motor ability with drug administration and side effects should be monitored. Care planning should include rest periods to prevent excessive fatigue [39].

Incomplete eyelid closure can develop, with corneal abrasion or ulceration a potential problem. Routine eye care is necessary, with normal saline every four to six hours and as needed. Protective eye shields may be used [39].

Patients on long-term anticholinesterase drug therapy may have periods of nausea, diarrhea, abdominal cramping, and constipation. These symptoms may indicate anticholinesterase toxicity. Antiemetics and antidiarrheal agents may be necessary. For patients with constipation, enemas should be avoided; mild cathartics or suppositories may be used. Alterations in diet and fluid regimen may be helpful. Patient education should include information about actions of drugs and specific factors that might precipitate an increase in muscle weakness [39].

MULTIPLE SCLEROSIS

Multiple sclerosis is the most common immune-mediated (inflammatory) demyelinating disorder of the CNS. Most cases are diagnosed in persons between 15 and 45 years of age, and it is a common cause of permanent disability in this segment of the population.

The disease is triggered by events that permit auto-activated T-cells to breach the blood-brain barrier and cross-react with myelin components within the white matter of the brain and spinal cord. This precipitates a cascade of immune-mediated inflammatory tissue injury. As seen on radiographic imaging and pathologic examination, the hallmark of the disease is this well-defined, focal zone of injury ("plaque") containing elements of inflammation, demyelination, and axon degeneration [149; 150].

Such lesions may be single or multiple, and over time, they may be partially reparative, relapsing, or recurrent in new locations.

Clinical Manifestations

The early signs and symptoms of multiple sclerosis are typically mild and difficult to detect. They differ in duration and severity from one individual to another and at different times in the same individual. However, at first clinical presentation, most patients report multiple symptoms. Patients generally experience either acute attacks of neurologic compromise or are afflicted by a steadily progressive deterioration in functional capabilities, as will be discussed in detail later in this course [151].

Primary symptoms of multiple sclerosis are caused by the inflammation and demyelination that arises within focal areas of the CNS. The clinical presentation is varied but, in general, consists of some disturbance in vision, sensation, and/or motor function. The most common primary symptoms in patients with multiple sclerosis are:

- Fatigue
- Heat sensitivity
- Muscle spasms
- Dizziness
- Pain
- Paresthesias
- Ataxia
- Cognitive changes
- Visual complaints
- Bowel or bladder dysfunction
- Sexual dysfunction
- Gait problems
- Nausea/vomiting
- Speech problems
- Tremor
- Weakness

Secondary symptoms arise as a result of the presence of certain primary symptoms. For example, pressure ulcers may form as a complication of paralysis, a primary symptom. Bladder problems or urinary incontinence can cause frequent, recurring urinary tract infections. These symptoms are treatable, but ideally they should be avoided by treating the primary symptoms.

Tertiary symptoms may be described as the “trickle down” effects of multiple sclerosis and include the social, psychological, and vocational complications associated with the primary and secondary symptoms [152]. Depression is a frequent tertiary symptom present among people with multiple sclerosis. Social isolation, job loss, marital or interpersonal conflict, and anxiety may all develop as a result of various primary and secondary symptoms of multiple sclerosis.



EVIDENCE-BASED
PRACTICE
RECOMMENDATION

In the absence of a clear-cut typical clinically isolated syndrome, the International Panel on Diagnosis of Multiple Sclerosis asserts that caution should be exercised in making the diagnosis of MS, and the diagnosis

should be confirmed by further clinical and radiologic follow-up. In such cases, the clinician should consider postponing making a definitive diagnosis and initiation of long-term disease-modifying therapies, pending longer follow-up to accumulate additional evidence supporting the diagnosis.

([https://www.thelancet.com/journals/lanneur/article/PIIS1474-4422\(17\)30470-2/fulltext](https://www.thelancet.com/journals/lanneur/article/PIIS1474-4422(17)30470-2/fulltext). Last accessed November 18, 2021.)

Level of Evidence: Expert Opinion/Consensus Statement

Therapeutic Measures

There is no cure for multiple sclerosis. However, effective treatment strategies are available to modify the disease course, treat or reduce exacerbations, prevent relapses, manage signs and symptoms, improve overall function and safety, and provide psychological support. The treatment strategy depends on the patient's clinical condition and disease course. In cases of mild multiple sclerosis without relapses, usually no treatment is necessary. If a patient experiences relapses or if symptoms become more severe, treatment should be initiated as soon as possible.

Treatment of the acute exacerbations seen with relapsing types of multiple sclerosis relies primarily on corticosteroids and, in patients unable to tolerate steroids, adrenocorticotropic hormone (ACTH). These agents have been found to promote speedier resolution of the neurologic deficits, lessen the severity of an attack, and effectively reduce the risk of permanent residual deficits. Both corticosteroids and ACTH are capable of restoring the breakdown of the blood-brain barrier, reducing inflammation, and immunomodulating mononuclear trafficking mechanisms. Corticosteroids also promote quick recovery from disability [153; 154].

It is now known that B-cell immunity also plays a key role in the pathogenesis of multiple sclerosis. Plasma exchange may be beneficial for relapsing forms of multiple sclerosis in which severe neurologic exacerbations prove refractory to parenteral corticosteroid therapy. It may also be beneficial for some patients with severe, rapidly progressive multiple sclerosis and similar disorders.

The use of disease-modifying drugs has been shown to decrease the relapse rate, reduce progression of disability, and slow the accumulation of lesions for patients with relapsing-remitting multiple sclerosis [63; 71; 154; 155]. The exact mechanism of action of these drugs is still not clear, but it is believed to be the result of immunomodulation regulating the activation of impaired immune cells. Additionally, the blood-brain barrier becomes less permeable with

immunomodulation, allowing fewer immune cells to enter the brain and reducing the autoimmune reaction between the immune cells and neuronal tissue.

Specific Nursing Measures

The primary goal of symptomatic multiple sclerosis therapy is to improve quality of life by eliminating or reducing symptoms affecting patients' functional abilities. The approaches to symptomatic treatment focus on controlling the symptom rather than the underlying disease process. Neurorehabilitation together with occupational therapy is the best approach.

INFECTIOUS AND INFLAMMATORY CNS DISORDERS

There are four major routes by which pathogens gain access to the CNS. The most common route is via the bloodstream (hematogenous) from septicemia or a septic embolus from endocarditis, lung infection, or pelvic abscess. Pathogens may also enter after trauma, including skull fracture, penetrating wounds, and operative procedures. Non-traumatic infections can occur from otitis media, mastoiditis, sinusitis, and osteomyelitis. Finally, pathogens may enter in a retrograde manner via nerve trunks (e.g., rabies) or through the cerebrospinal route from lumbar or ventricular puncture [13].

MENINGITIS

Meningitis is an inflammation of the meninges caused by a viral, bacterial, or fungal organism. There are three major types of meningitis: aseptic, septic, and tuberculous. Aseptic meningitis is thought to occur from viral inflammation or meningeal irritation. Septic meningitis is caused by infection from pus-forming bacteria (e.g., *Neisseria meningitidis*). Tuberculosis meningitis occurs when the chance location, progression, and rupture of a tubercle (developed following active infection with *Mycobacterium tuberculosis*) enters the subarachnoid space [23; 24].

After a pathogen enters the subarachnoid space, the infection spreads because of the open communication over the brain's convexity along the blood vessels of the pia and then penetrates the sulci. It is not uncommon for affected blood vessels to become engorged, leading to thrombosis or rupture. The accumulation of exudate over the convexities, in the cisterns or the ventricles, can cause obstruction of CSF flow. The exudate may ascend to involve the spinal cord. If the brain surface adjacent to the meninges becomes involved, secondary encephalitis and neuron degeneration can occur [23; 24].

Clinical Manifestations

The clinical course of meningitis can be acute, subacute, chronic, or recurrent. Headache, fever, meningeal irritation, and mental status changes are the most common symptoms. Patients often complain of severe headaches, the worst they have ever experienced. Meningeal signs include nuchal rigidity; photophobia; pain down the back and limbs; alterations in mental status and level of consciousness; restlessness; confusion; hallucinations; and delirium. There may be generalized seizures and increased ICP due to cerebral edema and communicating hydrocephalus. Opisthotonos, a sign of meningeal irritation, is manifested by arching of the neck and back. There also may be medullary signs, such as vomiting, respiratory difficulties, and a weak, rapid pulse. Cranial nerve involvement causes visual disturbances, ptosis, pupil abnormalities, strabismus, deafness, nystagmus, and vertigo. In cases of meningococcal meningitis, a skin rash may be present [23; 24].

Diagnosis is based on history of prior infection or exposure, symptoms and signs of an existing infection, clinical neurologic signs, and diagnostic tests. Skull x-rays are ordered to evaluate for possible fracture and/or infected sinuses or mastoids. Chest x-rays are ordered to assess for pneumonia and lung abscesses. CSF pressure is often increased to 700 mm/H₂O. The appearance of the CSF, as evalu-

ated by lumbar puncture, varies according to the infecting organism. In bacterial infection, the CSF is turbid to purulent. In tubercular infection, the CSF is clear, xanthochromic, or like ground glass. In viral infection, the CSF is usually clear. Glucose levels are low in bacterial and tubercular infections, but normal in viral infections [23; 24].

Therapeutic Measures

In bacterial meningitis, culture confirms the type of bacteria and results of sensitivity tests indicate appropriate drug therapy [24]. Prognosis is good with antibiotic therapy.

In the acute phase of meningococcal meningitis, it is possible to infect others by exposure to nasopharyngeal and droplet secretions from the respiratory tract. Isolation procedures to protect others should be maintained until cultures are negative [24].

Specific Nursing Measures

The major burden of care of patients with meningitis occurs in the acute phase of infection. During this period, level of consciousness and neurologic signs should be frequently assessed. As noted, appropriate infection control precautions are important. Patients with meningococcal meningitis or meningitis of unknown etiology are kept in isolation [33].

Patients will have a febrile period during which temperature is monitored every one to two hours if higher than 101°F (38°C). Tepid sponge baths, frequent skin care, axillary and groin compresses, and antipyretic drugs may be ordered. If shivering develops, chlorpromazine (Thorazine) may be administered [33].

