

# UNIT 15

## Neurologic Function

### Case Study

#### DEVELOPING A TEAM-BASED PLAN OF CARE

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You are the nurse caring for a 70-year-old man with Parkinson's disease who was recently admitted to the skilled nursing facility where you work. The patient was diagnosed with Parkinson's

disease 5 years ago but has only recently had a significant decline in function. He can no longer dress himself because he lacks the ability to control fine motor movements in his hands. In addition, he has lost 10 lb in the past 3 months as a result of difficulty feeding himself because of the constant tremors. The patient's wife has reported feeling exhausted prior to her husband's admission; she had not been sleeping well due to her husband having nightmares and trying to get up during the night. You form a task force to help develop a plan of care that is focused on increasing the patient's functionality so that he might be discharged home. Specifically, you invite a staff nutritionist, pharmacist, physical therapist, occupational therapist, and social worker to work with you in developing the plan of care for this patient.

### **QSEN Competency Focus: Teamwork and Collaboration**

The complexities inherent in today's health care system challenge nurses to demonstrate integration of specific interdisciplinary core competencies. These competencies are aimed at ensuring the delivery of safe, quality patient care (Institute of Medicine, 2003). The Quality and Safety Education for Nurses project (Cronenwett, Sherwood, Barnsteiner, et al., 2007; QSEN, 2020) provides a framework for the knowledge, skills, and attitudes (KSAs) required for nurses to demonstrate competency in these key areas, which include ***patient-centered care, interdisciplinary teamwork and collaboration, evidence-based practice, quality improvement, safety, and informatics.***

**Teamwork and Collaboration Definition:** Function effectively within nursing and interprofessional teams, fostering open communication, mutual respect, and shared decision-making to achieve quality patient care.

SELECT PRE-LICENSURE KSAs	APPLICATION AND REFLECTION
	<b>Knowledge</b>
<p>Describe scopes of practice and roles of health care team members</p> <p>Describe strategies for identifying and managing overlaps in team member roles and accountabilities</p> <p>Recognize contributions of other individuals and groups in helping patient/family achieve health goals</p>	<p>How can you help the patient and his wife identify health goals to manage his Parkinson's disease? Based upon your knowledge of their scopes of practice, identify why you decided which team members to include when you formed the task force. Identify the contributions of the different members of the team in meeting the patient's and wife's goals.</p>
	<b>Skills</b> <p>Assume role of team member or leader based on the situation</p> <p>Initiate requests for help when appropriate to situation</p> <p>Clarify roles and accountabilities under conditions of potential overlap in team member functioning</p> <p>Integrate the contributions of others who play a role in helping patient/family achieve health goals</p> <p>What skills will you need to assume the role of team leader and advocate for the needs of both the patient and his wife?</p> <p>What subjective and objective assessment criteria can you use to determine which team members need to be consulted for which specific problem or to help the patient and his wife meet specific goals?</p> <p>Once the consults have been made, what is your role in ensuring that the plan of care for this patient is being implemented?</p>

Attitudes	
Value the perspectives and expertise of all health team members	
Respect the centrality of the patient/family as core members of any health care team	How will you demonstrate to the patient and his wife the value of the various team members? How can team members ensure respect for each other? How will each member of the team maintain accountability for the plan of care?
Respect the unique attributes that members bring to a team, including variations in professional orientations and accountabilities	

Cronenwett, L., Sherwood, G., Barnsteiner, J., et al. (2007). Quality and safety education for nurses. *Nursing Outlook*, 55(3), 122–131; Institute of Medicine. (2003). *Health professions education: A bridge to quality*. Washington, DC:

National Academies Press; QSEN Institute. (2020). *QSEN competencies: Definitions and pre-licensure KSAs; Teamwork and collaboration*. Retrieved on 8/15/2020 at: [qsen.org/competencies/pre-licensure-ksas/#teamwork\\_collaboration](https://qsen.org/competencies/pre-licensure-ksas/#teamwork_collaboration)

# 60 Assessment of Neurologic Function

## LEARNING OUTCOMES

*On completion of this chapter, the learner will be able to:*

1. Describe the structures and functions of the central and peripheral nervous systems.
2. Differentiate between pathologic changes that affect motor control and those that affect sensory pathways.
3. Compare and contrast the functioning of the sympathetic and parasympathetic nervous systems.
4. Explain the significance of physical assessment to the diagnosis of neurologic dysfunction.
5. Discuss diagnostic tests used for assessment of suspected neurologic disorders and the related nursing implications.

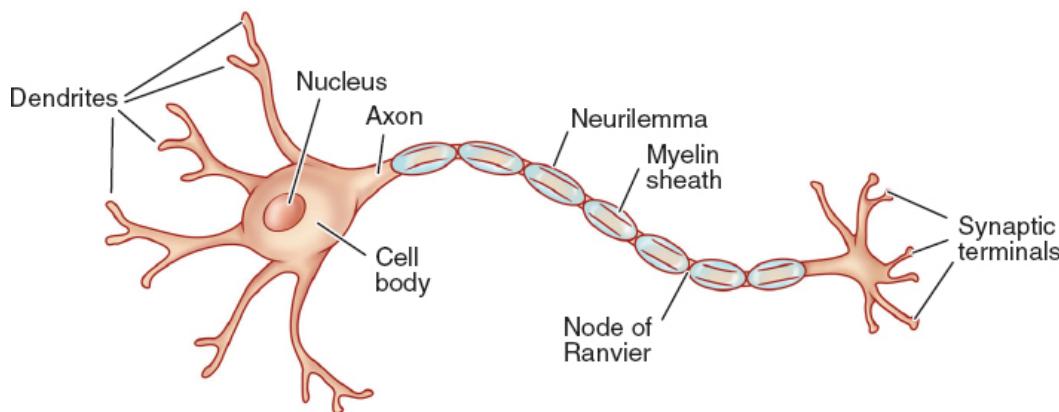
## NURSING CONCEPTS

- Assessment
- Intracranial Regulation
- Mobility
- Mood and Affect
- Patient Education
- Sensory Perception

## GLOSSARY

- agnosia:** loss of ability to recognize objects through a particular sensory system; may be visual, auditory, or tactile
- ataxia:** inability to coordinate muscle movements, resulting in difficulty in walking, talking, and performing self-care activities
- autonomic nervous system:** division of the nervous system that regulates the involuntary body functions
- axon:** portion of the neuron that conducts impulses away from the cell body
- Babinski reflex (sign):** a reflex action of the toes; in adults is indicative of abnormalities in the motor control pathways leading from the cerebral cortex
- clonus:** abnormal movement marked by alternating contraction and relaxation of a muscle occurring in rapid succession
- delirium:** an acute, confused state that begins with disorientation and if not recognized and treated early can progress to changes in level of consciousness, irreversible brain damage, and sometimes death
- dendrite:** portion of the neuron that conducts impulses toward the cell body
- flaccidity:** displaying lack of muscle tone; limp, floppy
- parasympathetic nervous system:** division of the autonomic nervous system active primarily during nonstressful conditions, controlling mostly visceral functions
- position (postural) sense:** awareness of position of parts of the body without looking at them (*synonym:* proprioception)
- reflex:** an automatic response to stimuli
- rigidity:** increase in muscle tone at rest characterized by increased resistance to passive stretch
- Romberg test:** test for cerebellar dysfunction that can be done with the patient seated or standing; inability to maintain position for 20 seconds is a positive test
- spasticity:** sustained increase in tension of a muscle when it is passively lengthened or stretched
- sympathetic nervous system:** division of the autonomic nervous system with predominantly excitatory responses (*synonym:* the “fight-or-flight” system)
- vertigo:** illusion of movement in which the individual or the surroundings are sensed as moving

Nurses in many practice settings encounter patients with altered neurologic function. Disorders of the nervous system can occur at any time during the lifespan and can vary from mild, self-limiting symptoms to devastating, life-threatening disorders. Nurses must be skilled in the general assessment of neurologic function and be able to focus on specific areas as needed. Assessment requires knowledge of the anatomy and physiology of the nervous system and an understanding of the array of tests and procedures used to diagnose neurologic disorders. Knowledge about the nursing implications and interventions related to assessment and diagnostic testing is also essential.



**Figure 60-1 •** Neuron.

## Anatomic and Physiologic Overview

The nervous system consists of two major parts: the central nervous system (CNS), including the brain and spinal cord, and the peripheral nervous system, which includes the cranial nerves, spinal nerves, and autonomic nervous system. The function of the nervous system is to control motor, sensory, autonomic, cognitive, and behavioral activities. The brain itself contains more than 100 billion cells that link the motor and sensory pathways, monitor the body's processes, respond to the internal and external environment, maintain homeostasis, and direct all psychological, biologic, and physical activity through complex chemical and electrical messages (Klein & Stewart-Amidei, 2017).

## Cells of the Nervous System

The basic functional unit of the brain is the neuron (see Fig. 60-1). It is composed of dendrites, a cell body, and an axon. The **dendrites** are branch-type structures for receiving electrochemical messages. The **axon** is a long projection that carries electrical impulses away from the cell body. Some axons

have a myelinated sheath that increases speed of conduction. Nerve cell bodies occurring in clusters are called *ganglia* or *nuclei*. A cluster of cell bodies with the same function is called a *center* (e.g., the respiratory center). Neurons are supported, protected, and nourished by glial cells, which are 50 times greater in number than neurons (Hickey & Strayer, 2020).

## Neurotransmitters

Neurotransmitters communicate messages from one neuron to another or from a neuron to a target cell, such as muscle or endocrine cells. Neurotransmitters are manufactured and stored in synaptic vesicles. As an electrical action potential moves along the axon and reaches the nerve terminal, neurotransmitters are released into the synapse. The neurotransmitter is transported across the synapse, binding to receptors on the postsynaptic cell membrane. A neurotransmitter can either excite or inhibit activity of the target cell. Usually, multiple neurotransmitters are at work in the neural synapse. The source and action of major neurotransmitters are described in [Table 60-1](#). Once released, enzymes either destroy the neurotransmitter or reabsorb it into the neuron for future use.

Many neurologic disorders are due, at least in part, to an imbalance in neurotransmitters. For example, Parkinson's disease develops from decreased availability of dopamine, whereas acetylcholine binding to muscle cells is impaired in myasthenia gravis (Norris, 2019). All brain functions are modulated through neurotransmitter receptor site activity, including memory and other cognitive processes (Hickey & Strayer, 2020).

**TABLE 60-1** Major Neurotransmitters

Neurotransmitter	Source	Action
Acetylcholine (major transmitter of the parasympathetic nervous system)	Neurons in many areas of the brain; autonomic nervous system	Usually excitatory; parasympathetic effects sometimes inhibitory (stimulation of heart by vagal nerve)
Serotonin	Brain stem, hypothalamus, dorsal horn of the spinal cord	Inhibitory; helps control mood and sleep, inhibits pain pathways
Dopamine	Neurons on the substantia nigra and basal ganglia	Usually inhibitory; affects behavior (attention, emotions) and fine movement
Norepinephrine (major transmitter of the sympathetic nervous system)	Brain stem, hypothalamus, postganglionic neurons of the sympathetic nervous system	Usually excitatory; affects mood and overall activity
Gamma-aminobutyric acid	Nerve terminals of the spinal cord, cerebellum, basal ganglia, some cortical areas	Inhibitory
Enkephalin, endorphin	Nerve terminals in the spine, brain stem, thalamus and hypothalamus, pituitary gland	Excitatory; pleasurable sensation inhibits pain transmission

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Ongoing research is evaluating diagnostic tests that can detect abnormal levels of neurotransmitters in the brain. Positron emission tomography (PET), for example, can detect dopamine, serotonin, and acetylcholine. Single-photon emission computed tomography (SPECT), similar to PET, can detect changes in some neurotransmitters, such as dopamine in Parkinson's disease (Fischbach & Fischbach, 2018). Both PET and SPECT are discussed in more detail later in this chapter.

## The Central Nervous System

The CNS consists of the brain and the spinal cord.

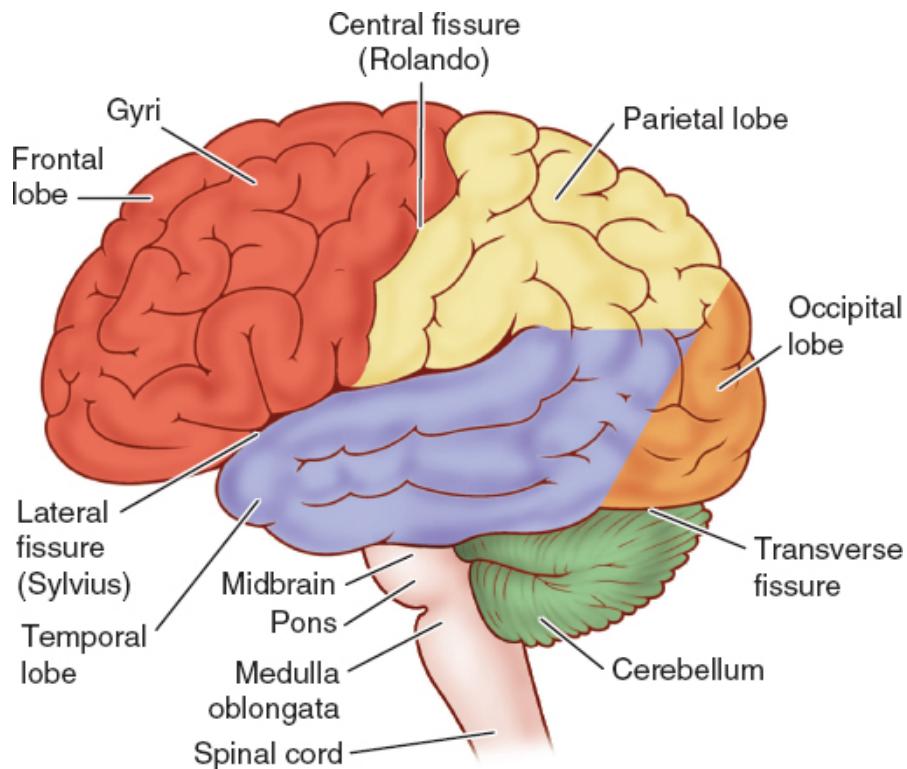
### The Brain

The brain accounts for approximately 2% of the total-body weight; in an average young adult, the brain weighs approximately 1400 g, whereas in an average older adult, the brain weighs approximately 1200 g (Hickey & Strayer,

2020). The brain is divided into three major areas: the cerebrum, the brain stem, and the cerebellum. The cerebrum is composed of two hemispheres, the thalamus, the hypothalamus, and the basal ganglia. The brain stem includes the midbrain, pons, and medulla. The cerebellum is located under the cerebrum and behind the brain stem (see [Fig. 60-2](#)).

### Cerebrum

The outside surface of the hemispheres has a wrinkled appearance that is the result of many folded layers or convolutions called *gyri*, which increase the surface area of the brain, accounting for the high level of activity carried out by such a small-appearing organ. Between each gyrus is a sulcus or fissure that serves as an anatomic division. In between the cerebral hemispheres is the great longitudinal fissure that separates the cerebrum into the right and left hemispheres. The two hemispheres are joined at the lower portion of the fissure by the corpus callosum. The external or outer portion of the hemispheres (the cerebral cortex) is made up of gray matter approximately 2 to 5 mm in depth; it contains billions of neuron cell bodies, giving it a gray appearance. White matter makes up the innermost layer and is composed of myelinated nerve fibers and neuroglia cells that form tracts or pathways connecting various parts of the brain with one another. These pathways also connect the cortex with lower portions of the brain and spinal cord. The cerebral hemispheres are divided into pairs of lobes as follows (see [Fig. 60-2](#)):

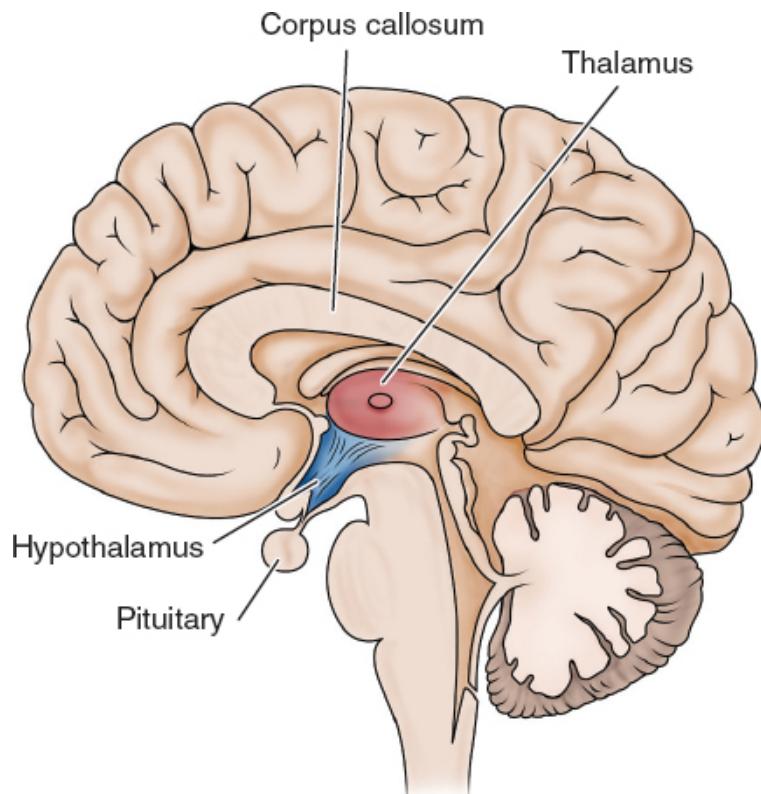


**Figure 60-2 •** View of the external surface of the brain showing lobes, cerebellum, and brain stem.

- *Frontal*—the largest lobe, located in the front of the brain. The major functions of this lobe are concentration, abstract thought, information storage or memory, and motor function. It contains Broca area, which is in the left hemisphere and is critical for motor control of speech. The frontal lobe is also responsible in large part for a person's affect, judgment, personality, and inhibitions (Hickey & Strayer, 2020).
- *Parietal*—a predominantly sensory lobe posterior to the frontal lobe. This lobe analyzes sensory information and relays the interpretation of this information to other cortical areas and is essential to a person's awareness of body position in space, size and shape discrimination, and right–left orientation (Hickey & Strayer, 2020).
- *Temporal*—located inferior to the frontal and parietal lobes, this lobe contains the auditory receptive areas and plays a role in memory of sound and understanding of language and music.
- *Occipital*—located posterior to the parietal lobe, this lobe is responsible for visual interpretation and memory.

The corpus callosum (see Fig. 60-3), a thick collection of nerve fibers that connects the two hemispheres of the brain, is responsible for the transmission of information from one side of the brain to the other. Information transferred includes sensation, memory, and learned discrimination. Right-handed people

and some left-handed people have cerebral dominance on the left side of the brain for verbal, linguistic, arithmetic, calculation, and analytic functions. The nondominant hemisphere is responsible for geometric, spatial, visual, pattern, and musical functions. Nuclei for cranial nerves I and II are also located in the cerebrum.



**Figure 60-3 •** Medial view of the brain.

The thalami lie on either side of the third ventricle and act primarily as a relay station for all sensation except smell. All memory, sensation, and pain impulses pass through this section of the brain. The hypothalamus (see Fig. 60-3) is located anterior and inferior to the thalamus, and beneath and lateral to the third ventricle. The infundibulum of the hypothalamus connects it to the posterior pituitary gland. The hypothalamus plays an important role in the endocrine system because it regulates the pituitary secretion of hormones that influence metabolism, reproduction, stress response, and urine production. It works with the pituitary to maintain fluid balance through hormonal release and maintains temperature regulation by promoting vasoconstriction or vasodilatation. In addition, the hypothalamus is the site of the hunger center and is involved in appetite control. It contains centers that regulate the sleep-wake cycle, blood pressure, aggressive and sexual behavior, and emotional responses (e.g., blushing, rage, depression, panic, fear). The hypothalamus also controls and regulates the autonomic nervous system. The optic chiasm (the

point at which the two optic tracts cross) and the mammillary bodies (involved in olfactory reflexes and emotional response to odors) are also found in this area.

The basal ganglia are masses of nuclei located deep in the cerebral hemispheres that are responsible for control of fine motor movements, including those of the hands and lower extremities.

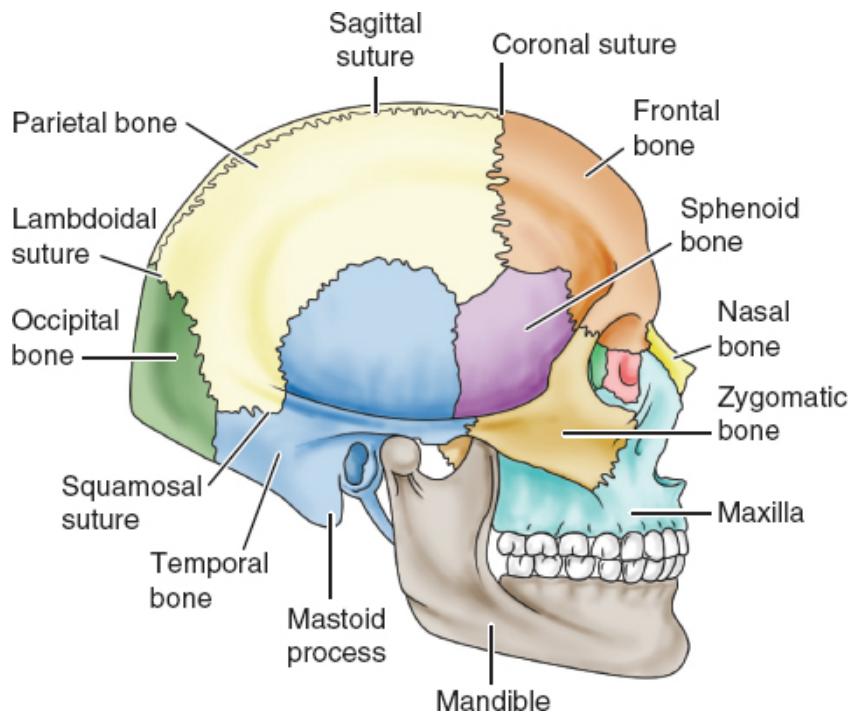
## Brain Stem

The brain stem consists of the midbrain, pons, and medulla oblongata (see [Fig. 60-2](#)). The midbrain connects the pons and the cerebellum with the cerebral hemispheres; it contains sensory and motor pathways and serves as the center for auditory and visual reflexes. Cranial nerves III and IV originate in the midbrain. The pons is situated in front of the cerebellum between the midbrain and the medulla and is a bridge between the two halves of the cerebellum, and between the medulla and the midbrain. Cranial nerves V through VIII originate in the pons. The pons also contains motor and sensory pathways. Portions of the pons help regulate respiration.

Motor fibers from the brain to the spinal cord and sensory fibers from the spinal cord to the brain are in the medulla. Most of these fibers cross, or decussate, at this level. Cranial nerves IX through XII originate in the medulla. Reflex centers for respiration, blood pressure, heart rate, coughing, vomiting, swallowing, and sneezing are also located in the medulla. The reticular formation, responsible for arousal and the sleep-wake cycle, begins in the medulla and connects with numerous higher structures.

## Cerebellum

The cerebellum is posterior to the midbrain and pons, and below the occipital lobe (see [Fig. 60-2](#)). The cerebellum integrates sensory information to provide smooth coordinated movement. It controls fine movement, balance, and **position (postural) sense** or proprioception (awareness of position of extremities without looking at them).



**Figure 60-4 •** Bones and sutures of the skull.

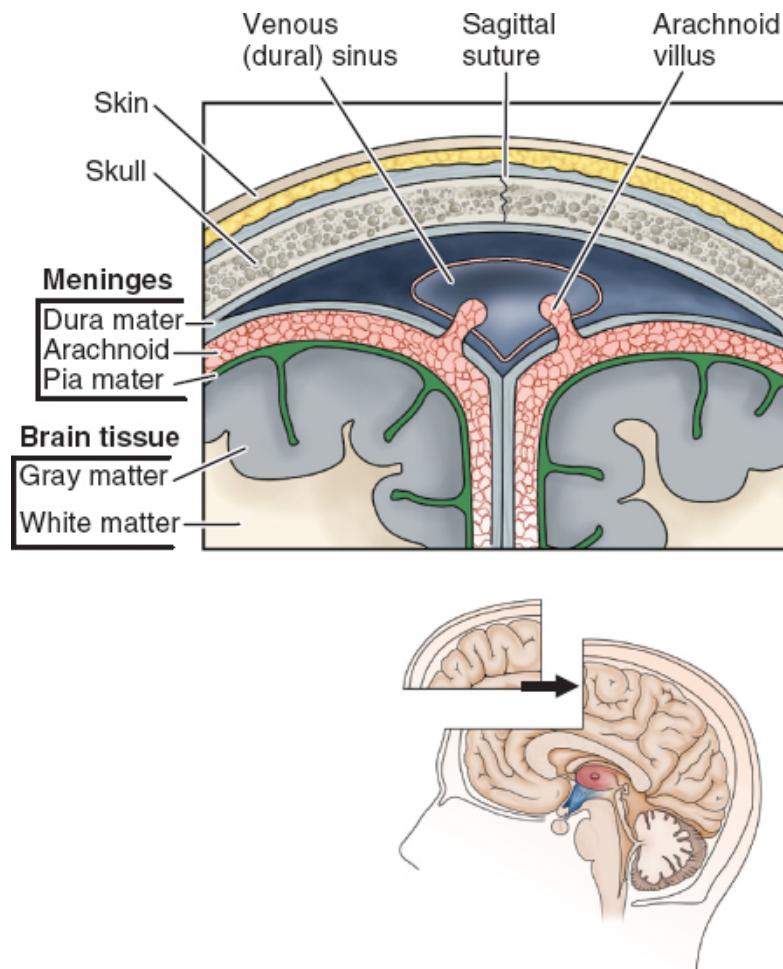
### Structures Protecting the Brain

The brain is contained in the rigid skull, which protects it from injury. The major bones of the skull are the frontal, temporal, parietal, occipital, and sphenoid bones. These bones join at the suture lines (see Fig. 60-4) and form the base of the skull. Indentations in the skull base are known as fossae. The anterior fossa contains the frontal lobe, the middle fossa contains the temporal lobe, and the posterior fossa contains the cerebellum and brain stem.

The meninges (fibrous connective tissues that cover the brain and spinal cord) provide protection, support, and nourishment. The layers of the meninges are the dura mater, arachnoid, and pia mater (see Fig. 60-5):

- *Dura mater*—the outermost layer; covers the brain and the spinal cord. It is tough, thick, inelastic, fibrous, and gray. There are three major extensions of the dura: the falx cerebri, which folds between the two hemispheres; the tentorium, which folds between the occipital lobe and cerebellum to form a tough, membranous shelf; and the falx cerebelli, which is located between the right and left side of the cerebellum. When excess pressure occurs in the cranial cavity, brain tissue may be compressed against these dural folds or displaced around them, a process called *herniation*. A potential space exists between the dura and the skull, and between the periosteum and the dura in the vertebral column, known as the epidural space. Another potential space, the subdural space, also

exists below the dura. Blood or an abscess can accumulate in these potential spaces.



**Figure 60-5 •** Meninges and related structures.

- *Arachnoid*—the middle membrane; an extremely thin, delicate membrane that closely resembles a spider web (hence the name *arachnoid*). The arachnoid membrane has cerebrospinal fluid (CSF) in the space below it, known as the subarachnoid space. This membrane has arachnoid villi, which are unique finger-like projections that absorb CSF into the venous system. When blood or bacteria enter the subarachnoid space, the villi become obstructed and *communicating hydrocephalus* (increased size of ventricles) may result.
- *Pia mater*—the innermost, thin, transparent layer that hugs the brain closely and extends into every fold of the brain's surface.

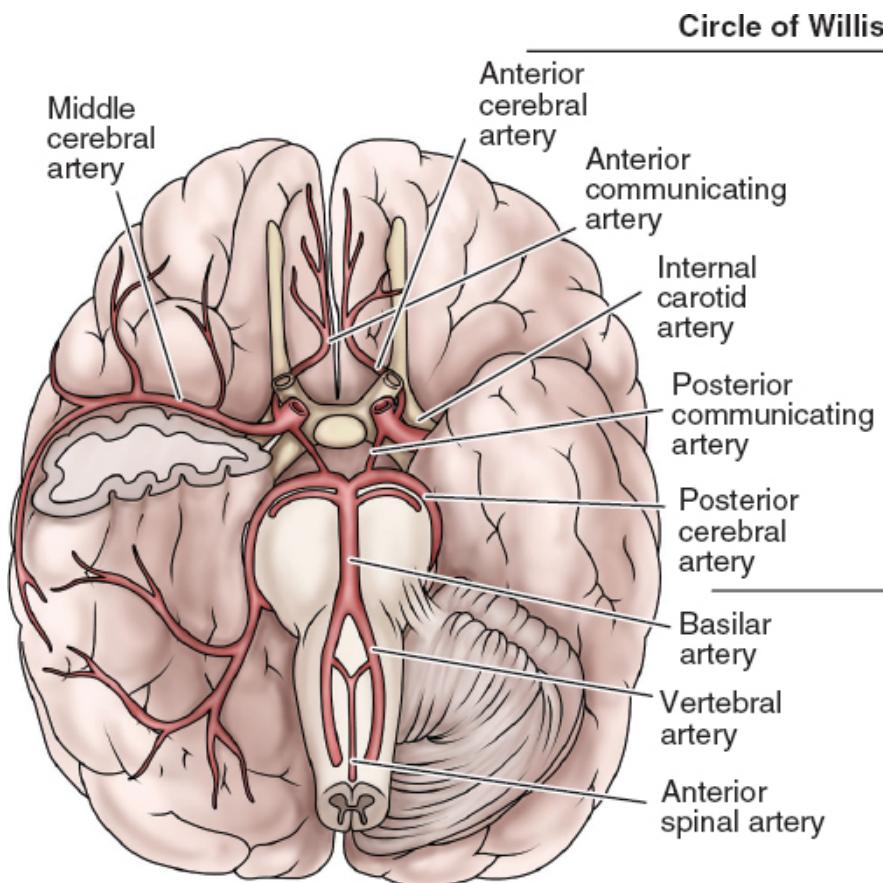
## Cerebrospinal Fluid

CSF is a clear and colorless fluid that is produced in the choroid plexus of the ventricles and circulates around the surface of the brain and the spinal cord. There are four ventricles: the right and left lateral and the third and fourth ventricles. The two lateral ventricles open into the third ventricle at the interventricular foramen (also known as the foramen of Monro). The third and fourth ventricles connect via the aqueduct of Sylvius. The fourth ventricle drains CSF into the subarachnoid space on the surface of the brain and spinal cord, where it is absorbed by the arachnoid villi. Blockage of the flow of CSF anywhere in the ventricular system produces *obstructive* hydrocephalus.

CSF is important in immune and metabolic functions in the brain. It is produced at a rate of about 500 mL/day; the ventricles and subarachnoid space contain approximately 125 to 150 mL of fluid (Hickey & Strayer, 2020). The composition of CSF is similar to other extracellular fluids (such as blood plasma), but the concentrations of the various constituents differ. A laboratory analysis of CSF indicates color (clear), specific gravity (normal 1.007), protein count, cell count, glucose, and other electrolyte levels (see Table A-5 in Appendix A on [thePoint](#)). Normal CSF contains a minimal number of white blood cells and no red blood cells. The CSF may also be tested for immunoglobulins or the presence of bacteria. A CSF sample may be obtained through a lumbar puncture or intraventricular catheter (Hickey & Strayer, 2020).

## Cerebral Circulation

The brain does not store nutrients and requires a constant supply of oxygen. These needs are met through cerebral circulation; the brain receives approximately 15% of the cardiac output, or 750 mL per minute of blood flow. Brain circulation is unique in several aspects. First, arterial and venous vessels are not parallel as in other organs in the body; this is due in part to the role the venous system plays in CSF absorption. Second, the brain has collateral circulation through the circle of Willis (see later discussion), allowing blood flow to be redirected on demand. Third, blood vessels in the brain have two rather than three layers, which may make them more prone to rupture when weakened or under pressure.



**Figure 60-6 •** Arterial blood supply of the brain, including the circle of Willis, as viewed from the ventral surface.

## Arteries

Arterial blood supply to the anterior brain originates from the common carotid artery, which is the first bifurcation of the aorta. The internal carotid arteries arise at the bifurcation of the common carotid. Branches of the internal carotid arteries (the anterior and middle cerebral arteries) and their connections (the anterior and posterior communicating arteries) form the circle of Willis (see Fig. 60-6).

The vertebral arteries branch from the subclavian arteries to supply most of the posterior circulation of the brain. At the level of the brain stem, the vertebral arteries join to form the basilar artery. The basilar artery divides to form the two branches of the posterior cerebral arteries. Functionally, the posterior and anterior portions of the circulation usually remain separate. However, the circle of Willis can provide collateral circulation through communicating arteries if one of the vessels supplying it becomes occluded or is ligated.

The bifurcations along the circle of Willis are frequent sites of aneurysm formation. Aneurysms are outpouchings of the blood vessel due to vessel wall

weakness. Aneurysms can rupture and cause a hemorrhagic stroke. See [Chapter 62](#) for a more detailed discussion of aneurysms.

## Veins

Venous drainage for the brain does not follow the arterial circulation as in other body structures. The veins reach the brain's surface, join larger veins, and then cross the subarachnoid space and empty into the dural sinuses, which are the vascular channels embedded in the dura (see [Fig. 60-5](#)). The network of the sinuses carries venous outflow from the brain and empties into the internal jugular veins, returning the blood to the heart. Cerebral veins are unique, because unlike other veins in the body, they do not have valves to prevent blood from flowing backward and depend on both gravity and blood pressure for flow.

## Blood–Brain Barrier

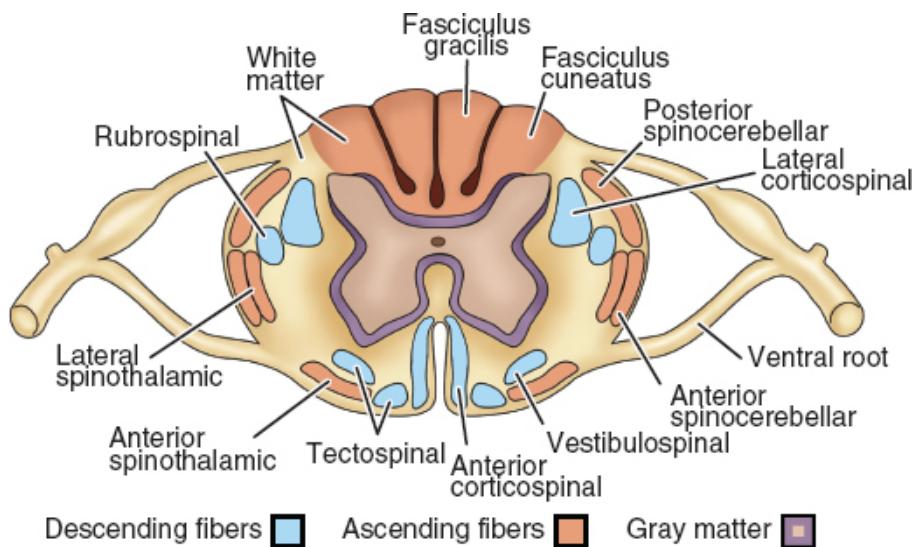
The CNS is inaccessible to many substances that circulate in the blood plasma (e.g., dyes, medications, antibiotic agents) because of the blood–brain barrier. This barrier is formed by the endothelial cells of the brain's capillaries, which form continuous tight junctions, creating a barrier to macromolecules and many compounds. All substances entering the CSF must filter through the capillary endothelial cells and astrocytes. The blood–brain barrier has a protective function but can be altered by trauma, cerebral edema, and cerebral hypoxemia; this has implications for treatment and selection of medications for CNS disorders (Hickey & Strayer, 2020).

## The Spinal Cord

The spinal cord is continuous with the medulla, extending from the cerebral hemispheres and serving as the connection between the brain and the periphery. Approximately 45 cm (18 inches) long and about the thickness of a finger, it extends from the foramen magnum at the base of the skull to the lower border of the first lumbar vertebra, where it tapers to a fibrous band called the *conus medullaris*. Continuing below the second lumbar space are the nerve roots that extend beyond the conus, which are called the *cauda equina* because they resemble a horse's tail. Meninges surround the spinal cord.

In a cross-sectional view, the spinal cord has an H-shaped central core of nerve cell bodies (gray matter) surrounded by ascending and descending tracts (white matter) (see [Fig. 60-7](#)). The lower portion of the H is broader than the upper portion and corresponds to the anterior horns. The anterior horns contain cells with fibers that form the anterior (motor) root and are essential for the voluntary and reflex activity of the muscles they innervate. The thinner posterior (upper horns) portion contains cells with fibers that enter over the

posterior (sensory) root and thus serve as a relay station in the sensory/reflex pathway.



**Figure 60-7 •** Cross-sectional diagram of the spinal cord showing major spinal tracts.

The thoracic region of the spinal cord has a projection from each side at the crossbar of the H-shaped structure of gray matter called the *lateral horn*. It contains the cells that give rise to the autonomic fibers of the sympathetic division. The fibers leave the spinal cord through the anterior roots in the thoracic and upper lumbar segments.

### The Spinal Tracts

The white matter of the spinal cord is composed of myelinated and unmyelinated nerve fibers. The fast-conducting myelinated fibers form bundles; fiber bundles with a common function are called *tracts*.

There are six ascending tracts (see Fig. 60-7). Two tracts, known as the fasciculus cuneatus and gracilis or the posterior columns, conduct sensations of deep touch, pressure, vibration, position, and passive motion from the same side of the body. Before reaching the cerebral cortex, these fibers cross to the opposite side in the medulla. The anterior and posterior spinocerebellar tracts conduct sensory impulses from muscle spindles, providing necessary input for coordinated muscle contraction. They ascend uncrossed and terminate in the cerebellum. The anterior and lateral spinothalamic tracts are responsible for conduction of pain, temperature, proprioception, fine touch, and vibratory sense from the upper body to the brain. They cross to the opposite side of the cord and then ascend to the brain, terminating in the thalamus (Klein & Stewart-Amidei, 2017).

There are eight descending tracts (see Fig. 60-7). The anterior and lateral corticospinal tracts conduct motor impulses to the anterior horn cells from the opposite side of the brain, cross in the medulla, and control voluntary muscle activity. The three vestibulospinal tracts descend uncrossed and are involved in some autonomic functions (sweating, pupil dilation, and circulation) and involuntary muscle control. The corticobulbar tract conducts impulses responsible for voluntary head and facial muscle movement and crosses at the level of the brain stem. The rubrospinal and reticulospinal tracts conduct impulses involved with involuntary muscle movement.

### Vertebral Column

The bones of the vertebral column surround and protect the spinal cord and normally consist of 7 cervical, 12 thoracic, and 5 lumbar vertebrae, as well as the sacrum (a fused mass of 5 vertebrae) and terminate in the coccyx. Nerve roots exit from the vertebral column through the intervertebral foramina (openings). The vertebrae are separated by discs, except for the first and second cervical, the sacral, and the coccygeal vertebrae. Each vertebra has a ventral solid body and a dorsal segment or arch, which is posterior to the body. The arch is composed of two pedicles and two laminae supporting seven processes. The vertebral body, arch, pedicles, and laminae all encase and protect the spinal cord.

## The Peripheral Nervous System

The peripheral nervous system includes the cranial nerves, the spinal nerves, and the autonomic nervous system.

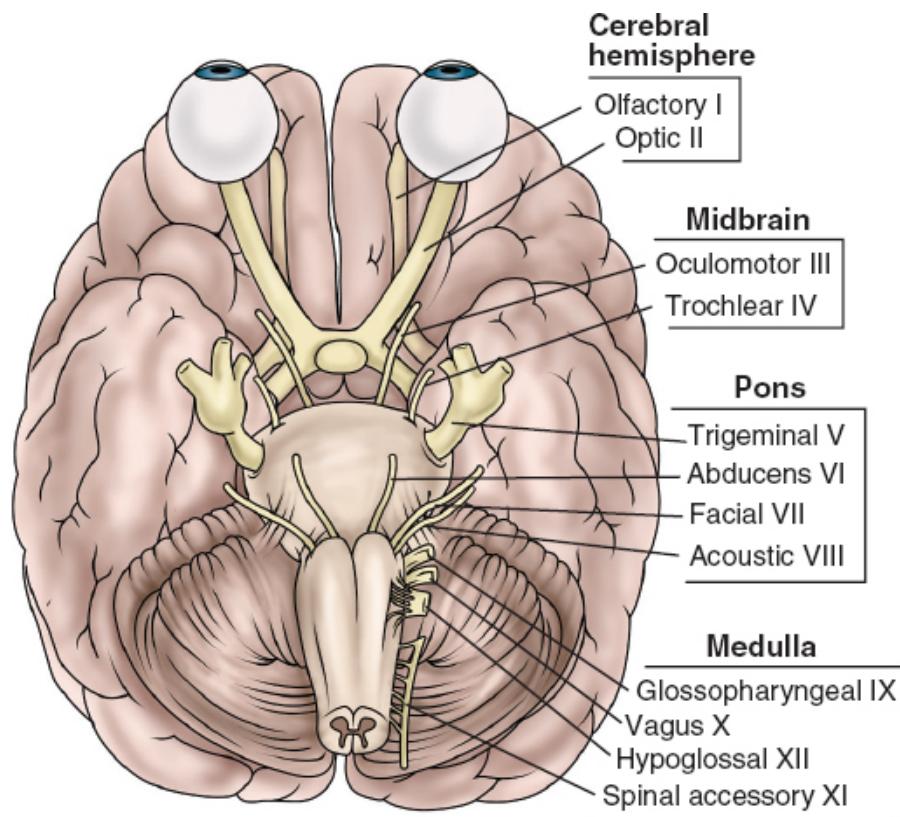
### Cranial Nerves

Twelve pairs of cranial nerves emerge from the lower surface of the brain and pass through openings in the base of the skull. Three cranial nerves are entirely sensory (I, II, VIII), five are motor (III, IV, VI, XI, and XII), and four are mixed sensory and motor (V, VII, IX, and X). The cranial nerves are numbered in the order in which they arise from the brain (see Fig. 60-8). The cranial nerves innervate the head, neck, and special sense structures. Table 60-2 provides a summary of the cranial nerves.