Fluid volume deficits can occur due to fever and inadequate intake. As such, serum electrolytes should be monitored daily during febrile periods. Oral fluids, intravenous fluids, and/or tube feedings are given as needed. Intermittent or in-dwelling bladder catheterization may be necessary for patients with impaired levels of consciousness [33].

Sensory perceptual deficits can occur because of photophobia, hyperalgesia, and hyperirritability. A quiet, dark, non-stimulating environment and limited visiting hours can help reduce photophobia. The potential impact of hypoxia or bladder distention on irritability should be ruled out before considering it a neurologic problem. For headache management, keep the head of the bed elevated, unless contraindicated, with good body alignment and repositioning every two hours [33; 64]. Pneumococcal and influenza immunizations are the best preventive measures [33].

ENCEPHALITIS

Encephalitis is an infection of brain tissue caused by a virus (most common), pyogenic bacteria, fungi, or parasite. Epidemic encephalitis begins in a reservoir and is transmitted to humans. For example, equine encephalitis begins in squirrels, horses, wild birds, chickens, frogs, or garter snakes; a mosquito or tick bites the reservoir animal and transmits the virus when it bites a human host. Incubation periods vary according to the host's susceptibility to, reaction to, and strength of the pathogen [23; 24].

Encephalitis begins with the pathogen gaining access to the CNS. This is followed by degeneration and destruction of cortical neurons with demyelination. Patches of hemorrhage, necrosis, and cavitation can occur, depending on the type of pathogen involved. Diffuse cerebral edema results [23; 24].

Viruses are the most common pathogens. Herpes simplex virus has the potential to cause acute encephalitis and acute neonatal encephalitis from exposure during vaginal delivery or in the first days of life. Herpes simplex virus type-1 is the most common cause of herpes encephalitis [159]. Other latent viruses that can cause encephalitis include herpes zoster virus, cytomegalovirus, Epstein-Barr virus, mumps, rabies, and measles [23; 24].

Clinical Manifestations

A prodromal illness often precedes the neurologic signs associated with encephalitis. Usual initial symptoms are headache, fever, malaise, sore throat, and myalgia. This is often followed by marked alteration in level of consciousness ranging from lethargy to coma. EEG abnormalities and CSF lymphocytic pleiocytosis occur in more than 80% of patients with herpes encephalitis [159]. Confusion and disorientation with abrupt behavioral disturbances may occur. Objective signs include motor and sensory deficits, tremor, and ataxia. Hyperirritability, meningeal signs, and seizures are possible [23; 24].

Therapeutic Measures

Clinical management of encephalitis is mainly symptomatic and supportive. There is no curative drug therapy, but steroids such as dexamethasone (Decadron) may be used to combat cerebral edema. Herpes encephalitis is treated with the antiviral agents acyclovir and valacyclovir; however, antivirals are ineffective for other viral causes [159; 160]. Not all patients recover completely. Those surviving an acute episode can have residual neurologic deficits, including seizures, dysphasia, memory loss, and/or personality changes [24].

Specific Nursing Measures

Nursing priorities for patients with encephalitis are similar to those for patients with meningitis, with several major differences. Patients with acute encephalitis have more marked alterations in level of consciousness and behavioral manifestations; restlessness, agitation, and dementia are more severe [33].

Neurologic deficits may increase rapidly due to cerebral edema and necrosis. Neurologic signs are monitored frequently during the acute stage, with any changes reported promptly. There is potential for fluid volume overload related to intravenous antiviral administration. To combat this, drugs are administered on a strict schedule and intake and output are maintained. Patients should be observed

for other possible drug side effects, including nausea, vomiting, diarrhea, weight loss, and transient alterations in blood cell and liver function tests. Other nursing measures include elevating the head of the bed and monitoring electrolyte levels and respiratory and cardiac status [33].

MYELITIS

Myelitis is an inflammation of the spinal cord. The most common viral diseases causing myelitis are poliomyelitis and herpes zoster [70].

Clinical Manifestations

The viruses that cause myelitis usually have an affinity for motor and sensory neurons rather than spinal tracts. Clinical findings in patients experiencing spinal cord involvement include paresis, numbness of the feet and legs (more than upper extremities), dysuria, and sometimes headache and stiff neck. Neurologic involvement can extend to the brain stem, cerebellum, cerebrum, and optic nerves in some patients [70].

Therapeutic and Nursing Measures

Treatment is supportive. Nursing care is aimed at preventing hazards of immobility and alterations in comfort. Bed rest is usually indicated during the acute phase of the illness. It is important that rehabilitation measures be instituted early [33].

INTRACRANIAL ABSCESS

An abscess may form around or within the brain as a result of a local or systemic foci of infection. Intracranial abscesses are purulent, usually encapsulated collections. Although abscesses can form anywhere in the brain, the most common sites are the temporal lobes, the frontal lobe, and the cerebellum [13].

Most brain abscesses develop secondary to a primary source of infection. Of these, at least 40% are caused by mastoiditis, otitis media, or sinusitis. A smaller percentage result from direct invasion by traumatic injury, such as gunshot wound, basilar skull fractures, or compound skull fractures with dural tears [13].

Clinical Manifestations

During the initial stage of organism invasion of the brain, the patient experiences chills, fever, malaise, and appetite loss. The almost common presenting symptom of an intracranial abscess is headache, which may be associated with vomiting and papilledema. Other common presenting symptoms are alterations in level of consciousness (especially drowsiness and confusion) and partial or generalized seizures. Focal neurologic deficits vary according to the anatomic location of the abscess. These include various motor, sensory, and speech disturbances [13].

In contrast, a subdural abscess tends to produce even more profound symptoms than brain abscesses. A subdural abscess affects the cortical blood vessels, causing thrombosis, arteritis, and eventually ischemia. The abscess usually arises from sinusitis. Headache occurs, with rapid deterioration in neurologic status including seizures, hemiplegia, and dysphasia. Without treatment, brain compression or abscess rupture into the ventricle or subarachnoid space can be fatal [13].

Diagnosis is usually made by history of a previous infection, neurologic exam, and CT scan. A lumbar puncture is not recommended, because a brain abscess acts as a mass lesion and the negative pressure created by the procedure can lead to a brain shift and herniation. The most important diagnostic test is CT scan, which can demonstrate displacement of the lateral ventricles and locate the abscess, which is observed as an area of decreased density [13].

Therapeutic Measures

Interventions are aimed at diagnosing and managing the primary infection source, providing for abscess drainage, and administering an effective antibiotic regimen. Most intracranial abscesses can be drained via bur hole aspiration. Some may require more than one aspiration. If the abscess has ruptured into the ventricles, ventricular drainage is used. Antibiotic therapy is given for at least six weeks to reduce virulence of the organism, eliminate the pathogen, and

penetrate the cavity. Prognosis is usually good after an effective regimen of antibiotics and/or surgery [13].

Specific Nursing Measures

Management of the acute infection is a priority of nursing care for patients with intracranial abscess. Intravenous drug administration sites should be inspected and rotated every 48 to 72 hours to prevent thrombosis phlebitis. During antibiotic therapy, patients are at risk and should be assessed for the development of opportunistic infections, especially in the mouth and gastrointestinal tract [39].

Sudden increases in ICP can result from cerebral edema that may surround an acute abscess. Frequent assessment of neurologic and vital signs, head of bed elevation to 30°, restricted fluid intake, and avoiding any stimulants that can raise ICP are necessary components of care [39].

Seizures are also a concern. Anticonvulsants may be ordered, with periodic checking of serum blood levels. During actual seizures, the patient should be protected from self-injury and helped to maintain a patent airway. Interventions for headache include providing a quiet environment, changing position to promote comfort, and administering mild analgesics, as ordered. Patient education should focus on preparing patients psychologically and physically for surgery (to aspirate the abscess and to provide intrathecal medication). Discharge teaching should include methods of preventing future abscesses if caused by an infected tooth, otitis media, or sinus problem [39].

HERPES ZOSTER

Herpes zoster (also known as shingles) is a viral disorder that affects the posterior root ganglia. It is characterized by cutaneous eruptions of vesicles along the distribution of involved spinal or cranial nerve roots. The highest percentage of cases involve spinal ganglia. Outbreaks occur mainly in adults and are more common in women than men and during the spring and fall [8; 32; 34].

Herpes zoster develops from reactivation of varicella virus, which is responsible for chickenpox. Children without immunization can develop chickenpox if exposed to an adult with shingles [8; 32; 34].

Clinical Manifestations

Mild-to-severe neuralgic pain in the affected nerve root distribution is the most common presenting symptom of shingles. The pain may be burning, tingling, sharp, or dull. Pain may be concurrent with or followed by skin reddening and an eruption of vesicles. Over the next one to two weeks, these lesions become pustules and then develop a crust. After healing, a pigmented scar may appear. If an infection or ulceration accompanies the vesicles, the scarring may be permanent [8; 32; 34].

Diagnosis is usually based on the sudden onset of root pain followed by the characteristic distribution of shingles. The lesions are unilateral and do not cross the midline of the body [8; 32; 34].

There are potential complications from a herpes zoster outbreak, most commonly scarring of the skin, facial palsies, and postherpetic neuralgia. In elderly or immunocompromised patients, the neuralgia can persist for months or years. The skin may also be hypersensitive to touch. Unfortunately, this variant does not respond well to treatment. Less commonly, some individuals develop Guillain-Barré syndrome following a herpes zoster outbreak [8; 32; 34].

Therapeutic Measures

Most treatment of herpes zoster outbreaks is aimed at giving local care to the vesicles. Topical corticosteroids can alleviate local pain and itching and may shorten the stage of vesicle eruption. Antibiotics may be administered to prevent or treat secondary infections. During the acute phase, bed rest and analgesics can be supportive [8; 14].

Antiviral medications (e.g., acyclovir) may be used to limit duration of outbreaks and help prevent postherpetic neuralgia [21]. Early treatment (within 72 hours of symptom onset) is most effective. Antiviral therapy should continue for 7 to 21 days.

Postherpetic neuralgia is a difficult condition to treat. Capsaicin preparations may be prescribed but take several days to weeks to provide significant relief [161]. Intractable cases may require neurosurgical sectioning of affected nerve roots or occasionally irradiation to the site. Treatment results are variable [8; 14].