### Spinal Nerves

The spinal cord is composed of 31 pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal. Each spinal nerve has a ventral root and a dorsal root. The dorsal roots are sensory and transmit sensory impulses from specific areas of the body known as dermatomes (see Fig. 60-9) to the dorsal horn ganglia. The sensory fiber may be somatic, carrying

information about pain, temperature, touch, and position sense (proprioception) from the tendons, joints, and body surfaces; or visceral, carrying information from the internal organs.



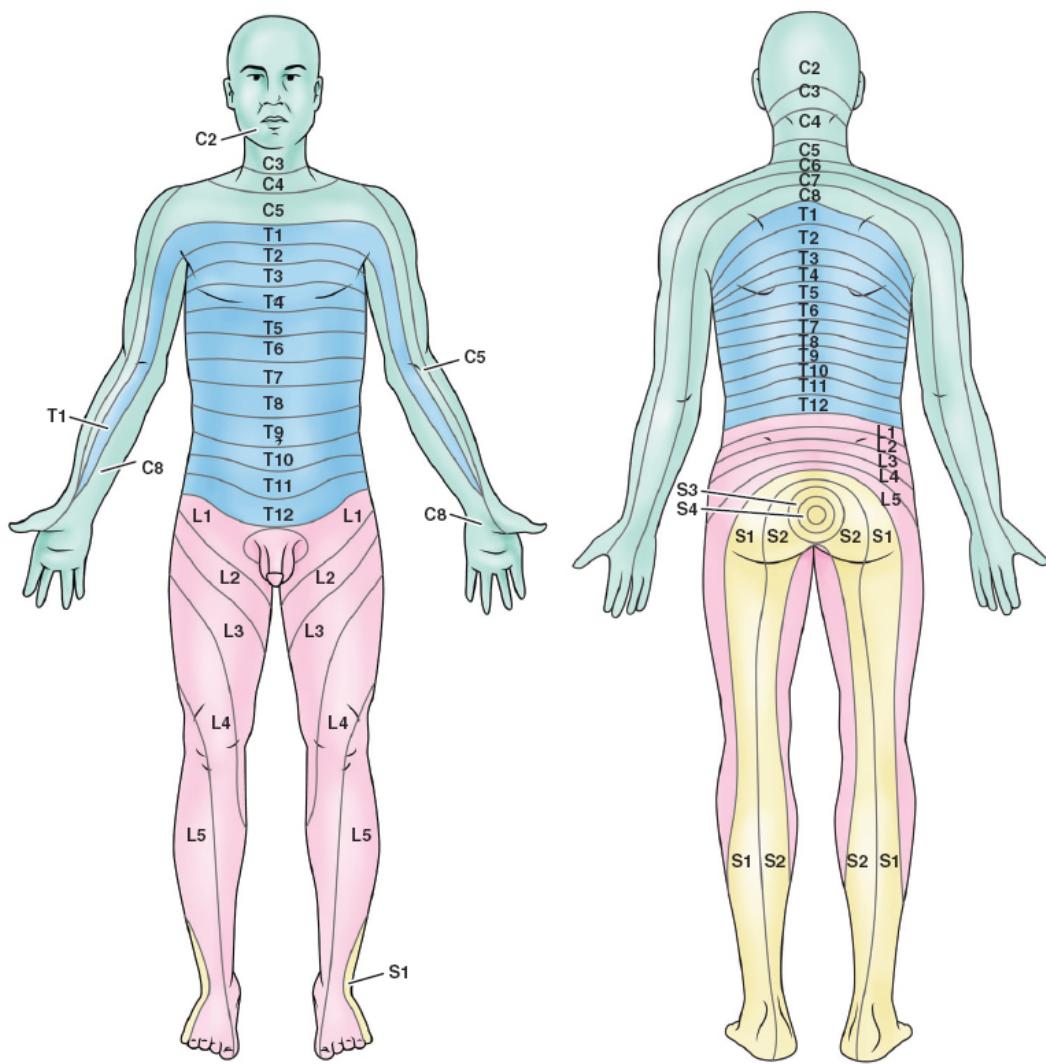
**Figure 60-8 •** Diagram of the base of the brain showing location of the cranial nerves.

**TABLE 60-2** Summary of Cranial Nerves

Nerve (Number)	Type	Functions	Methods for Examining Nerve
Olfactory (I)	Sensory	Sense of smell	Test each nostril for smell reception with various agents and interpretation
Optic (II)	Sensory	Sense of vision	Test vision for acuity and visual fields
Oculomotor (III)	Motor	Pupil constriction	
	Raise eyelids	Test pupillary reaction to light and ability to open and close eyelids	
Trochlear (IV)	Motor/proprioceptor	Downward, inward eye movement	Test for downward and inward movement of the eye
Trigeminal (V)	Motor	Jaw movements —chewing and mastication	Ask patient to open and clench jaws while you palpate the jaw muscles
	Sensory	Sensation on the face and neck	Test face and neck for pain sensations, light touch, and temperature
Abducens (VI)	Motor	Lateral movement of the eyes	Test ocular movement in all directions
Facial (VII)	Motor	Muscles of the face	Ask the patient to raise eyebrows, smile, show teeth, and puff out cheeks
	Sensory	Sense of taste on the anterior two thirds of the tongue	Test for the taste sensation with various agents
Acoustic (VIII)	Sensory	Sense of hearing	Test hearing ability
Glossopharyngeal (IX)	Motor	Pharyngeal movement and swallowing	Ask the patient to say “ah,” and have patient yawn to observe upward movement of the soft palate; elicit gag

			response; note ability to swallow
	Sensory	Sense of taste on the posterior one third of the tongue	Test for taste with various agents
Vagus (X)	Motor/sensory	Swallowing and speaking	Ask the patient to swallow and speak; note hoarseness
Accessory (XI)	Motor/sensory	Movement of shoulder muscles	Ask the patient to shrug shoulders against your resistance
Hypoglossal (XII)	Motor	Movement of the tongue; strength of the tongue	Ask the patient to protrude tongue; ask patient to push tongue against cheek

Reprinted with permission from Taylor, C., Lynn, P., & Bartlett, J. L. (2019). *Fundamentals of nursing: The art and science of person-centered care* (9th ed., Table 26-6). Philadelphia, PA: Wolters Kluwer.



**Figure 60-9 •** Dermatome distribution.

The ventral roots are motor and transmit impulses from the spinal cord to the body; these fibers are also either somatic or visceral. The visceral fibers include autonomic fibers that control the cardiac muscles and glandular secretions.

### Autonomic Nervous System

The **autonomic nervous system** regulates the activities of internal organs such as the heart, lungs, blood vessels, digestive organs, and glands (see Fig. 60-10). Maintenance and restoration of internal homeostasis is largely the responsibility of the autonomic nervous system. There are two major divisions: the **sympathetic nervous system**, with predominantly excitatory responses (i.e., the “fight-or-flight” response), and the parasympathetic nervous system, which controls mostly visceral functions.

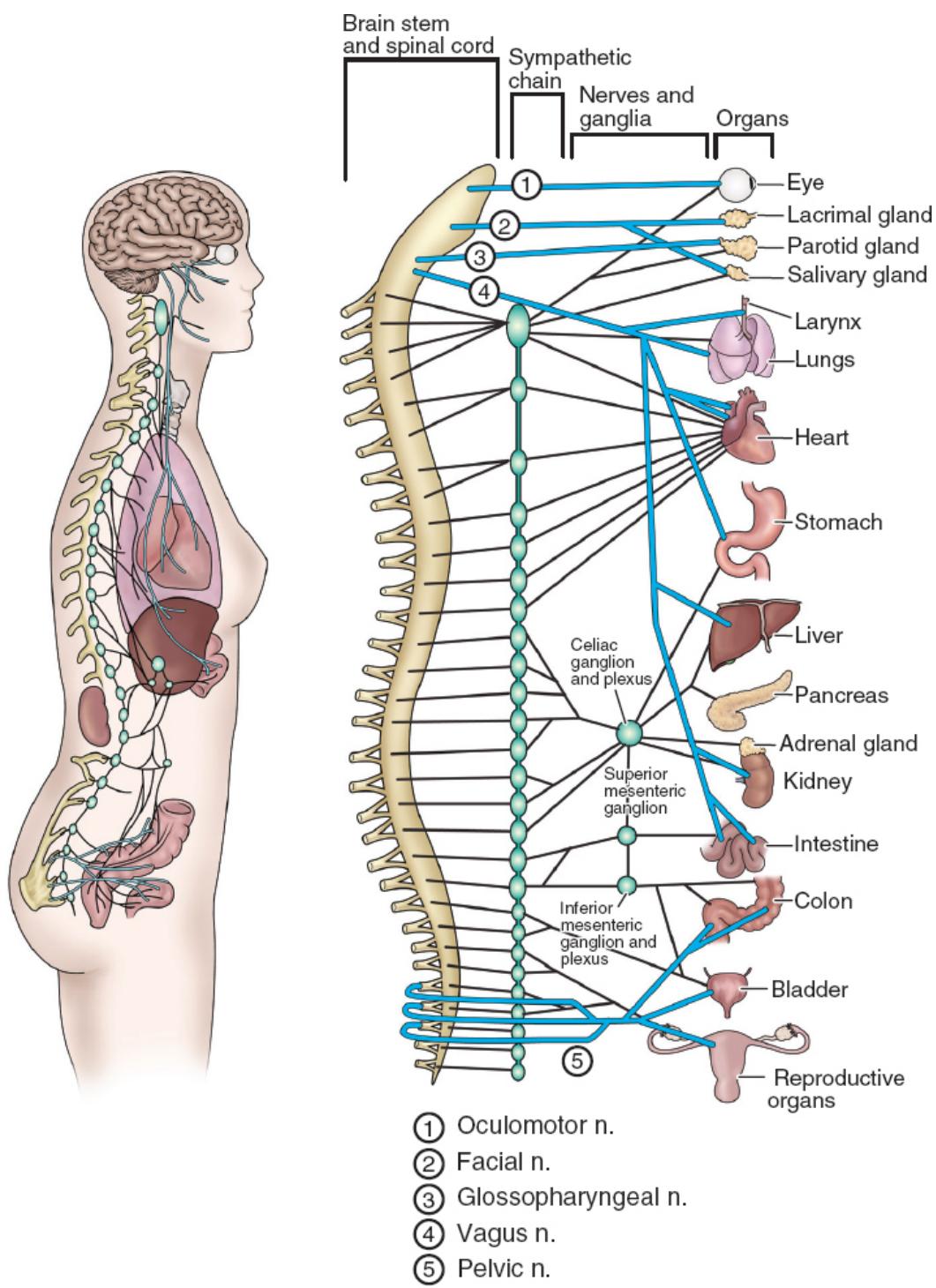
The autonomic nervous system innervates most body organs. Although usually considered part of the peripheral nervous system, this system is regulated by centers in the spinal cord, brain stem, and hypothalamus.

The hypothalamus is the major subcortical center for the regulation of autonomic activities, serving an inhibitory–excitatory role. The hypothalamus has connections that link the autonomic system with the thalamus, the cortex, the olfactory apparatus, and the pituitary gland. Located here are the mechanisms for the control of visceral and somatic reactions that were originally important for defense or attack and are associated with emotional states (e.g., fear, anger, anxiety); for the control of metabolic processes, including fat, carbohydrate, and water metabolism; for the regulation of body temperature, arterial pressure, and all muscular and glandular activities of the gastrointestinal tract; for control of genital functions; and for the sleep cycle.

The autonomic nervous system is separated into the anatomically and functionally distinct sympathetic and parasympathetic divisions. Most of the tissues and the organs under autonomic control are innervated by both systems. For example, the parasympathetic division causes contraction (stimulation) of the urinary bladder muscles and a decrease (inhibition) in heart rate, whereas the sympathetic division produces relaxation (inhibition) of the urinary bladder and an increase (stimulation) in the rate and force of the heartbeat. [Table 60-3](#) compares the sympathetic and the parasympathetic effects on the different systems of the body.

### Sympathetic Nervous System

The sympathetic division of the autonomic nervous system is best known for its role in the body's fight-or-flight response. Under stress from either physical or emotional causes, sympathetic impulses increase greatly. As a result, the bronchioles dilate for easier gas exchange; the heart's contractions are stronger and faster; the arteries to the heart and voluntary muscles dilate, carrying more blood to these organs; peripheral blood vessels constrict, making the skin feel cool but shunting blood to essential organs; the pupils dilate; the liver releases glucose for quick energy; peristalsis slows; hair stands on end; and perspiration increases. The main sympathetic neurotransmitter is norepinephrine (noradrenaline). A sympathetic discharge releases epinephrine (adrenalin)—hence, the term *adrenergic* is often used to refer to this division.



**Figure 60-10 • Anatomy of the autonomic nervous system.**

Sympathetic neurons are located primarily in the thoracic and lumbar segments of the spinal cord, and their axons, or the preganglionic fibers, emerge by way of anterior nerve roots from the eighth cervical or first thoracic segment to the second or third lumbar segment. A short distance from the cord,

these fibers diverge to join a chain, composed of 22 linked ganglia, that extends the entire length of the spinal column, adjacent to the vertebral bodies on both sides. Some form multiple synapses with nerve cells within the chain. Others traverse the chain without making connections or losing continuity to join large “prevertebral” ganglia in the thorax, the abdomen, or the pelvis or one of the “terminal” ganglia in the vicinity of an organ, such as the bladder or the rectum at the end of the colon (see Fig. 60-10). Postganglionic nerve fibers originating in the sympathetic chain rejoin the spinal nerves that supply the extremities and are distributed to blood vessels, sweat glands, and smooth muscle tissue in the skin. Postganglionic fibers from the prevertebral plexuses (e.g., the cardiac, pulmonary, splanchnic, pelvic plexuses) supply structures in the head and neck, thorax, abdomen, and pelvis, respectively, having been joined in these plexuses by fibers from the parasympathetic division.

**TABLE 60-3**

Effects of the Autonomic Nervous System

Structure or Activity	Parasympathetic Effects	Sympathetic Effects
<b>Pupil of the Eye</b>	Constricted	Dilated
<b>Circulatory System</b>		
Rate and force of heartbeat	Decreased	Increased
Blood vessels		
In heart muscle	Constricted	Dilated
In skeletal muscle	<i>a</i>	Dilated
In abdominal viscera and the skin	<i>a</i>	Constricted
Blood pressure	Decreased	Increased
<b>Respiratory System</b>		
Bronchioles	Constricted	Dilated
Rate of breathing	Decreased	Increased
<b>Gastrointestinal System</b>		
Peristaltic movements of digestive tube	Increased	Decreased
Muscular sphincters of digestive tube	Relaxed	Contracted
Secretion of salivary glands	Thin, watery saliva	Thick, viscid saliva
Secretions of stomach, intestine, and pancreas	Increased	<i>a</i>
Conversion of liver glycogen to glucose	<i>a</i>	Increased
<b>Genitourinary System</b>		
Urinary bladder		
Muscle walls	Contracted	Relaxed
Sphincters	Relaxed	Contracted
Muscles of the uterus	Relaxed; variable	Contracted under some conditions; varies with menstrual cycle and pregnancy
Blood vessels of external genitalia	Dilated	<i>a</i>
<b>Integumentary System</b>		
Secretion of sweat	<i>a</i>	Increased
Pilomotor muscles	<i>a</i>	Contracted (goose flesh)
<b>Adrenal Medulla</b>	<i>a</i>	Secretion of epinephrine and norepinephrine

<sup>a</sup>No direct effect.

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological and neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.

The adrenal glands, kidneys, liver, spleen, stomach, and duodenum are under the control of the giant celiac plexus, commonly known as the solar plexus. This receives its sympathetic nerve components by way of the three splanchnic nerves, composed of preganglionic fibers from nine segments of the spinal cord (T4 to L1), and is joined by the vagus nerve, representing the parasympathetic division. From the celiac plexus, fibers of both divisions travel along the course of blood vessels to their target organs.

Certain syndromes are distinctive to the sympathetic nervous system. For example, sympathetic storm is a syndrome associated with changes in level of consciousness, altered vital signs, diaphoresis, and agitation that may result from hypothalamic stimulation of the sympathetic nervous system following traumatic brain injury (Fischbach & Fischbach, 2018).

## Parasympathetic Nervous System

The **parasympathetic nervous system** functions as the dominant controller for most visceral functions; the primary neurotransmitter is acetylcholine. During quiet, nonstressful conditions, impulses from parasympathetic fibers (cholinergic) predominate. The fibers of the parasympathetic system are located in two sections: one in the brain stem and the other from spinal segments below L2. Because of the location of these fibers, the parasympathetic system is referred to as the craniosacral division, as distinct from the thoracolumbar (sympathetic) division of the autonomic nervous system.

The parasympathetic nerves arise from the midbrain and the medulla oblongata. Fibers from cells in the midbrain travel with the third oculomotor nerve to the ciliary ganglia, where postganglionic fibers of this division are joined by those of the sympathetic system, creating controlled opposition, with a delicate balance always maintained between the two systems.

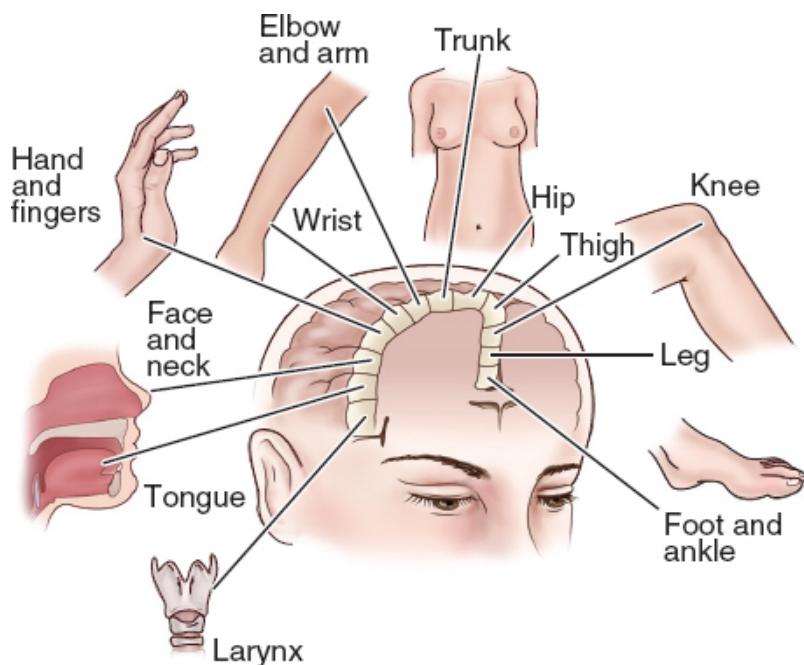
## Motor and Sensory Pathways of the Nervous System



Motor pathways within the CNS are responsible for voluntary, involuntary, and coordination of movement. Sensory pathways receive, integrate, and transmit a wide variety of sensations within the CNS.

### Motor Pathways

The corticospinal tract begins in the motor cortex, a vertical band within each frontal lobe, and controls voluntary movements of the body. The exact locations within the brain at which the voluntary movements of the muscles of the face, thumb, hand, arm, trunk, and leg originate are known (see Fig. 60-11). To initiate movement, these particular cells must send the stimulus along their fibers. Stimulation of these cells with an electric current also results in muscle contraction. En route to the pons, the motor fibers converge into a tight bundle known as the internal capsule. A comparatively small injury to the internal capsule results in a more severe paralysis than does a larger injury to the cortex itself.



**Figure 60-11 •** Diagrammatic representation of the cerebrum showing locations for control of motor movement of various parts of the body.

At the medulla, the corticospinal tracts cross to the opposite side, continuing to the anterior horn of the spinal cord, in proximity to a motor nerve cell. Until this point, neurons are known as upper motor neurons. As they connect to motor fibers of the spinal nerves, they become lower motor neurons. The lower motor neurons receive the impulse in the posterior part of the cord and run to the myoneural junction located in the peripheral muscle.

Involuntary motor activity is also possible and is mediated through reflex arcs. Synaptic connections between anterior horn cells and sensory fibers that have entered adjacent or neighboring segments of the spinal cord serve as protective mechanisms. These connections are seen during deep tendon reflex testing.

## Upper and Lower Motor Neurons

The voluntary motor system consists of two groups of neurons: upper motor neurons and lower motor neurons. Upper motor neurons originate in the cerebral cortex, the cerebellum, and the brain stem. Their fibers make up the descending motor pathways, are located entirely within the CNS, and modulate the activity of the lower motor neurons. Lower motor neurons are located either in the anterior horn of the spinal cord gray matter or within cranial nerve nuclei in the brain stem. Axons of lower motor neurons in both sites extend through peripheral nerves and terminate in skeletal muscle. Lower motor neurons are located in both the CNS and the peripheral nervous system.

The motor pathways from the brain to the spinal cord, as well as from the cerebrum to the brain stem, are formed by upper motor neurons. They begin in the cortex of one side of the brain, descend through the internal capsule, cross to the opposite side in the brain stem, descend through the corticospinal tract, and synapse with the lower motor neurons in the cord. The lower motor neurons receive the impulse in the posterior part of the cord and run to the myoneural junction located in the peripheral muscle. The clinical features of lesions of upper and lower motor neurons are discussed in the following sections and in [Table 60-4](#).

**TABLE 60-4** Comparison of Upper Motor Neuron and Lower Motor Neuron Lesions

Upper Motor Neuron Lesions	Lower Motor Neuron Lesions
Loss of voluntary control	Loss of voluntary control
Increased muscle tone	Decreased muscle tone
Muscle spasticity	Flaccid muscle paralysis
No muscle atrophy	Muscle atrophy
Hyperactive and abnormal reflexes	Absent or decreased reflexes

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological and neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.

## Upper Motor Neuron Lesions

Upper motor neuron lesions can involve the motor cortex, the internal capsule, the spinal cord gray matter, and other structures of the brain through which the corticospinal tract descends. If the upper motor neurons are damaged or destroyed, as frequently occur with stroke or spinal cord injury, paralysis (loss of voluntary movement) results. However, because the inhibitory influences of intact upper motor neurons are impaired, **reflex** (involuntary) movements are uninhibited, and hence hyperactive deep tendon reflexes, diminished or absent superficial reflexes, and pathologic reflexes such as a Babinski response occur. Severe leg spasms can occur as the result of an upper motor neuron lesion; the spasms result from the preserved reflex arc, which lacks inhibition along the

spinal cord below the level of injury. There is little or no muscle atrophy, and muscles remain permanently tense, exhibiting spastic paralysis.

Paralysis associated with upper motor neuron lesions can affect a whole extremity, both extremities, or an entire half of the body. *Hemiplegia* (paralysis of an arm and leg on the same side of the body) can be the result of an upper motor neuron lesion. If hemorrhage, an embolus, or a thrombus destroys the fibers from the motor area in the internal capsule, the arm and the leg of the opposite side become stiff, weak, or paralyzed, and the reflexes are hyperactive. If both legs are paralyzed, the condition is called *paraplegia*. If all four extremities are paralyzed, the condition is called *tetraplegia* (quadriplegia). See [Chapter 63](#) for additional discussion of these disorders.

### **Lower Motor Neuron Lesions**

A patient is considered to have lower motor neuron damage if a motor nerve is damaged between the spinal cord and muscle. The result of lower motor neuron damage is muscle paralysis. Reflexes are lost, and the muscle becomes flaccid (limp) and atrophied from disuse. If the patient has injured the spinal trunk and it can heal, the use of muscles connected to that section of the spinal cord may be regained. However, if the anterior horn motor cells are destroyed, the nerves cannot regenerate, and the muscles are never useful again.

Flaccid paralysis and atrophy of the affected muscles are the principal signs of lower motor neuron disease. Lower motor neuron lesions can be the result of trauma, infection (poliomyelitis), toxins, vascular disorders, congenital malformations, degenerative processes, and neoplasms. Compression of nerve roots by herniated intervertebral discs is a common cause of lower motor neuron dysfunction.

### **Coordination of Movement**

The motor system is complex, and motor function depends not only on the integrity of the corticospinal tracts but also on other pathways from the basal ganglia and cerebellum that control and coordinate voluntary motor function. The smoothness, accuracy, and strength that characterize the muscular movements of a normal person are attributable to the influence of the cerebellum and the basal ganglia.

Through the action of the cerebellum, the contractions of opposing muscle groups are adjusted in relation to each other to maximal mechanical advantage; muscle contractions can be sustained evenly at the desired tension and without significant fluctuation, and reciprocal movements can be reproduced at high and constant speed, in stereotyped fashion and with relatively little effort.

The basal ganglia play an important role in planning and coordinating motor movements and posture. Complex neural connections link the basal ganglia with the cerebral cortex. The major effect of these structures is to inhibit unwanted muscular activity.

Impaired cerebellar function, which may occur as a result of an intracranial injury or some type of an expanding mass (e.g., a hemorrhage, an abscess, or a tumor), results in loss of muscle tone, weakness, and fatigue. Depending on the area of the brain affected, the patient has different motor symptoms or responses. The patient may demonstrate abnormal flexion, abnormal extension, or flaccid posturing. **Flaccidity** (lack of muscle tone) preceded by abnormal posturing in a patient with cerebral injury indicates severe neurologic impairment, which may herald brain death (Klein & Stewart-Amidei, 2017; Posner, Saper, Schiff, et al., 2019). See [Chapter 61](#), Figure 61-1 for further explanation of posturing.

Destruction or dysfunction of the basal ganglia leads not to paralysis but to muscle rigidity, disturbances of posture, and difficulty initiating or changing movement. The patient tends to have involuntary movements. These may take the form of coarse tremors, most often in the upper extremities, particularly in the distal portions; athetosis, which is movement of a slow, squirming, writhing, twisting type; or chorea, marked by spasmodic, purposeless, irregular, uncoordinated motions of the trunk and the extremities, and facial grimacing. Disorders affecting basal ganglia activity include Parkinson's and Huntington diseases (see [Chapter 65](#)).

## Sensory System Function

### Receiving Sensory Impulses

Afferent impulses travel from their points of origin to their destinations in the cerebral cortex via the ascending pathways directly, or they may cross at the level of the spinal cord or in the medulla, depending on the type of sensation carried. Knowledge of these pathways is important for neurologic assessment and for understanding symptoms and their relationship to various lesions.

Sensory impulses convey sensations of heat, cold, and pain; position; and vibration. The axons enter the spinal cord by way of the posterior root, specifically in the posterior gray columns of the spinal cord, where they connect with the cells of secondary neurons. Pain and temperature fibers (located in the spinothalamic tract) cross immediately to the opposite side of the cord and course upward to the thalamus. Fibers carrying sensations of touch, light pressure, and localization do not connect immediately with the second neuron but ascend the cord for a variable distance before entering the gray matter and completing this connection. The axon of the secondary neuron traverses the cord, crosses in the medulla, and proceeds upward to the thalamus.

Position and vibratory sensations are produced by stimuli arising from muscles, joints, and bones. These stimuli are conveyed, uncrossed, all the way to the brain stem by the axon of the primary neuron. In the medulla, synaptic

connections are made with cells of the secondary neurons, whose axons cross to the opposite side and then proceed to the thalamus.

### Integrating Sensory Impulses

The thalamus integrates all sensory impulses except olfaction. It plays a role in the conscious awareness of pain and the recognition of variation in temperature and touch. The thalamus is responsible for the sense of movement and position as well as the ability to recognize the size, shape, and quality of objects. Sensory information is relayed from the thalamus to the parietal lobe for interpretation.

### Sensory Losses

Destruction of a sensory nerve results in total loss of sensation in its area of distribution (see Fig. 60-9). Lesions affecting the posterior spinal nerve roots may impair tactile sensation, causing intermittent severe pain that is referred to their areas of distribution. Destruction of the spinal cord yields complete anesthesia below the level of injury. Selective destruction or degeneration of the posterior columns of the spinal cord is responsible for a loss of position and vibratory sense in segments distal to the lesion, without loss of touch, pain, or temperature perception. A cyst in the center of the spinal cord causes dissociation of sensation—loss of pain at the level of the lesion. This occurs because the fibers carrying pain and temperature cross within the cord immediately on entering; thus, any lesion that divides the cord longitudinally divides these fibers. Other sensory fibers ascend the cord for variable distances, some even to the medulla, before crossing, thereby bypassing the lesion and avoiding destruction. Lesions in the thalamus or parietal lobe result in impaired touch, pain, temperature, and proprioceptive sensations.

## Assessment of the Nervous System

An assessment of the nervous system involves conducting a health history and physical assessment.

### Health History

An important aspect of the neurologic assessment is the history of the present illness. The initial interview provides an excellent opportunity to systematically explore the patient's current condition and related events while simultaneously observing overall appearance, mental status, posture, movement, and affect. Depending on the patient's condition, the nurse may need to rely on yes-or-no answers to questions, a review of the medical record, input from witnesses or the family, or a combination of these.

Neurologic disorders may be stable or progressive, characterized by symptom-free periods as well as fluctuations in symptoms. The health history therefore includes details about the onset, character, severity, location, duration, and frequency of symptoms and signs; associated complaints; precipitating, aggravating, and relieving factors; progression, remission, and exacerbation; and the presence or absence of similar symptoms among family members.

## Common Symptoms

The symptoms of neurologic disorders are as varied as the disease processes. Symptoms may be subtle or intense, fluctuating or permanent, inconvenient or devastating. This chapter discusses the most common signs and symptoms associated with neurologic disease; the relationship of specific signs and symptoms to a disorder is presented in later chapters in this unit.

### Pain

Pain is considered an unpleasant sensory perception and emotional experience associated with actual or potential tissue damage or described in terms of such damage. Pain is therefore considered multidimensional and entirely subjective. Pain can be acute or chronic. In general, acute pain lasts for a relatively short period of time and remits as the pathology resolves. In neurologic disease, acute pain may be associated with brain hemorrhage, spinal disc disease (Jarvis, 2020), or trigeminal neuralgia. In contrast, chronic or persistent pain extends for long periods of time and may represent a broader pathology. This type of pain can occur with many degenerative and chronic neurologic conditions (e.g., multiple sclerosis). See [Chapter 9](#) for a more detailed discussion of pain.

### Seizures

Seizures are the result of abnormal electrical discharges in the cerebral cortex, which then manifest as an alteration in sensation, behavior, movement, perception, or consciousness. The alteration may be short, such as in a blank stare that lasts only a second, or of longer duration, such as a tonic-clonic grand mal seizure that can last several minutes. The seizure activity reflects the area of the brain affected. Seizures can occur as isolated events, such as when induced by a high fever, alcohol or drug withdrawal, or hypoglycemia. A seizure may also be the first obvious sign of a brain lesion (Hickey & Strayer, 2020).

### Dizziness and Vertigo

Dizziness is an abnormal sensation of imbalance or movement. It is common in the older adult and a common complaint encountered by health

professionals (Jarvis, 2020). Dizziness can have a variety of causes, including viral syndromes, hot weather, roller-coaster rides, and middle ear infections, to name a few. One difficulty confronting health care providers when assessing dizziness is the vague and varied terms that patients use to describe the sensation.

About 50% of all patients with dizziness have **vertigo**, or the illusion of movement in which the individual or the surroundings are sensed as moving, usually as rotation (Jarvis, 2020). Vertigo is usually a manifestation of vestibular dysfunction. It can be so severe as to result in spatial disorientation, lightheadedness, loss of equilibrium (staggering), and nausea and vomiting.

### Visual Disturbances

Visual defects that cause people to seek health care can range from the decreased visual acuity associated with aging to sudden blindness caused by glaucoma. Normal vision depends on functioning visual pathways through the retina and optic chiasm and the radiations into the visual cortex in the occipital lobes. Lesions of the eye itself (e.g., cataract), lesions along the pathway (e.g., tumor), or lesions in the visual cortex (e.g., stroke) interfere with normal visual acuity. Abnormalities of eye movement (as in the nystagmus associated with multiple sclerosis) can also compromise vision by causing diplopia or double vision. See [Chapter 58](#) for a more detailed discussion of disorders that affect vision.

### Muscle Weakness

Muscle weakness is a common manifestation of neurologic disease. It frequently coexists with other symptoms of disease and can affect a variety of muscles, causing a wide range of disability. Weakness can be sudden and permanent, as in stroke, or progressive, as in neuromuscular diseases such as amyotrophic lateral sclerosis. Any muscle group can be affected.

### Abnormal Sensation

Abnormal sensation is a neurologic manifestation of both central and peripheral nervous system disease. Altered sensation can affect small or large areas of the body. It is frequently associated with weakness or pain and is potentially disabling. Lack of sensation places a person at risk for falls and injury.

### Past Health, Family, and Social History

The nurse may inquire about any family history of genetic diseases (see [Chart 60-1](#)). A review of the medical history, including a system-by-system evaluation, is part of the health history. The nurse should be aware of any history of trauma or falls that may have involved the head or spinal cord.

Questions regarding the use of alcohol, medications, and illicit drugs are also relevant. The history-taking portion of the neurologic assessment is critical and, in many cases of neurologic disease, leads to an accurate diagnosis.

## Physical Assessment



The neurologic examination is a systematic process that includes a variety of clinical tests, observations, and assessments designed to evaluate the neurologic status of a complex system. Many neurologic rating scales exist (Herndon, 2006), and some of the more common ones are discussed in this chapter.

The brain and spinal cord cannot be examined as directly as other systems of the body. Therefore, much of the neurologic examination is an indirect evaluation that assesses the function of the specific body part or parts controlled by the nervous system. A neurologic assessment is divided into five components: consciousness and cognition, cranial nerves, motor system, sensory system, and reflexes. One or more components may become the priority assessment, depending on the patient's condition. For example, motor, sensory, and reflex assessments are the priority in patients with spinal injury, whereas in a patient who is comatose, the cranial nerves and level of consciousness become the priority.

### Assessing Consciousness and Cognition

Cerebral abnormalities may cause disturbances in mental status, intellectual functioning, thought content, and emotional status. There may also be alterations in language abilities as well as lifestyle. The examiner must also be aware of the patient's overall level of consciousness and any changes over time (Posner et al., 2019).

The examiner records and reports specific observations regarding mental status, intellectual function, thought content, and emotional status, all of which permit comparison by others over time. Alterations should be described in specific and nonjudgmental terms. The use of terms such as "inappropriate" or "demented" is avoided, because they often mean different things to different people and are therefore not useful when describing behavior. Analysis and the conclusions that may be drawn from these findings usually depend on the examiner's knowledge of neuroanatomy, neurophysiology, and neuropathology.

**Chart 60-1**



## **GENETICS IN NURSING PRACTICE**

## **Neurologic Disorders**

Several neurologic disorders are associated with genetic abnormalities. Neurologic impairment is noted with many other genetic illnesses. Some examples include:

Autosomal Dominant:

- Cerebral arteriopathy
- Familial Alzheimer's disease
- Huntington disease
- Myotonic dystrophies
- Neurofibromatosis
- Von Hippel–Lindau syndrome

Autosomal Recessive:

- Canavan disease
- Familial dysautonomia
- Friedreich ataxia

X Linked:

- Duchenne muscular dystrophy
- Fragile X syndrome

Inheritance pattern is not distinct; however, there is a genetic predisposition for the disease:

- Amyotrophic lateral sclerosis (ALS)
- Epilepsy
- Neural tube defects (e.g., spina bifida, anencephaly)
- Parkinson's disease
- Tourette syndrome

Other genetic disorders that also impact the neurologic system:

- Bipolar disease
- Down syndrome
- Phenylketonuria (PKU)
- Schizophrenia
- Tay–Sachs disease
- Tuberous sclerosis complex

## **Nursing Assessments**

Refer to [Chapter 4, Chart 4-2: Genetics in Nursing Practice: Genetic Aspects of Health Assessment](#)

[Family History Assessment Specific to Neurologic Disorders](#)

- Assess for other similarly affected relatives with neurologic impairment.
- Inquire about age of onset (e.g., present at birth—spina bifida; developed in childhood—Duchenne muscular dystrophy; developed in adulthood—Huntington disease, Alzheimer’s disease, ALS).
- Inquire about the presence of related conditions such as intellectual disability or learning disabilities (neurofibromatosis type 1).

### Patient Assessment

- Assess for the presence of other physical features suggestive of an underlying genetic condition, such as skin lesions seen in neurofibromatosis (*café-au-lait* spots).
- Assess attention span, and the presence of hyperactivity or withdrawn behavior.
- Assess for other congenital abnormalities (e.g., cardiac, ocular).
- Inspect for presence of freckles in the axillary or inguinal areas.
- Assess for presence of uncoordinated movement of extremities, muscle twitching, or history of seizures.
- Assess for poor or hyperactive muscle tone.
- Assess for episodes of forgetfulness or uncharacteristic changes in behavior or mood.
- Inspect for disproportionate facial features (fragile X or Down syndrome).
- Observe for presence of “tics” or uncontrolled body movement.
- Ask about history of seizures or head trauma.

### Genetics Resources

Epilepsy Foundation, [www.epilepsy.com/learn/diagnosis/genetic-testing](http://www.epilepsy.com/learn/diagnosis/genetic-testing)

Huntington’s Disease Society of America, [hdsa.org](http://hdsa.org)

Muscular Dystrophy Association, [www.mda.org](http://www.mda.org)

See [Chapter 6, Chart 6-7](#) for components of genetic counseling.

### Mental Status

An assessment of mental status begins by observing the patient’s appearance and behavior, noting dress, grooming, and personal hygiene. Posture, gestures, movements, and facial expressions often provide important information about the patient. Does the patient appear to be aware of and interact with the surroundings?

Assessing orientation to time, place, and person assists in evaluating mental status. Does the patient know what day it is, what year it is, and the name of the president of the United States? Is the patient aware of where they are? Is the patient aware of who the examiner is and of their purpose for being in the room? Assessment of immediate and remote memory is also important. Is the capacity for immediate memory intact?

## Intellectual Function

A person with an average intelligence quotient (IQ) can repeat seven digits without faltering and can recite five digits backward. The examiner might ask the patient to count backward from 100 or to subtract 7 from 100, then 7 from that, and so forth (referred to as serial 7s). The capacity to interpret well-known proverbs tests abstract reasoning, which is a higher intellectual function—for example, does the patient know what is meant by “a stitch in time saves nine”? The intellectual function of patients with damage to the frontal cortex appears intact until one or more tests of intellectual capacity are performed. Questions designed to assess this capacity might include the ability to recognize similarities—for example, how are a mouse and dog or pen and pencil alike? Can the patient make judgments about situations—for example, if the patient arrives home without a house key, what alternatives are there?

## Thought Content

During the interview, it is important to assess the patient’s thought content. Are the patient’s thoughts spontaneous, natural, clear, relevant, and coherent? Does the patient have any fixed ideas, illusions, or preoccupations? What are their insights into these thoughts? Preoccupation with death or morbid events, hallucinations, and paranoid ideation are examples of unusual thoughts or perceptions that require further evaluation.

## Emotional Status

An assessment of consciousness and cognition also includes the patient’s emotional status. Is the patient’s affect (external manifestation of mood) natural and even, or irritable and angry, anxious, apathetic or flat, or euphoric? Does their mood fluctuate normally, or does the patient unpredictably swing from joy to sadness during the interview? Is affect appropriate to words and thought content? Are verbal communications consistent with nonverbal cues?

## Language Ability

The person with normal neurologic function can understand and communicate in spoken and written language. Does the patient answer questions appropriately? Can they read a sentence from a newspaper and explain its meaning? Can the patient write their name or copy a simple figure that the examiner has drawn? A deficiency in language function is called *aphasia*. Different types of aphasia result from injury to different parts of the brain (see Table 60-5). See [Chapter 62](#) for a detailed discussion of aphasia.

## Impact on Lifestyle

The nurse assesses the impact of any impairment on the patient’s lifestyle. Issues to consider include the limitations imposed on the patient by any

cognitive deficit and the patient's role in society, including family and community roles. The plan of care that the nurse develops needs to address and support adaptation to the neurologic deficit and continued function to the extent possible within the patient's support system.

### Level of Consciousness

Consciousness is the patient's wakefulness and ability to respond to the environment. Level of consciousness is the most sensitive indicator of neurologic function. To assess level of consciousness, the examiner observes for alertness and ability to follow commands.

If the patient is not alert or able to follow commands, the examiner observes for eye opening; verbal response and motor response to stimuli, if any; and the type of stimuli needed to obtain a response. Noxious stimuli should be used first, then painful stimuli if no response is observed. In the patient with decreased level of consciousness, motor and cranial nerve functions become the priority assessments, because abnormalities can indicate the area of involvement in the absence of responsiveness. See [Chapter 61](#) for further discussion of changes in level of consciousness.

**TABLE 60-5** Types of Aphasia and Region of Brain Involved

Type of Aphasia	Brain Area Involved
Auditory receptive	Temporal lobe
Visual receptive	Parietal and occipital area
Expressive speaking	Inferior–posterior frontal areas
Expressive writing	Posterior frontal area

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

### Unfolding Patient Stories: Marilyn Hughes • Part 2



Recall from [Chapter 37](#) Marilyn Hughes, who came to the hospital after falling on icy stairs. She sustained a left midshaft tibia–fibula fracture, which requires surgery. Her husband informs the nurse that she also hit her head and did not respond to him for a short time after the fall. Describe the neurologic assessment performed by the nurse. Why should the nurse report this information promptly to the health care team?

Care for Marilyn and other patients in a realistic virtual environment: [vSim\(the-point.lww.com/vSimMedicalSurgical\)](https://the-point.lww.com/vSimMedicalSurgical). Practice documenting these patients' care in DocuCare ([the-point.lww.com/DocuCareEHR](https://the-point.lww.com/DocuCareEHR)).

## Examining the Cranial Nerves

Cranial nerves are assessed when level of consciousness is decreased, with brain stem pathology, or in the presence of peripheral nervous system disease (Weber & Kelley, 2018). Right and left cranial nerve functions are compared throughout the examination.

See [Table 60-2](#) for methods of examining the cranial nerves.

## Examining the Motor System

### Motor Ability

A thorough examination of the motor system includes an assessment of muscle size and tone as well as strength, coordination, and balance. The patient is instructed to walk across the room, if possible, while the examiner observes posture and gait. The muscles are inspected, and palpated if necessary, for their size and symmetry. Any evidence of atrophy or involuntary movements (tremors, tics) is noted. Muscle tone (the tension present in a muscle at rest) is evaluated by palpating various muscle groups at rest and during passive movement. Resistance to these movements is assessed and documented. Abnormalities in tone include **spasticity** (increased muscle tone), **rigidity** (resistance to passive stretch), and flaccidity.

### Muscle Strength

Assessing the patient's ability to flex or extend the extremities against resistance tests muscle strength. The function of an individual muscle or group of muscles is evaluated by placing the muscle at a disadvantage. The quadriceps, for example, is a powerful muscle responsible for straightening the leg. Once the leg is straightened, it is exceedingly difficult for the examiner to flex the knee. If the knee is flexed and the patient is asked to straighten the leg against resistance, weakness can be elicited. The evaluation of muscle strength compares the sides of the body to each other. For example, the right upper extremity is compared to the left upper extremity. Subtle differences in strength may be evaluated by testing for drift. For example, both arms are out in front of the patient with palms up; drift is seen as pronation of the palm, indicating a subtle weakness that may not have been detected on the resistance examination.

Clinicians use a 5-point scale to rate muscle strength. A 5 indicates full power of contraction against gravity and resistance or normal muscle strength; 4 indicates fair but not full strength against gravity and a moderate amount of resistance or slight weakness; 3 indicates just sufficient strength to overcome the force of gravity or moderate weakness; 2 indicates the ability to move but not to overcome the force of gravity or severe weakness; 1 indicates minimal contractile power (weak muscle contraction can be palpated but no movement is noted) or very severe weakness; and 0 indicates no movement (Jarvis, 2020).



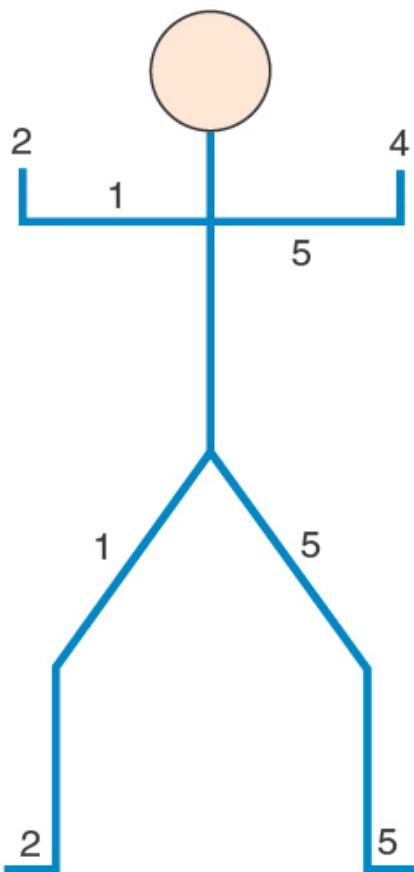
### Concept Mastery Alert

When recording muscle strength, a stick figure is used as a precise form to document findings. The five-point scale is used to rate and record distal and proximal strength in both upper and lower extremities. Figure 60-12 provides further details.

Assessment of muscle strength may be as detailed as necessary. One may quickly test the strength of the proximal muscles of the upper and lower extremities, always assessing both sides by comparing one side to the other. The strength of the finer muscles that control the function of the hand (hand grasp) and the foot (dorsiflexion and plantar flexion) can then be assessed.

### Balance and Coordination

Cerebellar and basal ganglia influence on the motor system is reflected in balance control and coordination. Coordination in the hands and upper extremities is tested by having the patient perform rapid, alternating movements and point-to-point testing. First, the patient is instructed to pat their thigh as fast as possible with each hand separately. Then, the patient is instructed to alternately pronate and supinate the hand as rapidly as possible. Last, the patient is asked to touch each of the fingers with the thumb in a consecutive motion. Speed, symmetry, and degree of difficulty are noted. Point-to-point testing is accomplished by having the patient touch the examiner's extended finger and then their own nose. This is repeated several times.



**Figure 60-12 •** A stick figure may be used to record muscle strength.

Coordination in the lower extremities is tested by having the patient run the heel down the anterior surface of the tibia of the other leg. Each leg is tested in turn. **Ataxia** is an incoordination of voluntary muscle action, particularly of the muscle groups used in activities such as walking or reaching for objects. Tremors (rhythmic, involuntary movements) noted at rest or during movement suggest a problem in the anatomic areas responsible for balance and coordination.

The **Romberg test** is a screening test for balance that can be done with the patient seated or standing. The patient can be seated or stand with feet together and arms at the side, first with eyes open and then with both eyes closed for 20 seconds (Weber & Kelley, 2018). The examiner stands close to support the standing patient if they begin to fall. Slight swaying is normal, but a loss of balance is abnormal and is considered a positive Romberg test. Additional cerebellar tests for balance in the patient who is ambulatory include hopping in place, alternating knee bends, and heel-to-toe walking (both forward and backward).

## Examining the Sensory System

The sensory system is even more complex than the motor system, because sensory modalities are more widespread throughout the central and peripheral nervous systems. The sensory examination is largely subjective and requires the cooperation of the patient. The examiner should be familiar with dermatomes that represent the distribution of the peripheral nerves that arise from the spinal cord (see [Fig. 60-9](#)) (Jarvis, 2020).

Assessment of the sensory system involves tests for tactile sensation, superficial pain, temperature, vibration, and position sense (proprioception). During the sensory assessment, the patient's eyes are closed. Simple directions and reassurance that the examiner will not hurt or startle the patient encourage the cooperation of the patient.

Tactile sensation is assessed by lightly touching a cotton wisp or fingertip to corresponding areas on each side of the body. The sensitivity of proximal parts of the extremities is compared with that of distal parts, and the right and left sides are compared.

Pain and temperature sensations are transmitted together in the lateral part of the spinal cord, so it is unnecessary to test for temperature sense in most circumstances. Determining the patient's sensitivity to a sharp object can assess superficial pain perception. However, pain sensation is usually reserved for patients who do not respond to or cannot discriminate touch stimulation. The patient is asked to differentiate between the sharp and dull ends of a broken wooden cotton swab or tongue blade; using a safety pin is inadvisable because it breaks the integrity of the skin. Both the sharp and dull sides of the object are applied with equal intensity at all times, and the two sides are compared. In the patient with an altered level of consciousness alternative methods of assessing pain may need to be used (Poulsen, Brix, Andersen, et al., 2016).

Vibration and proprioception are transmitted together in the posterior part of the spinal cord. Vibration may be evaluated through the use of a low-frequency (128 or 256 Hz) tuning fork. The handle of the vibrating fork is placed against a bony prominence, and the patient is asked if a sensation is felt; the patient is then instructed to signal the examiner when the sensation ceases. Common locations used to test for vibratory sense include the distal joint of the great toe and the proximal thumb joint. If the patient does not perceive the vibrations at the distal bony prominences, the examiner progresses upward with the tuning fork until the patient perceives the vibrations. As with all measurements of sensation, a side-to-side comparison is made.

Position sense or proprioception may be determined by asking the patient to close both eyes and indicate, as the great toe or index finger is alternately moved up and down, in which direction movement has taken place. Vibration and position sense are often lost together, frequently in circumstances in which all other sensation remains intact.

Integration of sensation in the brain is evaluated by testing two-point discrimination. When the patient is touched with two sharp objects simultaneously, are they perceived as two or as one? If touched simultaneously on opposite sides of the body, the patient should normally report being touched in two places. If only one site is reported, the one not being recognized is said to demonstrate extinction. Another test of higher cortical sensory ability is tactile identification. The patient is instructed to close both eyes and identify an object (e.g., key, coin) that is placed in one hand by the examiner; inability to identify an object by touch is known as tactile agnosia or astereognosis. **Agnosia** is the general loss of ability to recognize objects through a particular sensory system. The patient can also be shown a familiar object and asked to identify it by name; inability to identify a visualized object is known as visual agnosia. Each of these dysfunctions implicates a different part of the brain (see [Table 60-6](#)).

Decreased or absent sensations occur with problems anywhere along the sensory pathway. Sensory deficits resulting from peripheral neuropathy or spinal cord injury follow anatomic dermatomes. Destructive lesions of the brain may affect sensation on an entire side of the body. Stroke affecting a portion of the sensory cortex will produce altered sensory discrimination.

**TABLE 60-6** Types of Agnosia and Corresponding Sites of Lesions

Type of Agnosia	Affected Cerebral Area
Visual	Occipital lobe
Auditory	Temporal lobe (lateral and superior portions)
Tactile	Parietal lobe
Body parts and relationships	Parietal lobe (posteroinferior regions)

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

## Examining the Reflexes

Reflexes are involuntary contractions of muscles or muscle groups in response to a stimulus. Reflexes are classified as deep tendon, superficial, or pathologic. Testing reflexes enables the examiner to assess involuntary reflex arcs that depend on the presence of afferent stretch receptors, spinal or brain stem synapses, efferent motor fibers, and a variety of modifying influences from higher levels.

### Deep Tendon Reflexes

A reflex hammer is used to elicit a deep tendon reflex. The handle of the hammer is held loosely between the thumb and index finger, allowing a full swinging motion. The wrist motion is similar to that used during percussion. The extremity is positioned so that the tendon is slightly stretched. This

requires a sound knowledge of the location of muscles and their tendon attachments. The tendon is then struck briskly (see Fig. 60-13), and the response is compared with that on the opposite side of the body. A wide variation in reflex response may be considered normal; however, it is more important that the reflexes be symmetrically equivalent. When the comparison is made, both sides should be equivalently relaxed, and each tendon struck with equal force.

Valid findings depend on several factors: proper use of the reflex hammer, proper positioning of the extremity, and a patient who is relaxed (Jarvis, 2020). If the reflexes are symmetrically diminished or absent, the examiner may use isometric contraction of other muscle groups to increase reflex activity. For example, if lower extremity reflexes are diminished or absent, the patient is instructed to lock the fingers together and pull in opposite directions. Having the patient clench the jaw or press the heels against the floor or examining table may similarly elicit more reliable biceps, triceps, and brachioradialis reflexes.

The absence of reflexes is significant, although ankle jerks (Achilles reflex) may be normally absent in older adults. Deep tendon reflex responses are often graded on a scale of 0 to 4+, with 2+ considered normal (see Chart 60-2) but scale ratings are highly subjective. Findings can be recorded as a fraction, indicating the scale range (e.g., 2/4). Some examiners prefer to use the terms *present*, *absent*, and *diminished* when describing reflexes. As with muscle strength recording, a stick figure may be used to record numerical findings.

### Biceps Reflex

The biceps reflex is elicited by striking the biceps tendon over a slightly flexed elbow (see Fig. 60-13A). The examiner supports the forearm at the elbow with one arm while placing the thumb against the tendon and striking the thumb with the reflex hammer. The normal response is flexion at the elbow and contraction of the biceps.

### Triceps Reflex

To elicit a triceps reflex, the patient's arm is flexed at the elbow and hanging freely at the side. The examiner supports the patient's arm and identifies the triceps tendon by palpating 2.5 to 5 cm (1 to 2 inches) above the elbow. A direct blow on the tendon (see Fig. 60-13B) normally produces contraction of the triceps muscle and extension of the elbow.



**Figure 60-13 •** Techniques for eliciting major reflexes. **A.** Eliciting the biceps reflex. **B.** Eliciting the triceps reflex. **C.** Eliciting the patellar reflex. **D.** Eliciting the Achilles reflex. Parts A–D reprinted with permission from Weber, J., & Kelley, J. (2018). *Health assessment in nursing* (6th ed., Figs. 25-31, 25-32, 25-33A, 25-34A). Philadelphia, PA: Lippincott Williams & Wilkins.

### Brachioradialis Reflex

With the patient's forearm resting on the lap or across the abdomen, the brachioradialis reflex is assessed. A gentle strike of the hammer 2.5 to 5 cm (1 to 2 inches) above the wrist results in flexion and supination of the forearm (Jarvis, 2020).

### **Patellar Reflex**

The patellar reflex is elicited by striking the patellar tendon just below the patella. The patient may be in a sitting or a lying position. If the patient is supine, the examiner supports the legs to facilitate relaxation of the muscles (see Fig. 60-13C). Contractions of the quadriceps and knee extension are normal responses.

### **Achilles Reflex**

To elicit an Achilles reflex, the foot is dorsiflexed at the ankle and the hammer strikes the stretched Achilles tendon (see Fig. 60-13D). This reflex normally produces plantar flexion. If the examiner cannot elicit the ankle reflex and suspects that the patient cannot relax, the patient is instructed to kneel on a chair or similar elevated, flat surface. This position places the ankles in dorsiflexion and reduces any muscle tension in the gastrocnemius. The Achilles tendons are struck in turn, and plantar flexion is usually demonstrated (Jarvis, 2020).

### **Clonus**

When reflexes are hyperactive, a movement called **clonus** may be elicited. If the foot is abruptly dorsiflexed, it may continue to “beat” two or three times before it settles into a position of rest. Occasionally with CNS system disease, this activity persists, and the foot does not come to rest while the tendon is being stretched but persists in repetitive activity. The unsustained clonus associated with normal but hyperactive reflexes is not considered pathologic. Sustained clonus always indicates the presence of CNS disease and requires further evaluation.

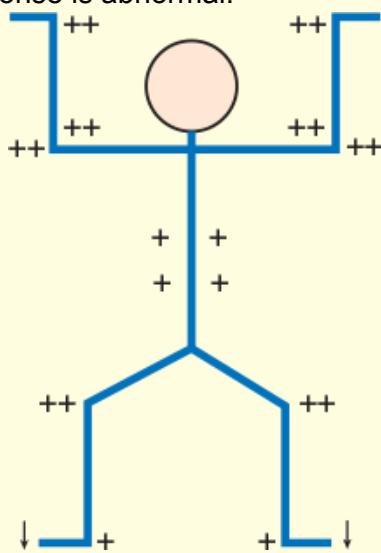
### **Chart 60-2**

## Documenting Reflexes

Deep tendon reflexes are graded on a scale of 0–4:

- 0 No response
- 1+ Diminished (hypoactive)
- 2+ Normal
- 3+ Increased (may be interpreted as normal)
- 4+ Hyperactive (hyperreflexia)

The deep tendon responses and plantar reflexes are commonly recorded on stick figures. The arrow points downward if the plantar response is normal and upward if the response is abnormal.



## Superficial Reflexes

The major superficial reflexes include corneal, palpebral, gag, upper/lower abdominal, cremasteric (men only), and perianal. These reflexes are graded differently than the motor reflexes and are noted to be present (+) or absent (−). Of these, only the corneal, gag, and plantar reflexes are commonly tested.

The corneal reflex is tested carefully using a clean wisp of cotton and lightly touching the outer corner of each eye on the sclera. The reflex is present if the action elicits a blink. A stroke or brain injury might result in loss of this reflex, either unilaterally or bilaterally. Loss of this reflex indicates the need for eye protection and possible lubrication to prevent corneal damage.

The gag reflex is elicited by gently touching the back of the pharynx with a cotton-tipped applicator, first on one side of the uvula and then the other. Positive response is an equal elevation of the uvula and “gag” with stimulation. Absent response on one or both sides can be seen following a stroke and requires careful evaluation and treatment of the resultant swallowing dysfunction to prevent aspiration of food and fluids.

## Pathologic Reflexes

Pathologic reflexes are seen in the presence of neurologic disease; they often represent emergence of earlier reflexes that disappeared with maturity of the nervous system. A pathologic reflex indicative of CNS disease affecting the corticospinal tract is the **Babinski reflex (sign)**. In a person with an intact CNS, if the lateral aspect of the sole of the foot is stroked, the toes contract and draw together. However, in a person who has CNS disease of the motor system, the toes fan out and draw back (Jarvis, 2020). This is normal in newborns but represents a serious abnormality in adults. Other pathologic reflexes include the suck (sucking motions in response to touching the lips), snout (lip pursing in response to touching the lips), palmar (grasp in response to stroking the palm), and palmonental (contraction of the facial muscle in response to stimulation of the thenar eminence near the thumb) reflexes in adults. These reflexes often signify progressive nervous system degeneration (Klein & Stewart-Amidei, 2017).



## Gerontologic Considerations

During the normal aging process, the nervous system undergoes many changes and is more vulnerable to illness. Age-related changes in the nervous system vary in degree and must be distinguished from those due to disease. It is important for clinicians not to attribute abnormality or dysfunction to aging without appropriate investigation. For example, although diminished strength and agility are a normal part of aging, localized weakness can only be attributed to disease.

### Structural and Physiologic Changes

As the brain ages, neurons are lost, leading to a decrease in the number of synapses and neurotransmitters. This results in slowed nerve conduction and response time. Brain weight decreases, and the ventricle size increases to maintain cranial volume leading to a decreased brain volume. These changes in brain volume accelerate even in healthy people between the ages of 60 and 70 years (Battaglini, Gentile, Luchetti, et al., 2019). Cerebral blood flow and metabolism are reduced, leading to slower mental functions. Temperature regulation becomes less efficient. In the peripheral nervous system, myelin is lost, resulting in a decrease in conduction velocity in some nerves. Visual and auditory nerves degenerate, leading to loss of visual acuity and hearing. Taste buds atrophy, and nerve cell fibers in the olfactory bulb degenerate (Jarvis, 2020). Nerve cells in the vestibular system of the inner ear, cerebellum, and proprioceptive pathways also degenerate, leading to balance difficulties. Deep tendon reflexes can be decreased or in some cases absent. Hypothalamic function is modified such that stage IV sleep is reduced. There is an overall

slowing of autonomic nervous system responses. Pupillary responses are reduced or may not appear at all in the presence of cataracts.

## **Motor Alterations**

Reduced nerve input into muscle contributes to an overall reduction in muscle bulk, with atrophy most easily noted in the hands. Changes in motor function often result in decreased strength and agility, with increased reaction time. Gait is often slowed and wide based. These changes can create difficulties in maintaining balance, predisposing the older person to falls.

## **Sensory Alterations**

Tactile sensation is dulled in the older adult due to a decrease in the number of sensory receptors. There may be difficulty in identifying objects by touch, because fewer tactile cues are received from the bottom of the feet and the person may become confused about body position and location.

Sensitivity to glare, decreased peripheral vision, and a constricted visual field occur due to degeneration of visual pathways, resulting in disorientation, especially at night when there is little or no light in the room. Because the older adult takes longer to recover visual sensitivity when moving from a light to dark area, nightlights and a safe and familiar arrangement of furniture are essential.

Loss of hearing can contribute to confusion, anxiety, disorientation, misinterpretation of the environment, feelings of inadequacy, and social isolation. A decreased sense of taste and smell may contribute to weight loss and disinterest in food. A decreased sense of smell may present a safety hazard, because older adults living alone may be unable to detect household gas leaks or fires. Smoke and carbon monoxide detectors—important in every residence—are critical for the older adult.

## **Temperature Regulation and Pain Perception**

The older adult patient may feel cold more readily than heat and may require extra covering when in bed; a room temperature somewhat higher than usual may be desirable. Reaction to painful stimuli may be decreased with age. Because pain is an important warning signal, caution must be used when hot or cold packs are used. The older patient may be burned or suffer frostbite before being aware of any discomfort. Complaints of pain such as abdominal discomfort or chest pain may be more serious than the patient's perception might indicate and thus require careful evaluation. In older adults, two common pain syndromes that affect the neurologic system are diabetic and postherpetic neuropathies. These frequently occur due to the high rate of these comorbid conditions in older adults. See [Chapter 46](#) for a discussion of diabetic neuropathy.

## Mental Status

Although mental processing time decreases with age, memory, language, and judgment capacities remain intact. Change in mental status should never be assumed to be a normal part of aging. **Delirium** is an acute confused state that begins with disorientation and if not recognized and treated early can progress to changes in level of consciousness, irreversible brain damage, and sometimes death. Older age is a risk, but delirium is also seen in patients who have underlying CNS damage or are experiencing an acute condition such as infection, adverse medication reaction, or dehydration. Drug toxicity and depression may produce impairment of attention and memory and should be evaluated as a possible cause of mental status change. Assessment with validated screening tool leads to improved detection of delirium (Smulter, Lingehall, Gustafson, et al., 2019). The Confusion Assessment Method (CAM) is a commonly used screening tool (Inouye, van Dyck, Alessi, et al., 1990) (see [Chapter 8, Chart 8-7](#)). Delirium must be differentiated from dementia, which is a chronic and irreversible deterioration of cognitive status. See [Chapter 61, Table 61-4](#) for further discussion of delirium and dementia.

## Nursing Implications

Nursing care for patients with age-related changes to the nervous system and for patients with long-term neurologic disability who are aging should include the modifications described previously. In addition, the consequences of any neurologic deficit and its impact on overall function such as activities of daily living, the use of assistive devices, and individual coping should be assessed and considered in planning patient care. Fall risk must be evaluated and fall prevention measures instituted for the patient who is hospitalized as well as in the home.

The nurse must understand the altered responses and the changing needs of the older adult patient before providing education. Visual and hearing deficits require adaptations in activities such as preoperative education, diet therapy, and education about new medications. When using visual materials for education or menu selection, adequate lighting without glare, contrasting colors, and large print are used to offset visual difficulties caused by rigidity and opacity of the lens in the eye and slower pupillary reaction. Procedures and preparations needed for diagnostic tests are explained, taking into account the possibility of impaired hearing and slowed responses in the older adult. Even with hearing loss, the older adult patient often hears adequately if the speaker uses a low-pitched, clear voice; shouting only makes it harder for the patient to understand the speaker. Providing auditory and visual cues aids understanding; if the patient has a significant hearing or visual loss, assistive devices, a signer, an interpreter, or a translator may be needed.

Providing education at an unrushed pace and using reinforcement enhance learning and retention. Material should be short, concise, and concrete. Vocabulary is matched to the patient's ability, and terms are clearly defined. The older adult patient requires adequate time to receive and respond to stimuli, learn, and react. These measures allow comprehension, memory, and formation of association and concepts.

## Diagnostic Evaluation

A wide range of diagnostic studies may be performed in patients with altered neurologic function. The nurse should educate the patient about the purpose, what to expect, and any possible side effects related to these examinations prior to testing. Women who are premenopausal are advised to practice effective contraception before and for several days after any diagnostic procedure using contrast, and the woman who is breast-feeding is instructed to stop for the time period recommended by the nuclear medicine department (Pagana & Pagana, 2018). The nurse should note trends in results, because they provide information about disease progression as well as the patient's response to therapy.

## Computed Tomography Scanning

Computed tomography (CT) scanning uses a narrow x-ray beam to scan body parts in successive layers. The images provide cross-sectional views of the brain, distinguishing differences in tissue densities of the skull, cortex, subcortical structures, and ventricles. An intravenous (IV) contrast agent may be used to highlight differences further. The brightness of each slice of the brain in the final image is proportional to the degree to which it absorbs x-rays. The image is displayed on an oscilloscope or television monitor and is photographed and stored digitally (Fischbach & Fischbach, 2018). CT scanning is usually performed first without contrast material and then with IV contrast, if needed. The patient lies on an adjustable table with the head in a headrest while the scanning system rotates around the head and produces cross-sectional images. The patient must lie with the head held perfectly still without talking or moving the face, because head motion distorts the image. CT scanning is quick and painless and uses a small amount of radiation to produce images; it has a high degree of sensitivity for detecting lesions.

Brain lesions have a different tissue density from the surrounding normal brain tissue. Abnormalities detected on brain CT include tumor or other masses, infarction, hemorrhage, displacement of the ventricles, and cortical atrophy (Fischbach & Fischbach, 2018). CT angiography allows visualization of blood vessels; in some situations, this eliminates the need for formal

angiography. Whole-body CT scanners allow cross-sections of the spinal cord to be visualized. The injection of a water-soluble iodinated contrast agent into the subarachnoid space through lumbar puncture improves the visualization of the spinal and intracranial contents on these images. The CT scan, along with magnetic resonance imaging (MRI), has largely replaced myelography as a diagnostic procedure for the diagnosis of herniated lumbar discs.

### Nursing Interventions

Essential nursing interventions include preparation for the procedure and patient monitoring. Preparation includes educating the patient about the need to lie quietly throughout the procedure. A review of relaxation techniques may be helpful for patients with claustrophobia. Sedation can be used if agitation, restlessness, or confusion interferes with a successful study. Ongoing patient monitoring during sedation is necessary. If a contrast agent is used, the patient must be assessed before the CT scan for an iodine/shellfish allergy, because the contrast agent used may be iodine based. Kidney function must also be evaluated because the contrast material is cleared through the kidneys. A suitable IV line for contrast injection and a period of fasting (usually 4 hours) are required prior to the study. Patients who receive an IV contrast agent are monitored during and after the procedure for allergic reactions and changes in kidney function (Fischbach & Fischbach, 2018). Fluid intake is also encouraged after IV contrast to facilitate contrast clearance through the kidney.

### Magnetic Resonance Imaging

MRI uses a powerful magnetic field to obtain images of different areas of the body. The magnetic field causes the hydrogen nuclei (protons) within the body to align like small magnets in a magnetic field. In combination with radiofrequency pulses, the protons emit signals, which are converted to images. An MRI scan can be performed with or without a contrast agent and can identify a cerebral abnormality earlier and more clearly than other diagnostic tests (Fischbach & Fischbach, 2018). It can provide information about the chemical changes within cells, allowing the clinician to monitor a tumor's response to treatment. It is particularly useful in the diagnosis of brain tumor, stroke, and multiple sclerosis and does not involve ionizing radiation. An MRI scan may take an hour or longer to complete, so its use in emergency situations is limited.

Several MRI applications allow imaging of brain blood flow and metabolism via special imaging techniques added to the MRI. Such techniques include diffusion-weighted imaging (DWI), perfusion-weighted imaging (PWI), magnetic resonance spectroscopy, and fluid attenuation inversion recovery (FLAIR) (Fischbach & Fischbach, 2018). Magnetic resonance angiography (MRA) allows separate visualization of the cerebral vasculature

without the administration of an arterial contrast agent. Both MRI and CT images are used as tools to plan and direct surgical intervention.

## Nursing Interventions

Patient preparation includes providing education and obtaining an adequate history. Ferromagnetic substances in the body may become dislodged by the magnet, so history of working with metal fragments must be reviewed. Patients with any type of cardiac implantable electronic device need to be screened to see if it is safe for the patient to undergo any type of MRI (Indik, Gimbel, Abe, et al., 2017). The patient is assessed for implants containing metal (e.g., aneurysm clips, orthopedic hardware, artificial heart valves, intrauterine devices). These objects could malfunction, be dislodged, or heat up as they absorb energy. Cochlear implants will be inactivated by MRI; therefore, other imaging procedures are considered. A complete list of metal compatibility may be found on MRI manufacturer Web sites.

Before the patient enters the room where the MRI is to be performed, all metal objects and credit cards (the magnetic field can erase them) must be removed. This includes medication patches that have a metal backing and metallic lead wires; these can cause burns if not removed (Fischbach & Fischbach, 2018). No metal objects may be brought into the room where the MRI is located; this includes oxygen tanks, IV poles, ventilators, or even stethoscopes. The magnetic field generated by the unit is so strong that any metal-containing items will be strongly attracted and literally can be pulled away with such force that they fly like projectiles toward the magnet. There is a risk of severe injury and death. Further, damage to expensive equipment may occur.



### Quality and Safety Nursing Alert

*For patient safety, the nurse prevents any patient care equipment containing metal or metal parts (e.g., portable oxygen tanks, wheelchairs) from entering the room where the MRI is located. The nurse also assesses for and removes any medication patches with foil backing (such as nicotine patches) that may cause a burn while an MRI scan is being performed.*

For the MRI, the patient lies with the head in a frame on a flat platform that is moved into a tube housing the magnet (see Fig. 60-14). The tube is narrow; people with a wide girth may not fit into the scanner. Patients who are unable to lie flat will not be able to tolerate an MRI. The scanning process is painless, but the patient hears loud thumping of the magnetic coils as the magnetic field is being pulsed. Patients may experience claustrophobia while inside the

narrow tube; sedation may be prescribed in these circumstances. “Open” MRI machines are less claustrophobic than the other devices and are available in many locations. However, the images produced on these machines are sometimes not so detailed, and traditional devices are preferred for accurate diagnosis. The patient may be educated about the use of relaxation techniques while in the scanner. The patient is informed that it will be possible to talk to the staff during the scan through a microphone inside the scanner (Fischbach & Fischbach, 2018).



**Figure 60-14 •** Technician explains what to expect during a magnetic resonance imaging procedure.

## Positron Emission Tomography

PET is a computer-based nuclear imaging technique that produces images of actual organ functioning. The patient either inhales a radioactive gas or is injected with a radioactive substance that emits positively charged particles. When these positrons combine with negatively charged electrons (normally found in the body’s cells), the resultant gamma rays can be detected by a scanning device that produces a series of two-dimensional views at various levels of the brain. This information is integrated by a computer and gives a composite picture of the brain at work.

PET permits the measurement of blood flow, tissue composition, and brain metabolism and thus indirectly evaluates brain function. The brain is one of the most metabolically active organs, consuming 80% of the glucose the body

uses (Hickey & Strayer, 2020). PET measures this activity in specific areas of the brain and can detect changes in glucose use.

PET is useful in showing metabolic changes in the brain (Alzheimer's disease), locating lesions (brain tumor, epileptogenic lesions), identifying blood flow and oxygen metabolism in patients with strokes, distinguishing tumor from areas of necrosis, and revealing biochemical abnormalities associated with mental illness. The isotopes used have a very short half-life and are expensive to produce, requiring specialized equipment for production. Improvement in the scanning procedure and production of isotopes, as well as the advent of reimbursement by third-party payers, has increased the clinical applications of PET studies.

### Nursing Interventions

Key nursing interventions include patient preparation, which involves explaining the test and educating the patient about inhalation techniques and the sensations (e.g., dizziness, lightheadedness, headache) that may occur. The IV injection of the radioactive substance produces similar side effects. Relaxation exercises may reduce anxiety during the test.

## Single-Photon Emission Computed Tomography

SPECT is a three-dimensional imaging technique that uses radionuclides and instruments to detect single photons. It is a perfusion study that captures a moment of cerebral blood flow at the time of injection of a radionuclide. Gamma photons are emitted from a radiopharmaceutical agent given to the patient and are detected by a rotating gamma camera or cameras; the image is sent to a minicomputer. This approach allows areas behind overlying structures or background to be viewed, greatly increasing the contrast between normal and abnormal tissue. It is relatively inexpensive, and the duration is similar to that of a CT scan.

SPECT is useful in detecting the extent and location of abnormally perfused areas of the brain, thus allowing detection, localization, and sizing of stroke (before it is visible by CT scan); localization of seizure foci in epilepsy; detection of tumor progression (Fischbach & Fischbach, 2018); and evaluation of perfusion before and after neurosurgical procedures.

### Nursing Interventions

The nursing interventions for SPECT primarily include patient preparation and patient monitoring. Providing education about what to expect before the test can allay anxiety and ensure patient cooperation during the test. Pregnancy and breast-feeding are contraindications to SPECT.

The nurse may need to accompany and monitor the patient during transport to the nuclear medicine department for the scan. Patients are monitored during and after the procedure for allergic reactions to the radiopharmaceutical agent.

## Cerebral Angiography

Cerebral angiography is an x-ray study of the cerebral circulation with a contrast agent injected into a selected artery. A valuable tool in investigating vascular disease or anomalies, it is used to determine vessel patency, identify presence of collateral circulation, and provide detail on vascular anomalies that can be used in planning interventions. With the advent of additional imaging techniques, formal cerebral angiography is less frequently performed.

Cerebral angiograms are performed by threading a catheter through the femoral artery in the groin or the radial artery of the wrist and up to the desired vessel. Alternatively, direct puncture of the carotid artery may be performed. X-ray images are obtained as the contrast agent flows through the vessels; the carotid and vertebral arterial systems are visualized, as well as venous drainage. Arterial access may also be used for interventional procedures, such as placing coils in an aneurysm or arteriovenous malformation.

## Nursing Interventions

Prior to the angiography, the patient's blood urea nitrogen and creatinine should be checked to ensure the kidneys will be able to excrete the contrast agent. The patient should be well hydrated, and clear liquids are usually permitted up to the time of the test. The patient is instructed to void immediately before the test, and locations of the appropriate peripheral pulses are marked with a felt-tip pen. The patient is instructed to remain immobile during the angiogram process and is told to expect a brief feeling of warmth in the face, behind the eyes, or in the jaw, teeth, tongue, and lips, and a metallic taste when the contrast agent is injected.

When the femoral artery is selected for access, the hair in the groin is clipped and prepared and a local anesthetic agent is given to minimize pain at the insertion site and to reduce arterial spasm. A catheter is introduced into the femoral artery, flushed with heparinized saline, and filled with contrast agent. When the radial artery is selected for access, the wrist will be prepared and accessed using medications to relax and dilate the vessel to allow the catheter to pass (Mason, Shah, Tamis-Holland, et al., 2018). Fluoroscopy is used to guide the catheter to the appropriate vessels. Neurologic assessment is conducted during and immediately following cerebral angiography to observe for embolism or arterial dissection that may occur during the test. Signs of these complications include new onset of alterations in the level of consciousness, weakness on one side of the body, motor or sensory deficits, and speech disturbances.

Nursing care after cerebral angiography includes observation of the injection site for bleeding or hematoma formation (a localized collection of blood). Because a hematoma at the puncture site or embolization to a distal artery affects peripheral pulses, the peripheral pulses that were marked prior to the test are monitored frequently. The color and temperature of the involved extremity are assessed to detect possible embolism (Fischbach & Fischbach, 2018; Mason et al., 2018). Fluids are encouraged to facilitate clearance of the contrast through the kidney. The nurse also monitors for an allergic reaction to the contrast agent, as well as hypotension if vasodilatory medications were used to facilitate a radial approach (Mason et al., 2018).

## Myelography

A myelogram is an x-ray of the spinal subarachnoid space taken after the injection of a contrast agent into the spinal subarachnoid space through a lumbar puncture. The water-based contrast agent disperses upward through the CSF to outline the spinal subarachnoid space and shows any distortion of the spinal cord or spinal dural sac caused by tumors, cysts, herniated vertebral discs, or other lesions. Myelography is often followed by CT scanning (Fischbach & Fischbach, 2018).

## Nursing Interventions

The patient is educated about what to expect during the procedure and made aware that changes in position may be made during the procedure. After myelography, the patient lies in bed with the head of the bed elevated 30 to 45 degrees. The patient is advised to remain in bed in the recommended position for 4 to 24 hours after testing. Drinking liberal amounts of fluid for rehydration and replacement of CSF may decrease the incidence of post-lumbar puncture headache. The blood pressure, pulse, respiratory rate, and temperature are monitored, as well as the patient's ability to void. Complications that may occur include nausea, vomiting, headache, fever, stiff neck, seizures, paralysis of one side of the body, and changes in level of consciousness (Fischbach & Fischbach, 2018).

## Noninvasive Carotid Flow Studies

Noninvasive carotid flow studies use ultrasound imagery and Doppler measurements of arterial blood flow to evaluate carotid and deep orbital circulation. The graph produced indicates blood velocity. Increased blood velocity can indicate stenosis or partial obstruction. These tests are often obtained before more invasive tests such as arteriography or are used as screening tools. Carotid Doppler, carotid ultrasonography, oculoplethysmography, and ophthalmodynamometry are four common

noninvasive vascular techniques that permit evaluation of arterial blood flow and detection of arterial stenosis, occlusion, and plaques. These vascular studies allow noninvasive imaging of extra- and intracranial circulation (Fischbach & Fischbach, 2018).

## Transcranial Doppler

Transcranial Doppler uses the same noninvasive techniques as carotid flow studies but records the blood flow velocities of the intracranial vessels. Arterial flow velocities can be measured through thin areas of the temporal and occipital bones of the skull. A handheld Doppler probe emits a pulsed beam; the signal is reflected by the moving red blood cells within the blood vessels. Transcranial Doppler is a noninvasive technique that is helpful in assessing vasospasm (a complication following subarachnoid hemorrhage), altered cerebral blood flow found in occlusive vascular disease, other cerebral pathologies, and brain death.

## Nursing Interventions

When a carotid flow study or transcranial Doppler is scheduled, the procedure is described to the patient. The patient is informed that this is a noninvasive test, that a handheld transducer will be placed over the neck and the orbits of the eyes, and that a water-soluble gel or lubricant is used on the transducer (Fischbach & Fischbach, 2018). Either of these two low-risk tests can be performed at the patient's bedside.

## Electroencephalography

An electroencephalogram (EEG) represents a record of the electrical activity generated in the brain (Hickey & Strayer, 2020). It is obtained through electrodes applied on the scalp or through microelectrodes placed within the brain tissue. It provides an assessment of cerebral electrical activity. It is useful for diagnosing and evaluating seizure disorders, coma, or organic brain syndrome. Tumors, brain abscesses, blood clots, and infection may cause abnormal patterns in electrical activity. The EEG is also used in making a determination of brain death.

Electrodes are applied to the scalp to record the electrical activity in various regions of the brain. The amplified activity of the neurons between any two of these electrodes is recorded on continuously moving paper; this record is called the *encephalogram*.

For a baseline recording, the patient lies quietly with both eyes closed. The patient may be asked to hyperventilate for 3 to 4 minutes or to look at a bright, flashing light for photic stimulation. These activation procedures are performed to evoke abnormal electrical discharges, such as seizure potentials.

A sleep EEG may be recorded after sedation because some abnormal brain waves are seen only when the patient is asleep. If the epileptogenic area is inaccessible to conventional scalp electrodes, nasopharyngeal electrodes may be used.

Chart 60-3



## NURSING RESEARCH PROFILE

## **Electroencephalographic Patterns During Nursing Interventions in Neurointensive Care**

Elf, K., Carlsson, T., Santeliz Rivas, L., et al. (2019). Electroencephalographic patterns during common nursing interventions in neurointensive care: A descriptive pilot study. *Journal of Neuroscience Nursing*, 51(1), 10–15.

### **Purpose**

The purpose of this study was to identify changes on electroencephalography (EEG) during standard neurointensive nursing care.

### **Design**

The study was a descriptive pilot study using a convenience sample of patients admitted to the neurointensive care unit with impaired consciousness due to a neurosurgical condition. The sample included 12 participants, with a mean age of 65 years with diagnoses of subarachnoid hemorrhage, intracerebral hemorrhage, acute subdural hematoma, meningitis, ischemic infarction, or traumatic brain injury. All participants were mechanically ventilated, with continuous sedations and intracranial monitoring. The study design included the monitoring of simultaneous continuous EEG and video recording. The nursing interventions monitored included airway suctioning, repositioning, and when professionally touched for assessment of hygienic interventions.

### **Findings**

Four participants had seizure activity during four nursing interventions (0.4% of nursing interventions); one participant had stimulus-induced rhythmic discharges during an intervention. All 12 participants showed muscle artifacts during 353 nursing interventions (36.3%), which may be a sign of stress. Muscle artifacts happened during all types of nursing interventions but occurred most often when more than one intervention was performed.

### **Nursing Implications**

Patients with neuroscience disorders in intensive care undergo many stressors, and the results of this study indicate that nursing interventions may cause stress in patients. Oral care, repositioning, suctioning, and hygienic care may cause stress. Nurses should be mindful of the comfort of the patient with a neuroscience disorder when delivering care at the bedside and consider shorter and fewer interventions in patients who are sensitive.

Depth recording of EEG is performed by introducing electrodes stereotactically (radiologically placed using instrumentation) into a target area of the brain, as indicated by the patient's seizure pattern and scalp EEG. It is used to identify patients who may benefit from surgical excision of epileptogenic foci. Special transsphenoidal, mandibular, and nasopharyngeal

electrodes can be used, and video recording combined with EEG monitoring and telemetry is used in hospital settings to capture epileptiform abnormalities and their sequelae. Some epilepsy centers provide long-term ambulatory EEG monitoring with portable recording devices. Some evidence suggests that continuous EEG may be a useful tool for nurses planning interventions in patients who are critically ill (Elf, Carlsson, Santeliz Rivas, et al., 2019). See the Nursing Research Profile in [Chart 60-3](#).

### Nursing Interventions

To increase the chances of recording seizure activity, it is sometimes recommended that the patient be deprived of sleep the night before the EEG. Anticonvulsant agents, tranquilizers, stimulants, and depressants should be withheld 24 to 48 hours before an EEG, because these medications can alter the EEG wave patterns or mask the abnormal wave patterns of seizure disorders (Pagana & Pagana, 2018). Coffee, tea, chocolate, and cola drinks are omitted from the meal before the test because of their stimulating effect. However, the meal itself is not omitted, because an altered blood glucose level can cause changes in brain wave patterns.

The patient is informed that the standard EEG takes 45 to 60 minutes; a sleep EEG requires 12 hours. The patient is assured that the procedure does not cause an electric shock and that the EEG is a diagnostic test, not a form of treatment. An EEG requires the patient to lie quietly during the test. Sedation is not advisable, because it may lower the seizure threshold in patients with a seizure disorder and it alters brain wave activity in all patients. The nurse needs to check the prescription regarding the administration of anticonvulsant medication prior to testing.

Routine EEGs use a water-soluble lubricant for electrode contact, which can be wiped off and removed by shampooing later. Sleep EEGs involve the use of collodion glue for electrode contact, which requires acetone for removal.

### Electromyography

An electromyogram (EMG) is obtained by inserting needle electrodes into the skeletal muscles to measure changes in the electrical potential of the muscles (Pagana & Pagana, 2018). The electrical potentials are shown on an oscilloscope and amplified so that both the sound and appearance of the waves can be analyzed and compared simultaneously.

An EMG is useful in determining the presence of neuromuscular disorders and myopathies. It helps distinguish weakness due to neuropathy (functional or pathologic changes in the peripheral nervous system) from weakness resulting from other causes.

## Nursing Interventions

The procedure is explained, and the patient is warned to expect a sensation similar to that of an intramuscular injection as the needle is inserted into the muscle. The muscles examined may ache for a short time after the procedure.

## Nerve Conduction Studies

Nerve conduction studies are performed by stimulating a peripheral nerve at several points along its course and recording the muscle action potential or the sensory action potential that results. Surface or needle electrodes are placed on the skin over the nerve to stimulate the nerve fibers. This test is useful in the study of peripheral neuropathies and is often included as part of the EMG.