Specific Nursing Measures

The primary objective of nursing management of patients with shingles is symptom management (to minimize discomfort) and care of interruptions in skin integrity. Patients should be instructed to avoid scratching vesicles to prevent spreading the virus and promoting infection. Open vesicles should be dressed with moist occlusive dressings. If systemic steroid therapy is used, it is important to monitor for side effects. Comfort measures to address localized pain and itching include positioning techniques (especially during bed rest), skin treatment, and analgesics as ordered [33].

NEOPLASTIC AND OBSTRUCTIVE CNS DISORDERS

Tumors within the cranium can be either primary or metastatic. Metastatic tumors are found predominantly within the substance of the brain, which they reach through the systemic circulation.

Tumors produce symptoms by invasion or compression of surrounding neural structures. The neurologic symptoms and signs of any tumor affecting the brain depend on the location of the tumor and its rate of growth. Disruption of neural structures can cause an insidious deterioration of neurologic function or an acute neurologic disturbance. Spinal cord tumors can cause spinal cord compression and related pain and other symptoms [27].

TRAUMATIC DISORDERS

Craniocerebral trauma is a major cause of death in persons between 1 and 44 years of age and contributes to more deaths than stroke in persons 45 to 64 years of age [60; 62]. Nearly 6.5 million head injuries occur yearly in the United States. The highest incidence occurs in young, previously healthy men. Serious injury leads to permanent disability and emotional devastation for the patient, and the cost of long-term rehabilitation and maintenance programs have a tremendous impact on the health-care system [60; 62].

HEAD TRAUMA

Serious cerebral injury can result from a number of factors, including motor vehicle, sports, and industrial accidents as well as assaults and falls. The skull is a rigid sphere filled to capacity with contents that are basically noncompressible. These components include CSF, the vascular system, and brain tissue. All maintain a fairly constant volume; an increase in the volume of one intracranial component occurs at the expense of the others or results in an increase in ICP. Early signs of increasing ICP include restlessness, irritability, and a gradual decrease in the level of consciousness. Lumbar punctures are contraindicated during periods of increasing ICP because herniation of brain structures into the foramen magnum may cause depression of vital centers in the brain [60; 62].

Among the methods for classifying head injuries are mechanism and severity of injury. Mechanisms include direct (acceleration or deceleration) and indirect causes. A direct acceleration injury occurs when the stationary head is struck by a moving object, such as a baseball. A direct deceleration injury occurs when the head in motion strikes an immovable object, such as when a person falls from a bicycle onto the pavement. In an indirect injury, the traumatic force is not directly applied to the head but is usually the result of shaking or slamming the body to the extent that the brain hits the skull [60; 62].

Another major classification is closed or open head injury, referring to whether the skull and the dura mater are intact. A closed head injury is a non-penetrating, blunt injury with no break in the integrity of the skull and dura mater. Brain concussions, contusions, and lacerations may occur with either type of injury. Brain stem compression from increasing ICP occurs late and can cause herniation and death [60; 62]

An important phenomenon in closed head injury is coup-contrecoup. A coup injury is bruising of the brain directly below the point of injury resulting from impact to the skull; there are visible signs of injury. Contrecoup refers to the rebound effect of injury—the mass movement of the brain opposite to the site of impact. For example, a blow to the frontal region (coup) causes damage to the occipital region (countercoup) of the brain [60; 62].

In open head trauma, a penetrating injury breaks the integrity of the skull or dura. Cerebral contusions or lacerations occur. The most frequent fractures are linear, at the base of the skull. Depressed and comminuted fractures are less frequent, but more serious, because of dural tears and lacerations of brain tissue. Infection is a high risk in open head injuries. Cerebral edema develops with any head injury and should be treated as early as possible [60; 62].

Clinical Manifestations

The location of a skull fracture is most crucial for determining damage to the underlying structure: the meninges, blood vessels, and the brain itself. Fractures are classified as linear, diastatic, depressed, compound, growing, or basilar [60; 62].

Linear skull fractures are usually simple breaks in bone continuity anywhere in the skull. These fractures are typically less complicated. Depressed skull fractures are caused by trauma from sharper,

penetrating injuries that commonly result in brain lacerations and infection. A compound skull fracture is a scalp laceration along with a depressed skull fracture. Debris (e.g., hair, dirt, foreign material) penetrates into the wound. The dura may or may not be torn. A basilar skull fracture involves injury to the base of the skull. Basilar skull fractures can indicate severe trauma and should be suspected in any significant head injury [60; 62]. Diastatic and growing fractures occur more often in children whose cranial sutures are not yet fused.

Traumatic brain injury is classified as a concussion, contusion, or laceration that may or may not be associated with vascular rupture and cardinal nerve damage. A concussion is considered the most benign form of brain injury. It is characterized by a loss of consciousness for five minutes or less and memory loss of events preceding and following trauma. Other symptoms may include dizziness, spots before the eyes, and a dazed state [60; 62].

A cerebral contusion is a bruising of the brain. There may be petechial hemorrhage of cortical tissue and white matter, with tearing of the pia mater. Contusions cause unconsciousness that persists for longer than five minutes. An initial period of shock is followed by signs of cerebral irritability [60; 62].

A cerebral laceration is a tearing of the brain tissue followed by intracerebral bleeding. Prolonged unconsciousness, immediate neurologic deficits, and a deterioration in condition can be expected [60; 62].

The major vascular hemorrhages from trauma include epidural, subdural, and intracerebral hematomas. Most epidural hematomas are from arterial bleeds. Classically, the trauma causes an initial loss of consciousness, followed by a lucid interval. Rapid and often unexpected unconsciousness follows. Epidural hematomas require prompt surgical intervention [60; 62].

Subdural hematomas are collections of blood from clots in the subdural space between the arachnoid and dura that usually involve venous bleeding. Acute hematoma occurs within 48 hours; subacute occurs within two weeks; and chronic types have an onset more than two weeks after injury. Subdural hematomas can develop over an entire hemisphere. Chronic subdural hematomas can increase in size over time, probably due to rebleeding [60; 62].

Acute subdural hematomas are often associated with massive cerebral or brain-stem injury. Although bleeding is mostly venous, it develops quickly, with a rapid onset of symptoms. Common clinical symptoms and signs include worsening headache, drowsiness, confusion, slow responses, and restlessness. A critical sign of deterioration is an ipsilateral dilation of the pupil that becomes unreactive [60; 62].

An intracerebral hematoma, a collection of blood in brain tissue, is a complication in a small percentage of all head traumas. The hematoma may accompany the contrecoup phenomenon [60; 62].

Therapeutic Measures

The treatment of head trauma often involves both medical and surgical approaches. The initial management of patients with head trauma includes diagnosis of injury and assessment for potential respiratory problems or injury to other systems. The primary focus is on maintenance of a patent airway and appropriate blood pressure, monitoring of ICP, and appropriate antibiotic therapy, as necessary. Hyperventilation therapy is employed in patients with severe head trauma to decrease PCO_2 , reducing cerebral vasodilation and ICP [60; 62]. Treatment of cerebral edema includes use of dehydrating agents, such as mannitol, that withdraw water from intracranial tissue. The presence of mental clarity and appropriate behavior indicates a positive response to therapy [60; 62].

Specific Nursing Measures

As noted, the initial nursing priority for the care of the patient with acute head injury is maintenance of effective airway clearance and breathing pattern. Compromised respiration leads to increased ICP, which causes ischemia. Patients with alterations in level of consciousness are at greatest risk for hypoventilation, and these patients should be monitored for airway patency and adequate pulmonary toilet. When transferring patients from one area to another, a bag-valve mask should be available if needed [39].

Patients who have sustained a traumatic head injury should be immobilized and not manipulated until cervical injury is ruled out by x-ray or CT scan. Neck hyperextension, flexion, and rotation should be avoided; manipulation can cause airway obstruction or can seriously complicate a cervical injury. If respiratory resuscitation is required, the jaw-thrust maneuver can be used. The mouth and oropharynx should be cleared of foreign bodies, and gentle oropharyngeal suctioning can be done to maintain an effective airway. Nasal suctioning is contraindicated. If the airway is not patent, endotracheal intubation or a tracheostomy will be indicated [39].

Patients with potential for respiratory complications may be repositioned after the cervical spine is stabilized and/or injury is ruled out. The semiprone lateral position facilitates drainage of secretions. Position should be changed at least every two hours. Chest excursions, breath sounds, and respiratory rate, rhythm, and pattern are assessed every one to two hours (or as necessary). Arterial blood gases are monitored initially, after four hours, and subsequently as necessary. Neurologic dysfunction can cause specific changes in respiratory patterns, such as Cheyne-Stokes respirations, and ataxic breathing patterns can indicate an impending respiratory arrest [39].

The neurologic status of the patient with acute head trauma should be assessed immediately after respiratory airway patency is established. Level of consciousness is the single most important aspect of the clinical nursing observation. Consciousness is assessed on a continuum from full reaction to no reaction to various kinds of stimuli. The bilateral corneal reflex should also be assessed on schedule [39].

Care of patients with basal skull fractures includes assessment for bilateral periorbital ecchymosis hemorrhages, Battle sign (an ecchymosis of the mastoid region), and hemotympanum (blood behind the eardrum). CSF leaks may present as rhinorrhea or otorrhea. The nose or ear should not be probed or irrigated if a CSF leak is suspected. If drainage occurs, collect the fluid to test for the presence of glucose. A glucose-positive result can confirm a CSF leak, as glucose is present in CSF but not in mucus. If drainage cannot be collected for testing, carefully inspect the patient's gown and linen. The "halo" sign, a combination of bloody or darker drainage encircled by a lighter yellowish stain, signifies a bloody leakage of CSF from the nose or ears. Basal skull fractures are considered serious head injurers due to the proximity of the fracture site to vital brain stem areas, damage to which can result in severe respiratory and cardiac dysfunction. Patients should be assessed frequently for alterations in urologic, respiratory, or cardiac status [39].