## Evoked Potential Studies

Evoked potential studies involve application of an external stimulus to specific peripheral sensory receptors with subsequent measurement of the electrical potential generated. Electrical changes are detected with the aid of computerized devices that extract the signal, display it on an oscilloscope, and store the data on magnetic tape or disc. In neurologic diagnosis, they reflect nerve conduction times in the peripheral nervous system. In clinical practice, the visual, auditory, and somatosensory systems are most often tested.

In visual evoked responses, the patient looks at a visual stimulus (flashing lights, a checkerboard pattern on a screen). The average of several hundred stimuli is recorded by EEG leads placed over the occipital lobe. The transit time from the retina to the occipital area is measured using computer-averaging methods.

Brainstem auditory evoked responses (BAERs) are measured by applying an auditory stimulus (repetitive auditory click) and measuring the transit time via the brain stem into the cortex. Specific lesions in the auditory pathway modify or delay the response. BAERs may be used in the diagnosis of brain stem abnormalities and in determination of brain death.

In somatosensory evoked responses (SERs), the peripheral nerves are stimulated (electrical stimulation through skin electrodes) and the transit time along the spinal cord to the cortex is measured and recorded from scalp electrodes. SERs are used to detect deficits in spinal cord or peripheral nerve conduction and to monitor spinal cord function during surgical procedures. It is also useful in the diagnosis of demyelinating diseases, such as multiple sclerosis and polyneuropathies, where nerve conduction is slowed.

## Nursing Interventions

The nurse explains the procedure and reassures the patient and encourages him or her to relax. The patient is advised to remain perfectly still throughout the recording to prevent artifacts (signals not generated by the brain) that interfere with the recording and interpretation of the test.

## Lumbar Puncture and Examination of Cerebrospinal Fluid

A lumbar puncture (spinal tap) is carried out by inserting a needle into the lumbar subarachnoid space to withdraw CSF (Schreiber, 2019). The test may be performed to obtain CSF for examination, to measure and reduce CSF pressure, to determine the presence or absence of blood in the CSF, and to administer medications intrathecally (into the spinal canal).

The needle is inserted into the subarachnoid space in the widest intervertebral spaces; between the second and third, the third and fourth, or fourth and fifth lumbar vertebrae (Schreiber, 2019). Because the spinal cord ends at the first lumbar vertebra, insertion of the needle below the level of the second lumbar vertebra prevents puncture of the spinal cord.

A lumbar puncture may be risky in the presence of an intracranial mass lesion because intraspinal pressure is decreased by removal of CSF, and the brain may herniate downward through the foramen magnum. A successful lumbar puncture requires that the patient be relaxed; a patient who is anxious is tense, and this may artificially alter the pressure reading. The nurse may be asked to assist with a lumbar puncture.



For the procedural guidelines for assisting with a lumbar puncture, go to [thepoint.lww.com/Brunner15e](http://thepoint.lww.com/Brunner15e).

### Cerebrospinal Fluid Analysis

The CSF should be clear and colorless. Pink, blood-tinged, or grossly bloody CSF may indicate a subarachnoid hemorrhage. The CSF may be bloody initially because of local trauma but becomes clearer as more fluid is drained (Hickey & Strayer, 2020; Schreiber, 2019). Specimens are obtained for cell count, culture, glucose, protein, and other tests as indicated. The specimens should be sent to the inhibitor laboratory immediately because changes will take place and alter the result if the specimens are allowed to stand. See Table A-5 in Appendix A on **thePoint** for the normal values of CSF.

### Post-Lumbar Puncture Headache

A post-lumbar puncture headache, ranging from mild to severe, may occur a few hours to several days after the procedure. It is a throbbing bifrontal or occipital headache that is dull and deep in character. It is particularly severe on sitting or standing but lessens or disappears when the patient lies down.

The headache is caused by CSF leakage at the puncture site (Schreiber, 2019). The fluid continues to escape into the tissues by way of the needle track from the spinal canal. As a result of a leak, the supply of CSF in the cranium is depleted to a point at which it is insufficient to maintain proper mechanical stabilization of the brain. When the patient assumes an upright position, tension and stretching of the venous sinuses and pain-sensitive structures occur.

Post-lumbar puncture headache may be avoided if a small-gauge needle (22 gauge) is used (Hickey & Strayer, 2020). A post-lumbar puncture headache is usually managed with analgesic agents, encouraging hydration, ingestion of caffeine, and lying supine (Schreiber, 2019).

### **Other Complications of Lumbar Puncture**

Herniation of the intracranial contents, spinal epidural abscess, spinal epidural hematoma, and meningitis are rare but serious complications of lumbar puncture. Other complications include temporary voiding problems, slight elevation of temperature, backache or spasms, and stiffness of the neck.

## **Promoting Home, Community-Based, and Transitional Care**



### **Educating Patients About Self-Care**

Many diagnostic tests are carried out in short-procedure units or outpatient testing settings or units. As a result, family members often provide the postprocedure care. Therefore, the patient and family must receive adequate education about precautions to take after the procedure, complications to watch for, and steps to take if complications occur. Because many patients undergoing neurologic diagnostic studies are older adults or have neurologic deficits, provisions must be made to ensure that transportation, postprocedure care, and appropriate monitoring are available.

### **Continuing and Transitional Care**

Contacting the patient and family after diagnostic testing enables the nurse to determine whether they have any questions about the procedure or whether the patient had any untoward results. Education is reinforced and the patient and family are reminded to make and keep follow-up appointments. Patients,

family members, and health care providers are focused on the immediate needs, issues, or deficits that necessitated the diagnostic testing.

## CRITICAL THINKING EXERCISES

**1 pq** Identify the priorities, approach, and techniques you would use to perform a neurologic assessment on a 32-year-old patient with headaches. How will your priorities, approaches, and techniques differ if the patient has a visual impairment, is hard of hearing, or has lower extremity weakness?

**2 ebp** A 60-year-old patient is scheduled for an MRI scan and tells you he has a pacemaker. What resources would you use to identify whether it is safe for this patient to undergo MRI? What is the evidence base for these practices? Identify the criteria used to evaluate the strength of the evidence for these practices.

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference.

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## Resources

- American Headache Society, [www.americanheadachesociety.org](http://www.americanheadachesociety.org)
- American Stroke Association, [www.stroke.org](http://www.stroke.org)
- Brain Trauma Foundation, [www.braintrauma.org](http://www.braintrauma.org)
- Epilepsy Foundation, [www.epilepsy.com](http://www.epilepsy.com)
- Harvard Health Publications, Harvard Medical School Office of Public Affairs, [www.health.harvard.edu/diagnostic-tests/#brain](http://www.health.harvard.edu/diagnostic-tests/#brain)
- National Headache Foundation, [www.headaches.org](http://www.headaches.org)

# 61 Management of Patients with Neurologic Dysfunction

## LEARNING OUTCOMES

*On completion of this chapter, the learner will be able to:*

1. Describe the causes, clinical manifestations, and medical management of various neurologic dysfunctions.
2. Use the nursing process as a framework for care of the patient with altered level of consciousness.
3. Identify the early and late clinical manifestations of increased intracranial pressure and apply the nursing process as a framework for care of the patient with increased intracranial pressure.
4. Compare and contrast the indications for intracranial or transsphenoidal surgery and use the nursing process as a framework for care of the patient undergoing intracranial or transsphenoidal surgery.
5. Explain the various types and causes of seizures and develop a plan of care for the patient experiencing seizures.
6. Recognize the causes, clinical manifestations, and medical and nursing management of the patient experiencing various types of headaches.

## NURSING CONCEPTS

- Comfort
- Development
- Family
- Health, Wellness, and Illness
- Infection
- Intracranial Regulation
- Mobility
- Patient Education

## GLOSSARY

**akinetic mutism:** unresponsiveness to the environment; the patient makes no movement or sound but sometimes opens the eyes

**altered level of consciousness (LOC):** when a patient is not oriented, does not follow commands, or needs persistent stimuli to achieve a state of alertness

**brain death:** irreversible loss of all functions of the entire brain, including the brain stem

**coma:** prolonged state of unconsciousness

**craniectomy:** a surgical procedure that involves removal of a portion of the skull

**craniotomy:** a surgical procedure that involves entry into the cranial vault

**Cushing's response:** the brain's attempt to restore blood flow by increasing arterial pressure to overcome the increased intracranial pressure (*synonym:* Cushing's reflex)

**decerebration:** an abnormal body posture associated with severe brain injury, characterized by extreme extension of the upper and lower extremities

**decortication:** an abnormal posture associated with severe brain injury, characterized by abnormal flexion of the upper extremities and extension of the lower extremities

**delirium:** an acute, confused state that begins with disorientation and if not recognized and treated early can progress to changes in level of consciousness, irreversible brain damage, and sometimes death

**dementia:** broad term for a syndrome characterized by a general decline in higher brain functioning, such as reasoning, with a pattern of eventual decline in the ability to perform even basic activities of daily living, such as toileting and eating

**epilepsy:** at least two unprovoked seizures occurring more than 24 hours apart

**herniation:** abnormal protrusion of tissue through a defect or natural opening

**intracranial pressure (ICP):** pressure exerted by the volume of the intracranial contents within the cranial vault

**locked-in syndrome:** condition resulting from a lesion in the pons in which the patient lacks all distal motor activity (paralysis) but cognition is intact

**migraine:** a severe, unrelenting headache often accompanied by symptoms such as nausea, vomiting, and visual disturbances

**minimally conscious state:** a state in which the patient demonstrates awareness but cannot communicate thoughts or feelings

**Monro–Kellie hypothesis:** theory that states that due to limited space for expansion within the skull, an increase in any one of the cranial contents—brain tissue, blood, or cerebrospinal fluid (CSF)—causes a change in the volume of the others (*synonym:* Monro–Kellie doctrine)

**persistent vegetative state:** condition in which the patient is wakeful but devoid of conscious content, without cognitive or affective mental function

**primary headache:** a headache for which no specific organic cause can be found

**pseudobulbar affect:** emotional disturbance characterized by uncontrollable episodes of crying or laughing, or other emotional displays

**secondary headache:** headache identified as a symptom of another organic disorder (e.g., brain tumor, hypertension)

**seizures:** paroxysmal transient disturbance of the brain resulting from a discharge of abnormal electrical activity

**status epilepticus:** episode in which the patient experiences multiple seizures with no recovery time in between

**Sudden Unexpected Death in Epilepsy (SUDEP):** nontraumatic, nondrowning unexpected death of patient with epilepsy

**transsphenoidal:** surgical approach to the pituitary via the sphenoid sinuses

This chapter presents an overview of care of the patient with an altered level of consciousness (LOC); the patient with increased intracranial pressure (ICP); and the patient who is undergoing neurosurgical procedures, experiencing seizures, or experiencing headaches. Some of the disorders in this chapter, such as headaches and seizures, may be symptoms of dysfunction in another body system. Alternatively, headaches and seizures can be symptoms of a disruption of the neurologic system. These disorders can also be diagnosed at times as “idiopathic,” or without an identifiable cause. The commonalities of these

disorders are often the behaviors and needs of the patient and the approaches nurses use to support the patient.

The central nervous system (CNS) contains a vast network of neurons that control the body's vital functions. However, this system is vulnerable, and its optimal function depends on several key factors. First, the neurologic system relies on its structural integrity for support and homeostasis, but this integrity may be disrupted. Examples of structural disruption include head injury, brain tumor, intracranial hemorrhage, infection, and stroke. As brain tissue expands in the inflexible cranium, **intracranial pressure (ICP)** (pressure exerted by the volume of the intracranial contents within the cranial vault) rises, and cerebral perfusion is impaired. Further expansion places pressure on vital centers, which can cause permanent neurologic deficits or lead to brain death.

Second, the neurologic system relies on the body's ability to maintain a homeostatic environment. It requires the delivery of the essential elements of oxygen and glucose, as well as filtration of substrates that are toxic to the neurons. The functions of the neurologic system may be decreased or absent because of the effect of toxic substrates or the body's inability to provide essential substrates. Sepsis, hypovolemia, myocardial infarction, cardiopulmonary arrest, hypoglycemia, electrolyte imbalance, drug and/or alcohol overdose, encephalopathy, and ketoacidosis are examples of such circumstances. Some conditions can be treated and reversed; others result in permanent neurologic deficits and disabilities.

Although the specialty of neuroscience nursing requires an understanding of neuroanatomy, neurophysiology, neurodiagnostic testing, critical-care nursing, and rehabilitation nursing, nurses in all settings care for patients with neurologic disorders (Hickey & Strayer, 2020). Ongoing assessment of the patient's neurologic function and health needs, identification of problems, mutual goal setting, development and implementation of care plans (including education, counseling, and coordinating activities), and evaluation of the outcomes of care are nursing actions integral to the recovery of the patient. The nurse also collaborates with other members of the health care team to provide essential care, offer a variety of solutions to problems, help the patient and family gain control of their lives, and explore the educational and supportive resources available in the community. The goals are to achieve as high a level of function as possible and to enhance the quality of life for the patient with neurologic impairment and their family.

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## ALTERED LEVEL OF CONSCIOUSNESS

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An **altered level of consciousness (LOC)** is present when the patient is not oriented, does not follow commands, or needs persistent stimuli to achieve a state of alertness. LOC is gauged on a continuum, with a normal state of alertness and full cognition (consciousness) on one end and coma on the other end. **Coma** is a clinical state of unarousable unresponsiveness in which there are no purposeful responses to internal or external stimuli, although nonpurposeful responses to painful stimuli and brain stem reflexes may be present. The usual duration of coma is variable. **Akinetic mutism** is a state of unresponsiveness to the environment in which the patient makes no voluntary movement. **Persistent vegetative state** is a condition in which the patient who is unresponsive resumes sleep–wake cycles after coma but is devoid of cognitive or affective mental function. A **minimally conscious state** differs from persistent vegetative state in that the patient has inconsistent but reproducible signs of awareness (Rohaut, Eliseyev, & Claassen, 2019). **Locked-in syndrome** results from a lesion affecting the pons and results in paralysis and the inability to speak, but vertical eye movements and lid elevation remain intact and are used to indicate responsiveness. The level of responsiveness and consciousness is the most important indicator of the patient's condition (Owen, 2019).

### Pathophysiology

Altered LOC is not a disorder itself; rather, it is a result of multiple pathophysiologic phenomena. The cause may be neurologic (head injury, stroke), toxicologic (drug overdose, alcohol intoxication), or metabolic (hepatic or kidney injury, diabetic ketoacidosis).

The underlying cause of neurologic dysfunction is disruption in the cells of the nervous system, neurotransmitters, or brain anatomy (see [Chapter 60](#)). Disruptions result from cellular edema or other mechanisms, such as disruption of chemical transmission at receptor sites by antibodies.

Intact anatomic structures of the brain are needed for normal function. The two hemispheres of the cerebrum must communicate, via an intact corpus callosum, and the lobes of the brain (frontal, parietal, temporal, and occipital) must communicate and coordinate their specific functions (see [Chapter 60](#)). Other

anatomic structures of importance are the cerebellum and the brain stem. The cerebellum has both excitatory and inhibitory actions and is largely responsible for coordination of movement. The brain stem contains areas that control the heart rate, respiration, and blood pressure. Disruptions in the anatomic structures result from trauma, edema, pressure from tumors, or other mechanisms, such as an increase or decrease in the circulation of blood or CSF.

## Clinical Manifestations

Alterations in LOC occur along a continuum, and the clinical manifestations depend on where the patient is on this continuum. As the patient's state of alertness and consciousness decreases, changes occur in the pupillary response, eye opening response, verbal response, and motor response. However, initial alterations in LOC may be reflected by subtle behavioral changes, such as restlessness or increased anxiety. The pupils, normally round and quickly reactive to light, become sluggish (response is slower); as the patient becomes comatose, the pupils become fixed (no response to light). The patient in a coma does not open the eyes to voice or command, respond verbally, or move the extremities in response to a request to do so.

## Assessment and Diagnostic Findings

The patient with an altered LOC is at risk for alterations in every body system. A complete assessment is performed, with particular attention to the neurologic system. The neurologic examination should be as complete as the LOC allows. It includes an evaluation of mental status, cranial nerve function, cerebellar function (balance and coordination), reflexes, and motor and sensory function. LOC, a sensitive indicator of neurologic function, is assessed based on the criteria in the Glasgow Coma Scale: eye opening, verbal response, and motor response (Hickey & Strayer, 2020). The patient's responses are rated on a scale from 3 to 15. A score of 3 indicates severe impairment of neurologic function, brain death, or pharmacologic inhibition of the neurologic response. A score of 15 indicates that the patient is fully responsive (see [Chapter 63, Chart 63-4](#)).

If the patient is comatose and has localized signs such as abnormal pupillary and motor responses, it is assumed that neurologic disease is present until proven otherwise. If the patient is comatose but pupillary light reflexes are preserved, a toxic or metabolic disorder is suspected. Common diagnostic procedures used to identify the cause of unconsciousness include computed tomography (CT) scanning, perfusion CT (PCT), magnetic resonance imaging (MRI), magnetic resonance spectroscopy (MRS), and electroencephalography (EEG). Additional procedures include positron emission tomography (PET) and single-photon emission computed tomography (SPECT) (see [Chapter 60](#)). Ongoing research confirms EEG, MRI, and PET as important technologies in determining brain function through the evaluation of metabolic and electrical activity (Rohaut et al., 2019). Laboratory tests include analysis of blood glucose, electrolytes, serum ammonia, and liver function tests; blood urea nitrogen (BUN) levels; serum osmolality; calcium level; and partial thromboplastin and prothrombin times. Other studies may be used to evaluate serum ketones, alcohol and drug concentrations, and arterial blood gases.

## Medical Management

The first priority of treatment for the patient with altered LOC is to obtain and maintain a patent airway. The patient may be orally or nasally intubated, or a tracheostomy may be performed. Until the ability of the patient to breathe is determined, a mechanical ventilator is used to maintain adequate oxygenation and ventilation. The circulatory status (blood pressure, heart rate) is monitored to ensure adequate perfusion to the body and brain. An intravenous (IV) catheter is inserted to provide access for IV fluids and medications. Neurologic care focuses on the specific neurologic pathology, if known. Nutritional support, via a feeding tube or a gastrostomy tube, is initiated as soon as possible. In addition to measures designed to determine and treat the underlying causes of altered LOC, other medical interventions are aimed at pharmacologic management and prevention of complications.

## NURSING PROCESS

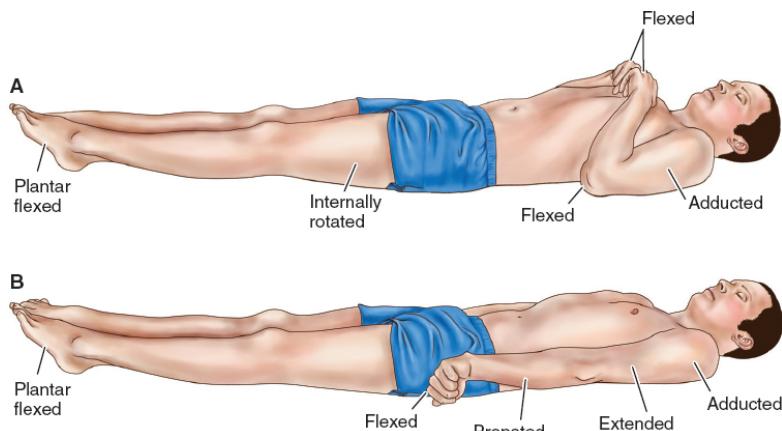
### The Patient with an Altered Level of Consciousness

#### Assessment

Assessment of the patient with an altered LOC often starts with assessing the verbal response through determining the patient's orientation to time, person, and place. Patients are asked to identify the day, date, or season of the year, as well as where they are or the clinicians, family members, or visitors present. Other questions such as "Who is the president?" or "What is the next holiday?" may be helpful in determining the patient's processing of information. Verbal response cannot be evaluated if the patient is intubated or has a tracheostomy, and this should be clearly documented.

Alertness is measured by the patient's ability to open the eyes spontaneously or in response to a vocal or noxious stimulus (pressure or pain). Patients with severe neurologic dysfunction cannot do this. The nurse assesses for periorbital edema (swelling around the eyes) or trauma, which may prevent the patient from opening the eyes, and documents any such condition that interferes with eye opening.

Motor response includes spontaneous, purposeful movement (e.g., the awake patient can move all four extremities with equal strength on command), movement only in response to painful stimuli, or abnormal posturing. If the patient is not responding to commands, the motor response is tested by applying a painful stimulus (firm but gentle pressure) to the nail bed or by squeezing a muscle. If the patient attempts to push away or withdraw, the response is recorded as purposeful or appropriate ("Patient withdraws to painful stimulus"). This response is considered purposeful if the patient can cross the midline from one side of the body to the other in response to a painful stimulus. An inappropriate or nonpurposeful response is random and aimless. Posturing may be decorticate or decerebrate (see Fig. 61-1). The most severe neurologic impairment results in flaccidity. The motor response cannot be elicited or assessed when the patient has been given pharmacologic paralyzing agents (i.e., neuromuscular-blocking agents).



**Figure 61-1 •** Abnormal posture response to stimuli. **A.** Decorticate posturing and flexion of the upper extremities, internal rotation of the lower extremities, and plantar flexion of the feet. **B.** Decerebrate posturing, involving extension and outward rotation of upper extremities and plantar flexion of the feet. Adapted from Posner, J. B., Saper, C. B., Schiff, N. D., et al. (2007). *Plum and Posner's diagnosis of stupor and coma* (4th ed.). Oxford, UK: Oxford University Press.

In addition to LOC, the nurse monitors parameters such as respiratory status, eye signs, and reflexes on an ongoing basis. Table 61-1 summarizes the assessment and the clinical significance of the findings. Body functions (circulation, respiration, elimination, fluid and electrolyte balance) are examined in a systematic and ongoing manner.

#### Diagnosis

#### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired breathing due to neurologic impairment
- Risk for injury associated with lack of adaptive and defensive resources due to decreased LOC

- Risk for hypovolemia associated with inability to take fluids by mouth
- Risk for impaired nutritional intake associated with inability to ingest nutrients to meet metabolic needs
- Impaired oral mucous membrane integrity associated with mouth breathing, absence of pharyngeal reflex, and altered fluid intake
- Risk for impaired skin integrity associated with prolonged immobility
- Risk for injury associated with diminished or absent corneal reflex
- Impaired thermoregulation associated with damage to hypothalamic center
- Impaired urination associated with altered impairment in neurologic sensing and control
- Bowel incontinence associated with impairment in neurologic sensing and control and also associated with changes in nutritional delivery methods
- Impaired health maintenance associated with neurologic impairment
- Interrupted family process associated with health crisis

#### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Respiratory distress or failure
- Pneumonia
- Aspiration
- Pressure injury
- Venous thromboembolism (VTE)
- Contractures

#### **Planning and Goals**

The patient with altered LOC is subject to all of the complications associated with immobility. Therefore, the goals of care for the patient with altered LOC include normalization of breathing, protection from injury, attainment of fluid volume balance, maintenance of nutritional needs, achievement of intact oral mucous membranes, maintenance of normal skin integrity, absence of corneal injury, attainment of effective thermoregulation, and effective urinary elimination. Additional goals include bowel continence, restoration of health maintenance, maintenance of intact family or support system, and absence of complications.

Because the protective reflexes of the patient who is unconscious are impaired, the quality of nursing care provided may mean the difference between life and death. The nurse must assume responsibility for the patient until the basic reflexes (coughing, blinking, and swallowing) return and the patient becomes conscious and oriented. Therefore, the major nursing goal is to compensate for the absence of these protective reflexes.

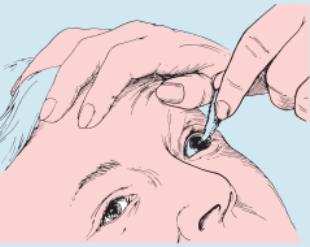
#### **Nursing Interventions**

##### **ACHIEVING AN ADEQUATE BREATHING PATTERN**

The most important consideration in managing the patient with altered LOC is to establish an adequate airway and ensure normalization of the breathing pattern. Obstruction of the airway is a risk because the epiglottis and tongue may relax, occluding the oropharynx, or the patient may aspirate vomitus or nasopharyngeal secretions.

**TABLE 61-1**

## Nursing Assessment of the Patient Who Is Unconscious

Examination	Clinical Assessment	Clinical Significance
Level of responsiveness or consciousness	Eye opening; verbal and motor responses; pupils (size, equality, reaction to light)	Obeying commands is a favorable response and demonstrates a return to consciousness
Pattern of respiration	Respiratory pattern Cheyne–Stokes respiration Hyperventilation Ataxic respiration with irregularity in depth/rate	Disturbances of respiratory center of brain may result in various respiratory patterns Suggests lesions deep in both hemispheres; area of basal ganglia and upper brain stem Suggests onset of metabolic problem or brain stem damage Ominous sign of damage to medullary center
Eyes		
Pupils (size, equality, reaction to light)	 Equal, normally reactive pupils Equal or unequal diameter Progressive dilation Fixed dilated pupils	Suggests that coma is toxic or metabolic in origin Helps determine location of lesion Indicates increasing intracranial pressure Indicates injury at level of midbrain
Eye movements	Normally, eyes should move from side to side	Functional and structural integrity of brain stem is assessed by inspection of extraocular movements; usually absent in deep coma
Corneal reflex	 When cornea is touched with a wisp of clean cotton, blink response is normal	Tests cranial nerves V and VII; helps determine location of lesion if unilateral; absent in deep coma
Facial symmetry	Asymmetry (sagging, decrease in wrinkles)	Sign of paralysis
Swallowing reflex	Drooling versus spontaneous swallowing	Absent in coma Paralysis of cranial nerves X and XII
Neck	Stiff neck Absence of spontaneous neck movement	Subarachnoid hemorrhage, meningitis Fracture or dislocation of cervical spine
Response of extremity to noxious stimuli	Firm pressure on a joint of the upper and lower extremities Observe spontaneous movements	Asymmetric response in paralysis Absent in deep coma
Deep tendon reflexes	Tap patellar and biceps tendons	Brisk response may have localizing value. Asymmetric response in paralysis Absent in deep coma
Pathologic reflexes	 Firm pressure with blunt object on sole of foot, moving along lateral margin and crossing to the ball of foot	Flexion of the toes, especially the great toe, is normal except in newborn Dorsiflexion of toes (especially great toe) indicates contralateral pathology of corticospinal tract (Babinski reflex) Helps determine location of lesion in brain
Abnormal posture	Observation for posturing (spontaneous or in response to noxious stimuli) Flaccidity with absence of motor response	Deep extensive brain lesion Seen with cerebral hemisphere pathology and metabolic depression of brain function Decerebrate posturing indicates deeper and more severe dysfunction than does

Decorticate posture (flexion and internal rotation of forearms and hands)	decorticate posturing; implies brain pathology; poor prognostic sign
Decerebrate posture (extension and external rotation)	

The accumulation of secretions in the pharynx presents a serious problem. Because the patient cannot swallow and lacks pharyngeal reflexes, these secretions must be removed to eliminate the danger of aspiration. Elevating the head of the bed to 30 degrees helps prevent aspiration. Positioning the patient in a lateral or semiprone position also helps because it allows the jaw and tongue to fall forward, thus promoting drainage of secretions.

Positioning alone is not always adequate, however. Suctioning and oral hygiene may be required. Suctioning is performed to remove secretions from the posterior pharynx and upper trachea. Before and after suctioning, the patient is adequately ventilated to prevent hypoxia (Hickey & Strayer, 2020). Chest physiotherapy and postural drainage may be initiated to promote pulmonary hygiene, unless contraindicated by the patient's underlying condition. The chest should be auscultated at least every 8 hours to detect adventitious breath sounds or absence of breath sounds.

Despite these measures, or because of the severity of impairment, the patient with altered LOC often requires intubation and mechanical ventilation. Nursing actions for the patient who is mechanically ventilated include maintaining the patency of the endotracheal tube or tracheostomy, providing frequent oral care, monitoring arterial blood gas measurements, and maintaining ventilator settings (see Chapter 19).

#### PROTECTING THE PATIENT

For the protection of the patient, side rails are padded. Two rails are kept in the raised position during the day and three at night; however, raising all four side rails is considered a restraint by The Joint Commission if the intent is to limit the patient's mobility. Care should be taken to prevent injury from invasive lines and equipment, and other potential sources of injury should be identified, such as restraints, tight dressings, environmental irritants, damp bedding or dressings, and tubes and drains.

Protection also includes ensuring the patient's dignity during altered LOC. Simple measures such as providing privacy and speaking to the patient during nursing care activities preserve the patient's dignity. Not speaking negatively about the patient's condition or prognosis is also important, because patients in a coma may be able to hear. The patient who is comatose has an increased need for advocacy, and the nurse is responsible for seeing that these advocacy needs are met.



#### Quality and Safety Nursing Alert

*If the patient begins to emerge from unconsciousness, every measure that is available and appropriate for calming and quieting the patient should be used. Any form of restraint is likely to be countered with resistance, leading to self-injury or to a dangerous increase in ICP. Therefore, physical restraints are avoided, if possible; a written prescription must be obtained if their use is essential for the patient's well-being.*

#### MAINTAINING FLUID BALANCE AND MANAGING NUTRITIONAL NEEDS

Hydration status is assessed by examining tissue turgor and mucous membranes, assessing intake and output trends, and analyzing laboratory data. Fluid needs are met initially by administering the required IV fluids. However, IV solutions (and blood component therapy) for patients with intracranial conditions must be given slowly. If they are given too rapidly, they can increase ICP. The quantity of fluids given may be restricted to minimize the possibility of cerebral edema.

If the patient does not recover quickly and sufficiently enough to take adequate fluids and calories by mouth, a feeding or gastrostomy tube will be inserted for the administration of fluids and enteral feedings. Research suggests that patients fed within 48 hours of injury have improved outcomes over those in whom nutrition is delayed (Lucke-Wold, Logsdon, Nguyen, et al., 2018).

#### PROVIDING MOUTH CARE

The mouth is inspected for dryness, inflammation, and crusting. The patient who is unconscious requires careful oral care, because there is a risk of parotitis if the mouth is not kept scrupulously clean. The mouth is cleansed and rinsed carefully to remove secretions and crusts and to keep the mucous membranes moist. A thin coating of petrolatum on the lips prevents drying, cracking, and encrustations.

If the patient has an endotracheal tube, the tube should be moved to the opposite side of the mouth daily to prevent ulceration of the mouth and lips. If the patient is intubated and mechanically ventilated, good oral care is also necessary. Research suggests that comprehensive mouth care with antiseptic such as chlorhexidine and head of bed elevation decreases ventilator-associated pneumonia and improves the oral health in patients who are intubated (Malhan, Usman, Trehan, et al., 2019).

#### **MAINTAINING SKIN AND JOINT INTEGRITY**

Preventing skin breakdown requires continuing nursing assessment and intervention. Special attention is given to patients who are unconscious, because they cannot respond to external stimuli. Assessment includes a regular schedule of turning to avoid pressure, which can cause breakdown and necrosis of the skin. Turning also provides kinesthetic (sensation of movement), proprioceptive (awareness of position), and vestibular (equilibrium) stimulation. After turning, the patient is carefully repositioned to prevent ischemic necrosis over pressure areas. Dragging or pulling the patient up in bed must be avoided, because this creates a shearing force and friction on the skin surface.

Maintaining correct body position is important; equally important is passive exercise of the extremities to prevent contractures. The use of splints or foam boots aids in the prevention of footdrop and eliminates the pressure of bedding on the toes. The use of trochanter rolls to support the hip joints keeps the legs in proper alignment. The arms are in abduction, the fingers lightly flexed, and the hands in slight supination. The heels of the feet are assessed for pressure areas. Specialty beds, such as fluidized or low-air-loss beds, may be used to decrease pressure on bony prominences (Hickey & Strayer, 2020).

#### **PRESERVING CORNEAL INTEGRITY**

Some patients who are unconscious have their eyes open and have inadequate or absent corneal reflexes. The cornea may become irritated, dry, or scratched, leading to ulceration. The eyes may be cleansed with cotton balls moistened with sterile normal saline to remove debris and discharge. Artificial tears or methylcellulose may be prescribed to provide lubrication. Periorbital edema often occurs after cranial surgery. If cold compresses are prescribed, care must be exerted to avoid contact with the cornea. Eye patches should be used cautiously because of the potential for corneal abrasion from contact with the patch; eye shields may provide eye protection with less risk of injury.

#### **MAINTAINING BODY TEMPERATURE**

High fever in the patient who is unconscious may be caused by infection of the respiratory or urinary tract, drug reactions, or damage to the hypothalamic temperature-regulating center. A slight elevation of temperature may be caused by dehydration. The environment can be adjusted, depending on the patient's condition, to promote a normal body temperature. If body temperature is elevated, a minimum amount of bedding is used. The room may be cooled to 18.3°C (65°F). However, if the patient is an older adult and does not have an elevated temperature, a warmer environment is needed.

Because of damage to the temperature-regulating center in the brain or severe intracranial infection, patients who are unconscious often develop very high temperatures. Such temperature elevations must be controlled, because the increased metabolic demands of the brain can exceed cerebral circulation and oxygen delivery, potentially resulting in cerebral deterioration (Hickey & Strayer, 2020). Studies suggest that hyperthermia may contribute to poor outcome after brain injury but not through a decreased brain oxygen level (Rincon, 2018). Persistent hyperthermia with no identified clinical source of infection indicates brain stem damage and a poor prognosis.



#### **Quality and Safety Nursing Alert**

*The body temperature of a patient who is unconscious is never taken by mouth. Rectal, tympanic (if not contraindicated), or core temperature measurement is preferred to the less accurate axillary temperature.*

Strategies for reducing fever include:

- Removing all bedding over the patient (with the possible exception of a light sheet, towel, or small drape)
- Administering acetaminophen or ibuprofen as prescribed
- Giving cool sponge baths
- Using a hypothermia blanket

- Monitoring temperature frequently to assess the patient's response to the therapy and to prevent an excessive decrease in temperature and shivering

#### **PREVENTING URINARY RETENTION**

The patient with an altered LOC is often incontinent or has urinary retention. The bladder is palpated or scanned at intervals to determine whether urinary retention is present, because a full bladder may be an overlooked cause of overflow incontinence. A portable bladder ultrasound instrument is a useful tool in bladder management and retraining programs.

If the patient is not voiding, a program of intermittent catheterization should be devised in order to reduce the patient's risk of urinary tract infection. A catheter may be inserted during the acute phase of illness to monitor urinary output. Because catheters are a major cause of urinary tract infection, the patient is observed for fever and cloudy urine. The area around the urethral orifice is inspected for drainage and cleansed routinely. The urinary catheter is usually removed if the patient has a stable cardiovascular system and if no diuresis, sepsis, or voiding dysfunction existed before the onset of coma. Although many patients who are unconscious urinate spontaneously after catheter removal, the bladder should be scanned with a portable bladder ultrasound device periodically for urinary retention (see [Chapter 47](#), [Fig. 47-8](#)).

An external catheter (condom catheter) for the male patient and absorbent pads or female incontinence device for the female patient can be used for patients who are unconscious and can urinate spontaneously, although involuntarily. As soon as consciousness is regained, a bladder training program is initiated (Hickey & Strayer, 2020). The patient who is incontinent is monitored frequently for skin irritation and skin breakdown. Appropriate skin care is implemented to prevent these complications.

#### **PROMOTING BOWEL FUNCTION**

The abdomen is assessed for distention by listening for bowel sounds and measuring the girth of the abdomen with a tape measure. There is a risk of diarrhea from infection, antibiotic agents, and hyperosmolar fluids. Frequent loose stools may also occur with fecal impaction. Commercial fecal collection bags are available for patients with fecal incontinence (see [Chapter 41](#), [Fig. 41-1](#)).

Immobility and lack of dietary fiber can cause constipation. The nurse monitors the number and consistency of bowel movements and performs a rectal examination for signs of fecal impaction. Stool softeners may be prescribed and can be given with tube feedings. To facilitate bowel emptying, a glycerin suppository or bowel stimulant may be indicated. The patient may require an enema routinely to empty the lower colon.

#### **RESTORING HEALTH MAINTENANCE**

Once increased ICP is not a problem, the nurse assists the patient and family to restore the health of the patient who is unconscious. This involves using auditory, visual, olfactory, gustatory, tactile, and kinesthetic activities to stimulate the patient emerging from coma (Gattuta, Coralo, Lo Buono, et al., 2018). Efforts are made to restore the sense of daily rhythm by maintaining usual day and night patterns for activity and sleep. The nurse touches and talks to the patient and encourages family members and friends to do so. Communication is extremely important and includes touching the patient and spending enough time with the patient to become sensitive to their needs. It is also important to avoid making any negative comments about the patient's status or prognosis in the patient's presence.

The nurse orients the patient to time and place at least once every 8 hours. Sounds from the patient's usual environment may be introduced using a recording. Family members can read to the patient from a favorite book and may suggest radio and television programs that the patient previously enjoyed as a means of enriching the environment and providing familiar input.

When arousing from coma, many patients experience a period of agitation, indicating that they are becoming more aware of their surroundings but still cannot react or communicate in an appropriate fashion. Although this is disturbing for many family members, it is actually a positive clinical sign. At this time, it is necessary to minimize stimulation by limiting background noises, having only one person speak to the patient at a time, giving the patient a longer period of time to respond, and allowing for frequent rest or quiet times. After the patient has regained consciousness, recordings of family or social events may assist the patient in recognizing family and friends and allow them to experience missed events.

Programs of sensory stimulation for patients with brain injury have been developed in an effort to improve outcomes. Although these are controversial programs with inconsistent results, some support the concept of providing structured neurostimulation (Hickey & Strayer, 2020).

#### **MEETING THE FAMILY'S NEEDS**

The family of the patient with altered LOC may be thrown into a sudden state of crisis and go through the process of severe anxiety, denial, anger, remorse, grief, and reconciliation. Depending on the disorder that caused the altered LOC and the extent of the patient's recovery, the family may be unprepared for the changes in the cognitive and physical status of their loved one. If the patient has significant residual deficits, the family may require considerable time, assistance, and support to come to terms with these changes. To help family members mobilize resources and coping skills, the nurse reinforces and clarifies information about the patient's condition, encourages the family to be involved in care, and listens to and encourages sharing of feelings and concerns while supporting decision making about management and placement after hospitalization. Families may benefit from participation in support services offered through the hospital, rehabilitation facility, or community organizations.

The family may need to face the death of their loved one. The patient with a neurologic disorder is often pronounced brain dead before the heart stops beating. The term **brain death** describes irreversible loss of all functions of the entire brain and absence of brain stem reflexes (Milliken & Uveges, 2020). The term may be misleading to the family because, although brain function has ceased, the patient appears to be alive, with the heart rate and blood pressure sustained by vasoactive medications and breathing continued by mechanical ventilation. When discussing a patient who is brain dead with family members, it is important to provide accurate, timely, understandable, and consistent information. See [Chapter 13](#) for discussion of end-of-life care.

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Pneumonia, aspiration, and respiratory failure are potential complications in any patient who has a depressed LOC and who cannot protect the airway or turn, cough, and take deep breaths. The longer the period of unconsciousness, the greater the risk of pulmonary complications.

Vital signs and respiratory function are monitored closely to detect any signs of respiratory failure or distress. Complete blood count and arterial blood gas measurements are assessed to determine whether there are adequate red blood cells to carry oxygen and whether ventilation is effective. Chest physiotherapy and suctioning are initiated to prevent respiratory complications such as pneumonia. Oral care interventions are performed for patients receiving mechanical ventilation to maintain oral health and decrease the incidence of pneumonia (Malhan et al., 2019). If pneumonia develops, cultures are obtained to identify the organism so that appropriate antibiotic agents can be given.

The patient with altered LOC is monitored closely for evidence of impaired skin integrity, and strategies to prevent skin breakdown and pressure injuries are continued through all phases of care, including hospitalization, rehabilitation, and home care. Factors that contribute to impaired skin integrity (e.g., incontinence, inadequate dietary intake, pressure on bony prominences, edema) are addressed. If pressure injuries develop, strategies to promote healing are undertaken. Care is taken to prevent bacterial contamination of pressure injuries, which may lead to sepsis and septic shock. See [Chapter 56](#) for assessment and management of pressure injuries.

The patient should also be monitored for signs and symptoms of VTE, which may manifest as a deep vein thrombosis (DVT) or pulmonary embolism (PE). Prophylaxis with subcutaneous heparin or low-molecular-weight heparin (dalteparin, danaparoid) as well as anti-embolism stockings or pneumatic compression devices are prescribed according to the patient's risk factors for thrombosis and bleeding (Galan, Egea-Guerrero, Diaz, et al., 2016). The nurse observes for signs and symptoms of DVT or PE.

Patients with a prolonged decrease in LOC are at risk for developing contractures. During acute care, the patient is turned every 2 hours and passive range of motion performed at least twice a day. Splints, provided by occupational therapy, are applied to the hands and feet in a rotating manner to maintain functional joint alignment. Hand splints have been reported to be safe and beneficial for patients in decreasing spasticity and improving hand opening (Khan, Amatya, Bensmail, et al., 2019).

#### **Evaluation**

Expected patient outcomes may include:

1. Attains optimal breathing pattern
2. Experiences no injuries
3. Attains or maintains adequate fluid balance and nutritional status
  - a. Has no clinical signs or symptoms of dehydration
  - b. Demonstrates normal range of serum electrolytes
  - c. Has no clinical signs or symptoms of overhydration or malnutrition
4. Achieves healthy oral mucous membranes

5. Maintains intact skin
6. Has no corneal injury
7. Attains or maintains thermoregulation
8. Has no urinary retention
9. Has no diarrhea or fecal impaction
10. Receives appropriate sensory stimulation
11. Has family members who cope with crisis
  - a. Verbalize fears and concerns
  - b. Participate in patient's care and provide sensory stimulation by talking and touching
12. Is free of complications
  - a. Has arterial blood gas values or oxygen saturation levels within normal range
  - b. Displays no signs or symptoms of pneumonia
  - c. Exhibits intact skin over pressure areas
  - d. Does not develop VTE such as DVT or PE



## INCREASED INTRACRANIAL PRESSURE

The rigid cranial vault contains brain tissue (1400 g), blood (75 mL), and CSF (75 mL). The volume and pressure of these three components are usually in a state of equilibrium and produce the ICP. ICP is usually measured in the lateral ventricles, with the normal pressure being 0 to 10 mm Hg, and 15 mm Hg being the upper limit of normal (Hickey & Strayer, 2020).