SPINAL CORD INJURY

The vast majority of spinal pathology results from traumatic injury. The highest incidence is in young men in the second and third decades of life, and the leading causes are motor vehicle accidents, sports accidents (e.g., football, diving), and penetrating injuries (e.g., gunshot or stab wounds) [70].

Fracture-dislocations can occur anywhere along the spine. Cervical injuries, the most common, are usually related to flexion-extension maneuvers during traumatic injury. Fracture-dislocations in the thoracic and lumbar areas usually arise from compression injuries, as in falls from high places. Spinal column fracture-dislocations may also result from pathologic bone processes [70].

Dislocation fractures of the high cervical vertebrae (C-1 to C-2) can occur as a result of fractures or congenital defects from arthritic changes that weaken the ligaments in this area. A forward movement of the skull and C-1 and C-2 vertebrae can compress the cervical cord [70].

Clinical Manifestations

The extent of a patient's functional loss following injury depends on the degree of spinal cord injury. Complete spinal cord transection causes a total and irreversible loss of motor and sensory function below the level of injury [70].

In the cervical areas, millimeters can be crucial for spinal nerve-root function. With cervical cord transection, quadriplegia (paralysis of all four extremities) results, with varying degrees of respiratory and arm paralysis, depending on the injury level. Cord transection of the thoracic spine through L-1 and L-2 causes paraplegias (paralysis of both legs). In a complete cord transection, the individual sustains an immediate flaccid paralysis, loss of sensation, and usually loss of reflexes below the level of injury. Some reflexes may be intact initially and then disappear within a few days. The loss can last for days, weeks, or several months. As paralysis subsides, reflexes usually return and flaccidity changes to involuntary spastic movement. Recovery of any motor or sensory function is rare when there is complete paralysis of these functions for several days after injury [70].

With incomplete spinal-cord injuries, degrees of sensory and motor defect below the level of damage vary, resulting in one of several spinal cord syndromes [70]. A number of factors are examined to assess the degree of spinal cord damage. The first consideration may be the mechanism of injury. A compression of the cord can occur from ligaments, bone, and herniated disk material or hematomas. A contusion causes a bruising of the cord. In transection injuries, the spinal cord is actually or physiologically severed. Hematomyelia can occur within the cord substance [70].

Another important factor influencing the degree of spinal injury is the diameter of the spinal canal, or the amount of space the spinal cord has to move while avoiding compression. This factor is especially crucial in the cervical area. Spinal cord necrosis can result from a number of factors, including disturbances in circulation causing poor cord perfusion, edema, and progressive hemorrhage of central gray matter [70]. Neurons do not regenerate within the cord substance.

Therapeutic Measures

In cases of acute spinal cord injury, the patient's spine should be handled with extreme caution. Patients with spinal cord trauma and neurologic deficits need special attention during transport to prevent further deterioration in neurologic status. Spinal precautions should include the use of a backboard and hard cervical collar. Pressure sore development is a common complication during long transports and care should be taken to pad all bony prominences. Administration of methylprednisolone or similar medication for spinal cord injury is a requisite and should be initiated and subsequently continued throughout transport. Any life-threatening injury to another body system or signs of systemic shock should be treated immediately. For emergency life support, the neck should not be hyperextended. In most trauma units, early fixation and spinal traction have replaced decompression laminectomies [70].



The American Association of Neurological Surgeons recommends expeditious and careful transport of patients with acute cervical spine or spinal cord injuries from the site of injury by the most appropriate mode of transportation available to the nearest capable definitive care medical facility. Whenever possible, the transport of these patients to specialized acute spinal cord injury treatment centers is recommended.

(https://academic.oup.com/neurosurgery/article/72/suppl_3/35/2557442. Last accessed November 18, 2021.)

Level of Evidence: III (Strategy for patient management for which the clinical utility is uncertain.)

Another goal of therapy is to reverse the effects of vertebral body fractures. Neurosurgery replaces the injured vertebra with bone, acrylic, or a combination. The advantage of acrylic is early mobilization for the patient, whereas bone fusion requires some internal fixation. For patients who remain unstable, a halo frame and body jacket vest are applied for several months, until stabilization is achieved. In the lower thoracic and lumbar areas, internal fixation can be achieved using rods [70].

Patients with spinal cord injury are at greatest risk in the first 7 to 10 days after trauma. During this time, shock, pulmonary dysfunction, infection, and paralytic ileus can be major problems. Patients who sustain quadriplegic injuries require intensive total medical and nursing management. The posttraumatic care of these patients mainly involves management of bladder and bowel dysfunction, skin care, nutrition maintenance, and physical therapy [70].

Specific Nursing Measures

Continuous monitoring of the patient's neurologic status, including respiratory, motor, and sensory functions, is essential. Level of consciousness and pupillary response should also be assessed. Progression of any neurologic deficit should be reported immediately [39].

With high cervical lesions, there will be ineffective airway clearance and alterations in breathing patterns. Many patients require endotracheal or tracheostomy tubes with ventilator support. Oxygen administration will be ordered by tracheostomy tube, tracheostomy collar, or ventilator [39].

High cervical injuries frequently cause respiratory arrest. During this emergency, the jaw-thrust maneuver is used for resuscitation. The patient then requires careful nasotracheal intubation or an emergency tracheostomy. For patients admitted with respiratory function intact, respiration should be carefully observed. During spinal shock, it is not uncommon for the level of injury to ascend one or two levels above actual damage due to massive spinal cord edema. When this occurs, patients may develop respiratory dysfunction and/or arrest, even if they did not have the problem initially. These patients are at high risk for pulmonary complications such as pneumonia and atelectasis (hypoventilation). Other possible complications include decreased cardiac output, decreased venous pressure, and severe hypotension [39].

To alleviate early cerebral or spinal-cord edema, large doses of intravenous or intramuscular glucocorticoids are often given. This may cause gastric distress or other side effects, such as behavioral changes, elevated glucose levels, and an acne-like rash [39].

Patients who have sustained a spinal injury are also at risk for abdominal distention and paralytic ileus. Nothing should be given by mouth until bowel sounds return. If necessary and not contraindicated, a rectal tube may be used to relieve abdominal distention. For patients with ulcer histories, cimetidine (Tagamet) may be ordered with steroids to prevent gastrointestinal bleeding [39].

The plan of care should include measures to prevent hazards of immobility, emboli, skin breakdown, and bowel and bladder dysfunction. It may be helpful to begin a bowel management program by administering a cleansing enema. To establish a bowel reflex, a combination of bulk in the diet, stool softeners, prune juice, and daily suppositories is provided. When the patient resumes eating, an oropharyngeal suction machine may be kept at the bedside for safety [39].

Patients should be assessed for symptoms and signs of thrombophlebitis and pulmonary embolism; in many cases, patients are unable to give subjective reports of calf pain [39]. One of the most important steps to prevent pressure ulcers is physically repositioning the patient frequently. Repositioning should be done every one to two hours, depending on the patient's condition. Every time the patient is repositioned, look for areas of redness and make sure that the new position does not put weight on these areas.

Perhaps the most sensitive area of nursing care is the patient's psychological reaction to paralysis. Body image and the patient's future plans have been suddenly and irrevocably altered. It is vital to assess available support systems and prevent sleep deprivation and sensory overload [39]. The range of emotion the patient experiences should be recognized and validated, with the goal of supporting the patient in progressing toward eventual acceptance and resolution. Patients should be encouraged to make as many decisions about their care as possible to help regain a sense of control over self and environment [39].

The acceptance of permanent disability may take a long time and should not be forced; denial can be beneficial for a time. Nurses can be empathetic listeners who can accurately reflect the patient's comments and feelings [39].

After vertebral fractures fuse, patients can be transferred to stretcher chairs. Hypotension and dizziness may occur during this process, so mobilization should be done progressively. Abdominal binders and anti-embolism stockings can help decrease hypotensive episodes and periods of dizziness [39].

Sexual dysfunction is a major concern that patients may or may not verbalize. Factors that determine future potential for sexual function include the anatomic level of injury, the patient's personality and former sexual experience, and the effectiveness of sexual counseling. The most important fact to convey is that a caring, loving relationship is possible after injury [39].

Autonomic dysreflexia is possible with patients who sustain high thoracic lesions (above the T-6 level). This condition results from abnormal autonomic responses (usually pain or discomfort below the level of injury). Bladder or bowel distention or skin stimulation can trigger the response. Autonomic dysreflexia is an emergency that may lead to increased ICP and severe hypertension. When symptoms occur, the patient's head should be elevated to lower blood pressure, the patency of the indwelling catheter should be assessed, and patients should be evaluated for a possible fecal impaction. Antihypertensive drugs may be necessary. The nurse can help prevent episodes of dysreflexia by preventing conditions that result in stimulus overload, fecal impaction, or bladder distention [39].

CONCLUSION

With knowledge of CNS structure and function and the dynamic pathology that intrudes and impedes normal function, nurses are more prepared to provide quality and often lifesaving care to patients. An awareness of symptoms' precipitating events leads to quicker reporting of changes in the patient's condition, and immediate interventions can be performed based on standing orders and the patient's needs. This changes what could be only technical care to professional care through the use of informed decision-making skills.

CASE STUDIES

CEREBROVASCULAR ACCIDENT

Patient A, 85 years of age, wakes in the morning to paralysis on his whole right side and no sensation in his arm or leg. He tries to get up with help from his wife but cannot. Mrs. A calls their physician and describes the situation. The physician instructs her to call 911 to have her husband taken to the hospital by ambulance, where he will meet them. Patient A protests but ultimately cooperates with emergency medical services when they arrive. He is quickly evaluated upon arrival in the emergency department and is admitted to the critical care unit (CCU).

Past Medical History

Patient A lives with his wife in their own home in a lower-middle class neighborhood. They have four sons who are living in different parts of the country. Until five years ago, Patient A had worked as a house painter.

Patient A's wife provides the patient's medical history. Patient A has not had any significant illness until two years ago, when he began to develop bilateral cataracts. Since then, he has consumed increasing amounts of alcohol as the encroaching cataracts impaired his ability to pursue his hobby of building ship models. Six months ago, the cataracts were successfully removed and lenses implanted. Since then, Patient A's alcohol consumption has decreased and he has resumed work on his models.