The **Monro–Kellie hypothesis**, also known as the Monro–Kellie doctrine, explains the dynamic equilibrium of cranial contents. The hypothesis states that because of the limited space for expansion within the skull, an increase in any one of the components causes a change in the volume of the others (Witherspoon & Ashby, 2017). Because brain tissue has limited space to expand, compensation typically is accomplished by displacing or shifting CSF, increasing the absorption or diminishing the production of CSF, or decreasing cerebral blood volume. Without such changes, ICP begins to rise. Under normal circumstances, minor changes in blood volume and CSF volume occur constantly as a result of alterations in intrathoracic pressure (coughing, sneezing, straining), posture, blood pressure, and systemic oxygen and carbon dioxide levels.

### Pathophysiology

Increased ICP affects many patients with acute neurologic conditions because pathologic conditions alter the relationship between intracranial volume and ICP. Although elevated ICP is most commonly associated with head injury, it also may be seen as a secondary effect in other conditions, such as brain tumors, subarachnoid hemorrhage, and toxic and viral encephalopathies. Increased ICP from any cause decreases cerebral perfusion, stimulates further edema (swelling), and may shift brain tissue, resulting in herniation—a dire and frequently fatal event.

### Decreased Cerebral Blood Flow

Increased ICP may reduce cerebral blood flow, resulting in ischemia and cell death. In the early stages of cerebral ischemia, the vasomotor centers are stimulated and the systemic pressure rises to maintain cerebral blood flow. Usually, this is accompanied by a slow bounding pulse and respiratory irregularities. These changes in blood pressure, pulse, and respiration are important clinically because they suggest increased ICP.

The concentration of carbon dioxide in the blood and in the brain tissue also plays a role in the regulation of cerebral blood flow. An increase in the partial pressure of arterial carbon dioxide ( $\text{PaCO}_2$ ) causes cerebral vasodilation, leading to increased cerebral blood flow and increased ICP. A decrease in

$\text{PaCO}_2$  has a vasoconstrictive effect, limiting blood flow to the brain. Decreased venous outflow may also increase cerebral blood volume, thus raising ICP.

### Cerebral Edema

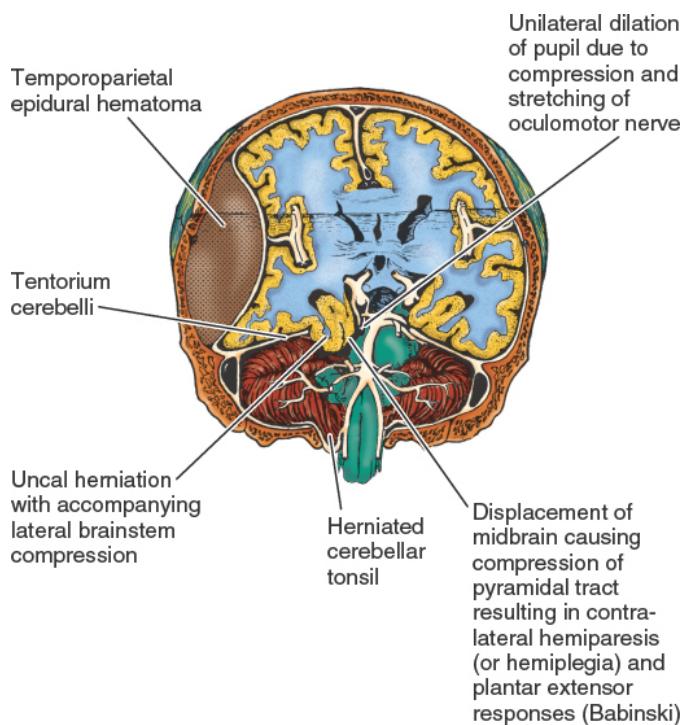
Cerebral edema or swelling is defined as an abnormal accumulation of water or fluid in the intracellular space, extracellular space, or both, associated with an increase in the volume of brain tissue. Edema can occur in the gray, white, or interstitial matter. As brain tissue swells within the rigid skull, several mechanisms attempt to compensate for the increasing ICP. These compensatory mechanisms include autoregulation as well as decreased production and flow of CSF. **Autoregulation** refers to the brain's ability to change the diameter of its blood vessels to maintain a constant cerebral blood flow during alterations in systemic blood pressure. This mechanism can be impaired in patients who are experiencing a pathologic and sustained increase in ICP.

### Cerebral Response to Increased Intracranial Pressure

As ICP rises, compensatory mechanisms in the brain work to maintain blood flow and prevent tissue damage. The brain can maintain a steady perfusion pressure if the arterial systolic blood pressure is 50 to 150 mm Hg and the ICP is less than 40 mm Hg. Changes in ICP are closely linked with cerebral perfusion pressure (CPP). The CPP is calculated by subtracting the ICP from the mean arterial pressure (MAP). For example, if the MAP is 100 mm Hg and the ICP is 15 mm Hg, then the CPP is 85 mm Hg. The normal CPP is 70 to 100 mm Hg (Hickey & Strayer, 2020). As ICP rises and the autoregulatory mechanism of the brain is overwhelmed, the CPP can increase to greater than 100 mm Hg or decrease to less than 50 mm Hg. Patients with a CPP of less than 50 mm Hg experience irreversible neurologic damage. Therefore, the CPP must be maintained at 70 to 80 mm Hg to ensure adequate blood flow to the brain. If ICP is equal to MAP, cerebral circulation ceases.

A clinical phenomenon known as the **Cushing's response** (also called Cushing's reflex) is seen when cerebral blood flow decreases significantly. When ischemic, the vasomotor center triggers an increase in arterial pressure in an effort to overcome the increased ICP. A sympathetically mediated response causes an increase in the systolic blood pressure with a widening of the pulse pressure and cardiac slowing. This response is seen clinically as an increase in systolic blood pressure, widening of the pulse pressure, and reflex slowing of the heart rate. It is a late sign requiring immediate intervention; however, perfusion may be recoverable if the Cushing's response is treated rapidly.

At a certain point, the brain's ability to autoregulate becomes ineffective and decompensation (ischemia and infarction) begins. When this occurs, the patient exhibits significant changes in mental status and vital signs. The bradycardia, hypertension, and bradypnea associated with this deterioration are known as Cushing's triad, which is a grave sign. At this point, herniation of the brain stem and occlusion of the cerebral blood flow occur if therapeutic intervention is not initiated. **Herniation** refers to the shifting of brain tissue from an area of high pressure to an area of lower pressure (see Fig. 61-2). The herniated tissue exerts pressure on the brain area into which it has shifted, which interferes with the blood supply in that area. Cessation of cerebral blood flow results in cerebral ischemia, infarction, and brain death.



**Figure 61-2 •** Cross-section of the brain showing herniation of part of the temporal lobe through the tentorium as a result of a temporoparietal epidural hematoma. Reprinted with permission from Kintzel, K. C. (1977). *Advanced concepts in clinical nursing*. Philadelphia, PA: J. B. Lippincott.

## Clinical Manifestations

If ICP increases to the point at which the brain's ability to adjust has reached its limits, neural function is impaired; this may be manifested at first by clinical changes in LOC and later by abnormal respiratory and vasomotor responses.



### Quality and Safety Nursing Alert

The earliest sign of increasing ICP is a change in LOC. Agitation, slowing of speech, and delay in response to verbal suggestions may be early indicators.

Any sudden change in the patient's condition, such as restlessness (without apparent cause), confusion, or increasing drowsiness, has neurologic significance. These signs may result from compression of the brain due to swelling from hemorrhage or edema, an expanding intracranial lesion (hematoma or tumor), or a combination of both.

As ICP increases, the patient becomes stuporous, reacting only to loud or painful stimuli. At this stage, serious impairment of brain circulation is probably taking place, and immediate intervention is required. As neurologic function deteriorates further, the patient becomes comatose and exhibits abnormal motor responses in the form of **decortication** (abnormal flexion of the upper extremities and extension of the lower extremities), **decerebration** (extreme extension of the upper and lower extremities), or flaccidity (see Fig. 61-1). If the coma is profound and irreversible with no known confounding factors, brain stem reflexes are absent, and respirations are impaired or absent, the patient may be evaluated for brain death (Milliken & Uveges, 2020).

## Assessment and Diagnostic Findings

The diagnostic studies used to determine the underlying cause of increased ICP are discussed in detail in Chapter 60. The most common diagnostic tests are CT scanning and MRI. The patient may also undergo

cerebral angiography, PET, or SPECT. Transcranial Doppler studies provide information about cerebral blood flow. The patient with increased ICP may also undergo electrophysiologic monitoring to observe cerebral blood flow indirectly. Evoked potential monitoring measures the electrical potentials produced by nerve tissue in response to external stimulation (auditory, visual, or sensory). Lumbar puncture is avoided in patients with increased ICP, because the sudden release of pressure in the lumbar area can cause the brain to herniate (Hickey & Strayer, 2020). See [Chapter 60](#) for further discussion of lumbar puncture and other diagnostic tests.

## Complications

Complications of increased ICP include brain stem herniation, diabetes insipidus, and syndrome of inappropriate antidiuretic hormone (SIADH).

Brain stem herniation results from an excessive increase in ICP in which the pressure builds in the cranial vault and the brain tissue presses down on the brain stem. This increasing pressure on the brain stem results in cessation of blood flow to the brain, leading to irreversible brain anoxia and brain death.

Neurogenic diabetes insipidus is the result of decreased secretion of antidiuretic hormone (ADH). The patient has excessive urine output, decreased urine osmolality, and serum hyperosmolarity (Tudor & Thompson, 2019). Therapy consists of administration of fluids, electrolyte replacement, and administration of a synthetic vasopressin (desmopressin). See [Chapters 10](#) and [45](#) for a discussion of diabetes insipidus.

SIADH is the result of increased secretion of ADH. The patient becomes volume overloaded, urine output diminishes, and serum sodium concentration becomes dilute. Treatment of SIADH includes fluid restriction (less than 800 mL/day with no free water), which is usually sufficient to correct the hyponatremia. In severe cases, careful administration of a 3% hypertonic saline solution may be therapeutic (Hickey & Strayer, 2020). The change in serum sodium concentration should not exceed a correction rate of approximately 1.3 mEq/L/h. See [Chapters 10](#) and [45](#) for further discussion of SIADH.

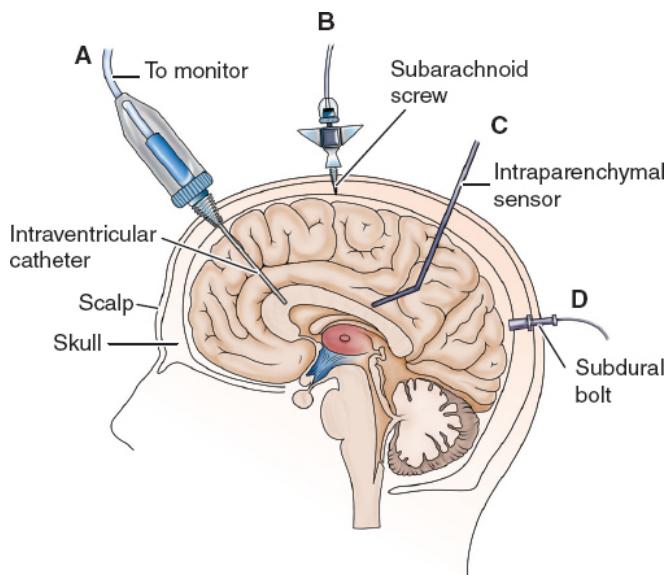
## Medical Management

Increased ICP is a true emergency and must be treated promptly. Invasive monitoring of ICP is an important component of management. Immediate management to relieve increased ICP requires decreasing cerebral edema, lowering the volume of CSF, or decreasing cerebral blood volume while maintaining cerebral perfusion. These goals are accomplished by administering osmotic diuretics, restricting fluids, draining CSF, controlling fever, maintaining systemic blood pressure and oxygenation, and reducing cellular metabolic demands. See [Chapter 63](#) for a discussion of the management of increased ICP.

### Monitoring Intracranial Pressure and Cerebral Oxygenation

The purposes of ICP monitoring are to identify increased pressure early in its course (before cerebral damage occurs), to quantify the degree of elevation, to initiate appropriate treatment, to provide access to CSF for sampling and drainage, and to evaluate the effectiveness of treatment. ICP can be monitored with the use of an intraventricular catheter (ventriculostomy), a subarachnoid bolt, an epidural or subdural catheter, or a fiberoptic transducer-tipped catheter placed in the subdural space or in the ventricle (see [Fig. 61-3](#)).

When a ventriculostomy or intraventricular catheter monitoring device is used for monitoring ICP, a fine-bore catheter is inserted into a lateral ventricle, preferably in the nondominant hemisphere of the brain (Hickey & Strayer, 2020). The catheter is connected by a fluid-filled system to a transducer, which records the pressure in the form of an electrical impulse. In addition to obtaining continuous ICP recordings, the ventricular catheter allows CSF to drain, particularly during acute increases in pressure. The ventriculostomy can also be used to drain blood from the ventricle. Continuous drainage of CSF under pressure control is an effective method of treating intracranial hypertension. Another advantage of a ventricular catheter is access for the intraventricular administration of medications and the occasional instillation of air or a contrast agent for ventriculography. Complications associated with its use include infection, meningitis, ventricular collapse, occlusion of the catheter by brain tissue or blood, and problems with the monitoring system.



**Figure 61-3 •** Intracranial pressure monitoring. A device may be placed in the ventricle (A), the subarachnoid space (B), the intraparenchymal space (C), or the subdural space (D).

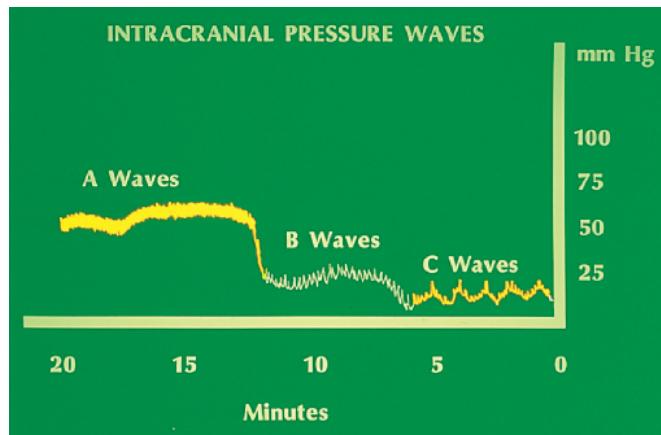
The subarachnoid screw or bolt is a hollow device that is inserted through the skull and dura mater into the cranial subarachnoid space (Hickey & Strayer, 2020). It has the advantage of not requiring a ventricular puncture. The subarachnoid screw is attached to a pressure transducer, and the output is recorded on an oscilloscope. The hollow screw technique also has the advantage of avoiding complications from brain shift and small ventricle size. Complications include infection and blockage of the screw by clot or brain tissue, which leads to a loss of pressure tracing and a decrease in accuracy at high ICP readings.

An epidural monitor uses a pneumatic flow sensor to detect ICP. The epidural ICP monitoring system has a low incidence of infection and complications and appears to read pressures accurately. Calibration of the system is maintained automatically, and abnormal pressure waves trigger an alarm system. One disadvantage of the epidural catheter is the inability to withdraw CSF for analysis.

A fiberoptic monitor, or transducer-tipped catheter, is an alternative to other intraventricular, subarachnoid, and subdural systems (Al-Mufti, Smith, Lander, et al., 2018). The miniature transducer reflects pressure changes, which are converted to electrical signals in an amplifier and displayed on a digital monitor. The catheter can be inserted into the ventricle, subarachnoid space, subdural space, or brain parenchyma or under a bone flap. If inserted into the ventricle, it can also be used in conjunction with a CSF drainage device.

### Interpreting Intracranial Pressure Waveforms

Waves of high pressure and troughs of relatively normal pressure indicate changes in ICP. Waveforms are captured and recorded on an oscilloscope. These waves have been classified as A waves (plateau waves), B waves, and C waves (see Fig. 61-4). The plateau waves (A waves) are transient, paroxysmal, recurring elevations of ICP that may last 5 to 20 minutes and range in amplitude from 40 to 100 mm Hg (Al-Mufti et al., 2018). Plateau waves have clinical significance and indicate changes in vascular volume within the intracranial compartment that are beginning to compromise cerebral perfusion. The A waves may increase in amplitude and frequency, reflecting cerebral ischemia and brain damage that can occur before overt signs and symptoms of raised ICP are seen clinically. B waves are shorter (30 seconds to 2 minutes) and have smaller amplitude (up to 50 mm Hg). They have less clinical significance, but if seen in a series in a patient with depressed consciousness, they may precede the appearance of A waves. B waves may be seen in patients with intracranial hypertension and decreased intracranial compliance. C waves are small, rhythmic oscillations with frequencies of 4 to 8 per minute and appear to be related to rhythmic variations of the systemic arterial blood pressure and respirations (Hickey & Strayer, 2020).



**Figure 61-4 •** Intracranial pressure waves. Composite diagram of A (plateau) waves, which indicate cerebral ischemia; B waves, which indicate intracranial hypertension and variations in the respiratory cycle; and C waves, which relate to variations in systemic arterial pressure and respirations.

### Other Neurologic Monitoring Systems

Another trend in neurologic monitoring is microdialysis of the patient with a brain injury (Zhou & Kalanuria, 2018). Cortical probes are placed near the injured area and are used to measure levels of glutamate, lactate, pyruvate, and glucose, substances that reflect the metabolic function of the brain. Some researchers theorize that direct measurements of glucose and energy by-products in the brain will lead to better management of these patients. Although cerebral microdialysis has reduced the mortality of patients who are brain injured, more study is needed to link it to improved outcomes (Zhou & Kalanuria, 2018).

An additional trend is monitoring of cerebral oxygenation through monitoring of the oxygen saturation in the jugular venous bulb ( $S_{jv}O_2$ ) or via a catheter in the brain. Cerebral oxygenation is thought to be important because changes in cerebral perfusion may reflect an increase in ICP. Readings taken from a catheter residing in the jugular outflow tract allow for a comparison of arterial and venous oxygen saturation, and the balance of cerebral oxygen supply and demand is demonstrated. Venous jugular desaturations can reflect early cerebral ischemia, alerting the clinician before an increase in ICP occurs. Minimizing cerebral desaturations can potentially improve outcomes. This type of monitoring is now widely available and has been successfully used to identify secondary brain insults. A limiting factor is that this saturation reflects overall perfusion of the brain rather than that of a specific injured area (Al-Mufti et al., 2018).

Another method of measuring cerebral oxygenation and temperature is by inserting a fiberoptic catheter into the brain matter. The most common system is Licox (see Fig. 61-5). The system includes a monitor with a screen for the display of oxygen and temperature values and cables that connect to the monitoring probes in the brain (Hickey & Strayer, 2020).

### Decreasing Cerebral Edema

Osmotic diuretics such as mannitol and hypertonic saline (3%) may be administered to decrease fluid in the brain tissue and reduce cerebral edema (Witherspoon & Ashby, 2017). These agents act by drawing water across intact membranes, thereby reducing the volume of the brain and extracellular fluid. An indwelling urinary catheter is usually inserted to monitor urinary output and to manage the resulting diuresis. If the patient is receiving osmotic diuretics, serum osmolality and electrolytes should be determined to assess hydration status. If a brain tumor is the cause of the increased ICP, corticosteroids (e.g., dexamethasone) help reduce the edema surrounding the tumor.



**Figure 61-5 •** The Licox brain tissue oxygen monitor. Permission granted by Integra LifeSciences Corporation, Princeton, New Jersey, USA.

Another method for decreasing cerebral edema is fluid restriction (Hickey & Strayer, 2020). Limiting overall fluid intake leads to dehydration and hemoconcentration, which draws fluid across the osmotic gradient and decreases cerebral edema. Conversely, overhydration of the patient with increased ICP is avoided, because it increases cerebral edema.

Researchers have long hypothesized that lowering body temperature would decrease cerebral edema by reducing the oxygen and metabolic requirements of the brain, thus protecting the brain from continued ischemia. If body metabolism can be reduced by lowering the body temperature, the collateral circulation in the brain may be able to provide an adequate blood supply to the brain. The effect of hypothermia on ICP requires more study; thus far, induced hypothermia has not consistently been shown to be beneficial for patients with brain injury. Inducing and maintaining hypothermia is a major clinical treatment and requires knowledge and skilled nursing observation and management. The type and length of rewarming techniques after hypothermia may also be factors in the outcome of patients with neurologic injuries (Rincon, 2018).

### Maintaining Cerebral Perfusion

Cardiac output may be manipulated to provide adequate perfusion to the brain. Improvements in cardiac output are made using fluid volume and inotropic agents such as dobutamine and norepinephrine. The effectiveness of the cardiac output is reflected in the CPP, which is maintained at greater than 70 mm Hg. A lower CPP indicates that the cardiac output is insufficient to maintain adequate cerebral perfusion. SjvO<sub>2</sub> and Licox, described earlier, assist in monitoring cerebral perfusion.

Decompressive hemicraniectomy may also be considered as a surgical strategy to assist in the management of refractory intracranial hypertension. The removal of a part of the skull allows the brain to expand without the pressure constraints exerted by the cranial vault. Complications of this procedure include infection and increased potential for injury to the unprotected underlying brain structures. Once the patient is no longer at risk for increased ICP, the bone flap may be surgically replaced (Hutchinson, Koliás, Tajsic, et al., 2019).

### Reducing Cerebrospinal Fluid and Intracranial Blood Volume

CSF drainage is frequently performed, because the removal of CSF with a ventriculostomy drain can dramatically reduce ICP and restore CPP. Caution should be used in draining CSF, however, because excessive drainage may result in collapse of the ventricles and herniation. The reduction in PaCO<sub>2</sub> may result in hypoxia, ischemia, and an increase in cerebral lactate levels. Maintaining the PaCO<sub>2</sub> at greater than 30 mm Hg may prove beneficial (Hickey & Strayer, 2020).

### Controlling Fever

Preventing a temperature elevation is critical, because fever increases cerebral metabolism and the rate at which cerebral edema forms. Strategies to reduce body temperature include administration of antipyretic medications, as prescribed, and the use of a hypothermia blanket. Additional strategies for reducing fever were discussed previously in the Nursing Process section on altered LOC. The patient's temperature is monitored closely, and the patient is observed for shivering, which should be avoided because it is associated with increased oxygen consumption, increased levels of circulating catecholamines, and increased vasoconstriction. Shivering is associated with decreased levels of brain oxygenation; however, the association between shivering and neurologic outcome is unknown.

### Maintaining Oxygenation and Reducing Metabolic Demands

Arterial blood gases and pulse oximetry are monitored to ensure that systemic oxygenation remains optimal. Metabolic demands may be reduced through the administration of high doses of barbiturates if the patient is unresponsive to conventional treatment. The mechanism by which barbiturates decrease ICP and protect the brain is uncertain, but the resultant comatose state is thought to reduce the metabolic requirements of the brain, thus providing cerebral protection.

Another method of reducing cellular metabolic demand and improving oxygenation is the administration of medications causing sedation. The patient who receives these agents cannot move; this decreases the metabolic demands and results in a decrease in cerebral oxygen demand. The patient cannot respond to or report pain either. The most common agents used for sedation are pentobarbital, thiopental, propofol, and dexmedetomidine (Opdenakker, Vanstraelen, De Sloovere, et al., 2019).

If sedative agents are used, the ability to perform serial neurologic assessments is lost. Therefore, other monitoring tools are needed to assess the patient's status and response to therapy. Important parameters that must be assessed include ICP, blood pressure, heart rate, respiratory rate, and the patient's response to ventilator therapy (e.g., patient-ventilator dyssynchrony; see [Chapter 19](#)). The level of pharmacologic paralysis is adjusted based on serum levels of the medications given and the assessed parameters. Potential complications of these medications include hypotension caused by decreased sympathetic tone and myocardial depression.

Patients receiving high doses of barbiturates or pharmacologic sedatives require continuous cardiac monitoring, endotracheal intubation, mechanical ventilation, and arterial pressure monitoring, as well as ICP monitoring.

## NURSING PROCESS

### The Patient with Increased Intracranial Pressure



#### Assessment

Initial assessment of the patient with increased ICP includes obtaining a history of events leading to the present illness and the pertinent past medical history. It is usually necessary to obtain this information from family or friends. The neurologic examination should be as complete as the patient's condition allows. It includes an evaluation of mental status, LOC, cranial nerve function, cerebellar function (balance and coordination), reflexes, and motor and sensory function. Because the patient is critically ill, ongoing assessment is more focused, including pupil checks, assessment of selected cranial nerves, frequent measurements of vital signs and ICP, and the use of the Glasgow Coma Scale (see [Table 61-1](#)).

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses include the following:

- Impaired breathing associated with neurologic dysfunction (brain stem compression, structural displacement)
- Risk for ineffective tissue perfusion associated with the effects of increased ICP
- Hypovolemia associated with fluid restriction
- Risk for infection associated with ICP monitoring system (fiberoptic or intraventricular catheter)

Other relevant nursing diagnoses are included in the earlier section on altered LOC.

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Brain stem herniation
- Diabetes insipidus
- SIADH

#### Planning and Goals

The goals for the patient include normalization of respiration, adequate cerebral tissue perfusion through reduction in ICP, restoration of fluid balance, absence of infection, and absence of complications.

#### Nursing Interventions

##### ACHIEVING AN ADEQUATE BREATHING PATTERN

In order to normalize breathing it is essential to maintain the airway. The patency of the airway is assessed. Secretions that are obstructing the airway must be suctioned with care, because transient elevations of ICP occur with suctioning (Hickey & Strayer, 2020). Hypoxia caused by poor oxygenation leads to cerebral ischemia and edema. Coughing is discouraged because it increases ICP. The lung fields are auscultated at least every 8 hours to determine the presence of adventitious sounds or any areas of congestion. Elevating the head of the bed may aid in clearing secretions and improve venous drainage of the brain.

The patient must be monitored for respiratory irregularities. Increased pressure on the frontal lobes or deep midline structures may result in Cheyne–Stokes respirations, whereas pressure in the midbrain can cause hyperventilation. If the lower portion of the brain stem (the pons and medulla) is involved, respirations become irregular and eventually cease.

There is ongoing controversy about the use of hyperventilation therapy in traumatic brain injury. This therapy is used in some circumstances to reduce ICP by causing cerebral vasoconstriction and a decrease in cerebral blood volume. The nurse collaborates with the respiratory therapist in monitoring the PaCO<sub>2</sub>, which is usually maintained at less than 30 mm Hg. Employing hyperventilation should follow guidelines for management of TBI as it involves risk of cerebral vasoconstriction and ischemia (Saherwala, Bader, Stutzman, et al., 2018). Patients undergoing hyperventilation therapy also benefit from multimodality monitoring to determine the overall effect of this therapy on brain perfusion (Hickey & Strayer, 2020).

A neurologic observation record (see Fig. 61-6) is maintained, and all observations are made in relation to the patient's baseline condition. Repeated assessments of the patient are made (sometimes minute by minute) so that improvement or deterioration may be noted immediately. If the patient's condition deteriorates, the primary provider is notified emergently and preparations are made for surgical intervention.

#### OPTIMIZING CEREBRAL TISSUE PERFUSION

In addition to ongoing nursing assessment, strategies are initiated to reduce factors contributing to the elevation of ICP (see Table 61-2).

Proper positioning helps reduce ICP. The patient's head is kept in a neutral (midline) position, maintained with the use of a cervical collar if necessary, to promote venous drainage. Elevation of the head is maintained at 30 to 45 degrees unless contraindicated. Extreme rotation of the neck and flexion of the neck are avoided, because compression or distortion of the jugular veins increases ICP. Extreme hip flexion is also avoided, because this position causes an increase in intra-abdominal and intrathoracic pressures, which can produce an increase in ICP. Relatively minor changes in position can significantly affect ICP. If monitoring reveals that turning the patient raises ICP, rotating beds, turning sheets, and holding the patient's head during turning may minimize the stimuli that increase ICP. Research suggests that patient response to position change is highly variable and requires close hemodynamic monitoring and individualized care (Hickey & Strayer, 2020).

The Valsalva maneuver, which can be produced by straining at defecation or even moving in bed, raises ICP and is to be avoided. Stool softeners may be prescribed. If the patient is alert and able to eat, a diet high in fiber may be indicated. Abdominal distention, which increases intra-abdominal and intrathoracic pressure and ICP, should be noted. Enemas and cathartics are avoided if possible. When moving or being turned in bed, the patient can be instructed to exhale (which opens the glottis) to avoid the Valsalva maneuver.

Mechanical ventilation presents unique problems for the patient with increased ICP. Before suctioning, the patient should be preoxygenated and briefly hyperventilated using 100% oxygen on the ventilator. Suctioning should not last longer than 15 seconds. High levels of positive end-expiratory pressure (PEEP) must be utilized cautiously, because they may decrease venous return to the heart and decrease venous drainage from the brain through increased intrathoracic pressure (Hickey & Strayer, 2020).

Activities that increase ICP, as indicated by changes in waveforms, should be avoided if possible. Spacing of nursing interventions may prevent transient increases in ICP. During nursing interventions, the ICP should not increase above 25 mm Hg, and it should return to baseline levels within 5 minutes. Patients with increased ICP should not demonstrate a significant increase in pressure or change in the ICP waveform. Patients with the potential for a significant increase in ICP may need sedation before initiation of nursing activities (Hickey & Strayer, 2020).

Emotional stress and frequent arousal from sleep are avoided. A calm atmosphere is maintained. Environmental stimuli (e.g., noise, conversation) should be minimal.

#### MAINTAINING NEGATIVE FLUID BALANCE

The administration of osmotic and loop diuretics is part of the treatment protocol to reduce ICP. Corticosteroids may be used to reduce cerebral edema (except when it results from trauma), and fluids may be restricted. All of these treatment modalities promote dehydration.

Skin turgor, mucous membranes, urine output, and serum and urine osmolality are monitored to assess fluid status. If IV fluids are prescribed, the nurse ensures that they are given at a slow to moderate rate with an IV infusion pump, to prevent too-rapid administration and avoid overhydration. For the patient receiving mannitol, the nurse observes for the possible development of heart failure and pulmonary edema. The intent of treatment is to promote a shift of fluid from the intracellular to the intravascular compartment and to control cerebral edema. However, this shift of fluid volume to the intravascular compartment may overwhelm the ability of the myocardium to increase workload sufficient to meet these demands, which may cause failure and pulmonary edema.

For patients undergoing dehydrating procedures, vital signs, including blood pressure, must be monitored to assess fluid volume status. An indwelling urinary catheter is inserted to permit assessment of renal function and fluid status. During the acute phase, urine output is monitored hourly. An output greater than 200 mL/h for 2 consecutive hours may indicate the onset of diabetes insipidus (Hickey & Strayer, 2020). These patients need careful oral hygiene, because mouth dryness occurs with dehydration. Frequently rinsing the mouth with nondrying solutions, lubricating the lips, and removing encrustations relieve dryness and promote comfort.

NURSING NEUROLOGIC CRITICAL CARE FLOWSHEET		ADDRESSOGRAPH									
		Date									
		Time									
		Initials									
Level of orientation (✓)	Person										
	Place										
	Date and time										
	No orientation										
Awakens to (✓)	Voice										
	Touch										
	Noxious stimuli										
	Painful stimuli										
Best verbal response (✓)	No response										
	Clear and appropriate										
	Clear and inappropriate										
	Difficulty speaking*										
	Perseveration										
	Aphasic expressive (non-fluent)										
	Aphasic receptive (fluent)										
	Sounds no speech										
	No verbal response										
	ETT/TRACH										
Best motor response (✓)	Moves all extremities purposefully										
	Withdraws and lifts to painful stimuli										
	Moves to painful stimuli										
	Decorticates (spinal reflex)										
	Decerebrates (spinal reflex)										
Best motor strength upper extremities (✓)	No motor response										
	No drifts (R/L)	R	L	R	L	R	L	R	L	R	L
	Drift (R/L)	R	L	R	L	R	L	R	L	R	L
	Can only lift forearm (R/L)	R	L	R	L	R	L	R	L	R	L
	Trace movement of hand or arm (R/L)	R	L	R	L	R	L	R	L	R	L
	Trace movement of fingers only (R/L)	R	L	R	L	R	L	R	L	R	L
	No motor response (R/L)	R	L	R	L	R	L	R	L	R	L
	Raises leg off bed (R/L)	R	L	R	L	R	L	R	L	R	L
	Drags heel on bed and lifts knee (R/L)	R	L	R	L	R	L	R	L	R	L
	Trace movement of foot or leg (R/L)	R	L	R	L	R	L	R	L	R	L
Best strength lower extremities (✓)	Trace movement of toes only (R/L)	R	L	R	L	R	L	R	L	R	L
	No response (R/L)	R	L	R	L	R	L	R	L	R	L
	Seizure activity (✓)	No seizure activity									
	With loss of consciousness*										
Ataxia (✓)	Without loss of consciousness*										
	Gross ataxia										
	Fine motor ataxia										
ICP monitoring	Does not apply										
	Ventriculostomy mL										
	ICP mm Hg										
	Not applicable										

\*= FURTHER DOCUMENTATION IS REQUIRED TO VALIDATE ASSESSMENT

PUPIL GAUGE (mm)									
• 2	• 3	● 4	● 5	● 6	● 7	● 8	● 9	● 10	● 11
B=Brisk, S=Sluggish, F=Fixed									
ADDRESSOGRAPH									
Date									
Time									
Initials									
Incision +/-	Dry and intact								
Drainage									
Pupils: refer to above gauge (+)=Present (-)Absent	R L	R L	R L	R L	R L	R L	R L	R L	R L
Regular (R/L)	R L	R L	R L	R L	R L	R L	R L	R L	R L
Irregular* (R/L)	R L	R L	R L	R L	R L	R L	R L	R L	R L
Reaction (R/L) (B) - (S) - (F)	R L	R L	R L	R L	R L	R L	R L	R L	R L
Ptosis (R/L) (+) (-)	R L	R L	R L	R L	R L	R L	R L	R L	R L
Gaze preference (R/L) (+)* (-)	R L	R L	R L	R L	R L	R L	R L	R L	R L
Meningeal signs (+)=Present (-)Absent	Headache								
Nuchal rigidity									
Photophobia									
Right upper outer									
Visual field (+)=Present (-)Absent* NA=Not applicable									
Right lower outer									
Left upper outer									
Left lower outer									
Nystagmus (+)=Present (-)Absent	R L	R L	R L	R L	R L	R L	R L	R L	R L
Vertical (R/L)	R L	R L	R L	R L	R L	R L	R L	R L	R L
Cranial nerves (+)=Present (-)Absent	III, IV, VI, Extraocular movements								
VII – Peripheral facial drop (R/L)	R L	R L	R L	R L	R L	R L	R L	R L	R L
XII – Tongue deviation (R/L)	R L	R L	R L	R L	R L	R L	R L	R L	R L
IX – Gag reflex									
V, VII – Corneal reflex (R/L)	R L	R L	R L	R L	R L	R L	R L	R L	R L
X, IX – Cough reflex									
Doll's eyes if appropriate									
Follows commands	Two step verbal command								
	One step verbal command								
	Unable to follow command								
* = FURTHER DOCUMENTATION IS REQUIRED TO VALIDATE ASSESSMENT									
Initials	Signature		Title	Initials	Signature		Title		

**Figure 61-6 •** A neurologic assessment flowsheet. The nurse fills these out online now in most institutions.

**TABLE 61-2** Increased Intracranial Pressure and Interventions

Factor	Physiology	Interventions	Rationale
Cerebral edema	Can be caused by contusion, tumor, or abscess; water intoxication (hypo-osmolality); alteration in the blood-brain barrier (protein leaks into the tissue, causing water to follow).	Administer osmotic diuretics as prescribed (monitor serum osmolality). Maintain head of bed elevation at 30 degrees. Maintain alignment of the head.	Promotes venous return. Prevents impairment of venous return through the jugular veins.
Hypoxia	A decrease in PaO <sub>2</sub> to <60 mm Hg causes cerebral vasodilation.	Maintain PaO <sub>2</sub> >60 mm Hg. Maintain oxygen therapy. Monitor arterial blood gas values. Suction when needed. Maintain a patent airway.	Prevents hypoxia and vasodilation.
Hypercapnia (elevated PaCO <sub>2</sub> )	Causes vasodilation.	Maintain PaCO <sub>2</sub> (normally 35–45 mm Hg) by establishing ventilation.	Normalizing PaCO <sub>2</sub> minimizes vasodilation and thus reduces the cerebral blood volume.
Impaired venous return	Increases the cerebral blood volume.	Maintain head alignment. Elevate head of bed 30 degrees.	Hyperextension, rotation, or hyperflexion of the neck causes decreased venous return.
Increase in intrathoracic or abdominal pressure	An increase in these pressures due to coughing, PEEP, or Valsalva maneuver causes a decrease in venous return.	Monitor arterial blood gas values, and keep PEEP as low as possible. Provide humidified oxygen. Administer stool softeners as prescribed.	To keep secretions loose and easy to suction or expectorate. Soft bowel movements will prevent straining or Valsalva maneuver.

PaCO<sub>2</sub>, partial pressure of arterial carbon dioxide; PaO<sub>2</sub>, partial pressure of arterial oxygen; PEEP, positive end-expiratory pressure.

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.

#### PREVENTING INFECTION

The risk of infection is greatest when ICP is monitored with an intraventricular catheter and increases with the duration of the monitoring. Most health care facilities have written protocols for managing these systems and maintaining their sterility; strict adherence to the protocols is essential.

Aseptic technique must be used when managing the system and changing the ventricular drainage bag. The drainage system is also checked for loose connections, because they can cause leakage and contamination of the CSF as well as inaccurate readings of ICP. The nurse observes the character of the CSF drainage and reports increasing cloudiness or blood. The patient is monitored for signs and symptoms of meningitis: fever, chills, nuchal (neck) rigidity, and increasing or persistent headache. See Chapter 64 for a discussion of meningitis.

#### MONITORING AND MANAGING POTENTIAL COMPLICATIONS

The primary complication of increased ICP is brain herniation resulting in death (see Fig. 61-2). Nursing management focuses on detecting early signs of increasing ICP, because medical interventions are usually ineffective once later signs develop (Hickey & Strayer, 2020). Frequent neurologic

assessments and documentation and analysis of trends will reveal the subtle changes that may indicate increasing ICP.

**Detecting Indications of Increasing Intracranial Pressure.** The nurse assesses for and immediately reports any signs or symptoms of increasing ICP (see Chart 61-1). The focus is on detecting early signs of increasing ICP.

**Monitoring Intracranial Pressure.** Because clinical assessment is not always a reliable guide in recognizing increased ICP, especially in patients who are comatose, monitoring of ICP and cerebral oxygenation is an essential part of management. ICP is monitored closely for continuous elevation or significant increase over baseline. The trend of ICP measurements over time is an important indication of the patient's underlying status. Vital signs are assessed when an increase in ICP is noted (Hickey & Strayer, 2020).

Careful attention to aseptic technique is needed when handling any part of the monitoring system (Hickey & Strayer, 2020). The insertion site is inspected for signs of infection. Temperature, pulse, and respirations are closely monitored for systemic signs of infection (Rincon, 2018). All connections and stopcocks are checked for leaks, because even small leaks can distort pressure readings and lead to infection (Hickey & Strayer, 2020).

When ICP is monitored with a fluid system, the transducer is calibrated at a particular reference point, usually 2.5 cm (1 inch) above the ear with the patient in the supine position; this point corresponds to the level of the foramen of Monro (see Fig. 61-7). CSF pressure readings depend on the patient's position. For subsequent pressure readings, the head should be in the same position relative to the transducer. Fiberoptic catheters are calibrated before insertion and do not require further referencing; they do not require the head of the bed to be at a specific position to obtain an accurate reading.

## Chart 61-1

### Detecting Increasing Intracranial Pressure (ICP)

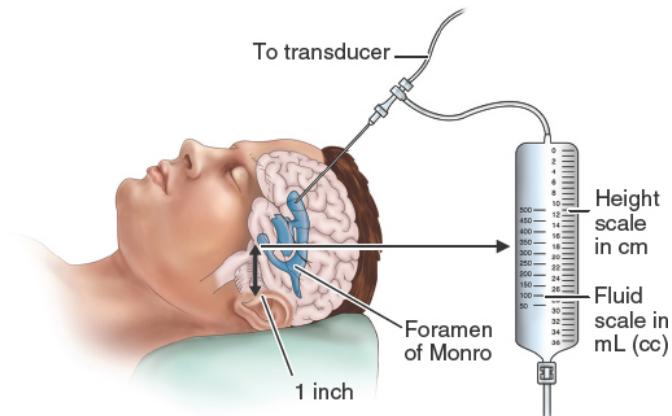
#### Early Signs and Symptoms of Increasing ICP

- *Disorientation, restlessness, increased respiratory effort, purposeless movements, and mental confusion.* These are early clinical indications of increasing ICP because the brain cells responsible for cognition are extremely sensitive to decreased oxygenation.
- *Pupillary changes and impaired extraocular movements.* These occur as the increasing pressure displaces the brain against the oculomotor and optic nerves (cranial nerves II, III, IV, and VI), which arise from the midbrain and brain stem (see Chapter 60).
- *Weakness in one extremity or on one side of the body.* This occurs as increasing ICP compresses the pyramidal tracts.
- *Headache that is constant, increasing in intensity, and aggravated by movement or straining.* This occurs as increasing ICP causes pressure and stretching of venous and arterial vessels in the base of the brain.

#### Later Signs and Symptoms of Increasing ICP

- The level of consciousness continues to deteriorate until the patient is comatose (Glasgow Coma Scale score ≤8).
- The pulse rate and respiratory rate decrease or become erratic, and the blood pressure and temperature increase. The pulse pressure (the difference between the systolic and diastolic pressures) widens. The pulse fluctuates rapidly, varying from bradycardia to tachycardia.
- Altered respiratory patterns develop, including Cheyne–Stokes breathing (rhythmic waxing and waning of rate and depth of respirations alternating with brief periods of apnea) and ataxic breathing (irregular breathing with a random sequence of deep and shallow breaths).
- Projectile vomiting may occur with increased pressure on the reflex center in the medulla.
- Hemiplegia or decorticate or decerebrate posturing may develop as pressure on the brain stem increases; bilateral flaccidity occurs before death.
- Loss of brain stem reflexes, including pupillary, corneal, gag, and swallowing reflexes, is an ominous sign of approaching death.