Mrs. A reports no knowledge of high blood pressure, heart disease, lung disease, kidney disease, cancer, or any other serious medical illness in the patient. He has no history of surgery or serious injuries during their 65 years of marriage.

PATIENT A'S PHYSICAL EXAM RESULTS	
Parameter	Findings
General appearance	Semiconscious and aphasic. Made no attempt to respond verbally to questions. Height: 5 feet 9 inches (175 cm) Weight: 163 pounds (74 kg)
Head and eyes	Face flushed Pupils equal, round, reactive to light, accommodation Corneal reflexes present Unable to test extraocular muscle function Nasal passage clear, with septum deviated to the left Edentulous, full dentures not in place, gums without lesions Tongue deviated to the left when protruded spontaneously Facial drooping on left
Ears	Tympanic membranes intact and clear No history of impaired hearing
Chest	Symmetrical excursion while lying in bed Lungs clear to auscultation and percussion Breath sounds diminished in the bases
Abdomen	Flat Bowel sounds present in all quadrants Soft and without masses or organomegaly on palpation
Extremities	Flaccid right arm and leg
Genitourinary system	Normal adult male with smooth, enlarged prostate gland
Neurologic status	Spontaneous respirations with regular variation in depth and rate Deep tendon reflexes in arms and femorals: 2+ Deep tendon reflexes in popliteals, posterior tibials, and dorsalis pedis: 1+ Spontaneous movement and active response to pain in left arm and leg Grimace but no movement with pain in right extremities
Cardiovascular system	Heart sounds consisted of normal S ₁ , S ₂ , and S ₃ Soft systolic ejection murmur heard at second intercostal space to right of sternum
Vital Signs	
Blood pressure	200/110 mm Hg
Temperature	100° F
Heart rate	86 bpm and regular
Respiratory rate	22 breaths per minute and stertorous Oxygen mask in place with flow at 8 L/min
Source: Author	

Table 1

Assessment and Diagnosis

Upon admittance to the CCU, a full physical exam is conducted (**Table 1**). Several laboratory tests are ordered, with the following results:

- Complete blood count with differential:
Within normal limits
- Serum electrolyte levels: Within normal limits
- Serum glucose level: Mildly elevated

Based on the results of the assessment, Patient A is diagnosed with:

- CVA (thrombosis or aneurysm of left middle cerebral artery), with right hemiparalysis and hemiparesis and questionable aphasia
- Benign prostatic hypertrophy

Management

When Patient A is admitted to the CCU, the nurse orients him and his wife to the physical layout and pertinent policies of the unit. The nurse also completes an initial physical assessment while carrying out the medical and nursing orders for supportive management. Nursing actions include:

- Continue oxygen by mask at 8 L/min and obtain arterial blood gas sample.
- Take vital signs every 15 minutes until stable, then every 30 minutes for two hours, then increasing interval until every four hours.
- Complete neurologic checks every hour.
- Insert IV devices and administer dextrose 5% in water (D5W) at a rate of 100 mL/hour.
- Insert 16F indwelling urinary catheter connected to a urinometer.
- Monitor and record intake and output every hour.
- Suction oropharynx to stimulate coughing and remove secretions.
- Frequent oral care, including Patient A's usual denture care routine.
- Repositioning every two hours, with body kept in functional alignment.
- Skin care and some passive range of motion with each turning so all joints are exercised every eight hours.
- Administer ordered medications:
 - Acetylsalicylic acid (aspirin): 650 mg every six hours
 - Sodium nitroprusside (Nipride) infusion: As needed to maintain systolic arterial pressure between 170 and 180 mm Hg

Twelve hours after admission, the nurse assessing Patient A notes that his eyes are half open, with ptosis of the right eyelid, and eye movements occur when the nurse or his wife speaks his name. Patient

A's right cheek is more flaccid than the left. His right arm and leg are limp with no muscle tone. There is some grasp strength in the patient's left hand, although he does not grasp on command. Patient A responds with grunts to painful stimuli but does not attempt to speak, follow commands, or answer questions.

Study Questions

1. Outline a complete neurologic status assessment.
2. How did the physician conclude that Patient A's CVA involved the left side cerebral artery?
3. What signs and symptoms would alert the nursing staff to occlusion of the left anterior or posterior cerebral artery?
4. Why did the physician order sodium nitroprusside to keep Patient A's systolic arterial pressure between 170 and 180 mm Hg?
5. What nursing diagnoses or nursing problems and outcomes assume priority in the acute care period of a CVA?
6. What other disciplines would be expected to assist in rehabilitation of a patient with a CVA? When should disciplines such as physical and occupational therapy be expected to begin working with the patient?

ACUTE PYOGENIC MENINGITIS

Patient B is a white woman, 67 years of age, who felt well until approximately one week ago, when she developed an upper respiratory tract infection. She has improved slowly, but during the past 48 hours she has developed a more severe cough with significant production of rust-colored sputum, fever with occasional shaking chills, and muscle aches. Patient B arrives at the hospital emergency department. She is transported by her husband, who was concerned when the patient woke in the morning mildly confused and complaining of a severe headache.

At the hospital, Patient B informs the physician (with some difficulty concentrating) that she has had a “bad cold” for about a week. She explains that her neck feels stiff, sore, and extremely painful when she tilts her head forward and bright lights hurt her eyes. She also tells the physician that she has had no skin rashes, nausea, or vomiting but has had some severe chills. She does not recall any of her recent contacts being ill, and she denies any difficulty breathing or chest pain.

Past Medical History

Patient B denies any past history of head trauma, sinus infection, immunodeficiency disorders, or medications that cause immunosuppression. She has smoked a half-pack of cigarettes each day for the last 45 years, was diagnosed with emphysema five years ago, and had several severe episodes of chronic bronchitis and one episode of pneumonia in the past two years. Her emphysema is being managed with ipratropium bromide delivered with a metered-dose inhaler (two to four puffs every six hours). She has never suffered from episodes of angina or symptoms of heart failure. She has an allergy to peanuts but not to any medications. She is taking no medications other than ipratropium and combined estrogen plus progestogen therapy for menopausal symptoms. The patient was vaccinated for influenza six months previously and pneumococcus when she turned 65 years of age.

Assessment and Diagnosis

Upon admittance to the CCU, a full physical exam is conducted (**Table 2**). A blood chemistry panel, chest x-rays, and lumbar puncture are ordered. Chest x-ray finds shadows on the right middle and lower lobe consistent with pneumonia; the left lung is clear but hyperinflated. Several laboratory tests are ordered, with the following results:

- Hematocrit: 41%
- Hemoglobin: 14.8 g/dL
- Red blood cells: 5.2 million/mL
- White blood cells: 14,000/mL (90% neutrophils)
- Platelets: 280,000/mL
- Sodium: 145 meq/L
- Potassium: 5.0 meq/L
- Chloride: 110 meq/L
- Calcium: 9.3 mg/dL
- Bicarbonate: 22 meq/L
- Fasting blood glucose: 123 mg/dL
- Blood urea nitrogen: 12 mg/dL
- Creatinine: 1.0 mg/dL
- CSF white blood cells: 1,100/mL (predominately neutrophils)
- CSF protein: 1,254 mg/dL
- CSF glucose: 40 mg/dL
- CSF gram stain: Positive for encapsulated diplococci
- CSF culture: Positive for *Streptococcus pneumoniae*
- Sputum gram stain: Positive for diplococci

Based on the physical examination and results of diagnostic testing, a preliminary diagnosis of meningitis is made. Patient B is admitted to the hospital for treatment and continued observation.

PATIENT B'S PHYSICAL EXAM RESULTS	
Parameter	Findings
General appearance	Slight female in acute distress, with headache, intermittent chills, and constant coughing. Appears older than her stated age. Height: 5 feet 0 inches (152.5 cm) Weight: 97 pounds (44 kg)
Head and eyes	Normocephalic with no signs of head injury Pupils equal at 3 mm, round and sluggishly reactive to light Difficult to view fundi due to photophobia, but no papilledema observed Nares slightly flared, purulent discharge visible Pharynx red with purulent postnasal drainage No tonsillar exudates Mucous membranes moist
Ears	Within normal limits
Neck	Stiff and painful with flexion Shows mild anterior cervical lymphadenopathy
Chest	Significant use of accessory muscles Breath sounds markedly decreased in right middle and lower lobes Crackles present at right posterior axillary line Clear left lung, both upper and lower lobes
Abdomen	Flat, soft, non-distended, with no tenderness to palpation Bowel sounds present in all four quadrants and within normal limits No masses, bruits, or organomegaly
Extremities	Peripheral pulses full and symmetric in all extremities No cyanosis, rashes, or edema upon careful inspection Mild clubbing
Genitourinary system	Normal adult female
Neurologic status	Oriented, but conversation is slightly confused Level of consciousness assessed at 14 on Glasgow Coma Scale Cranial nerves intact, including eye movements Strength 5/5 and symmetric throughout Deep tendon reflexes 2+ and symmetric Gait steady Positive Kernig and Brudzinski signs
Cardiovascular system	Distinct S ₁ and S ₂ with no murmurs or gallops Regular rate and rhythm Skin warm, moist, and pale
Vital Signs	
Blood pressure	160/74 mm Hg (right arm sitting)
Temperature	101.5° F
Heart rate	115 bpm and regular
Respiratory rate	24 breaths per minute and slightly labored Oxygen mask in place with flow at 8 L/min
Source: Author	

Table 2

Study Questions

1. List clinical manifestations that strongly suggest that a patient has developed meningitis.
2. Why is it appropriate for the physician to examine the patient for a head injury?
3. Define papilledema and explain the significance of lack of papilledema in this patient.
4. Explain the pathophysiology behind this patient's lymphadenopathy.
5. Is the patient's rating on the Glasgow Coma Scale normal or abnormal?
6. Based on all of the available test data, what is an appropriate neurologic diagnosis for Patient B?
7. How did this patient's neurologic condition probably develop?
8. Which type of white blood cell predominates in the blood and CSF of patients with acute bacterial meningitis?