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.



**Figure 61-7 •** Location of the foramen of Monro for calibration of the intracranial pressure monitoring system.

When technology is associated with patient management, the nurse must be certain that the technologic equipment is functioning properly and used correctly (Liu, Griffith, Jang, et al., 2020) (see the Nursing Research Profile in Chart 61-2). The most important concern must be the patient to whom equipment is attached. The patient and family must be informed about the technology and the goals of its use. The patient's response is monitored, and appropriate comfort measures are implemented to ensure that the patient's stress is minimized.

ICP measurement is only one parameter; repeated neurologic checks and clinical examinations remain important measures. Astute observation, comparison of findings with previous observations, and interventions can assist in preventing life-threatening ICP elevations.

**Monitoring for secondary complications.** The nurse also assesses for complications of increased ICP, including diabetes insipidus and SIADH (see Chapters 10 and 45). Urine output should be monitored closely. Diabetes insipidus requires fluid and electrolyte replacement, along with the administration of vasopressin, to replace and slow the urine output. Serum electrolyte levels are monitored for imbalances. SIADH requires fluid restriction and monitoring of serum electrolyte levels.

#### Chart 61-2



#### NURSING RESEARCH PROFILE

### Aspects of Intracranial Pressure Monitoring

Liu, X., Griffith, M., Jang, H., et al. (2020). Intracranial pressure monitoring via external ventricular drain: Are we waiting long enough before recording the real value? *Journal of Neuroscience Nursing*, 52(1), 37–42.

#### Purpose

The purpose of this study of intracranial pressure (ICP) recordings was to obtain an insight into how well the intermittent external ventricular drain (EVD) clamping procedure is performed for ICP documentation.

#### Design

This was a retrospective analysis of ICP recordings. For each recording of ICP, the mean and standard deviation were calculated. The duration of EVD closure, time interval between two adjacent EVD closures, and the total number of EVD closures were calculated for each patient. An algorithm to evaluate whether ICP reached a new equilibrium before the EVD was reopened for drainage was developed. The percentage of EVD closures that reached the equilibrium was calculated.

#### Findings

Data were obtained from 107 patients with subarachnoid hemorrhage who had 32,755 EVD closures in total. Only 65.9% of openings lasted less than 1 minute and 16.3% lasted longer than 5 minutes. The median duration of each EVD closure was 25 seconds. Only 22.9% of EVD closures reached ICP equilibrium before EVD reopening.

#### Nursing Implications

This research provides evidence for the need to properly train and provide a standard guideline for bedside nurses to correctly obtain and document ICP. Nurses working in settings where ICP is monitored need to work on clear guidelines that specify the need to wait the required time when opening and closing an EVD in order to get an accurate reading.

#### Evaluation

Expected patient outcomes may include:

1. Attains optimal breathing pattern
  - a. Maintains patent airway
  - b. Breathes in a regular pattern
  - c. Attains or maintains arterial blood gas values within acceptable range
2. Demonstrates optimal cerebral tissue perfusion
  - a. Increasingly oriented to time, place, and person
  - b. Follows verbal commands; answers questions correctly
3. Attains desired fluid balance
  - a. Maintains fluid restriction
  - b. Demonstrates serum and urine osmolality values within acceptable range
4. Has no signs or symptoms of infection
  - a. Has no fever
  - b. Shows no redness, swelling, or drainage at arterial, IV, and urinary catheter sites
  - c. Has no redness, swelling, or purulent drainage from invasive intracranial monitoring device
5. Absence of complications
  - a. Has ICP values that remain within normal limits
  - b. Demonstrates urine output and serum electrolyte levels within acceptable limits



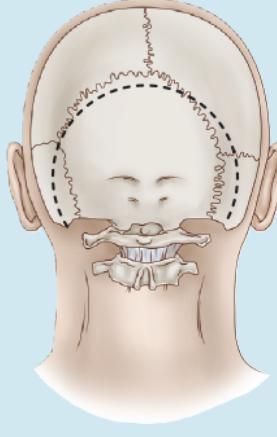
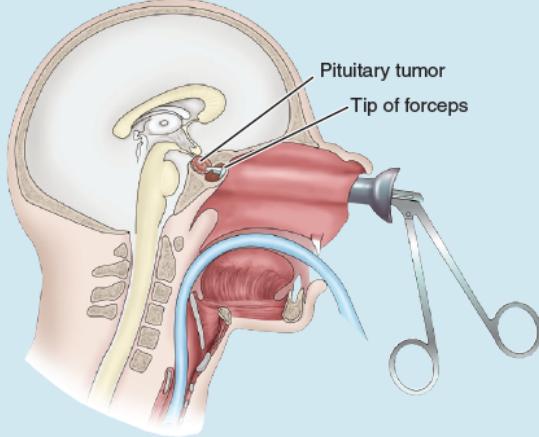
## INTRACRANIAL SURGERY

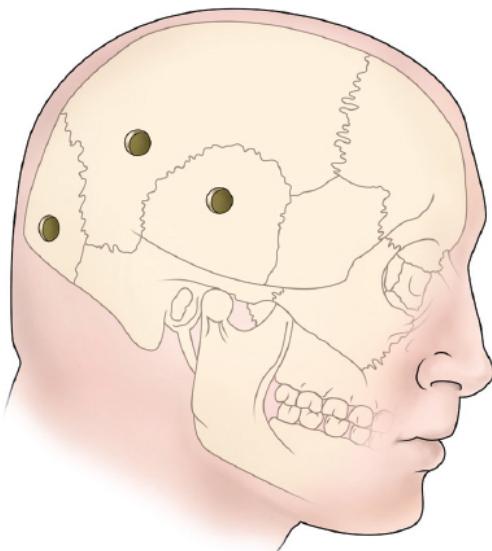
A **craniotomy** involves opening the skull surgically to gain access to intracranial structures. This procedure is performed to remove a tumor, relieve elevated ICP, evacuate a blood clot, or control

hemorrhage. The surgeon cuts the skull to create a bony flap, which can be repositioned after surgery and held in place by periosteal or wire sutures. One of two approaches through the skull is used: above the tentorium (supratentorial craniotomy) into the supratentorial compartment, or below the tentorium into the infratentorial (posterior fossa) compartment. A third approach, the **transsphenoidal** approach (through the mouth and nasal sinuses) is often used to gain access to the pituitary gland (Hickey & Strayer, 2020). Table 61-3 compares these three different surgical approaches.

Alternatively, intracranial structures may be approached through burr holes (see Fig. 61-8), which are circular openings made in the skull by either a hand drill or an automatic craniotome (which has a self-controlled system to stop the drill when the bone is penetrated). Burr holes may be used to determine the presence of cerebral swelling and injury and the size and position of the ventricles. They are also a means of evacuating an intracranial hematoma or abscess and for making a bone flap in the skull that allows access to the ventricles for decompression, ventriculography, or shunting procedures. Other cranial procedures include **craniectomy** (excision of a portion of the skull) and cranioplasty (repair of a cranial defect using a plastic or metal plate).

**TABLE 61-3** Comparison of Cranial Surgical Approaches

Supratentorial	Infratentorial	Transsphenoidal
		
<b>Site of Surgery</b>		
Above the tentorium	Below the tentorium, brain stem	Sella turcica and pituitary region
<b>Incision Location</b>		
Incision is made above the area to be operated on; usually located behind the hairline.	Incision is made at the nape of the neck, around the occipital lobe.	Incision is made beneath the upper lip to gain access into the nasal cavity.
<b>Select Nursing Interventions</b>		
Maintain head of bed elevated at 30 degrees, with neck in neutral alignment.	Maintain neck in straight alignment. Avoid flexion of the neck to prevent possible tearing of the suture line.	Maintain nasal packing in place and reinforce as needed. Instruct patient to avoid blowing the nose. Provide oral care according to institutional procedure.
Position patient on either side or back. (Avoid positioning patient on operative side if a large tumor has been removed.)	Position the patient on either side. (Check surgeon's preference for positioning of patient.)	Keep head of bed elevated to promote venous drainage and drainage from the surgical site.



**Figure 61-8 •** Burr holes may be used in neurosurgical procedures to make a bone flap in the skull, to aspirate a brain abscess, or to evacuate a hematoma.

## Supratentorial and Infratentorial Approaches

### Preoperative Management

#### Medical Management

Preoperative diagnostic procedures may include a CT scan to demonstrate the lesion and show the degree of surrounding brain edema, the ventricular size, and the displacement. An MRI scan provides information similar to that of a CT scan with improved tissue contrast, resolution, and anatomic definition. Cerebral angiography may be used to study a tumor's blood supply or obtain information about vascular lesions. Transcranial Doppler flow studies are used to evaluate the blood flow within intracranial blood vessels.

Patients may be prescribed an anticonvulsant medication such as phenytoin, levetiracetam, or a phenytoin metabolite (fosphenytoin sodium) before surgery to reduce the risk of postoperative **seizures** (paroxysmal transient disturbance of the brain resulting from a discharge of abnormal electrical activity) (Comerford & Durkin, 2020). Recent research suggests that anticonvulsant medications should not be used routinely, but only when the patient experiences seizures (Mirian, Pedersen, Sabers, et al., 2019). Before surgery, corticosteroids such as dexamethasone may be given to reduce cerebral edema if the patient has a brain tumor. Fluids may be restricted. A hyperosmotic agent (mannitol) and a diuretic agent such as furosemide may be administered IV immediately before and sometimes during surgery if the patient tends to retain fluid, as do many who have intracranial dysfunction. Antibiotic agents may be given if there is a chance of cerebral contamination; diazepam or lorazepam may be prescribed before surgery to allay anxiety.

#### Nursing Management

The preoperative assessment serves as a baseline against which postoperative status and recovery are compared. This assessment includes evaluating the LOC and responsiveness to stimuli and identifying any neurologic deficits, such as paralysis, visual dysfunction, alterations in personality or speech, and bladder and bowel disorders. Baseline distal and proximal motor strength in both upper and lower extremities is tested and recorded. See [Chapter 60](#) for a discussion of the testing of motor function.

The patient's and family's understanding of and reactions to the anticipated surgical procedure and its possible sequelae are assessed, as is the availability of support systems for the patient and family. Adequate preparation for surgery, with attention to the patient's physical and emotional status, can reduce the risk of

anxiety, fear, and postoperative complications. The patient is assessed for neurologic deficits and their potential impact after surgery. For motor deficits or weakness or paralysis of the arms or legs, trochanter rolls are applied to the extremities, and the feet are positioned against a footboard or the ankles are supported in a neutral position with orthotic boots. A patient who can ambulate is encouraged to do so. If the patient is aphasic, writing materials or picture and word cards showing the bedpan, glass of water, blanket, and other frequently used items may help improve communication.

Preparation of the patient and family includes providing education about what to expect during and after surgery. The patient should plan to shower and wash their hair prior to surgery using the preferred cleansing solution. Hair is removed with the use of clippers and the surgical site prepared immediately before surgery (usually in the operating room), and IV antibiotics are given 1 hour prior to the incision to decrease the chance of infection (American Association of Neuroscience Nurses [AANN], 2016b). An indwelling urinary catheter is inserted in the operating room to drain the bladder during the administration of diuretic agents and to permit monitoring of urinary output. The patient may have an arterial line placed for monitoring of pressures after surgery. The large head dressing applied after surgery may impair hearing temporarily. Vision may be limited if the eyes are swollen shut. If a tracheostomy or endotracheal tube remains in place, the patient will be unable to speak until the tube is removed, so an alternative method of communication must be established.

An altered cognitive state may make the patient unaware of the impending surgery. Even so, encouragement and attention to the patient's needs are necessary. Whatever the state of awareness of the patient, the family needs reassurance and support, because they usually recognize the seriousness of brain surgery.

### **Postoperative Management**

Postoperatively, an arterial line may be in place to monitor and manage blood pressure. The patient may be intubated and may receive supplemental oxygen therapy. Ongoing postoperative management is aimed at detecting and reducing cerebral edema, relieving pain and preventing seizures, and monitoring ICP and neurologic status.

### **Reducing Cerebral Edema**

Medications to reduce cerebral edema include mannitol, which increases serum osmolality and draws free water from areas of the brain (with an intact blood–brain barrier). The fluid is then excreted by osmotic diuresis. Dexamethasone may be administered IV every 6 hours for 24 to 72 hours; the route is changed to oral as soon as possible, and the dosage is typically tapered over 5 to 7 days; some patients may require an extended taper (Comerford & Durkin, 2020).

### **Relieving Pain and Preventing Seizures**

Acetaminophen is usually prescribed for temperatures exceeding 37.5°C (99.6°F) (Rincon, 2018) and for mild pain. The patient usually has a headache after a craniotomy as a result of stretching and irritation of nerves in the scalp during surgery. Codeine, administered IV or orally, is often sufficient to relieve headache. Morphine sulfate may also be used in the management of postoperative pain in patients who have undergone a craniotomy with the goal of a patient reporting acceptable pain level (AANN, 2016b).

Anticonvulsant medication (phenytoin, levetiracetam) is often prescribed prophylactically for patients who have undergone supratentorial craniotomy because of the high risk of seizures after these procedures (Hickey & Strayer, 2020). Serum levels are monitored to check that the medication levels are within the therapeutic range.

### **Monitoring Intracranial Pressure**

A patient undergoing intracranial surgery may have an ICP or cerebral oxygenation monitor inserted during surgery. Strict adherence to written protocols for managing these systems is essential, as discussed earlier, for preventing infection and managing ICP. The system is removed after the ICP or cerebral oxygenation is normal and stable. The neurosurgeon must be notified immediately if the system is not functioning.

## NURSING PROCESS

### The Patient Who Has Undergone Intracranial Surgery

#### Assessment

After surgery, the frequency of postoperative monitoring is based on the patient's clinical status. Assessing respiratory function is essential, because even a small degree of hypoxia can increase cerebral ischemia. The respiratory rate and pattern are monitored, and arterial blood gas values are assessed frequently. Fluctuations in vital signs are carefully monitored and documented, because they may indicate increased ICP. The patient's temperature is measured to assess for hyperthermia secondary to infection or damage to the hypothalamus. Neurologic checks are made frequently to detect increased ICP resulting from cerebral edema or bleeding. A change in LOC or response to stimuli may be the first sign of increasing ICP.

The surgical dressing is inspected for evidence of bleeding and CSF drainage. The incision is monitored for redness, tenderness, bulging, separation, or foul odor. Sodium retention may occur in the immediate postoperative period. Serum and urine electrolytes, BUN, blood glucose, weight, and clinical status are monitored. Intake and output are measured in view of losses associated with fever, respiration, and CSF drainage. The nurse must be alert to the development of complications; all assessments are carried out with these problems in mind. Seizures are a potential complication, and any seizure activity is carefully recorded and reported. Restlessness may occur as the patient becomes more responsive, or restlessness may be caused by pain, confusion, hypoxia, or other stimuli.

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Risk for ineffective tissue perfusion associated with cerebral edema
- Risk for impaired thermoregulation associated with damage to the hypothalamus, dehydration, and infection
- Impaired gas exchange associated with hypoventilation, aspiration, and immobility
- Difficulty coping associated with sensory perception changes due to periorbital edema, head dressing, endotracheal tube, and effects of ICP
- Disturbed body image associated with change in appearance or physical disabilities

Other nursing diagnoses may include impaired communication (aphasia) associated with insult to brain tissue and high risk for impaired skin integrity associated with immobility, pressure, and incontinence; impaired mobility associated with a neurologic deficit secondary to the neurosurgical procedure or to the underlying disorder may also occur.

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Increased ICP
- Bleeding and hypovolemic shock
- Fluid and electrolyte disturbances
- Infection
- CSF leak
- Seizures

#### Planning and Goals

The major goals for the patient include maintaining or restoring neurologic homeostasis to improve cerebral tissue perfusion, adequate thermoregulation, normal ventilation and gas exchange, ability to cope with sensory deprivation, adaptation to changes in body image, and absence of complications.

#### Nursing Interventions

##### MAINTAINING CEREBRAL TISSUE PERfusion

Attention to the patient's respiratory status is essential, because even slight decreases in the oxygen level (hypoxia) or slight increases in the carbon dioxide level (hypercarbia) can affect cerebral perfusion, the clinical course, and the patient's outcome. The endotracheal tube is left in place until the patient shows signs of awakening and has adequate spontaneous ventilation, as evaluated clinically and by arterial blood gas analysis. Secondary brain damage can result from impaired cerebral oxygenation.

Some degree of cerebral edema occurs after brain surgery; it tends to peak 24 to 36 hours after surgery, potentially producing decreased responsiveness on the second postoperative day. The control of cerebral edema was discussed earlier. Nursing strategies used to control factors that may raise ICP were presented in the previous Nursing Process section on increased ICP. Intraventricular drainage is carefully monitored, using strict asepsis when any part of the system is handled.

Vital signs and neurologic status (LOC and responsiveness, pupillary and motor responses) are assessed every 15 to 60 minutes. Extreme head rotation is avoided, because this raises ICP. After supratentorial surgery, the patient is placed on their back or side (on the unoperated side if a large lesion was removed) with one pillow under the head. The head of the bed may be elevated 30 degrees, depending on the level of the ICP and the neurosurgeon's preference. After posterior fossa (infratentorial) surgery, the patient is kept flat on one side (off the back) with the head on a small, firm pillow. The patient may be turned on either side, keeping the neck in a neutral position. When the patient is being turned, the body should be turned as a unit to prevent placing strain on the incision and possibly tearing the sutures. The head of the bed may be elevated slowly as tolerated by the patient.

The patient's position is changed every 2 hours, and skin care is given frequently. During position changes, care is taken to prevent disruption of the ICP monitoring system. A turning sheet or lift sling placed under the patient's head to midthigh makes it easier to move and turn the patient safely.

#### **REGULATING TEMPERATURE**

Moderate temperature elevation can be expected after intracranial surgery because of the reaction to blood at the operative site or in the subarachnoid space. Injury to the hypothalamic centers that regulate body temperature can occur during surgery. Fever is treated vigorously to combat the effect of an elevated temperature on brain metabolism and function.

Nursing interventions include monitoring the patient's temperature and using the following measures to reduce body temperature: removing blankets, placing ice packs, and administering prescribed antipyretics to reduce fever (Rincon, 2018).

Conversely, hypothermia may be seen after lengthy neurosurgical procedures. Therefore, frequent measurements of rectal temperatures are necessary. Rewarming should occur slowly to prevent shivering, which increases cellular oxygen demands.

#### **IMPROVING GAS EXCHANGE**

The patient undergoing neurosurgery is at risk for impaired gas exchange and pulmonary infections due to immobility, immunosuppression, decreased LOC, and fluid restriction. Immobility compromises the respiratory system by causing pooling and stasis of secretions in dependent areas and the development of atelectasis. The patient whose fluid intake is restricted may be more vulnerable to atelectasis as a result of inability to expectorate thickened secretions. Pneumonia can develop due to aspiration and restricted mobility.

Repositioning the patient every 2 hours helps to mobilize pulmonary secretions and prevent stasis. After the patient regains consciousness, additional measures to expand collapsed alveoli can be instituted, such as yawning, sighing, deep breathing, incentive spirometry, and coughing (unless contraindicated). If necessary, the oropharynx and trachea are suctioned to remove secretions that cannot be raised by coughing; however, coughing and suctioning increase ICP. Therefore, suctioning should be used cautiously. Increasing the humidity in the oxygen delivery system may help to loosen secretions. The nurse and the respiratory therapist work together to monitor the effects of chest physiotherapy.

#### **COPING WITH SENSORY DEPRIVATION**

Periorbital edema is a common consequence of intracranial surgery, because fluid drains into the dependent periorbital areas when the patient has been positioned in a prone position during surgery. A hematoma may form under the scalp and spread down to the orbit, producing an area of ecchymosis (black eye).

Before surgery, the patient and family should be informed that one or both eyes may be edematous temporarily after surgery. After surgery, elevating the head of the bed (if not contraindicated) and applying cold compresses over the eyes will help reduce the edema. The surgeon is notified if periorbital edema increases significantly, because this may indicate that a postoperative clot is developing or that there is increasing ICP and poor venous drainage. Health care personnel should announce their presence when entering the room to avoid startling the patient whose vision is impaired due to periorbital edema or neurologic deficits.

Additional factors that can affect sensation include a bulky head dressing, the presence of an endotracheal tube, and effects of increased ICP. The first postoperative dressing change is usually

performed by the neurosurgeon. In the absence of bleeding or a CSF leak, every effort is made to minimize the size of the head dressing. If the patient requires an endotracheal tube for mechanical ventilation, every effort is made to extubate the patient as soon as clinical signs indicate it is possible. The patient is monitored closely for the effects of elevated ICP.

#### **ENHANCING SELF-IMAGE**

The patient is encouraged to verbalize feelings and frustrations about any change in appearance. Nursing support is based on the patient's reactions and feelings. Factual information may need to be provided if the patient has misconceptions about puffiness about the face, periorbital bruising, and hair loss. Attention to grooming, the use of the patient's own clothing, and covering the head with a turban (and later a wig until hair growth occurs) are encouraged. Social interaction with close friends, family, and hospital personnel may increase the patient's sense of self-worth.

The family and social support system can be of assistance while the patient recovers from surgery.

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

The nurse must be vigilant for complications that may develop within hours of surgery and require close collaboration with the neurosurgeon. These include increased ICP, bleeding and hypovolemic shock, altered fluid and electrolyte balance (e.g., water intoxication, diabetes insipidus), infection, identification of a CSF leak, and seizures.

**Monitoring for Increased Intracranial Pressure and Bleeding.** Increased ICP and bleeding are life-threatening to the patient who has undergone intracranial surgery. The following points must be kept in mind when caring for any patient who has undergone such surgery:

- An increase in blood pressure and decrease in pulse with respiratory failure may indicate increased ICP.
- An accumulation of blood under the bone flap (extradural, subdural, or intracerebral hematoma) may pose a threat to life. A clot must be suspected in any patient who does not awaken as expected or whose condition deteriorates. An intracranial hematoma is suspected if the patient has any new postoperative neurologic deficits (especially a dilated pupil on the operative side). In these circumstances, the patient is returned to the operating room immediately for evacuation of the clot, if indicated.
- Cerebral edema, infarction, metabolic disturbances, and hydrocephalus are conditions that may mimic the clinical manifestations of a clot.

The patient is monitored closely for indicators of complications, and early signs and trends in clinical status are reported to the surgeon. Treatments are initiated promptly, and the nurse assists in evaluating the patient's response to treatment. The nurse also provides support to the patient and family.



#### **Quality and Safety Nursing Alert**

*If signs and symptoms of increased ICP occur, efforts to decrease the ICP are initiated: alignment of the head in a neutral position without flexion to promote venous drainage, elevation of the head of the bed to 30 degrees (when prescribed), administration of mannitol (an osmotic diuretic), and possible administration of pharmacologic paralyzing agents.*

**Managing Fluid and Electrolyte Disturbances.** Fluid and electrolyte imbalances may occur because of the patient's underlying condition and its management or as complications of surgery. These disturbances can contribute to the development of cerebral edema.

The postoperative fluid regimen depends on the type of neurosurgical procedure and is determined on an individual basis. The volume and composition of fluids are adjusted based on daily serum electrolyte values, along with fluid intake and output. Fluids may have to be restricted in patients with cerebral edema.

Oral fluids are usually resumed after the first 24 hours. The presence of gag and swallowing reflexes must be checked before initiation of oral fluids. Some patients with posterior fossa tumors have impaired swallowing, so fluids may need to be given by alternative routes. The patient should be observed for signs and symptoms of nausea and vomiting as the diet is progressed (AANN, 2016b).

Patients undergoing surgery for brain tumors often receive large doses of corticosteroids and are at risk for hyperglycemia. Serum glucose levels are measured every 4 to 6 hours, and sliding scale insulin is prescribed as needed. These patients are prone to stress ulcers, so histamine-2 receptor antagonists

(H<sub>2</sub> blockers) or proton pump inhibitors are prescribed to suppress the secretion of gastric acid. Patients also are monitored for bleeding and assessed for gastric pain.

If the surgical site is near to (or causes edema to) the pituitary gland and hypothalamus, the patient may develop symptoms of diabetes insipidus, which is characterized by excessive urinary output, elevated serum osmolality, decreased urine osmolality, hypernatremia, and a low urine specific gravity. The urine specific gravity is measured hourly, and fluid intake and output are monitored. Fluid replacement must compensate for urine output, and serum potassium levels must be monitored.

SIADH, which results in water retention with hyponatremia and serum hypo-osmolality, occurs in a wide variety of CNS disorders (e.g., brain tumor, head trauma) causing fluid disturbances. Nursing management includes careful intake and output measurements, specific gravity determinations of urine, and monitoring of serum and urine electrolyte levels while following directives for fluid restriction. SIADH is usually self-limited.

**Preventing Infection.** The patient undergoing neurosurgery is at risk for infection associated with the neurosurgical procedure (brain exposure, bone exposure, wound hematomas) and the presence of IV and arterial lines for fluid administration and monitoring. Risk for infection is increased in patients who undergo lengthy intracranial operations and in those who have external ventricular drains in place.

The dressing is often stained with blood in the immediate postoperative period. Because blood is an excellent culture medium for bacteria, the dressing is reinforced with sterile pads so that contamination and infection are avoided. A heavily stained or displaced dressing should be reported immediately. A drain is sometimes placed in the craniotomy incision to facilitate drainage.

After suboccipital surgical procedures, CSF may leak through the incision. This complication is dangerous because of the possibility of meningitis. After a suboccipital craniotomy, the patient is instructed to avoid coughing, sneezing, or nose blowing, which can cause CSF leakage by creating increased pressure on the operative site.



#### **Quality and Safety Nursing Alert**

*Any sudden discharge of fluid from a cranial incision is reported at once, because a large leak often requires surgical repair. Attention should be paid to the patient who complains of a salty taste or “postnasal drip,” because this can be caused by CSF trickling down the throat.*

Aseptic technique is used when handling dressings, drainage systems, and IV and arterial lines. The patient is monitored carefully for signs and symptoms of infection, and cultures are obtained if infection is suspected. Appropriate antibiotic agents are given as prescribed. Other causes of infection in the patient undergoing intracranial surgery, such as pneumonia and urinary tract infections, are similar to those in other patients postoperatively.

**Monitoring for Seizure Activity.** Seizures may occur as complications after any intracranial neurosurgical procedure. Preventing seizures is essential to avoid further cerebral edema. Administering the prescribed anticonvulsant medication before and after surgery may prevent the development of seizures in subsequent months and years. **Status epilepticus** (prolonged seizures without recovery of consciousness in the intervals between seizures) may occur after craniotomy and also may be related to the development of complications (hematoma, ischemia). The management of status epilepticus is described later in this chapter.

**Monitoring and Managing Other Complications.** Other complications may occur during the first 2 weeks or later and may compromise the patient’s recovery. The most important of these are VTE (DVT, PE), pulmonary and urinary tract infection, and pressure injuries. Most of these complications may be avoided with frequent changes of position, adequate suctioning of secretions, thrombosis prophylaxis, early removal of indwelling urinary catheter, early ambulation, and skin care.

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



**Educating Patients About Self-Care.** The recovery at home of a patient who has had neurosurgery depends on the extent of the surgical procedure and its success. The patient’s strengths as well as limitations are assessed and explained to the family, along with the family’s part in promoting recovery. Because administration of postoperative medication is a priority, the patient and family are

educated to use a check-off system, pill boxes, and alarms to ensure that the medication is taken as prescribed.

The patient and family are educated about what to expect after intracranial surgery (see [Chart 61-3](#)). Dietary restrictions usually are not required unless another health problem necessitates a special diet. Although showering or tub bathing is permitted, the scalp should be kept dry until all sutures have been removed, unless the primary provider has specific wound care instructions. A clean scarf or cap may be worn until a wig or hairpiece is purchased. If skull bone has been removed, a protective helmet may be prescribed. The patient may require rehabilitation, depending on the postoperative level of function. The patient may require physical therapy for residual weakness and mobility issues. An occupational therapist is consulted to assist with self-care issues. If the patient has aphasia, speech therapy may be necessary.

**Continuing and Transitional Care.** The patient is discharged from the hospital as soon as possible. Patients with severe motor deficits require extensive physical therapy and rehabilitation. Those with postoperative cognitive and speech impairments require psychological evaluation, speech therapy, and rehabilitation. The nurse collaborates with the primary provider and other health care professionals during hospitalization and home or transitional care to achieve as complete a rehabilitation as possible and to assist the patient in living with residual disability.

If tumor, injury, or disease makes the prognosis poor, care is directed toward making the patient as comfortable as possible. With return of the tumor or cerebral compression, the patient becomes less alert and aware. Other possible consequences include paralysis, blindness, and seizures. The home health nurse, hospice nurse, and social worker collaborate with the family to plan for additional services or placement of the patient in an extended care facility (see the Cerebral Metastases section in [Chapter 65](#)). The patient and family are encouraged to discuss end-of-life preferences for care; the patient's end-of-life preferences must be respected (see [Chapter 13](#)). The nurse involved in home and continuing care of patients after cranial surgery also should remind patients and family members of the need for health promotion activities and recommended health screening.

#### Evaluation

Expected patient outcomes may include:

1. Achieves optimal cerebral tissue perfusion
  - a. Opens eyes on request; uses recognizable words, progressing to normal speech
  - b. Obeys commands with appropriate motor responses
2. Maintains normal body temperature
  - a. Registers normal body temperature
3. Has normal gas exchange
  - a. Has arterial blood gas values within normal ranges
  - b. Breathes easily; lung sounds clear without adventitious sounds
  - c. Takes deep breaths and changes position as directed
4. Copes with sensory deprivation
5. Demonstrates improving self-concept
  - a. Pays attention to grooming
  - b. Visits and interacts with others

Chart 61-3



#### HOME CARE CHECKLIST

## Discharge After Intracranial Surgery

### At the completion of education, the patient and/or caregiver will be able to:

- Name the procedure that was performed, any complications that occurred, and identify any permanent changes in anatomic structure or function as well as changes in ADLs, IADLs, roles, relationships, and spirituality.
- Identify interventions and strategies (e.g., durable medical equipment, adaptive equipment) used in recovery period.
- Describe ongoing postoperative therapeutic regimen, including diet and activities to perform (e.g., walking and breathing exercises) and to limit or avoid (e.g., lifting weights, driving a car, contact sports).
- State the name, dose, side effects, frequency, and schedule for all medications.
- State how to obtain medical supplies and carry out dressing changes, wound care, and other prescribed regimens.
- Identify durable medical equipment needs, proper usage, and maintenance necessary for safe utilization.
- Describe signs and symptoms of complications.
- State time and date of follow-up appointments.
- Relate how to reach primary provider with questions or complications.
- Identify community resources for peer and caregiver/family support:
  - Identify sources of support (e.g., friends, relatives, faith community).
  - Identify the contact details for support services for patients and their caregivers/families.
- Identify the need for health promotion (e.g., weight reduction, smoking cessation, stress management), disease prevention and screening activities.

ADL, activities of daily living; IADL, instrumental activities of daily living.

6. Exhibits absence of complications
  - a. Exhibits ICP within normal range
  - b. Has minimal bleeding at surgical site; surgical incision healing without evidence of infection
  - c. Shows fluid balance and electrolyte levels within desired ranges
  - d. Exhibits no evidence of seizures

## Transsphenoidal Approach

Tumors within the sella turcica and small adenomas of the pituitary can be removed through a transsphenoidal approach (see [Table 61-3](#)). Although an otorhinolaryngologist may make the initial opening, the neurosurgeon completes the opening into the sphenoidal sinus and exposes the floor of the sella. Microsurgical techniques provide improved illumination, magnification, and visualization so that nearby vital structures can be avoided.

The transsphenoidal approach offers direct access to the sella turcica with minimal risk of trauma and hemorrhage (Hickey & Strayer, 2020). It avoids many of the risks of craniotomy, and the postoperative discomfort is similar to that of other transnasal surgical procedures. It may also be used for pituitary ablation (destruction) in patients with metastatic breast or prostatic cancer.

## Complications

Manipulation of the posterior pituitary gland during surgery may produce transient diabetes insipidus of several days' duration (Hickey & Strayer, 2020). It is treated with vasopressin but occasionally persists. Other complications include CSF leakage, visual disturbances, postoperative meningitis, pneumocephalus (air in the intracranial cavity), and SIADH (see [Chapter 45](#)).

## Preoperative Management

## **Medical Management**

The preoperative evaluation includes a series of endocrine tests, rhinologic evaluation (to assess the status of the sinuses and nasal cavity), and neuroradiologic studies. Funduscopic examination and visual field determinations are performed, because the most serious effect of pituitary tumor is localized pressure on the optic nerve or chiasm. In addition, the nasopharyngeal secretions are cultured, because a sinus infection is a contraindication to an intracranial procedure using this approach. Corticosteroids may be given before and after surgery, because the surgery involves removal of the pituitary, which is the source of adrenocorticotrophic hormone (ACTH). Antibiotic agents may or may not be given prophylactically.

## **Nursing Management**

The patient is educated in deep breathing techniques before surgery. In addition, the patient is instructed that after the surgery, they will need to avoid vigorous coughing, blowing the nose, sucking through a straw, or sneezing, because these actions may place increased pressure at the surgical site and cause a CSF leak (Hickey & Strayer, 2020).

### **Postoperative Management**

## **Medical Management**

Because the procedure disrupts the oral and nasal mucous membranes, management focuses on preventing infection and promoting healing. Medications include antimicrobial agents (which are continued until the nasal packing inserted at the time of surgery is removed), corticosteroids, analgesic agents for discomfort, and agents for the control of diabetes insipidus, if necessary (Hickey & Strayer, 2020).

## **Nursing Management**

Vital signs are measured to monitor hemodynamic, cardiac, and ventilatory status. Because of the anatomic proximity of the pituitary gland to the optic chiasm, visual acuity and visual fields are assessed at regular intervals. One method is to ask the patient to count the number of fingers held up by the nurse. Evidence of decreasing visual acuity suggests an expanding hematoma.

The head of the bed is raised to decrease pressure on the sella turcica and to promote normal drainage. The patient is cautioned against blowing the nose or engaging in any activity that raises ICP, such as bending over or straining during urination or defecation.

Intake and output are measured as a guide to fluid and electrolyte replacement and to assess for diabetes insipidus. The urine specific gravity, serum sodium, and serum osmolality are measured and evaluated regularly. Daily weight is monitored. Fluids are usually given after nausea ceases, and the patient then progresses to a regular diet.

The nasal packing inserted during surgery is checked frequently for blood or CSF drainage. The major discomfort is related to the nasal packing and to mouth dryness and thirst caused by mouth breathing. Oral care is provided every 4 hours or more frequently. Usually, the teeth are not brushed until the incision above the teeth has healed. Warm saline mouth rinses and the use of a cool mist vaporizer are helpful. Petrolatum is soothing when applied to the lips. The packing is removed in 3 to 4 days, and only then can the area around the nares be cleaned with the prescribed solution to remove crusted blood and moisten the mucous membranes (Hickey & Strayer, 2020).

Home care considerations include advising the patient to use a room humidifier to keep the mucous membranes moist and to soothe irritation. The head of the bed is elevated at 30 degrees for at least 2 weeks after surgery. The patient is cautioned against blowing the nose or sneezing for at least 1 month, or as directed by their surgeon (Hickey & Strayer, 2020).

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## **OTHER NEUROLOGIC DYSFUNCTIONS**

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Three other types of neurologic dysfunctions the nurse should be aware of include delirium, dementia, and pseudobulbar affect.

## Delirium

**Delirium**, often called *acute confusional state*, begins with disorientation and if not recognized and treated can progress to changes in LOC, irreversible brain damage, and sometimes death. In fact, up to 80% of patients in intensive care units are affected, and the presence of delirium triples in-hospital mortality rates (Mulkey, Hardin, Munro, et al., 2019). Delirium is disturbing for the affected patient and their family, associated with worse outcomes, and a significant increase in medical care costs (Devlin, Skrobik, Gelinas, et al., 2018; Mulkey et al., 2019).

Careful clinical assessment is essential because delirium is sometimes mistaken for dementia and the two conditions may overlap; [Table 61-4](#) compares dementia and delirium. It helps to know an individual patient's usual mental status and whether the changes noted are long term, which probably represents dementia, or are abrupt in onset, which is more likely delirium.

There are numerous risk factors for delirium. Risk factors that are modifiable include the use of medications such as benzodiazepines and the administration of blood transfusions (Devlin et al., 2018). Nonmodifiable risk factors include age, presence of dementia, prior coma, as well as recent emergency surgery or trauma (Devlin et al., 2018). Older adults are particularly vulnerable to acute confusion if they are in a debilitated health state or take multiple medications.

Nurses must recognize the symptoms of delirium and report them immediately. The Confusion Assessment Method (CAM) is a commonly used screening tool (Devlin et al., 2018; Inouye, van Dyck, Alessi, et al., 1990). (See [Chapter 8, Chart 8-7](#).) Because of the acute and unexpected onset of symptoms, it is recommended that all patients who are critically ill receive routine screening for delirium at prescribed intervals (Devlin et al., 2018). If the delirium goes unrecognized and the underlying cause is not treated, permanent, irreversible brain damage or death can follow.

The most effective approach is prevention. Strategies include providing therapeutic activities for cognitive impairment, reorienting the patient as needed, ensuring early mobilization, controlling pain, minimizing the use of psychoactive drugs, preventing sleep deprivation, enhancing communication methods (particularly eyeglasses and hearing aids) for vision and hearing impairment, maintaining oxygen levels and fluid and electrolyte balance, and preventing surgical complications (Eliopoulos, 2018). Including the family in therapeutic activities, as appropriate, is encouraged but more research is needed to validate the effect of families (Devlin et al., 2018). There is some research evidence for the use of bright light therapy to reduce delirium in patients who are critically ill (Devlin et al., 2018).

Once delirium occurs, treatment of the underlying cause is most important. Therapeutic interventions vary depending on the cause. Delirium increases the risk of falls; therefore, management of patient safety and behavioral problems is essential. Because medication interactions and toxicity are often implicated, the nurse should alert the prescriber about any nonessential medications that could be discontinued. Nutritional and fluid intake should be supervised and monitored. The environment should be quiet and calm. To increase function and comfort, the nurse provides familiar environmental cues and encourages family members or friends to touch and talk to the patient (see [Fig. 61-9](#)). The nurse should provide for sleep hygiene measures in addition to assessing for and managing pain (Bennett, 2019). Ongoing mental status assessments using prior mental cognitive status as a baseline are helpful in evaluating responses to treatment and upon admission to a hospital or extended care facility. If the underlying problem is adequately treated, the patient often returns to baseline within several days.

## Dementia

The cognitive, functional, and behavioral changes that characterize dementia eventually destroy a person's ability to function. The symptoms are usually subtle in onset and often progress slowly until they are obvious and devastating. Dementia in older adults is typically caused by some degree of neurodegeneration (Gale, Acar, & Daffner, 2018). The most common type of **dementia** is Alzheimer's disease (AD) (see [Chapter 8](#) for discussion of AD). AD alone or in conjunction with other dementing disorders accounts for up to 75% of older adults with dementia (Hickey & Strayer, 2020). Other non-Alzheimer's dementias include degenerative, vascular, neoplastic, demyelinating, infectious, inflammatory, toxic, metabolic, and psychiatric disorders. It is important to identify reversible dementia, which occurs when pathologic conditions masquerade as dementia.

## Pseudobulbar Affect

The condition known as **pseudobulbar affect** involves inappropriate or exaggerated emotional expression, usually episodes of laughing or crying. It is associated with brain injury (e.g., stroke, traumatic brain injury, multiple sclerosis [MS], amyotrophic lateral sclerosis [ALS], AD, Parkinson's disease). The term "pseudobulbar" refers to damage that occurs in the corticobulbar tracts in the brain (see [Chapter 60](#)). The emotional outbursts can cause embarrassment, anxiety, and depression, and often impair quality of life (Hickey & Strayer, 2020).

Older adults with pseudobulbar affect can respond appropriately to treatment. Initial management involves evaluation and recognition that this condition can coexist with mood disorders, such as depression, although crying in these patients should not be considered indicative of depression. Pharmacology studies have reported effective management with dextromethorphan hydrobromide and quinidine sulfate in patients with ALS, MS, stroke, TBI, and dementia (Comerford & Durkin, 2020; Hakimi & Maurer, 2018).

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## SEIZURE DISORDERS

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Seizures are episodes of abnormal motor, sensory, autonomic, or psychic activity (or a combination of these) that result from sudden excessive discharge from cerebral neurons (Hickey & Strayer, 2020). A localized area or all of the brain may be involved. The International League Against Epilepsy (ILAE) has defined **epilepsy** as more than one unprovoked seizure (Fisher, Cross, French, et al., 2017). The ILAE differentiates between three main seizure types: focal onset, generalized onset, and unknown onset seizures (see [Chart 61-4](#)). Focal (or partial) seizures are thought to originate within a localized area of the brain. Generalized seizures occur in and rapidly engage bilaterally distributed networks. Unknown onset seizures can be described as "unclassified," so termed because of incomplete data surrounding the event, but they may also be described from their clinical features (Fisher et al., 2017). Seizures may also be characterized as "provoked," or related to acute, reversible conditions such as structural, metabolic, immune, infectious, or unknown etiologies.