Implicit Bias in Health Care

The role of implicit biases on healthcare outcomes has become a concern, as there is some evidence that implicit biases contribute to health disparities, professionals' attitudes toward and interactions with patients, quality of care, diagnoses, and treatment decisions. This may produce differences in help-seeking, diagnoses, and ultimately treatments and interventions. Implicit biases may also unwittingly produce professional behaviors, attitudes, and interactions that reduce patients' trust and comfort with their provider, leading to earlier termination of visits and/or reduced adherence and follow-up. Disadvantaged groups are marginalized in the healthcare system and vulnerable on multiple levels; health professionals' implicit biases can further exacerbate these existing disadvantages.

Interventions or strategies designed to reduce implicit bias may be categorized as change-based or control-based. Change-based interventions focus on reducing or changing cognitive associations underlying implicit biases. These interventions might include challenging stereotypes. Conversely, control-based interventions involve reducing the effects of the implicit bias on the individual's behaviors. These strategies include increasing awareness of biased thoughts and responses. The two types of interventions are not mutually exclusive and may be used synergistically.

Works Cited

1. Eunice Kennedy Shriver National Institute of Child Health and Human Development. What are Common Treatments for Cerebral Palsy? Available at <https://www.nichd.nih.gov/health/topics/cerebral-palsy/conditioninfo/treatments>. Last accessed October 25, 2021.
2. McCance KL, Huether SE. *Pathophysiology: The Biologic Basis for Disease in Adults and Children*. 8th ed. St. Louis, MO: Mosby; 2018.
3. Copstead LE. *Pathophysiology*. 6th ed. St. Louis, MO: Mosby; 2018.
4. Mitchell RN, Robbins SL. *Pocket Companion to Robbins and Coltran Pathologic Basis of Disease*. 8th ed. Philadelphia, PA: Saunders; 2012.
5. Rowland MD. *Merritt's Neurology*. 11th ed. Philadelphia, PA: Lippincott, Williams, and Wilkins; 2005.
6. Rae-Grant A. *Weiner and Levitt's Neurology*. 8th ed. Philadelphia, PA: Wolters Kluwer Health/ Lippincott Williams & Wilkins; 2008.
7. Nowak T. *Pathophysiology: Concepts and Applications for Health Care Professionals*. 3rd ed. Boston, MA: McGraw-Hill; 2011.
8. Biller J. *Practical Neurology*. 5th ed. Philadelphia, PA: Wolters Kluwer; 2017.
9. Galvagno SM. *Emergency Pathophysiology: Clinical Applications for Prehospital Care*. Jackson, WY: Teton NewMedia; 2013.
10. Hall J. *Guyton and Hall Textbook of Medical Physiology*. 13th ed. Philadelphia, PA: Elsevier; 2016.
11. Campbell WW, DeJong RN. *DeJong's The Neurologic Examination*. 7th ed. Philadelphia, PA: Wolters Kluwer; 2012.
12. Ellenbogen RG, Sekhar LN, Kitchen D. *Principles of Neurological Surgery*. 4th ed. Philadelphia, PA: Elsevier; 2018.
13. Berg SM, Bittner EA. *Massachusetts General Hospital Review of Critical Care Medicine*. Philadelphia, PA: Wolters Kluwer, Lippincott, Williams & Wilkins; 2014.
14. Hankey G, Wardlaw JM. *Clinical Neurology*. New York, NY: Demos Medical; 2008.
15. Singh A. *Case-Based Neurology*. New York, NY: Demos Medical; 2012.
16. Futrell N, Jamieson DG. *Vascular Neurology*. New York, NY: Demos Medical; 2008.
17. Gulanick. *Nursing Care Plans: Diagnoses, Interventions, and Outcomes*. 9th ed. St. Louis, MO: Elsevier Health Sciences; 2017.
18. Haberland C. *Clinical Neuropathology*. New York, NY: Demos Medical; 2007.
19. Araim RJ, Aghlar MR. *Neuromuscular Quick Pocket Reference*. New York, NY: Demos Medical; 2012.
20. Howard J. *Neurology Video Textbook*. New York, NY: Demos Medical; 2013.
21. Chen N, Li Q, Yang J, Zhou M, Zhou D, He L. Antiviral treatment for preventing postherpetic neuralgia. *Cochrane Database Syst Rev*. 2014;(2):CD006866.
22. DiPiro J. *Pharmacotherapy A Pathophysiologic Approach*. 10th ed. New York, NY: McGraw-Hill; 2017.
23. Zane RD, Kosowsky JM. *Pocket Emergency Medicine*. 3rd ed. Philadelphia, PA: Wolters Kluwer; 2015.
24. Markovchick VJ, Pons PT, Bakes KM, Buchanan JA. *Emergency Medicine Secrets*. 6th ed. Philadelphia, PA: Elsevier; 2016.
25. Grossman SC, Porth CM. *Pathophysiology: Concepts of Altered Health States*. Philadelphia, PA: Lippincott, Williams and Wilkins; 2014.
26. Bruyere HJ Jr. *100 Case Studies in Pathophysiology*. Philadelphia, PA: Lippincott, Williams and Wilkins; 2009.
27. Bope ET, Kellerman RD, Conn HF. *Conn's Current Therapy 2016*. Philadelphia, PA: Elsevier; 2016.
28. Marshall RS, Mayer SA. *On Call Neurology*. 3rd ed. Philadelphia, PA: Elsevier; 2007.
29. Nelms M. *Nutrition Therapy and Pathophysiology*. 3rd ed. Boston, MA: Cengage Learning; 2016.
30. Ackley BJ, Ladwig GB, Flynn Makic MB. *Nursing Diagnosis Handbook: An Evidence-Based Guide to Planning Care*. 11th ed. St. Louis, MO: Elsevier; 2017.
31. Zerwekh JA, Claborn JC. *Mosby's Pharmacology Memory Notecards*. 4th ed. St. Louis, MO: Elsevier; 2015.
32. Buttaro T, Trybulski J, Polgar-Bailey P, Sandberg-Cook J. *Primary Care: A Collaborative Practice*. 5th ed. St. Louis, MO: Elsevier; 2017.
33. Hickey JV. *The Clinical Practice of Neurological and Neurosurgical Nursing*. 7th ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2014.
34. Domino FJ. *The 5-Minute Clinical Consult Standard 2016*. 24th ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2015.
35. Ford SM, Roach SS. *Roach's Introductory Clinical Pharmacology*. 10th ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2014.
36. Caplan L. *Caplan's Stroke: A Clinical Approach*. 5th ed. Cambridge: Cambridge University Press; 2016.
37. Ferrell BR, Paice JA. *Oxford Textbook of Palliative Nursing*. 5th ed. New York, NY: Oxford University Press; 2019.
38. Ehrman J, Gordon PM, Visich PS, Keteyian SJ. *Clinical Exercise Physiology*. 3rd ed. Champaign, IL: Human Kinetics Publishing; 2013.
39. Baird MS. *Manual of Critical Care Nursing: Nursing Interventions and Collaborative Management*. 7th ed. St. Louis, MO: Mosby; 2016.
40. Ladwig GE, Ackley B, Flynn Makic MB. *Mosby's Guide to Nursing Diagnosis*. 5th ed. St. Louis, MO: Elsevier; 2016.
41. Barkauskas VH, Baumann LC, Darling-Fisher CS. *Health and Physical Assessment*. 3rd ed. St. Louis, MO: Mosby; 2002.
42. Black JM, Hawks JH, Luckmann J. *Medical Surgical Nursing: Clinical Management for Positive Outcomes*. 8th ed. Philadelphia, PA: W.B. Saunders; 2009.

43. Brashers VL. *Clinical Applications of Pathophysiology: Assessment, Diagnostic Reasoning and Management*. 2nd ed. St. Louis, MO: Mosby; 2002.
44. Hall JE, Guyton A. *Textbook of Medical Physiology*. 13th ed. Philadelphia, PA: Elsevier; 2016.
45. Huether SE, McCance KL. *Understanding Pathophysiology*. 6th ed. St. Louis, MO: Mosby; 2017.
46. LeMone P. *Medical Surgical Nursing: Critical Thinking in Client Care*. 5th ed. Boston, MA: Pearson; 2014.
47. Bullock BA. *Focus on Pathophysiology*. Philadelphia, PA: Lippincott; 2000.
48. Corwin EJ. *Handbook of Pathophysiology*. 3rd ed. Philadelphia, PA: Wolters Kluwer; 2015.
49. Price SA. *Pathophysiology: Clinical Concepts of Disease Processes*. 6th ed. St. Louis, MO: Mosby; 2003.
50. Patten JP. *Neurological Differential Diagnosis*. 2nd ed. New York, NY: Springer; 2000.
51. Johnson MV. *Principles of Drug Therapy in Neurology*. 2nd ed. New York, NY: Oxford University Press; 2008.
52. Wilson BA, Shannon MT, Shields KM. *Pearson Nurse's Drug Guide for 2017*. Boston, MA: Pearson Publishing; 2017.
53. Plum F, Posner JB. *Diagnosis of Stupor and Coma*. 3rd ed. New York, NY: Oxford University Press; 2000.
54. Wilson BA, Shannon MT, Shields KM. *Pearson Nurse's Drug Guide 2011*. New York, NY: Pearnson; 2011.
55. Alberstone CD, Steinmetz MP. *Anatomic Basis of Neurologic Diagnosis*. 2nd ed. New York, NY: Thieme Publishing; 2011.
56. Rubin D, Daube JR. *Clinical Neurophysiology*. 4th ed. Oxford, Oxford University Press; 2016.
57. Bedlack RS, Mitsumoto H. *Amyotrophic Lateral Sclerosis: A Patient Care Guide for Clinicians*. New York, NY: Demos Medical; 2013.
58. Harvey R, Stein J. *Stroke Recovery and Rehabilitation*. 2nd ed. New York, NY: Demos Medical; 2014.
59. Cristian A. *Medical Management of Adults with Neurologic Disabilities*. New York, NY: Demos Medical; 2009.
60. Cifu DX, Caruso D. *Traumatic Brain Injury*. New York, NY: Demos Medical; 2010.
61. Prahlow N, Kincaid JC, Buschbacher RM. *Neuromuscular Rehabilitation*. New York, NY: Demos Medical; 2014.
62. Zasler ND, Katz DI. *Brain Injury Medicine: Principles and Practice*. 2nd ed. New York, NY: Demos Medical; 2013.
63. MedlinePlus. Interferon Beta-1b Injection. Available at <https://medlineplus.gov/druginfo/meds/a601151.html>. Last accessed October 25, 2021.
64. Perret D, Chang EY, Hata J, Chen H, Tribuzio B. *The Pain Center Manual*. New York, NY: Demos Medical; 2014.
65. Berg AT, Berkovic SF, Brodie MJ, et al. Revised terminology and concepts for organization of seizures and epilepsies: report of the ILAE Commission on Classification and Terminology, 2005–2009. *Epilepsia*. 2010;51(4):676-685.
66. Mulley JC, Scheffer IE, Harkin LA, Berkovic SF, Dibbens LM. Susceptibility genes for complex epilepsy. *Hum Mol Genetics*. 2005;14(R2):R243-R249.
67. Engel J Jr, International League Against Epilepsy. A proposed diagnostic scheme for people with epileptic seizures and with epilepsy: report of the ILAE Task Force on Classification and Terminology. *Epilepsia*. 2001;42(6):796-803.
68. National Institute of Neurological Disorders and Stroke. The Epilepsies and Seizures: Hope Through Research. Available at <https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Hope-Through-Research/Epilepsies-and-Seizures-Hope-Through>. Last accessed October 25, 2021.
69. Fuller G, Manford M. *Neurology: An Illustrated Colour Text*. 3rd ed. Edinburgh: Churchill Livingstone Publisher; 2010.
70. Bryce TN. *Spinal Cord Injury*. New York, NY: Demos Medical; 2010.
71. MedlinePlus. Interferon Beta-1a Subcutaneous Injection. Available at <https://medlineplus.gov/druginfo/meds/a604005.html>. Last accessed October 25, 2021.
72. Blumenfeld H. *Neuroanatomy Through Clinical Cases*. 2nd ed. Sunderland, MA: Sinouer Associates; 2010.
73. Caplan LR. *Uncommon Causes of Stroke*. 3rd ed. Cambridge: Cambridge University Press; 2018.
74. Greer DM. *Acute Ischemic Stroke*. Hoboken, NJ: Wiley-Liss; 2007.
75. Hennerici MG. *Case Studies in Stroke: Common and Uncommon Presentations*. Cambridge: Cambridge University Press; 2007.
76. Benjamin EJ, Virani SS, Callaway CW, et al. Heart disease and stroke statistics—2018 update: a report from the American Heart Association. *Circulation*. 2018;137(12):e67-e492.
77. National Heart, Lung, and Blood Institute. 2012 NHLBI Incidence and Prevalence Chart Book on Cardiovascular and Lung Diseases. Available at https://www.nhlbi.nih.gov/files/docs/research/2012_ChartBook_508.pdf. Last accessed October 25, 2021.
78. Broderick JP, Brott T, Tomsick T, Miller R, Huster G. Intracerebral hemorrhage more than twice as common as subarachnoid hemorrhage. *J Neurosurg*. 1993;78(2):188-191.
79. Manno EM, Atkinson JL, Fulgham JR, Wijdicks EF. Emerging medical and surgical management strategies in the evaluation and treatment of intracerebral hemorrhage. *Mayo Clin Proc*. 2005;80(3):420-433.

80. Shah QA, Ezzeddine MA, Qureshi AI. Acute hypertension in intracerebral hemorrhage: pathophysiology and treatment. *J Neurol Sci.* 2007;261(1-2):74-79.
81. Morgenstern LB, Hemphill JC 3rd, Anderson C, et al. Guidelines for the management of spontaneous intracerebral hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association, American Heart Association Stroke Council and Council on Cardiovascular Nursing. *Stroke.* 2010;41(9):2108-2129.
82. Counsell C, Boonyakarnkul S, Dennis M, et al. Primary intracerebral hemorrhage in the Oxfordshire Community Stroke Project: prognosis. *Cerebrovasc Dis.* 1995;5:26-34.
83. Flaherty ML, Haverbusch M, Sekar PK, et al. Long-term mortality after intracerebral hemorrhage. *Neurology.* 2006;66(8):1182-1186.
84. Poon MT, Fonville AF, Al-Shahi Salman R. Long-term prognosis after intracerebral hemorrhage: systematic review and meta-analysis. *J Neurol Neurosurg Psychiatry.* 2014;85(6):660-667.
85. Sutherland GR, Auer RN. Primary intracerebral hemorrhage. *J Clin Neuroscience.* 2006;13:511-517.
86. Qureshi AI, Tuhim S, Broderick JP, Batjer H, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. *N Engl J Med.* 2001;344:1450-1460.
87. Ritter MA, Droste DW, Hegedüs, et al. Role of cerebral amyloid angiopathy in intracerebral hemorrhage in hypertensive patients. *Neurology.* 2005;64(7):1233-1237.
88. Kernan WN, Viscoli CM, Brass LM, et al. Phenylpropanolamine and the risk of hemorrhagic stroke. *N Engl J Med.* 2000;343:1826-1832.
89. Suarez JI, Tarr RW, Selman WR. Aneurysmal subarachnoid hemorrhage. *N Engl J Med.* 2006;354(4):387-396.
90. van Gijn J, Rinkel GJ. Subarachnoid haemorrhage: diagnosis, causes and management. *Brain.* 2001;124(pt 2):249-278.
91. Niewwkamp DJ, de Wilde A, Wermer MJ, Algra A, Rinkel GJ. Long-term outcome after aneurysmal subarachnoid hemorrhage—risks of vascular events, death from cancer and all-cause death. *J Neurol.* 2014;261(2):309-315.
92. Mayer SA, Kreiter KT, Copeland D, et al. Global and domain-specific cognitive impairment and outcome after subarachnoid hemorrhage. *Neurology.* 2002;59(11):1750-1758.
93. MedlinePlus. Cerebral Arteriovenous Malformation. Available at <https://medlineplus.gov/ency/article/000779.htm>. Last accessed October 25, 2021.
94. Flaherty ML, Haverbusch M, Kissela B, et al. Perimesencephalic subarachnoid hemorrhage: incidence, risk factors, and outcome. *J Stroke Cerebrovasc Dis.* 2005;14(6):267-271.
95. Konczalla J, Kashefiolasl S, Brawanski N, Senft C, Seifert V, Platz J. Increasing numbers of nonaneurysmal subarachnoid hemorrhage in the last 15 years: antithrombotic medication as reason and prognostic factor? *J Neurosurg.* 2016;124(6):1731-1731.
96. Muir KW, Buchan A, von Kummer R, Rother J, Baron JC. Imaging of acute stroke. *Lancet Neurol.* 2006;5(9):755-768.
97. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2019;50(12):e344-e418.
98. Jauch EC, Al Kasab S. Ischemic Stroke. Available at <https://emedicine.medscape.com/article/1916852-overview>. Last accessed October 25, 2021.
99. Powers WJ, Derdeyn CP, Biller J, et al. 2015 American Heart Association/American Stroke Association focused update of the 2013 guidelines for the early management of patients with acute ischemic stroke regarding endovascular treatment: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2015;46(10):3020-3035.
100. Birschel P, Ellul J, Barer D. Progressing stroke: towards an internationally agreed definition. *Cerebrovasc Dis.* 2004;17(2-3):242-252.
101. Hayashi K, Kitagawa N, Takahata H, et al. Endovascular treatment for cervical carotid artery stenosis presenting with progressing stroke: three case reports. *Surg Neurol.* 2002;58(2):148-154.
102. Du Mesnil de Rochemont R, Sitzler M, Neumann-Haefelin T, Harmjanz A, Berkefeld J. Endovascular recanalization of acute atherothrombotic carotid artery occlusion holds up progressive stroke. *Neuroradiology.* 2004;46(7):583-586.
103. Rerkasem K, Rothwell PM. Systematic review of the operative risks of carotid endarterectomy for recently symptomatic stenosis in relation to the timing of surgery. *Stroke.* 2009;40:e564-e572.
104. Kleindorfer DO, Towfighi A, Chaturvedi S, et al. 2021 guideline for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline from the American Heart Association/American Stroke Association. *Stroke.* 2021;52(7):e364-e467.
105. Winstein CJ, Stein J, Arena R, et al. AHA/ASA guidelines for adult stroke rehabilitation and recovery: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2016;47(6):e98-e169.
106. Langhorne P, Stott DJ, Robertson L, et al. Medical complications after stroke: a multicenter study. *Stroke.* 2000;31(6):1223-1229.
107. Giles MF, Rothwell PM. Risk of stroke early after transient ischemic attack: a systematic review and meta-analysis. *Lancet Neurol.* 2007;6(12):1063-1072.