**TABLE 61-4**

Summary of Differences Between Dementia and Delirium

	Dementia	Delirium	
	Alzheimer's Disease (AD)	Vascular (Multi-Infarct) Dementia	
Etiology	Early onset (familial, genetic [chromosomes 14, 19, 21]) Late-onset sporadic—etiology unknown	Cardiovascular (CV) disease Cerebrovascular disease Hypertension	Medication toxicity and interactions; acute disease; trauma; chronic disease exacerbation Fluid and electrolyte disorders
Risk factors	Advanced age; genetics	Preexisting CV disease	Preexisting cognitive impairment
Occurrence	75% of dementias	10–20% of dementias	Up to 80% among hospitalized people
Onset	Slow	Often abrupt Follows a stroke or transient ischemic attack	Rapid, acute onset A harbinger of acute medical illness
Age of onset	Early-onset AD: 40–65 yrs Late-onset AD: 65+ yrs Most commonly: 85+ yrs	Most commonly 50–70 yrs	Any age, although predominantly in older adults
Gender	Males and females equally	Predominantly males	Males and females equally
Course	Chronic, irreversible; progressive, regular, downhill	Chronic, irreversible Fluctuating, stepwise progression	Acute onset Hypoalert—hypoactive Hyperalert—hyperactive Mixed hypo—hyper
Duration	2–20 yrs	Variable; years	Lasts 1 day to 1 mo
Symptom progress	Onset insidious: <i>Early</i> —mild and subtle <i>Middle and late</i> —intensified Progression to death (infection or malnutrition) <i>Early</i> —no motor deficits	Depends on location of infarct and success of treatment; death attributed to underlying CV disease	Symptoms are fully reversible with adequate treatment; can progress to chronicity or death if underlying condition is ignored
Mood	Depression common	Labile; mood swings	Variable
Speech/language	Speech remains intact until late in disease: <i>Early</i> —mild anomia (cannot name objects); deficits progress until speech lacks meaning; echoes and repeats words and sounds; mutism <i>Early</i> —no motor deficits	May have speech deficit/aphasia depending on location of lesion	Fluctuating; often cannot concentrate long enough to speak May be somnolent
Physical signs	<i>Middle</i> —apraxia (cannot perform purposeful movement) <i>Late</i> —Dysarthria (impaired speech) <i>End stage</i> —loss of all voluntary activity; positive neurologic signs	According to location of lesion: focal neurologic signs, seizures Commonly exhibits motor deficits	Signs and symptoms of underlying disease
Orientation	Becomes lost in familiar places (topographic disorientation) Has difficulty drawing three-dimensional objects (visual and spatial disorientation) Disorientation to time, place, and person—with disease progression		May fluctuate between lucidity and complete disorientation to time, place, and person
Memory	Loss is an early sign of dementia; loss of recent memory is soon followed by progressive decline in recent and remote memory		Impaired recent and remote memory; may fluctuate between lucidity and confusion
Personality	Apathy, indifference, irritability: <i>Early disease</i> —social behavior intact; hides cognitive deficits <i>Advanced disease</i> —disengages from activity and relationships; suspicious; paranoid delusions caused by memory loss; aggressive; catastrophic reactions		Fluctuating; cannot focus attention to converse; alarmed by symptoms (when lucid); hallucinations; paranoid
Functional status, activities of daily living	Poor judgment in everyday activities; has progressive decline in ability to handle money, use telephone, use computer and other electronic devices, function in home and workplace		Impaired
Attention span	Distractible; short attention span		Highly impaired; cannot maintain or shift attention
Psychomotor	Wandering, hyperactivity, pacing, restlessness, agitation		Variable; alternates between high

activity		agitation, hyperactivity, restlessness, and lethargy
Sleep-wake cycle	Often impaired; wandering and agitation at nighttime	Takes brief naps throughout day and night

Adapted from Devlin, J. W., Skrobik, Y., Gelinas, C., et al. (2018). Clinical practice guidelines for the prevention and management of pain, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. *Critical Care Medicine*, 46(9), e825–e873; Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.



**Figure 61-9 •** Talking to family members may increase the comfort of patients with delirium.

## Pathophysiology

The underlying cause is an electrical disturbance (arrhythmia) in the nerve cells in one section of the brain; these cells emit abnormal, recurring, uncontrolled electrical discharges. The characteristic seizure is a manifestation of this excessive neuronal discharge. Associated loss of consciousness, excess movement or loss of muscle tone or movement, and disturbances of behavior, mood, sensation, and perception may also occur.

### Chart 61-4

### Classification of Seizures: 2017 Basic Scheme

#### Focal

- Motor
- Nonmotor
- Awareness
  - Aware
  - Impaired awareness
  - Unknown awareness

#### Generalized

- Motor
- Absence

#### Unknown

- Motor
- Nonmotor
- Awareness
  - Aware
  - Impaired awareness
  - Unknown awareness
- Unclassified

Adapted from Fisher, R., Cross, H., French, J., et al. (2017). Operational classification of seizure types by the International League Against Epilepsy (ILAE). Retrieved on 5/25/2020 at: [www.ilae.org/files/dmfile/Operational-Classification–Fisher\\_et\\_al-2017-Epilepsia.pdf](http://www.ilae.org/files/dmfile/Operational-Classification–Fisher_et_al-2017-Epilepsia.pdf)

The specific causes of seizures are varied and can be categorized as genetic, due to a structural or metabolic condition, or the cause may be yet unknown etiologies (Fisher et al., 2017).

Causes of seizures include:

- Allergies
- Brain tumor
- Cerebrovascular disease
- CNS infections
- Drug and alcohol withdrawal
- Fever (childhood)
- Head injury
- Hypertension
- Hypoxemia of any cause, including vascular insufficiency
- Metabolic and toxic conditions (e.g., kidney injury, hyponatremia, hypocalcemia, hypoglycemia, pesticide exposure)

### Clinical Manifestations

Depending on the location of the discharging neurons, seizures may range from a simple staring episode (generalized absence seizure) to prolonged convulsive movements with loss of consciousness.

The initial pattern of the seizures indicates the region of the brain in which the seizure originates (see Chart 61-4). Only a finger or hand may shake, or the mouth may jerk uncontrollably. The person may talk unintelligibly; may be dizzy; and may experience unusual or unpleasant sights, sounds, odors, or tastes, but without loss of consciousness (Hickey & Strayer, 2020).

Generalized seizures often involve both hemispheres of the brain, causing both sides of the body to react. Intense rigidity of the entire body may occur, followed by alternating muscle relaxation and contraction (generalized tonic-clonic contraction). The simultaneous contractions of the diaphragm and chest muscles may produce a characteristic epileptic cry. The tongue is often chewed, and the patient can be incontinent of urine and feces. After 1 or 2 minutes, the convulsive movements begin to subside; the patient relaxes and lies in deep coma, breathing noisily. The respirations at this point are chiefly abdominal. In the postictal state (after the seizure), the patient is often confused and hard to arouse and

may sleep for hours. Many patients report headache, sore muscles, fatigue, and depression (AANN, 2016a). Other generalized seizures may be absence types of seizures (Hickey & Strayer, 2020).

Focal seizures are subdivided into events characterized by both motor and nonmotor symptoms. There may be an impairment of consciousness or awareness or other dyscognitive features, localization, and progression of symptoms (Fisher et al., 2017).

## Assessment and Diagnostic Findings

The diagnostic assessment is aimed at determining the type of seizures, their frequency and severity, and the factors that precipitate them. A developmental history is taken, including events of pregnancy and childbirth, to seek evidence of preexisting injury. The patient is also questioned about illnesses or head injuries that may have affected the brain. In addition to physical and neurologic evaluations, diagnostic examinations include biochemical, hematologic, and serologic studies. MRI is used to detect structural lesions such as focal abnormalities, cerebrovascular abnormalities, and cerebral degenerative changes (AANN, 2016a).

The EEG furnishes diagnostic evidence for a substantial proportion of patients with epilepsy and assists in classifying the type of seizure. Abnormalities in the EEG usually continue between seizures or, if not apparent, may be elicited by hyperventilation or during sleep (AANN, 2016a). Microelectrodes (depth electrodes) can be inserted deep in the brain to probe the action of single brain cells. Some people with clinical seizures have normal EEGs, whereas others who have never had seizures have abnormal EEGs. Telemetry and computerized equipment are used to monitor electrical brain activity while the patient pursues their normal activities and to store the readings on computer tapes for analysis. Video recording of seizures taken simultaneously with EEG telemetry is useful in determining the type of seizure as well as its duration and magnitude (Hickey & Strayer, 2020).

SPECT is an additional tool that is sometimes used in the diagnostic workup. It is useful for identifying the epileptogenic zone so that the area in the brain giving rise to seizures can be removed surgically (AANN, 2016a).

## Nursing Management

### During a Seizure

A major responsibility of the nurse is to observe and record the sequence of signs. The nature of the seizure usually indicates the type of treatment required (AANN, 2016a). Before and during a seizure, the patient is assessed and the following items are documented:

- Circumstances before the seizure (visual, auditory, or olfactory stimuli; tactile stimuli; emotional or psychological disturbances; sleep; hyperventilation)
- Occurrence of an aura (a premonitory or warning sensation, which can be visual, auditory, or olfactory)
- First thing the patient does in the seizure—where the movements or the stiffness begins, conjugate gaze position, and the position of the head at the beginning of the seizure. This information gives clues to the location of the seizure origin in the brain. (In recording, it is important to state whether the beginning of the seizure was observed.)
- Type of movements in the part of the body involved
- Areas of the body involved (turn back bedding to expose patient)
- Size of both pupils and whether the eyes are open
- Whether the eyes or head are turned to one side
- Presence or absence of automatisms (involuntary motor activity, such as lip smacking or repeated swallowing)
- Incontinence of urine or stool
- Duration of each phase of the seizure
- Unconsciousness, if present, and its duration
- Any obvious paralysis or weakness of arms or legs after the seizure
- Inability to speak after the seizure
- Movements at the end of the seizure
- Whether or not the patient sleeps afterward
- Cognitive status (confused or not confused) after the seizure

In addition to providing data about the seizure, nursing care is directed at preventing injury and supporting the patient, not only physically but also psychologically. Consequences such as anxiety, embarrassment, fatigue, and depression can be devastating to the patient.

### After a Seizure

After a patient has a seizure, the nurse's role is to document the events leading to and occurring during and after the seizure and to prevent complications (e.g., aspiration, injury). The patient is at risk for hypoxia, vomiting, and pulmonary aspiration. To prevent complications, the patient is placed in the side-lying position to facilitate drainage of oral secretions, and suctioning is performed, if needed, to maintain a patent airway and prevent aspiration (see [Chart 61-5](#)). Seizure precautions are maintained, including having available functioning suction equipment with a suction catheter. The bed is placed in a low position with two to three side rails up and padded, if necessary, to prevent injury to the patient. The floor may be padded as an additional safety measure. The patient may be drowsy and may wish to sleep after the seizure; they may not remember events leading up to the seizure and for a short time thereafter.

## The Epilepsies

Epilepsy is a group of syndromes characterized by unprovoked, recurring seizures (AANN, 2016a). Epileptic syndromes are classified by specific patterns of clinical features, including age at onset, family history, and seizure type. Epilepsy can be primary (idiopathic) or secondary (when the cause is known and the epilepsy is a symptom of another underlying condition, such as a brain tumor).

Epilepsy affects an estimated 3% of people during their lifetime, and most forms of epilepsy occur in children and older adults (Hickey & Strayer, 2020). The improved treatment for cerebrovascular disorders, head injuries, brain tumors, meningitis, and encephalitis has increased the number of patients at risk for seizures after recovery from these conditions. In addition, advances in EEG have aided in the diagnosis of epilepsy. The general public has been educated about epilepsy, which has reduced the stigma associated with it; as a result, more people are willing to acknowledge that they have epilepsy.

Although some evidence suggests that susceptibility to some types of epilepsy may be inherited, the cause of seizures in many people is idiopathic (unknown). Epilepsy can follow birth trauma, asphyxia neonatorum, head injuries, some infectious diseases (bacterial, viral, parasitic), toxicity (carbon monoxide and lead poisoning), circulatory problems, fever, metabolic and nutritional disorders, or drug or alcohol intoxication. It is also associated with brain tumors, abscesses, and congenital malformations.

## Pathophysiology

Messages from the body are carried by the neurons (nerve cells) of the brain by discharges of electrochemical energy that sweep along them. These impulses occur in bursts whenever a nerve cell has a task to perform. Sometimes, these cells or groups of cells continue firing after a task is finished. During the period of unwanted discharges, parts of the body controlled by the errant cells may perform erratically. Resultant dysfunction ranges from mild to incapacitating and often causes loss of consciousness (Hickey & Strayer, 2020). If these uncontrolled, abnormal discharges occur repeatedly, a person is said to have an epileptic syndrome. Epilepsy is not associated with intellectual level. People who have epilepsy without other brain or nervous system disabilities fall within the same intelligence ranges as the overall population. Epilepsy is not synonymous with intellectual or developmental disabilities, but many people who have these types of disabilities, because of serious neurologic damage, also have epilepsy.

### Chart 61-5

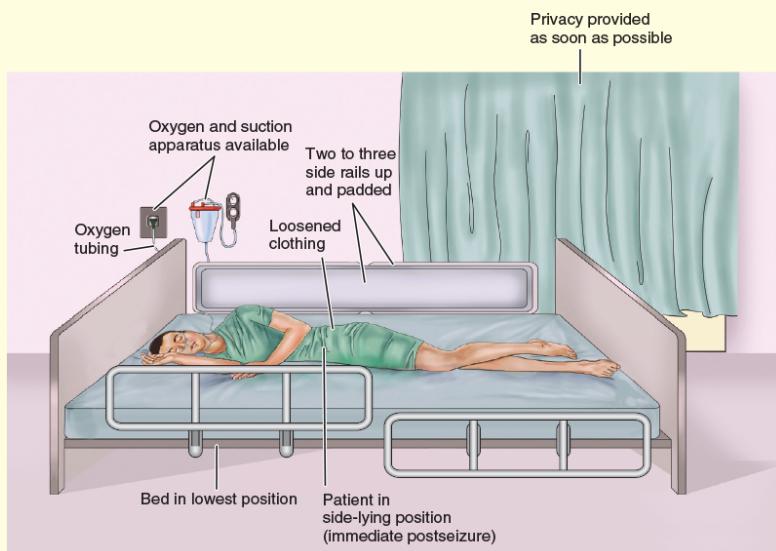
## Care of the Patient During and After a Seizure

### Nursing Care During a Seizure

- Provide privacy, and protect the patient from curious onlookers. (The patient who has an aura may have time to seek a safe, private place.)
- Ease the patient to the floor, if possible.
- Protect the head with a pad to prevent injury (from striking a hard surface).
- Loosen constrictive clothing and remove eyeglasses.
- Push aside any furniture that may injure the patient during the seizure.
- If the patient is in bed, remove pillows and raise side rails.
- Do not attempt to pry open jaws that are clenched in a spasm or attempt to insert anything in the mouth during a seizure.* Broken teeth and injury to the lips and tongue may result from such an action.
- Do not attempt to restrain the patient during the seizure, because muscular contractions are strong and restraint can produce injury.
- If possible, place the patient on one side with head flexed forward, which allows the tongue to fall forward and facilitates drainage of saliva and mucus. If suction is available, use it if necessary to clear secretions.

### Nursing Care After the Seizure

- Keep the patient on one side to prevent aspiration. Make sure the airway is patent.
- On awakening, reorient the patient to the environment.
- If the patient is confused or wandering, guide the patient gently to a bed or chair.
- If the patient becomes agitated after a seizure (postictal), stay a distance away, but close enough to prevent injury until the patient is fully aware.



Adapted from American Association of Neuroscience Nurses (AANN). (2016a). *Care of adults and children with seizures and epilepsy: AANN clinical practice guideline series*. Chicago, IL: Author.

Patients with epilepsy, particularly those with generalized events that are medically refractory, are at serious risk for **Sudden Unexpected Death in Epilepsy (SUDEP)**, defined as nontraumatic, nondrowning unexpected death of a patient with epilepsy. These events may be witnessed or unwitnessed and postmortem examination reveals no anatomic or toxicologic cause of death. Cardiac and respiratory abnormalities have been implicated in these deaths. SUDEP may or may not be related to a seizure event (Barot & Nei, 2019).

## Epilepsy in Women

More than one million American women have epilepsy, and they face particular needs associated with the syndrome. Women with epilepsy often note an increase in seizure frequency during menses; this has been

linked to the increase in sex hormones that alter the excitability of neurons in the cerebral cortex. The effectiveness of contraceptives is decreased by anticonvulsant medications. Therefore, patients should be encouraged to discuss family planning with their primary provider and to obtain preconception counseling if they are considering childbearing (Stephen, Harden, Tomson, et al., 2019).

Women of childbearing age who have epilepsy require special care and guidance before, during, and after pregnancy. Many women note a change in the pattern of seizure activity during pregnancy. The risk of congenital fetal anomaly is two to three times higher in women with epilepsy. Maternal seizures, anticonvulsant medications, and genetic predisposition all contribute to possible malformations. Women who take certain anticonvulsant medications for epilepsy are at risk and need careful monitoring, including blood studies to detect the level of anticonvulsant medications taken throughout pregnancy. Mothers who are at high risk (teenagers, women with histories of difficult deliveries, women who use illicit drugs [e.g., “crack” cocaine, heroin], and women with diabetes or hypertension) should be identified and monitored closely during pregnancy, because damage to the fetus during pregnancy and delivery can increase the risk of epilepsy. All of these issues need further study (Stephen et al., 2019).

Because of bone loss associated with the long-term use of anticonvulsant medications, patients receiving anticonvulsant agents should be assessed for low bone mass and osteoporosis. They should be educated about strategies to reduce their risks of osteoporosis (AANN, 2016a).



## Gerontologic Considerations

Older adults have a high incidence of new-onset epilepsy (Hickey & Strayer, 2020). Cerebrovascular disease is the leading cause of seizures in the older adult but they are also associated with head injury, dementia, infection, alcoholism, and aging. Treatment depends on the underlying cause. Because many older adults have chronic health problems, they may be taking other medications that can interact with medications prescribed for seizure control. In addition, the absorption, distribution, metabolism, and excretion of medications are altered in the older adult as a result of age-related changes in renal and liver function. Therefore, older adult patients must be monitored closely for adverse and toxic effects of anticonvulsant medications and for osteoporosis.

## Prevention

Society-wide efforts are the key to prevention of epilepsy. Head injury is one of the main causes of epilepsy that can be prevented. Through highway safety programs and occupational safety precautions, lives can be saved and epilepsy due to head injury prevented; these programs are discussed in [Chapter 63](#).

## Medical Management

The management of epilepsy is individualized to meet the needs of each patient and not just to manage and prevent seizures. Management differs from patient to patient, because some forms of epilepsy arise from brain damage and others result from altered brain chemistry.

### Pharmacologic Therapy

Many medications are available to control seizures, although the exact mechanisms of action are unknown. The objective is to achieve seizure control with minimal side effects. Medication therapy controls—rather than cures—seizures. Medications are selected on the basis of the type of seizure being treated and the effectiveness and safety of the medications. If properly prescribed and taken, medications control seizures in 70% to 80% of patients with seizures. However, 20% of patients with generalized seizures and 30% of those with focal seizures do not demonstrate improvement with any prescribed medication or may be unable to tolerate the side effects of medications (AANN, 2016a). [Table 61-5](#) lists select anticonvulsant medications.

Treatment usually starts with a single medication. The starting dose and the rate at which the dosage is increased depend on the occurrence of side effects. The medication levels in the blood are monitored, because the rate of drug absorption varies among patients. Changing to another medication may be necessary if seizure control is not achieved or if toxicity makes it impossible to increase the dosage. The medication may need to be adjusted because of concurrent illness, weight changes, or increases in stress. Side effects of anticonvulsant medications may be divided into three groups: idiosyncratic or allergic

disorders, which manifest primarily as skin reactions; acute toxicity, which may occur when the medication is initially prescribed; and chronic toxicity, which occurs late in the course of therapy.

The manifestations of drug toxicity are variable, and any organ system may be involved. For example, gingival hyperplasia (swollen and tender gums) can be associated with long-term use of phenytoin (Comerford & Durkin, 2020). Periodic physical and dental examinations and laboratory tests are performed for patients receiving medications that are known to have hematopoietic, genitourinary, or hepatic effects.

### Surgical Management

Surgery is indicated for patients whose epilepsy results from intracranial tumors, abscesses, cysts, or vascular anomalies. Some patients have intractable seizure disorders that do not respond to medication. A focal atrophic process may occur secondary to trauma, inflammation, stroke, or anoxia. If the seizures originate in a reasonably well-circumscribed area of the brain that can be excised without producing significant neurologic deficits, the removal of the area generating the seizures may produce long-term control and improvement (AANN, 2016a).

This type of neurosurgery has been aided by several advances, including microsurgical techniques, EEGs with depth electrodes, improved illumination and hemostasis, and the introduction of neuroleptanalgesic agents (droperidol and fentanyl). These techniques, combined with the use of local anesthetic agents, enable the neurosurgeon to perform surgery on an alert and cooperative patient. Using special testing devices, electrocortical mapping, and the patient's responses to stimulation, the boundaries of the epileptogenic focus (i.e., abnormal area of the brain) are determined. Any abnormal epileptogenic focus is then excised (AANN, 2016a). Resection surgery significantly reduces the incidence of seizures in patients with refractory epilepsy.

**TABLE 61-5**  Select Anticonvulsant Medications

Medication	Dose-Related Side Effects	Toxic Effects
carbamazepine	Dizziness, drowsiness, unsteadiness, nausea and vomiting, diplopia, mild leukopenia	Severe skin rash, blood dyscrasias, hepatitis
clonazepam	Drowsiness, behavior changes, headache, hirsutism, alopecia, palpitations	Hepatotoxicity, thrombocytopenia, bone marrow failure, ataxia
ethosuximide	Nausea and vomiting, headache, gastric distress	Skin rash, blood dyscrasias, hepatitis, systemic lupus erythematosus
felbamate	Cognitive impairments, insomnia, nausea, headache, fatigue	Aplastic anemia, hepatotoxicity
gabapentin	Dizziness, drowsiness, somnolence, fatigue, ataxia, weight gain, nausea	Leukopenia, hepatotoxicity
lamotrigine	Drowsiness, tremor, nausea, ataxia, dizziness, headache, weight gain	Severe rash (Stevens–Johnson syndrome)
levetiracetam	Somnolence, dizziness, fatigue	Unknown
oxcarbazepine	Dizziness, somnolence, double vision, fatigue, nausea, vomiting, loss of coordination, abnormal vision, abdominal pain, tremor, abnormal gait	Hepatotoxicity
phenobarbital	Sedation, irritability, diplopia, ataxia	Skin rash, anemia
phenytoin	Visual problems, hirsutism, gingival hyperplasia, arrhythmias, dysarthria, nystagmus	Severe skin reaction, peripheral neuropathy, ataxia, drowsiness, blood dyscrasias
primidone	Lethargy, irritability, diplopia, ataxia, impotence	Skin rash
tiagabine	Dizziness, fatigue, nervousness, tremor, difficulty concentrating, dysarthria, weak or buckling knees, abdominal pain	Unknown
topiramate	Fatigue, somnolence, confusion, ataxia, anorexia, depression, weight loss	Nephrolithiasis
valproate	Nausea and vomiting, weight gain, hair loss, tremor, menstrual irregularities	Hepatotoxicity, skin rash, blood dyscrasias, nephritis
zonisamide	Somnolence, dizziness, anorexia, headache, nausea, agitation, rash	Leukopenia, hepatotoxicity

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

When seizures are refractory to medication in adolescents and adults with focal seizures, a vagal nerve stimulator (VNS) may be implanted under the clavicle. The device is connected to the vagus nerve in the

cervical area, where it delivers electrical signals to the brain to control and reduce seizure activity. An external programming system is used by the primary provider to change stimulator settings (Tzadok, Harush, Nissenkorn, et al., 2019). Patients can activate the stimulator with a magnet at the time of a seizure or aura. Some patients report that use of the VNS diminishes the severity or duration of the seizure. Complications such as infection, cardiac arrhythmias, hoarseness, cough, and laryngeal spasm can occur with the use of this device (AANN, 2016a).

Another surgical option for patients with refractory seizure activity is the responsive neurostimulation system (RNS). This is a surgically implanted device with electrodes that sense and record brain electrical activity. Electrodes deliver an electrical stimulation to the location of seizure origination within the brain. The RNS works by interrupting brainwave activity before a clinical seizure can occur (Wong, Mani, & Danish, 2019).

For patients with well-defined or anatomically deep epileptogenic lesions, MRI-guided stereotactic laser interstitial thermal therapy (LiTT) offers a less invasive treatment option. This treatment involves computer-assisted placement of a laser probe into the brain and delivery of heat therapy. Decisions about epilepsy surgery are complex, and these patients should be referred to epilepsy centers for further evaluation (Crepeau & Sirven, 2017).

More research is needed to determine the effects of the various surgical approaches on complication rates, quality of life, anxiety, and depression, all of which are issues for patients with epilepsy.

## NURSING PROCESS

### The Patient with Epilepsy

#### Assessment

The nurse elicits information about the patient's seizure history. The patient is asked about the factors or events that may precipitate the seizures. Alcohol intake is documented. The nurse determines whether the patient has an aura before an epileptic seizure, which may indicate the origin of the seizure (e.g., seeing a flashing light may indicate that the seizure originated in the occipital lobe). Observation and assessment during and after a seizure assist in identifying the type of seizure and its management.

The effects of epilepsy on the patient's lifestyle are assessed (AANN, 2016a). What limitations are imposed by the seizure disorder? Does the patient participate in any recreational activities? Have any social contacts? Is the patient working, and is it a positive or stressful experience? What coping mechanisms are used?

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Risk for injury associated with seizure activity
- Fear associated with the possibility of seizures
- Difficulty coping associated with stresses imposed by epilepsy
- Lack of knowledge associated with epilepsy and anticonvulsant medications

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

The major potential complications for patients with epilepsy are status epilepticus and medication side effects (toxicity).

#### Planning and Goals

The major goals for the patient may include prevention of injury, control of seizures, achievement of a satisfactory psychosocial adjustment, acquisition of knowledge and understanding about the condition, and absence of complications.

#### Nursing Interventions

##### PREVENTING INJURY

Injury prevention for the patient with seizures is a priority. Patients for whom seizure precautions are instituted should have pads applied to the side rails while in bed. Steps to prevent or minimize injury are presented in [Chart 61-5](#).

##### REDUCING FEAR OF SEIZURES

Fear that a seizure may occur unexpectedly can be reduced by the patient's adherence to the prescribed treatment regimen. Cooperation of the patient and family and their trust in the prescribed regimen are essential for control of seizures. The nurse emphasizes that the prescribed anticonvulsant medication must be taken on a continuing basis and that drug dependence or addiction does not occur. Periodic monitoring is necessary to ensure the adequacy of the treatment regimen, to prevent side effects, and to monitor for drug resistance (Hickey & Strayer, 2020).

In an effort to control seizures, factors that may precipitate them are identified, such as emotional disturbances, new environmental stressors, onset of menstruation in female patients, or fever (AANN, 2016a). The patient is encouraged to follow a regular and moderate routine in lifestyle, diet (avoiding excessive stimulants), exercise, and rest (sleep deprivation may lower the seizure threshold). Moderate activity is therapeutic, but excessive exercise should be avoided. An additional dietary intervention, referred to as the ketogenic diet or the Modified Atkins diet, may be helpful for control of seizures in some patients. This high-protein, low-carbohydrate, high-fat diet is most effective in children whose seizures have not been controlled with two anticonvulsant medications and has shown some success in adults with poor seizure control. Dietary therapy is not without risk and requires close monitoring and medical follow-up for possible side effects of therapy such as hyperlipidemia, malnutrition, weight loss, and osteoporosis (Crepeau & Sirven, 2017).

Photic stimulation (e.g., bright flickering lights, television viewing) may precipitate seizures; wearing dark glasses or covering one eye may be preventive. Tension states (anxiety, frustration) induce seizures in some patients. Classes in stress management may be of value. Because seizures are known to occur with alcohol intake, alcoholic beverages should be avoided.

#### **IMPROVING COPING MECHANISMS**

The social, psychological, and behavioral problems that frequently accompany epilepsy can be more of a disability than the actual seizures. Epilepsy may be accompanied by feelings of stigmatization, alienation, depression, and uncertainty (Hickey & Strayer, 2020). The patient must cope with the constant fear of a seizure and the psychological consequences (AANN, 2016a). Children with epilepsy may be ostracized and excluded from school and peer activities. These problems are compounded during adolescence and add to the challenges of dating, not being able to drive, and feeling different from other people. Adults face these problems in addition to the burden of finding employment, concerns about relationships and childbearing, insurance problems, and legal barriers. Substance use disorders may complicate matters. Family reactions may vary from outright rejection of the person with epilepsy to overprotection.

Counseling assists the patient and family to understand the condition and the limitations it imposes. Social and recreational opportunities are necessary for good mental health. Nurses can improve the quality of life for patients with epilepsy by educating them and their families about symptoms and their management (AANN, 2016a).



#### **PROVIDING PATIENT AND FAMILY EDUCATION**

Perhaps the most valuable facets of care contributed by the nurse to the person with epilepsy are education and efforts to modify the attitudes of the patient and family toward the disorder. The person who experiences seizures may consider every seizure a potential source of humiliation and shame. This may result in anxiety, depression, hostility, and secrecy on the part of the patient and family. Ongoing education and encouragement should be given to patients to enable them to overcome these reactions. The patient with epilepsy should carry an emergency medical identification card or wear a medical information bracelet. The patient and family need to be educated about medications as well as care during a seizure.

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Status epilepticus is the major potential complication and is described later in this chapter. Another complication is the toxicity of medications. The patient and family are educated about side effects and are given specific guidelines to assess and report signs and symptoms that indicate medication overdose. Anticonvulsant medications require careful monitoring for therapeutic levels. The patient should plan to have serum drug levels assessed at regular intervals. Many known drug interactions occur with anticonvulsant medications. A complete pharmacologic profile should be reviewed with the patient to avoid interactions that either potentiate or inhibit the effectiveness of the medications.



#### **Quality and Safety Nursing Alert**

*Patients with epilepsy are at risk for status epilepticus from having their medication regimen interrupted.*

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



**Educating Patients About Self-Care.** Thorough oral hygiene after each meal, gum massage, daily flossing, and regular dental care are essential to prevent or control gingival hyperplasia in patients receiving phenytoin. The patient is also educated to inform all health care providers of the medication being taken, because of the possibility of drug interactions. An individualized comprehensive education plan is needed to assist the patient and family to adjust to this chronic disorder. Written patient education materials must be appropriate for the patient's reading level and must be provided in alternative formats if warranted.

**Continuing and Transitional Care.** Because epilepsy can be lifelong, health promotion is important. See [Chart 61-6](#) for health promotion strategies for the patient with epilepsy.

For many patients with epilepsy, overcoming employment problems is a challenge. State vocational rehabilitation agencies can provide information about job training. The EFA has a training and placement service. If seizures are not well controlled, information about sheltered workshops or home employment programs may be obtained. Federal and state agencies and federal legislation may be of assistance to people with epilepsy who experience job discrimination. As a result of the Americans

With Disabilities Act, the number of employers who knowingly hire people with epilepsy is increasing, but barriers to employment still exist.

Chart 61-6



## HEALTH PROMOTION

### Strategies for the Patient with Epilepsy

- Take anticonvulsant medications daily as prescribed to keep the drug level constant to prevent seizures. Never discontinue medications, even if there is no seizure activity.
- Keep a medication and seizure record (in electronic or paper format), noting when medications are taken and any seizure activity.
- Notify the primary provider if unable to take medications due to illness.
- Have anticonvulsant medication serum levels checked regularly. When testing is prescribed, report to the laboratory for blood sampling before taking morning medication.
- Avoid activities that require alertness and coordination (driving, operating machinery) until after the effects of the medication have been evaluated.
- Report signs of toxicity so that dosage can be adjusted. Common signs include drowsiness, lethargy, dizziness, difficulty walking, hyperactivity, confusion, inappropriate sleep, and visual disturbances.
- Avoid over-the-counter medications unless approved by the primary provider.
- Carry a medical alert bracelet or identification card specifying the name of the anticonvulsant medication and primary provider.
- Avoid seizure triggers, such as alcoholic beverages, electrical shocks, stress, caffeine, constipation, fever, hyperventilation, and hypoglycemia.
- Take showers rather than tub baths to avoid drowning if seizure occurs; never swim alone.
- Exercise in moderation in a temperature-controlled environment to avoid excessive heat.
- Develop regular sleep patterns to minimize fatigue and insomnia.
- Be aware of and use the Epilepsy Foundation of America (EFA) special services, including help in obtaining medications, vocational rehabilitation, and coping with epilepsy.

Patients who have uncontrollable seizures accompanied by psychological and social difficulties should be referred as early as possible to a comprehensive epilepsy center where continuous audio-video and EEG monitoring, specialized treatment, and rehabilitation services are available (AANN, 2016a). Patients and their families need to be reminded of the importance of participating in health promotion activities and recommended health screenings to promote a healthy lifestyle. Genetic and preconception counseling is advised.

### Evaluation

Expected patient outcomes may include:

1. Sustains no injury during seizure activity
  - a. Adheres to treatment regimen and identifies the hazards of stopping the medication
  - b. Can identify appropriate care during seizure; caregivers can do so as well
2. Indicates a decrease in fear
3. Displays effective individual coping
4. Exhibits knowledge and understanding of epilepsy
  - a. Identifies the side effects of medications
  - b. Avoids factors or situations that may precipitate seizures (e.g., flickering lights, hyperventilation, alcohol)

- c. Follows a healthy lifestyle by getting adequate sleep and eating meals at regular times to avoid hypoglycemia
  - 5. Absence of complications
- 

## Status Epilepticus

Status epilepticus (acute prolonged seizure activity) can be defined as a seizure lasting 5 minutes or longer or serial seizures occurring without full recovery of consciousness between attacks (Hickey & Strayer, 2020). The term has been broadened to include continuous clinical or electrical seizures (on EEG) lasting at least 30 minutes, even without impairment of consciousness. It is considered a medical emergency. Status epilepticus produces cumulative effects. Vigorous muscular contractions impose a heavy metabolic demand and can interfere with respirations. Some respiratory arrest at the height of each seizure produces venous congestion and hypoxia of the brain. Repeated episodes of cerebral anoxia and edema may lead to irreversible and fatal brain damage. Factors that precipitate status epilepticus include interruption of anticonvulsant medication, fever, concurrent infection, or other illness.

## Medical Management

The goals of treatment are to stop the seizures as quickly as possible, to ensure adequate cerebral oxygenation, and to maintain the patient in a seizure-free state. An airway and adequate oxygenation are established. If the patient remains unconscious and unresponsive, an endotracheal tube is inserted. IV diazepam, lorazepam, or fosphenytoin is given slowly in an attempt to halt seizures immediately. Other medications (phenytoin, phenobarbital) are given later to maintain a seizure-free state.

An IV line is established, and blood samples are obtained to monitor serum electrolytes, glucose, and phenytoin levels. EEG monitoring may be useful in determining the nature of the seizure activity. Vital signs and neurologic signs are monitored on a continuing basis. An IV infusion of dextrose is given if the seizure is caused by hypoglycemia. If initial treatment is unsuccessful, general anesthesia with a short-acting barbiturate may be used. The serum concentration of the anticonvulsant medication is measured, because a low level suggests that the patient was not taking the medication or that the dosage was too low. Cardiac involvement or respiratory depression may be life-threatening. The potential for postictal cerebral edema also exists.

## Nursing Management

The nurse initiates ongoing assessment and monitoring of respiratory and cardiac function because of the risk for delayed depression of respiration and blood pressure secondary to administration of anticonvulsant medications and sedatives to halt the seizures. Nursing assessment also includes monitoring and documenting the seizure activity and the patient's responsiveness.

The patient is turned to a side-lying position, if possible, to assist in draining pharyngeal secretions. Suction equipment must be available because of the risk of aspiration. The IV line is closely monitored, because it may become dislodged during seizures.

A person who has received long-term anticonvulsant therapy has a significant risk for fractures resulting from bone disease (osteoporosis, osteomalacia, and hyperparathyroidism), which is a side effect of therapy (Comerford & Durkin, 2020). Therefore, during seizures, the patient is protected from injury with the use of seizure precautions and is monitored closely. The patient having seizures can inadvertently injure nearby people, so nurses should protect themselves. Additional nursing interventions for the person having seizures are presented in [Chart 61-5](#).

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## HEADACHE

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Headache, or cephalgia, is one of the most common of all human physical complaints. Headache is a symptom rather than a disease entity; it may indicate organic disease (neurologic or other disease), a stress response, vasodilation (migraine), skeletal muscle tension (tension headache), or a combination of factors. A **primary headache** is one for which no organic cause can be identified. This type of headache includes migraine, tension-type, and cluster headaches (Hickey & Strayer, 2020). Cranial arteritis is another

common cause of headache. A classification of headaches was issued first by the Headache Classification Committee of the International Headache Society (IHS) in 1988. The IHS revised the headache classification in 2018; an abbreviated list is shown in [Chart 61-7](#).

### Chart 61-7

#### International Headache Society Classification of Headache

- Migraine
- Tension-type headache
- Trigeminal autonomic cephalgias
- Other primary disorders
- Headache attributed to trauma or injury to the head and/or neck
- Headache attributed to cranial or cervical vascular disorder
- Headache attributed to nonvascular intracranial disorder
- Headache attributed to a substance or its withdrawal
- Headache attributed to infection
- Headache attributed to disorder of homeostasis
- Headache or facial pain attributed to disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures
- Headache attributed to psychiatric disorder
- Painful cranial neuropathies and other facial pains
- Other headache disorders

Adapted from Headache Classification Committee of the International Headache Society (IHS). (2018). *The International Classification of Headache Disorders*, 3rd edition. *Cephalgia*, 38(1), 1–211.

**Migraine** is a complex of symptoms characterized by periodic and recurrent attacks of severe headache lasting from hours to days in adults. The cause of migraine has not been clearly demonstrated, but it is primarily a vascular disturbance that has a strong familial tendency. The typical time of onset is at puberty, and the incidence is higher in women than men (Hickey & Strayer, 2020).

There are many subtypes of migraine headache, including migraine with and without aura. *Tension-type headaches* tend to be chronic and less severe and are probably the most common type of headache. *Trigeminal autonomic cephalgias* include cluster headaches and paroxysmal hemicrania. Cluster headaches are relatively uncommon and seen more frequently in men than in women (Norris, 2019). Types of headaches not subsumed under these categories fall into the *other primary headache* group and include headaches triggered by cough, exertion, and sexual activity (IHS, 2018).

*Cranial arteritis* is a cause of headache in the older population, reaching its greatest incidence in those older than 70 years of age. Inflammation of the cranial arteries is characterized by a severe headache localized in the region of the temporal arteries. The inflammation may be generalized (in which case cranial arteritis is part of a vascular disease) or focal (in which case only the cranial arteries are involved).

A **secondary headache** is a symptom associated with other causes, such as a brain tumor, an aneurysm, or lumbar puncture. Although most headaches do not indicate serious disease, persistent headaches require further investigation. Serious disorders related to headache include brain tumors, subarachnoid hemorrhage, stroke, severe hypertension, meningitis, and head injuries.

### Pathophysiology

The cerebral signs and symptoms of migraine result from a hyperexcitable brain that is susceptible to a phenomenon known as cortical spreading depression, a wave of depolarization over the cerebral cortex, cerebellum, and hippocampus. This depolarization activates inflammatory neuropeptides and other neurotransmitters (including serotonin), resulting in the stimulation of meningeal nociceptors. Vascular changes, inflammation, and a continuation of pain signal stimulation occur (Goadsby & Holland, 2019). The initial phase of this process is known as the premonitory phase and may include light, sound, and smell sensitivity. If treatment is initiated at this point, the migraine may be fully terminated. As the attack progresses, central sensitization occurs, and the migraine becomes much harder to treat.

Attacks can be triggered by hormonal changes associated with menstrual cycles, bright lights, stress, depression, sleep deprivation, fatigue, or odors. Certain foods containing tyramine (aged cheese, red wine,

beer), monosodium glutamate, and chocolate may be food triggers (Hickey & Strayer, 2020). The use of oral contraceptives may be associated with increased frequency and severity of attacks in some women.

Emotional or physical stress may cause contraction of the muscles in the neck and scalp, resulting in tension headache. The pathophysiology of cluster headache is not fully understood. One theory is that it is caused by dilation of orbital and nearby extracranial arteries. Cranial arteritis is thought to represent an immune vasculitis in which immune complexes are deposited within the walls of affected blood vessels, producing vascular injury and inflammation. A biopsy may be performed on the involved artery to make the diagnosis.

## Clinical Manifestations

### Migraine

The migraine with aura can be divided into four phases: premonitory, aura, the headache, and recovery (headache termination and postdrome).

#### Premonitory Phase

The premonitory phase is experienced by more than 80% of adult migraine sufferers, with symptoms that occur hours to days before a migraine headache. Symptoms may include depression, irritability, feeling cold, food cravings, anorexia, change in activity level, increased urination, diarrhea, or constipation. Patients may experience the same prodrome with each migraine headache. A current theory regarding premonitory symptoms is that they involve the neurotransmitter dopamine.

#### Aura Phase

An aura may be a variable feature for patients who experience migraines and can be seen in about 30% of patients (Goadsby & Holland, 2019). An aura is characterized by focal neurologic symptoms. Visual disturbances (i.e., light flashes and bright spots) are most common and may be hemianopic (affecting only half of the visual field). Other symptoms that may follow include numbness and tingling of the lips, face, or hands; mild confusion; slight weakness of an extremity; drowsiness; and dizziness.

This period of aura was thought to correspond to the phenomenon of cortical spreading depression that is associated with reduced metabolic demand in abnormally functioning neurons. This can be associated with decreased blood flow; however, cerebral blood flow studies performed during migraine headaches demonstrate that although changes in blood vessels occur during phases of migraine, cerebral blood flow is not the main abnormality. In fact, some studies suggest that the aura and headache phases may occur simultaneously (Goadsby & Holland, 2019).

#### Headache Phase

Migraine headache is severe and incapacitating and is often associated with photophobia (light sensitivity), phonophobia (sound sensitivity), or allodynia (abnormal perception of innocuous stimuli) (Goadsby & Holland, 2019). Research differs in the role of vascular changes (either vasodilatory or vasoconstrictive) with respect to migraine pathophysiology and the experience of migraine headache. Symptoms of migraine can also include nausea and vomiting.

#### Postdrome Phase

In the postdrome phase, the pain gradually subsides, but patients may experience tiredness, weakness, cognitive difficulties, and mood changes for hours to days. Muscle contraction in the neck and scalp is common, with associated muscle ache and localized tenderness. Physical exertion may exacerbate the headache pain. During this postheadache phase, patients may sleep for extended periods.

### Other Headache Types

The tension-type headache is characterized by a steady, constant feeling of pressure that usually begins in the forehead, temple, or back of the neck. It is often bandlike or may be described as “a weight on top of my head.”