108. Albers GW, Caplan LR, Easton JD, et al. Transient ischemic attack-proposal for a new definition. *N Engl J Med*. 2002;347(21):1713-1716.
109. Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. *Stroke*. 2009;40(6):2276-2293.
110. Gladstone DJ. Toward an emergency response to transient ischemic attacks. *Postgrad Med J*. 2005;117(1):9-14.
111. Fisher CM. Transient ischemic attacks. *N Engl J Med*. 2002;347(21):1642-1643.
112. Johnston SC, Gress DR, Browner WS, Sidney S. Short-term prognosis after emergency department diagnosis of TIA. *JAMA*. 2000;284(22):2901-2906.
113. Levy DE. How transient are transient ischemic attacks? *Neurology*. 1988;38(5):647-677.
114. Henry GL, Johnston SC, Pinkowish MD. Acute neurologic symptoms—TIA, migraine, or something else? *Patient Care*. 2004;38(3):50-56.
115. Nanda A, Singh NN. Transient Ischemic Attack. Available at <https://emedicine.medscape.com/article/1910519-overview>. Last accessed October 25, 2021.
116. Ko DY, Sahai-Srivastava S. Generalized Tonic-Clonic Seizures. Available at <https://emedicine.medscape.com/article/1184608-overview>. Last accessed October 25, 2021.
117. Epilepsy Foundation. Types of Seizures. Available at <https://www.epilepsy.com/learn/types-seizures>. Last accessed October 25, 2021.
118. Johns Hopkins Medicine. Generalized Seizures. Available at <https://www.hopkinsmedicine.org/health/conditions-and-diseases/epilepsy/generalized-seizures>. Last accessed October 25, 2021.
119. Centers for Disease Control and Prevention. Epilepsy. Available at <https://www.cdc.gov/epilepsy/index.html>. Last accessed October 25, 2021.
120. Ko DY. Epilepsy and Seizures. Available at <https://emedicine.medscape.com/article/1184846-overview>. Last accessed October 25, 2021.
121. Adamolekun B. Seizure Disorders. Available at <https://www.merckmanuals.com/professional/neurologic-disorders/seizure-disorders/seizure-disorders>. Last accessed October 25, 2021.
122. St. Louis EK, Rosenfeld WE, Bramley T. Antiepileptic drug monotherapy: the initial approach in epilepsy management. *Curr Neuropharmacol*. 2009;7:77-82.
123. Scottish Intercollegiate Guidelines Network. *Diagnosis and Management of Headache in Adults: A National Clinical Guideline*. Edinburgh: Scottish Intercollegiate Guidelines Network; 2008.
124. World Health Organization. Headache Disorders. Available at <http://www.who.int/en/news-room/fact-sheets/detail/headache-disorders>. Last accessed October 25, 2021.
125. International Headache Society. The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalalgia*. 2013;33(9):629-808.
126. MedlinePlus. Tension Headache. Available at <https://medlineplus.gov/ency/article/000797.htm>. Last accessed October 25, 2021.
127. National Institute on Aging. Basics of Alzheimer's Disease and Dementia. Available at <https://www.nia.nih.gov/health/alzheimers/basics>. Last accessed October 25, 2021.
128. Alzheimer's Association. 2021 Alzheimer's Disease Facts and Figures. Available at <https://www.alz.org/media/Documents/alzheimers-facts-and-figures.pdf>. Last accessed October 25, 2021.
129. National Institute on Aging. What Causes Alzheimer's Disease? Available at <https://www.nia.nih.gov/health/what-causes-alzheimers-disease>. Last accessed October 25, 2021.
130. Alzheimer's Association. Stages of Alzheimer's. Available at <https://www.alz.org/alzheimers-dementia/stages>. Last accessed October 25, 2021.
131. Kuller LH, Lopez OL, Becker JT, et al. Risk of dementia and death in the long-term follow-up of the Pittsburgh Cardiovascular Health Study—Cognition Study. *Alzheimers Dement*. 2016;12(2):170-183.
132. Solfrizzi V, Frisardi V, Seripa D, et al. Mediterranean diet in predementia and dementia syndromes. *Curr Alzheimer Res*. 2011;8(5):520-542.
133. Winslow BT, Onysko MK, Stob CM, Hazlewood KA. Treatment of Alzheimer disease. *Am Fam Physician*. 2011;83(12):1403-1412.
134. Seltzer B. Cholinesterase inhibitors in the clinical management of Alzheimer's disease: importance of early and persistent treatment. *J Int Med Res*. 2006;34(4):339-347.
135. National Institute on Aging. How is Alzheimer's Disease Treated? Available at <https://www.nia.nih.gov/health/how-alzheimers-disease-treated>. Last accessed October 25, 2021.

136. Alzheimer's Association. Medications for Memory. Available at <https://www.alz.org/alzheimers-dementia/treatments/medications-for-memory>. Last accessed October 25, 2021.
137. Beier MT. Treatment strategies for the behavioral symptoms of Alzheimer's disease: focus on early pharmacologic intervention. *Pharmacotherapy*. 2007;27(3):399-411.
138. DeMaagd G, Philip A. Parkinson's disease and its management. Part 1: disease entity, risk factors, pathophysiology, clinical presentation, and diagnosis. *PT*. 2015;40(8):504-532.
139. Gazewood JD, Richards DR, Clebak K. Parkinson disease: an update. *Am Fam Physician*. 2013;87(4):267-273.
140. Williams DR, Litvan I. Parkinsonian syndromes. *Continuum (Minneapolis Minn)*. 2013;19(5):11890-11212.
141. Braak H, Ghebremedhin E, Rüb U, Bratzke H, Del Tredici K. Stages in the development of Parkinson's disease-related pathology. *Cell Tissue Res*. 2004;318(1):121-134.
142. Schrag A, Horsfall L, Walters K, Noyce A, Petersen I. Prediagnostic presentations of Parkinson's disease in primary care: a case-control study. *Lancet Neurol*. 2015;14(1):57-64.
143. Braak H, Bohl JR, Muller CM, et al. Stanley Fahn Lecture 2005: the staging procedure for the inclusion body pathology associated with sporadic Parkinson's disease reconsidered. *Mov Disord*. 2006;21(12):2042-2051.
144. Williams DR, Lees AJ. How do patients with parkinsonism present? A clinicopathological study. *Intern Med J*. 2009;39(1):7-12.
145. Massano J, Bhatia KP. Clinical approach to Parkinson's disease: features, diagnosis, and principles of management. *Cold Spring Harb Perspect Med*. 2012;2(6):a008870.
146. Mhyre TR, Boyd JT, Hamill RW, Maguire-Zeiss KA. Parkinson's disease. *Subcell Biochem*. 2012;65:389-455.
147. Postuma RB, Berg D, Stern M, et al. MDS clinical diagnostic criteria for Parkinson's disease. *Mov Disord*. 2015;30(12):1591-1599.
148. Jenner P. Treatment of the later stages of Parkinson's disease: pharmacological approaches now and in the future. *Transl Neurodegener*. 2015;4:3.
149. Compston A, Coles A. Multiple sclerosis. *Lancet*. 2008;372(9648):1502-1517.
150. Koch-Henriksen N, Sørensen PS. The changing demographic pattern of multiple sclerosis epidemiology. *Lancet Neurol*. 2010;9(5):520-532.
151. Frohman TC, O'Donoghue DL, Northrop D (eds). *Multiple Sclerosis for the Physician Assistant: A Practical Primer*. New York, NY: National Multiple Sclerosis Society; 2011.
152. National Multiple Sclerosis Society. Symptoms and Diagnosis. Available at <https://www.nationalmssociety.org/Symptoms-Diagnosis>. Last accessed October 25, 2021.
153. Hersh CM, Fox RJ. Disease Management: Multiple Sclerosis. Available at https://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/neurology/multiple_sclerosis/Default.htm. Last accessed October 25, 2021.
154. Rae-Grant A, Day GS, Marrie RA, et al. Practice guideline recommendations summary: Disease-modifying therapies for adults with multiple sclerosis: report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology. *Neurology*. 2018;90(17):777-788.
155. MedlinePlus. Interferon Beta-1a Intramuscular Injection. Available at <https://medlineplus.gov/druginfo/meds/a693040.html>. Last accessed October 25, 2021.
156. Saitoh Y, Takahashi Y. Riluzole for the treatment of amyotrophic lateral sclerosis. *Neurodegener Dis Manag*. 2020;10(6):343-355.
157. National Institute on Neurological Disorders and Stroke. Myasthenia Gravis Fact Sheet. Available at <https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Myasthenia-Gravis-Fact-Sheet>. Last accessed October 25, 2021.
158. Dhillon S. Eculizumab: a review in generalized myasthenia gravis. *Drugs*. 2018;78(3):367-376.
159. Genetic and Rare Diseases Information Center. Herpes Simplex Encephalitis. Available at <https://rarediseases.info.nih.gov/diseases/6649/herpes-simplex-encephalitis>. Last accessed October 25, 2021.
160. Icahn School of Medicine at Mount Sinai. Viral Encephalitis. Available at <https://www.mountsinai.org/health-library/report/viral-encephalitis>. Last accessed October 25, 2021.
161. LexiComp Online. Available at <https://online.lexi.com>. Last accessed October 25, 2021.

Evidence-Based Practice Recommendations Citations

- Brown MD, Burton JH, Nazarian DJ, Promes SB, American College of Emergency Physicians. Clinical policy: use of intravenous tissue plasminogen activator for the management of acute ischemic stroke in the emergency department. *Ann Emerg Med.* 2015;66(3):322-333. Available at https://www.acep.org/globalassets/new-pdfs/clinical-policies/tpa-for-stroke_new.pdf. Last accessed November 18, 2021.
- Krumholz A, Wiebe S, Gronseth GS, et al. Evidence-based guideline: management of an unprovoked first seizure in adults: report of the Guideline Development Subcommittee of the American Academy of Neurology and the American Epilepsy Society. *Neurology.* 2015;84(16):1705-1713. Available at <https://www.aan.com/Guidelines/home/GetGuidelineContent/688>. Last accessed November 18, 2021.
- Douglas AC, Wippold FJ II, Broderick DF, et al. *ACR Appropriateness Criteria: Headache*. Reston, VA: American College of Radiology; 2019. Available at <https://acsearch.acr.org/docs/69482/Narrative>. Last accessed November 18, 2021.
- Fletcher K. Nursing Standard of Practice Protocol: Recognition and Management of Dementia. Abstract available from Guideline Central at <https://www.guidelinecentral.com/summaries/recognition-and-management-of-dementia-in-evidence-based-geriatric-nursing-protocols-for-best-practice>. Last accessed November 18, 2021.
- Preissner K. *Occupational Therapy Practice Guidelines for Adults with Neurodegenerative Diseases*. Bethesda, MD: American Occupational Therapy Association, Inc.; 2014. Abstract available from Guideline Central at <https://www.guidelinecentral.com/summaries/occupational-therapy-practice-guidelines-for-adults-with-neurodegenerative-diseases>. Last accessed November 18, 2021.
- Thompson AJ, Banwell BL, Barkhof F, et al. Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria. *Lancet Neurol.* 2018;17(2):162-173. Available at [https://www.thelancet.com/journals/lanneur/article/PIIS1474-4422\(17\)30470-2/fulltext](https://www.thelancet.com/journals/lanneur/article/PIIS1474-4422(17)30470-2/fulltext). Last accessed November 18, 2021.
- Theodore N, Aarabi B, Dhall SS, et al. Transportation of patients with acute traumatic cervical spine injuries. In: Guidelines for the management of acute cervical spine and spinal cord injuries. *Neurosurgery.* 2013;72(Suppl 2):35-39. Available at https://academic.oup.com/neurosurgery/article/72/suppl_3/35/2557442. Last accessed November 18, 2021.