Cluster headaches are unilateral and come in clusters of one to eight daily, with excruciating pain localized to the eye and orbit and radiating to the facial and temporal regions. The pain is accompanied by watering of the eye and nasal congestion. Each attack lasts 15 minutes to 3 hours and may have a crescendo-decrescendo pattern (Hickey & Strayer, 2020). The headache is often described as penetrating.

Cranial arteritis often begins with general manifestations, such as fatigue, malaise, weight loss, and fever. Clinical manifestations associated with inflammation (heat, redness, swelling, tenderness, or pain over the involved artery) usually are present. Sometimes a tender, swollen, or nodular temporal artery is visible. Visual problems are caused by ischemia of the involved structures.

## Assessment and Diagnostic Findings

The diagnostic evaluation includes a detailed history, a physical assessment of the head and neck, and a complete neurologic examination. Headaches may manifest differently in the same person over the course of a lifetime, and the same type of headache may manifest differently from patient to patient. The health history focuses on assessing the headache itself, with emphasis on the factors that precipitate or provoke it. The patient is asked to describe the headache in their own words.

Because headache is often the presenting symptom of a wider variety of physiologic and psychological disturbances, a general health history is an essential component of the patient database. Therefore, questions addressed in the health history should cover major medical and surgical illness as well as a body systems review.

The medication history can provide insight into the patient's overall health status and indicate medications that may be provoking headaches. Antihypertensive agents, diuretic medications, anti-inflammatory agents, and monoamine oxidase (MAO) inhibitors are a few of the categories of medications that can provoke headaches. Daily use of over-the-counter or prescribed pain medications for 8 to 10 days out of a month can lead to a chronic headache due to medication overuse (Comerford & Durkin, 2020). Emotional factors can play a role in precipitating headaches. Stress is thought to be a major initiating factor in migraine headaches; therefore, sleep patterns, level of stress, recreational interests, appetite, emotional problems, and family stressors are relevant. There is a strong familial tendency for headache disorders, and a positive family history may help in making a diagnosis.

A direct relationship may exist between exposure to toxic substances and headache. Careful questioning may uncover chemicals to which a worker has been exposed. Under the Right-to-Know Law, employees have access to the material safety data sheets (commonly referred to as MSDSs) for all substances with which they come in contact in the workplace (see [Chapter 68](#)). The occupational history also includes assessment of the workplace as a possible source of stress and for a possible ergonomic basis of muscle strain and headache.

A complete description of the headache itself is crucial. The nurse reviews the age at onset of headaches; this particular headache's frequency, location, and duration; the type of pain; factors that relieve and precipitate the event; and associated symptoms (Starling, 2018). The data obtained should include the patient's own words about the headache in response to the following questions:

- What is the location? Is it unilateral or bilateral? Does it radiate?
- What is the quality—dull, aching, steady, boring, burning, intermittent, continuous, paroxysmal?
- How many headaches occur during a given period of time?
- What are the precipitating factors, if any—environmental (e.g., sunlight, weather change), foods, exertion, other?
- What makes the headache worse (e.g., coughing, straining)?
- What time (day or night) does it occur?
- How long does a typical headache last?
- Are there any associated symptoms, such as facial pain, lacrimation (excessive tearing), or scotomas (blind spots in the field of vision)?
- What usually relieves the headache (aspirin, nonsteroidal anti-inflammatory drugs [NSAIDs], ergot preparation, food, heat, rest, neck massage)?
- Does nausea, vomiting, weakness, or numbness in the extremities accompany the headache?
- Does the headache interfere with daily activities?
- Do you have any allergies?
- Do you have insomnia, poor appetite, loss of energy?
- Is there a family history of headache?
- What is the relationship of the headache to your lifestyle or physical or emotional stress?
- What medications are you taking?

Diagnostic testing often is not helpful in the investigation of headache, because usually there are few objective findings. In patients who demonstrate abnormalities on the neurologic examination, CT scan, cerebral angiography, or MRI scan may be used to detect underlying causes, such as tumor or aneurysm.

Electromyography (EMG) may reveal a sustained contraction of the neck, scalp, or facial muscles. Laboratory tests may include complete blood count, erythrocyte sedimentation rate, electrolytes, glucose, creatinine, and thyroid hormone levels.

## Prevention

Prevention begins by having the patient avoid specific triggers that are known to initiate the headache syndrome. Preventive medical management of migraine involves the daily use of one or more agents that are thought to block the physiologic events leading to an attack. Treatment regimens vary greatly, as do patient responses; therefore, close monitoring is indicated.

Alcohol, nitrates, vasodilators, and histamines may precipitate cluster headaches. Elimination of these factors helps prevent the headaches.

## Medical Management

Therapy for migraine headache is divided into abortive (symptomatic) and preventive approaches. The abortive approach, best used in those patients who have less frequent attacks, is aimed at relieving or limiting a headache at the onset or while it is in progress. The preventive approach is used in patients who experience more frequent attacks at regular or predictable intervals and may have a medical condition that precludes the use of abortive therapies (Starling, 2018). Medical management of migraine during pregnancy and lactation includes nonpharmacologic strategies in addition to safe medication practices. Nonpharmacologic treatments include mainly avoidance of triggers (Hickey & Strayer, 2020) (see [Chart 61-8](#)). Noninvasive neuromodulation devices may also provide some relief with minimal side effects (Tepper, 2019).

The triptans, which are serotonin receptor agonists, are the most specific antimigraine agents available. These agents cause vasoconstriction, reduce inflammation, and may reduce pain transmission. The triptans in routine clinical use include sumatriptan, naratriptan, rizatriptan, zolmitriptan, almotriptan, eletriptan, and frovatriptan (Comerford & Durkin, 2020). Many of the triptan medications are available in a variety of formulations, such as nasal sprays, inhalers, conventional tablet, disintegrating tablet, suppositories, or injections. The nasal sprays may be useful for patients experiencing nausea and vomiting (Tepper, 2019).

Chart 61-8



### PATIENT EDUCATION

#### Migraine Headaches

The nurse instructs the patient to:

- Be aware of the definition of migraine headaches along with the characteristics and manifestations.
- Recognize triggers of migraine headaches and how to avoid such triggers as:
  - Foods that contain tyramine, such as chocolate, cheese, coffee, dairy products
  - Dietary habits that result in long periods between meals
  - Menstruation and ovulation (caused by hormone fluctuation)
  - Alcohol (causes vasodilation of blood vessels)
  - Fatigue and fluctuations in sleep patterns
- Develop and use a paper or electronic headache diary.
- Implement stress management and lifestyle changes to minimize the frequency of headaches.
- Ensure correct pharmacologic management: acute therapy and prophylaxis to include medication regimen and side effects.
- Use comfort measures during headache attacks, such as resting in a quiet and dark environment, applying cold compresses to the painful area, and elevating the head.
- Seek out resources for education and support, such as the National Headache Foundation.

The triptans are considered first-line treatment of the management of moderate to severe migraine pain. Best results are achieved with early use of triptans; oral dosing takes effect within 20 to 60 minutes of taking the drug and if needed may be repeated in 2 to 4 hours. Triptans are contraindicated in patients with ischemic heart disease. Careful administration and dosing instructions to patients are important to prevent

adverse reactions such as increased blood pressure, drowsiness, muscle pain, sweating, and anxiety. Interactions are possible if the medication is taken in conjunction with St. John's wort (Comerford & Durkin, 2020).

Ergotamine preparations (taken orally, sublingually, subcutaneously, intramuscularly, by rectum, or by inhalation) may be effective in aborting the headache if taken early in the migraine process. They are low in cost. Ergotamine tartrate acts on smooth muscle, causing prolonged constriction of the cranial blood vessels. Each patient's dosage is based on individual needs. Side effects include aching muscles, paresthesias (numbness and tingling), nausea, and vomiting. Pretreatment with antiemetic agents may be required. None of the triptan medications should be taken concurrently with medications containing ergotamine because of the potential for a prolonged vasoactive reaction (Comerford & Durkin, 2020).

Other nonspecific medications are also used in the treatment of migraine and include NSAIDs, antispasmodic agents, and neuroleptics. Neuroleptic agents can be used alone or in conjunction with triptans and/or NSAIDs (Tepper, 2019).

Prophylactic treatment of migraine includes the use of beta-blockers, antiepileptics, antidepressants, angiotensin-converting enzyme (ACE) inhibitors, and angiotensin receptor blockers. Calcitonin gene-related peptides (CGRPs) have been found in increased levels in patients with migraine, and three CGRP monoclonal antibodies have been approved by the U.S. Food and Drug Administration (FDA) for migraine prevention: erenumab, fremanezumab, and galcanezumab (Hickey & Strayer, 2020).

The medical management of an acute attack of cluster headaches may include 100% oxygen by facemask for 15 minutes, subcutaneous sumatriptan, or intranasal zolmitriptan (Hickey & Strayer, 2020).

The medical management of cranial arteritis consists of early administration of a corticosteroid to prevent the possibility of loss of vision due to vascular occlusion or rupture of the involved artery (Starling, 2018). The patient is instructed not to stop the medication abruptly, because this can lead to relapse. Analgesic agents are prescribed for comfort.

## Nursing Management

When migraine or the other types of headaches have been diagnosed, the goal of nursing management is pain relief. It is reasonable to try nonpharmacologic interventions first, but the use of medications should not be delayed. The first priority is to treat the acute event of the headache and the second is to prevent recurrent episodes. Prevention involves patient education regarding precipitating factors, possible lifestyle or habit changes that may be helpful, and pharmacologic measures.

### Relieving Pain

Individualized treatment depends on the type of headache and differs for migraine, cluster headaches, cranial arteritis, and tension headache. Nursing care is directed toward treatment of the acute episode. A migraine or a cluster headache in the early phase requires abortive medication therapy instituted as soon as possible. Some headaches can be prevented if the appropriate medications are taken before the onset of pain. Nursing care during an attack includes comfort measures such as a quiet, dark environment; elevation of the head of the bed to 30 degrees; and symptomatic treatment (i.e., administration of antiemetic medication) (Hickey & Strayer, 2020).

Symptomatic pain relief for tension headache may be obtained by application of local heat or massage. Additional strategies may include administration of analgesic agents, antidepressant medications, and muscle relaxants.

### Promoting Home, Community-Based, and Transitional Care



#### Educating Patients About Self-Care

Headaches, especially migraines, are more likely to occur when the patient is ill, overly tired, or stressed. Nonpharmacologic therapies are important and include patient education about the type of headache, its mechanism (if known), and appropriate changes in lifestyle to avoid triggers. Regular sleep, meals, exercise, relaxation, and avoidance of dietary triggers may be helpful in avoiding headaches (Starling, 2018).

The patient with tension headaches needs education and reassurance that the headache is not the result of a brain tumor or other intracranial disorder. Stress reduction techniques, such as biofeedback, exercise programs, and meditation, are examples of nonpharmacologic therapies that may prove helpful. The patient

and family need to be educated about the importance of following the prescribed treatment regimen for headache and keeping follow-up appointments. In addition, the patient is reminded of the importance of participating in health promotion activities and recommended health screenings to promote a healthy lifestyle. **Chart 61-8** presents educational topics for the patient with migraine headaches.

### Continuing and Transitional Care

The National Headache Foundation (see the Resources section) provides a list of clinics in the United States and the names of primary providers who specialize in headache and who are members of the American Headache Society.

### CRITICAL THINKING EXERCISES

- 1 pq** A patient is admitted to your unit for a supratentorial cranial procedure. Identify the nursing priorities before, during, and after the procedure. What are the priorities for patient and caregiver education in preparation for discharge?
- 2 ebp** As a member of your unit's practice council, you are working on identifying interventions to assess and manage delirium. Using your knowledge of evidence-based practice guidelines, list the most important assessments and interventions for nurses to implement. Compare and contrast the options supported by the guidelines.
- 3 ipc** You are a nurse working in an outpatient neurology clinic. A 28-year-old woman is newly diagnosed with epilepsy. What nursing and interprofessional assessments are indicated? What interventions, including patient education, will you implement? What interprofessional referrals would be appropriate?

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\*Asterisk indicates nursing research.  
\*\*Double asterisk indicates classic reference.

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## Resources

American Headache Society, [www.americanheadachesociety.org](http://www.americanheadachesociety.org)

Brain Injury Association, [www.biausa.org](http://www.biausa.org)

Brain Trauma Foundation (BTF), [www.braintrauma.org](http://www.braintrauma.org)

Epilepsy Foundation, [www.epilepsy.com](http://www.epilepsy.com)  
Hydrocephalus Association, [www.hydroassoc.org](http://www.hydroassoc.org)  
National Headache Foundation, [www.headaches.org](http://www.headaches.org)

# 62 Management of Patients with Cerebrovascular Disorders

## LEARNING OUTCOMES

*On completion of this chapter, the learner will be able to:*

1. Describe the incidence of, risk factors and preventive measures for, and impact of cerebrovascular disorders.
2. Compare the various types of cerebrovascular disorders: their causes, clinical manifestations, and medical management.
3. Explain the principles of nursing management as they relate to the care of a patient in the acute stage of an ischemic stroke.
4. Use the nursing process as a framework for care of the patient recovering from an ischemic stroke or from a hemorrhagic stroke.
5. Discuss essential elements for family education and preparation for home care of the patient who has had a stroke.

## NURSING CONCEPTS

Family  
Functional Ability  
Intracranial Regulation  
Patient Education  
Perfusion  
Sensory Perception

## GLOSSARY

**agnosia:** loss of ability to recognize objects through a particular sensory system; may be visual, auditory, or tactile

**aneurysm:** a weakening or bulge in an arterial wall

**aphasia:** inability to express oneself or to understand language

**apraxia:** inability to perform previously learned purposeful motor acts on a voluntary basis

**dysarthria:** defects of articulation due to neurologic causes

**dysphagia:** difficulty swallowing

**expressive aphasia:** inability to express oneself; often associated with damage to the left frontal lobe area

**hemianopsia:** blindness of half of the field of vision in one or both eyes

**hemiparesis:** weakness of one side of the body, or part of it, due to an injury in the motor area of the brain

**hemiplegia:** paralysis of one side of the body, or part of it, due to an injury in the motor area of the brain

**infarction:** tissue necrosis in an area deprived of blood supply

**penumbra region:** area of low cerebral blood flow

**receptive aphasia:** inability to understand what someone else is saying; often associated with damage to the temporal lobe area

*Cerebrovascular disorder* is an umbrella term that refers to a functional abnormality of the central nervous system (CNS) that occurs when the blood supply to the brain is disrupted. Stroke is the primary cerebrovascular disorder in the United States, and while it dropped from

the fourth to the fifth leading cause of death, it is still a leading cause of serious, long-term disability. Approximately 795,000 people experience a stroke each year in the United States. Approximately 610,000 of these are new strokes, and 185,000 are recurrent strokes (Virani, Alonso, Benjamin, et al., 2020). About 7 million Americans over the age of 20 who have survived a stroke are alive today. The financial impact of stroke is profound, with estimated direct and indirect costs of \$45.5 billion from 2014 to 2015 (Virani et al., 2020).

Strokes can be divided into two major categories: ischemic (approximately 87%), in which vascular occlusion and significant hypoperfusion occur, and hemorrhagic (approximately 13%), in which there is extravasation of blood into the brain or subarachnoid space (Hickey & Strayer, 2020; Virani et al., 2020). Although there are some similarities between the two types of stroke, differences exist in etiology, pathophysiology, medical management, surgical management, and nursing care. [Table 62-1](#) compares ischemic and hemorrhagic strokes.

**TABLE 62-1** Comparison of Major Types of Stroke

Types of Stroke	Causes	Main Presenting Symptoms	Functional Recovery
<b>Ischemic</b>	<ul style="list-style-type: none"><li>• Large artery thrombosis</li><li>• Small penetrating artery thrombosis</li><li>• Cardiogenic embolic</li><li>• Cryptogenic (no known cause)</li><li>• Other</li></ul>	<ul style="list-style-type: none"><li>• Numbness or weakness of the face, arm, or leg, especially on one side of the body, aphasia, vision loss (homonymous hemianopsia)</li></ul>	Majority of recovery made in the first 3–6 mo, slower steps toward recovery may be made up to 1 yr and beyond with therapy.
<b>Hemorrhagic</b>	<ul style="list-style-type: none"><li>• Intracerebral hemorrhage</li><li>• Subarachnoid hemorrhage</li><li>• Cerebral aneurysm</li><li>• Arteriovenous malformation</li></ul>	<ul style="list-style-type: none"><li>• “Worst headache of my life”</li><li>• Decreased level of consciousness</li><li>• Seizure</li></ul>	Slower recovery, typically left with more disability.

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

## Ischemic Stroke

An ischemic stroke, formerly referred to as a cerebrovascular accident or “brain attack,” is a sudden loss of function resulting from disruption of the blood supply to a part of the brain. The term *brain attack* has been used to suggest to health care practitioners and the public that a stroke is an urgent health care issue similar to a heart attack. The only U.S. Food and Drug Administration (FDA)-approved thrombolytic therapy has a treatment window of 3 hours after the onset of a stroke, and scientific statements have endorsed its expanded use for up to 4.5 hours (Del Zoppo, Saver, Jauch, et al., 2009; Powers, Rabinstein, Ackerson, et al., 2019). Although the time frame for treatment has expanded, urgency is

needed on the part of the public and health care practitioners for rapid transport of the patient to a hospital for assessment and administration of the medication.

Ischemic strokes are subdivided into five different types based on the cause: large artery thrombotic strokes (20%), small penetrating artery thrombotic strokes (25%), cardiogenic embolic strokes (20%), cryptogenic strokes (30%), and others (5%) (see [Table 62-1](#)). Large artery thrombotic strokes are caused by atherosclerotic plaques in the large blood vessels of the brain. Thrombus formation and occlusion at the site of the atherosclerosis result in ischemia and **infarction** (tissue necrosis in an area deprived of blood supply) (Hickey & Strayer, 2020).

Small penetrating artery thrombotic strokes affect one or more vessels and are a common type of ischemic stroke. Small artery thrombotic strokes are also called *lacunar strokes* because of the cavity that is created after the death of infarcted brain tissue (Hickey & Strayer, 2020).

Cardiogenic embolic strokes are associated with cardiac arrhythmias, usually atrial fibrillation. Embolic strokes can also be associated with valvular heart disease and thrombi in the left ventricle. Emboli originate from the heart and circulate to the cerebral vasculature, most commonly the left middle cerebral artery, resulting in a stroke. Embolic strokes may be prevented by the use of anticoagulation therapy in patients with atrial fibrillation.

The last two classifications of ischemic strokes are cryptogenic strokes, which have no known cause, and strokes from other causes, such as illicit drug use (cocaine), coagulopathies, migraine/vasospasm, or spontaneous dissection of the carotid or vertebral arteries.



## COVID-19 Considerations

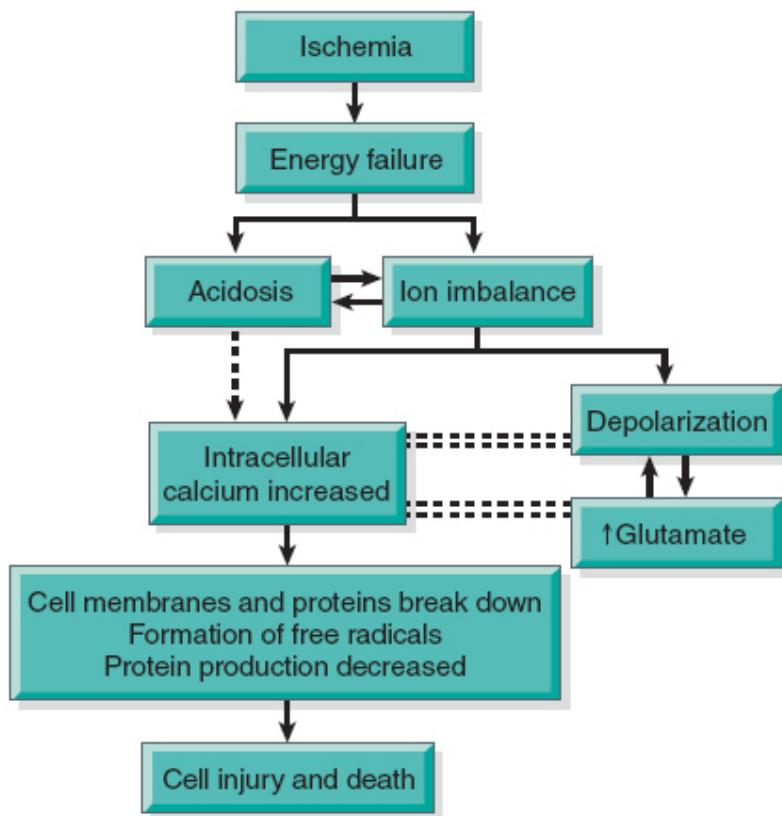
Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a community-acquired coronavirus whose primary pathologic evolution occurs within the respiratory system; however, due to abnormal blood clotting, one of the manifestations of coronavirus disease 2019 (COVID-19) can be ischemic stroke (Wadman, Couzin-Frankel, Kaiser, et al., 2020). The increase in blood clots in patients with COVID-19 is associated with laboratory findings of high D-dimer levels (Wadman et al., 2020). Case reports of patients who have had strokes and COVID-19 reveal that many are younger than 50 years of age and that the strokes occur in the large blood vessels of the brain, resulting in severe

neurologic deficits (see later discussion) (Oxley, Mocco, Majidi, et al., 2020).

## Pathophysiology

In an ischemic brain attack, there is disruption of the cerebral blood flow due to obstruction of a blood vessel. This disruption in blood flow initiates a complex series of cellular metabolic events referred to as the ischemic cascade (see Fig. 62-1).

### Physiology/Pathophysiology



**Figure 62-1 •** Some of the processes contributing to ischemic brain cell injury.

The ischemic cascade begins when cerebral blood flow decreases to less than 25 mL per 100 g of blood per minute. At this point, neurons are no longer able to maintain aerobic respiration. The mitochondria must then switch to anaerobic respiration, which generates large amounts of

lactic acid, causing a change in the pH. This switch to the less efficient anaerobic respiration also renders the neuron incapable of producing sufficient quantities of adenosine triphosphate (ATP) to fuel the depolarization processes. The membrane pumps that maintain electrolyte balance begin to fail, and the cells cease to function.

Early in the cascade, an area of low cerebral blood flow, referred to as the **penumbra region**, exists around the area of infarction. The penumbra region is ischemic brain tissue that may be salvaged with timely intervention. The ischemic cascade threatens cells in the penumbra because membrane depolarization of the cell wall leads to an increase in intracellular calcium and the release of glutamate. The influx of calcium and the release of glutamate, if continued, activate a number of damaging pathways that result in the destruction of the cell membrane, the release of more calcium and glutamate, vasoconstriction, and the generation of free radicals. These processes enlarge the area of infarction into the penumbra, extending the stroke. A person experiencing a stroke typically loses 1.9 million neurons each minute that a stroke is not treated, and the ischemic brain ages 3.6 years each hour without treatment (Saver, 2006).

Each step in the ischemic cascade represents an opportunity for intervention to limit the extent of secondary brain damage caused by a stroke. The penumbra area may be revitalized by administration of tissue plasminogen activator (t-PA). Medications that protect the brain from secondary injury are called *neuroprotectants*. A number of clinical trials have focused on neuroprotective medications and strategies to improve stroke recovery and survival; so far, none have shown positive results (Powers et al., 2019).

## Clinical Manifestations

An ischemic stroke can cause a wide variety of neurologic deficits, depending on the location of the lesion (which blood vessels are obstructed), the size of the area of inadequate perfusion, and the amount of collateral (secondary or accessory) blood flow. See [Chapter 60](#) for discussion of anatomy and brain blood supply. The patient may present with any of the following signs or symptoms:

- Numbness or weakness of the face, arm, or leg, especially on one side of the body
- Confusion or change in mental status
- Trouble speaking or understanding speech

- Visual disturbances
- Difficulty walking, dizziness, or loss of balance or coordination
- Sudden severe headache

Motor, sensory, cranial nerve, cognitive, and other functions may be disrupted. [Table 62-2](#) reviews the neurologic deficits frequently seen in patients with stroke. [Table 62-3](#) compares the symptoms and behaviors seen in right hemispheric stroke with those seen in left hemispheric stroke.

## **Motor Loss**

A stroke is an upper motor neuron lesion and results in loss of voluntary control over motor movements. Because the upper motor neurons decussate (cross), a disturbance of voluntary motor control on one side of the body may reflect damage to the upper motor neurons on the opposite side of the brain. The most common motor dysfunction is **hemiplegia** (paralysis of one side of the body, or part of it) caused by a lesion of the opposite side of the brain. **Hemiparesis**, or weakness of one side of the body, or part of it, is another sign. The concept of upper and lower motor neuron lesions is described in more detail in [Chapter 60, Table 60-4](#).

In the early stage of stroke, the initial clinical features may be flaccid paralysis and loss of or decrease in the deep tendon reflexes. When these deep reflexes reappear (usually by 48 hours), increased tone is observed along with spasticity (abnormal increase in muscle tone) of the extremities on the affected side.

## **Communication Loss**

Other brain functions affected by stroke are language and communication. In fact, stroke is the most common cause of **aphasia** (inability to express oneself or to understand language). The following are dysfunctions of language and communication:

- **Dysarthria** (difficulty in speaking) or dysphasia (impaired speech), caused by paralysis of the muscles responsible for producing speech
- Aphasia, which can be **expressive aphasia** (inability to express oneself), **receptive aphasia** (inability to understand language), or global (mixed) aphasia (see [Chapter 60, Table 60-5](#))
- **Apraxia** (inability to perform a previously learned action), as may be seen when a patient makes verbal substitutions for

desired syllables or words

## Perceptual Disturbances

Perception is the ability to interpret sensation. Stroke can result in visual-perceptual dysfunctions, disturbances in visual-spatial relations, and sensory loss.

Visual-perceptual dysfunctions are caused by disturbances of the primary sensory pathways between the eye and visual cortex. Homonymous **hemianopsia** (blindness in half of the visual field in one or both eyes) may occur from stroke and may be temporary or permanent. The affected side of vision corresponds to the paralyzed side of the body.

Disturbances in visual-spatial relations (perceiving the relationship of two or more objects in spatial areas) are frequently seen in patients with right hemispheric damage.

## Sensory Loss

Sensory losses from stroke may be mild, such as a slight impairment of touch, or more severe, with loss of proprioception (ability to perceive the position and motion of body parts) as well as difficulty in interpreting visual, tactile, and auditory stimuli. An **agnosia** is the loss of the ability to recognize objects through a particular sensory system; it may be visual, auditory, or tactile (see [Chapter 60, Table 60-6](#)).

## Cognitive Impairment and Psychological Effects

If damage has occurred to the frontal lobe, learning capacity, memory, or other higher cortical intellectual functions may be impaired. Such dysfunction may be reflected in a limited attention span, difficulties in comprehension, forgetfulness, and a lack of motivation. These changes can cause the patient to become easily frustrated during rehabilitation. Depression is common and may be exaggerated by the patient's natural response to this catastrophic event. Emotional lability, hostility, frustration, resentment, lack of cooperation, and other psychological problems may occur.

**TABLE 62-2** Neurologic Deficits of Stroke: Manifestations and Nursing Implications

Neurologic Deficit	Manifestation	Nursing Implications/Patient Education Applications
<b>Visual Field Deficits</b>		
Homonymous hemianopsia (loss of half of the visual field)	<ul style="list-style-type: none"> <li>• Unaware of persons or objects on side of visual loss</li> <li>• Neglect of one side of the body</li> <li>• Difficulty judging distances</li> </ul>	<p>Place objects within intact field of vision.</p> <p>Approach the patient from side of intact field of vision.</p> <p>Instruct/remind the patient to turn head in the direction of visual loss to compensate for loss of visual field.</p> <p>Encourage the use of eyeglasses if available.</p> <p>When educating the patient, do so within patient's intact visual field.</p>
Loss of peripheral vision	<ul style="list-style-type: none"> <li>• Difficulty seeing at night</li> <li>• Unaware of objects or the borders of objects</li> </ul>	<p>Place objects in center of patient's intact visual field.</p> <p>Encourage the use of a cane or other object to identify objects in the periphery of the visual field.</p>
Diplopia	<ul style="list-style-type: none"> <li>• Double vision</li> </ul>	<p>Ensure that the patient's driving ability is evaluated.</p> <p>Explain to the patient the location of an object when placing it near the patient.</p> <p>Consistently place patient care items in the same location.</p>
<b>Motor Deficits</b>		
Hemiparesis	<ul style="list-style-type: none"> <li>• Weakness of the face, arm, and leg on the same side (due to a lesion in the opposite hemisphere)</li> </ul>	<p>Place objects within the patient's reach on the nonaffected side.</p> <p>Instruct the patient to exercise and increase the strength on the unaffected side.</p>
Hemiplegia	<ul style="list-style-type: none"> <li>• Paralysis of the face, arm, and leg on the same side (due to a lesion in the opposite hemisphere)</li> </ul>	<p>Encourage the patient to provide range-of-motion exercises to the affected side.</p> <p>Reposition the patient every 2 h.</p> <p>Maintain body alignment in functional position.</p> <p>Exercise unaffected limb to increase mobility, strength, and use.</p>
Ataxia		
Support patient during the initial		

	<ul style="list-style-type: none"> <li>• Staggering, unsteady gait</li> <li>• Unable to keep feet together; needs a broad base to stand</li> </ul>	<p>ambulation phase.</p> <p>Provide supportive device for ambulation (walker, cane).</p> <p>Instruct the patient not to walk without assistance or supportive device.</p>
Dysarthria	<ul style="list-style-type: none"> <li>• Difficulty in forming words</li> </ul>	<p>Provide the patient with alternative methods of communicating.</p> <p>Allow the patient sufficient time to respond to verbal communication.</p> <p>Support patient and family to alleviate frustration related to difficulty in communicating.</p>
Dysphagia	<ul style="list-style-type: none"> <li>• Difficulty in swallowing</li> </ul>	<p>Test the patient's pharyngeal reflexes before offering food or fluids.</p> <p>Assist the patient with meals.</p> <p>Place food on the unaffected side of the mouth.</p> <p>Allow ample time to eat.</p>
<b>Sensory Deficits</b>		
Paresthesia (occurs on the side opposite the lesion)	<ul style="list-style-type: none"> <li>• Sensation of numbness, tingling, or a "pins and needles" sensation</li> <li>• Difficulty with proprioception</li> </ul>	<p>Instruct patient that sensation may be altered.</p> <p>Provide range of motion to affected areas and apply corrective devices as needed.</p> <p>If numbness is present, protect the affected areas from injury and burns.</p>
<b>Verbal Deficits</b>		
Expressive aphasia	<ul style="list-style-type: none"> <li>• Unable to form words that are understandable; may be able to speak in single-word responses</li> </ul>	<p>Encourage patient to repeat sounds of the alphabet.</p> <p>Explore the patient's ability to write as an alternative means of communication.</p>
Receptive aphasia	<ul style="list-style-type: none"> <li>• Unable to comprehend the spoken word; can speak but may not make sense</li> </ul>	<p>Speak clearly and in an unhurried manner to assist the patient in forming the sounds.</p> <p>Explore the patient's ability to read as an alternative means of communication.</p>
Global (mixed) aphasia	<ul style="list-style-type: none"> <li>• Combination of both receptive and</li> </ul>	<p>Speak clearly and in simple sentences; use gestures or pictures</p>

	expressive aphasia	when able. Establish alternative means of communication.
<b>Cognitive Deficits</b>	<ul style="list-style-type: none"> <li>• Short- and long-term memory loss</li> <li>• Decreased attention span</li> <li>• Impaired ability to concentrate</li> <li>• Poor abstract reasoning</li> <li>• Altered judgment</li> </ul>	<p>Reorient patient to time, place, and situation frequently.</p> <p>Use verbal and auditory cues to orient patient.</p> <p>Provide familiar objects (family photographs, favorite objects).</p> <p>Use noncomplicated language.</p> <p>Match visual tasks with a verbal cue; holding a toothbrush, simulate brushing of teeth while saying, “I would like you to brush your teeth now.”</p> <p>Minimize distracting noises and views when providing education to the patient.</p> <p>Repeat and reinforce instructions frequently.</p>
<b>Emotional Deficits</b>	<ul style="list-style-type: none"> <li>• Loss of self-control</li> <li>• Emotional lability</li> <li>• Decreased tolerance to stressful situations</li> <li>• Depression</li> <li>• Withdrawal</li> <li>• Fear, hostility, and anger</li> <li>• Feelings of isolation</li> </ul>	<p>Support patient during uncontrollable outbursts.</p> <p>Discuss with the patient and family that the outbursts are due to the disease process.</p> <p>Encourage patient to participate in group activity.</p> <p>Provide stimulation for the patient.</p> <p>Control stressful situations, if possible.</p> <p>Provide a safe environment.</p> <p>Encourage patient to express feelings and frustrations related to disease process.</p>

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

**TABLE 62-3** Comparison of Left and Right Hemispheric Strokes

Left Hemispheric Stroke	Right Hemispheric Stroke
Paralysis or weakness on right side of body	Paralysis or weakness on left side of body
Right visual field deficit	Left visual field deficit
Aphasia (expressive, receptive, or global)	Spatial-perceptual deficits
Altered intellectual ability	Increased distractibility
Slow, cautious behavior	Impulsive behavior and poor judgment Lack of awareness of deficits

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

## Assessment and Diagnostic Findings

Any patient with neurologic deficits needs a careful history eliciting the last time the patient was seen well and a rapid focused physical and neurologic examination. Initial assessment focuses on airway patency, which may be compromised by loss of gag or cough reflexes and altered respiratory pattern; cardiovascular status (including blood pressure, cardiac rhythm and rate, carotid bruit); and gross neurologic deficits.

Patients may present to the acute care facility with temporary neurologic symptoms. A transient ischemic attack (TIA) is a neurologic deficit that completely resolves in 24 hours (most last less than 1 hour). A TIA is manifested by a sudden loss of motor, sensory, or visual function. The symptoms result from temporary ischemia (impairment of blood flow) to a specific region of the brain; however, when brain imaging is performed, there is no evidence of ischemia. A TIA may serve as a warning of impending stroke. Approximately 3% to 15% of all strokes are preceded by a TIA and occur within the first 90 days after the TIA (Hickey & Strayer, 2020; Johnston, Easton, Farrant, et al., 2018). Lack of evaluation and treatment of a patient who has experienced previous TIAs may result in a stroke and irreversible deficits.

The initial diagnostic test for a stroke is a noncontrast computed tomography (CT) scan. This should be initiated within 20 minutes from the time the patient presents to the emergency department (ED) to determine if the event is ischemic or hemorrhagic, as the type of stroke determines treatment (Powers et al., 2019). Some cities now have mobile stroke units (an ambulance with a CT scanner) that can rapidly make this important distinction and can begin acute medical management. Further

diagnostic workup for ischemic stroke involves attempting to identify the source of the thrombi or emboli and to determine if the patient would benefit from mechanical intervention (clot removal). Studies may include CT angiography or CT perfusion; magnetic resonance imaging (MRI) and magnetic resonance angiography of the brain and neck vessels; transcranial Doppler flow studies; and transthoracic or transesophageal echocardiography (Hickey & Strayer, 2020; Powers et al., 2019). A 12-lead electrocardiogram (ECG) and a carotid ultrasound are other standard tests.

## Prevention

Primary prevention of ischemic stroke remains the best approach. A healthy lifestyle including not smoking, engaging in physical activity (at least 40 minutes a day, 3 to 4 days a week), maintaining a healthy weight, and following a healthy diet (including modest alcohol consumption), can reduce the risk of having a stroke (Virani et al., 2020). Specific diets that have decreased risk of stroke include the Dietary Approaches to Stop Hypertension (DASH) diet (high in fruits and vegetables, moderate in low-fat dairy products, and low in animal protein), the Mediterranean diet (supplemented with nuts), and overall diets that are rich in fruits and vegetables. Research findings suggest that low-dose aspirin may lower the risk of a first stroke for those who are at risk (Meschia, Bushnell, Boden-Albala, et al., 2014).

Stroke risk screenings are an ideal opportunity to lower stroke risk by identifying people or groups of people who are at high risk for stroke and by educating patients and the community about recognition and prevention of stroke. Stroke screenings are usually coordinated and run by nurses. Age, gender, and race are well-known nonmodifiable risk factors for stroke. High-risk groups include people older than 55 years, and the incidence of stroke more than doubles in each successive decade. Men have a higher age-adjusted rate of stroke than that of women in younger and middle age, but that difference narrows in the oldest age groups, in which the rate in women is almost equal to or sometimes even higher than in men. Each year, 55,000 more women than men have a stroke. Compared to Caucasian Americans, African Americans and some Hispanic/Latino Americans have a higher incidence of stroke and higher mortality (Virani et al., 2020).

There are many risk factors for ischemic stroke (see Chart 62-1). For people who are at high risk, interventions that alter modifiable factors,

such as treating hypertension and stopping smoking, reduce stroke risk (Meschia et al., 2014).

Chart 62-1



## MODIFIABLE RISK FACTORS

### Ischemic Stroke

- Asymptomatic carotid stenosis
- Atrial fibrillation
- Diabetes (associated with accelerated atherogenesis)
- Dyslipidemia
- Excessive alcohol consumption
- Hypercoagulable states
- Hypertension (controlling hypertension, the major risk factor, is the key to preventing stroke)
- Migraine
- Obesity
- Sedentary lifestyle
- Sleep apnea
- Smoking

Adapted from Meschia, J. F., Bushnell, C., Boden-Albala, B., et al. (2014). Guidelines for the primary prevention of stroke: A statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*, 45(12), 3754–3832.

Additional treatable conditions that increase risk of stroke include sickle cell diseases, cardiomyopathy (ischemic and nonischemic), and valvular heart disease (e.g., endocarditis, prosthetic heart valves). Lesser known and potentially modifiable risk factors for stroke are migraine (especially migraine with aura), sleep apnea, and inherited and acquired hypercoagulable states. Chronic inflammatory conditions that have been associated with an increased risk of stroke are systemic lupus erythematosus and rheumatoid arthritis (Norris, 2019).

Several methods of preventing recurrent stroke have been identified for patients with TIAs or ischemic stroke. Patients with moderate to severe carotid stenosis are treated with carotid endarterectomy (CEA) or

carotid angioplasty and stenting. In patients with atrial fibrillation, which increases the risk of emboli, administration of an anticoagulant that inhibits clot formation may prevent both thrombotic and embolic strokes (Kernan, Ovbiagele, Black, et al., 2014).

## Medical Management

Patients who have experienced a TIA or stroke should have medical management for secondary prevention. Those with atrial fibrillation (or cardioembolic strokes) are treated with dose-adjusted warfarin with a target international normalized ratio (INR) of 2 to 3. Other anticoagulants that may be prescribed as alternative drugs include dabigatran, apixaban, edoxaban, or rivaroxaban, unless they are contraindicated. These drugs are also known as direct oral anticoagulants (DOACs). If anticoagulants are contraindicated, aspirin alone is the best option, although the addition of clopidogrel to aspirin is also a reasonable therapy (Kernan et al., 2014).

Platelet-inhibiting medications, including aspirin, extended-release dipyridamole plus aspirin, and clopidogrel decrease the incidence of cerebral infarction in patients who have experienced TIAs and stroke from suspected embolic or thrombotic causes. The specific medication that is used is based on the patient's health history. If the patient has had a minor ischemic stroke or what is considered a TIA with a high risk of having stroke, and they did not receive thrombolytic therapy, they may receive two platelet-inhibiting medications (dual antiplatelet therapy). Typically this is clopidogrel and aspirin, and can be taken for a period of 21 to 90 days after the stroke or TIA (Powers et al., 2019).

Research suggests that medications known as statins reduce coronary events and ischemic strokes. The most current stroke prevention guideline now includes the recommendation of a statin even if the low-density lipoprotein (LDL) cholesterol is below 100 mg/dL and there is no evidence of atherosclerotic cardiovascular disease (coronary artery disease/myocardial infarction, hypertensive heart disease and peripheral arterial disease) (Kernan et al., 2014). The FDA has included indications for statin medications, such as atorvastatin and simvastatin, to include secondary stroke prevention.

After the acute stroke period, antihypertensive medications are also used, if indicated, for secondary stroke prevention. Preferred drugs include angiotensin-converting enzyme (ACE) inhibitors and diuretics, or a combination of both (Kernan et al., 2014).

Medical management of acute ischemic stroke needs to include consideration for endovascular treatment (Powers et al., 2019). The FDA has approved several devices that open the blocked artery and restore blood flow to the brain. These devices are used by specialists in the endovascular suite.

## Thrombolytic Therapy

Thrombolytic agents are used to treat ischemic stroke by dissolving the blood clot that is blocking blood flow to the brain. Recombinant t-PA is a genetically engineered form of t-PA (a thrombolytic substance made naturally by the body) (Comerford & Durkin, 2020). It works by binding to fibrin and converting plasminogen to plasmin, which stimulates fibrinolysis of the clot. Rapid diagnosis of stroke and initiation of thrombolytic therapy (within 3 hours) in patients with ischemic stroke leads to a decrease in the size of the stroke and an overall improvement in functional outcome after 3 months (National Institute of Neurological Disorders and Stroke [NINDS], 1995). The goal is for intravenous (IV) t-PA to be given within 45 minutes of the patient arriving to the ED (Powers et al., 2019).

Mechanical intervention (e.g., mechanical thrombectomy, endovascular thrombectomy, intra-arterial mechanical thrombectomy) can be used with t-PA or as an alternative to IV administration (Amatangelo & Thomas, 2019). Intra-arterial mechanical thrombectomy allows for higher concentrations of the drug to be given directly to the clot, and the time window for treatment may be extended up to 24 hours in patients that meet specific criteria (Powers et al., 2019). Those not eligible for IV delivery may be eligible for intra-arterial delivery, and these methods may also be combined. Treatment using intra-arterial delivery must occur in specialized centers with access to emergent cerebral angiogram and interventional operating rooms/suites (Powers et al., 2019). Ongoing clinical trials continue to investigate the efficacy of other thrombolytic agents.

To realize the full potential of early intervention, community education directed at recognizing the symptoms of stroke and obtaining appropriate emergency care is necessary to ensure rapid transport to a hospital and initiation of therapy within the recommended 3-hour period (which may be extended up to 4.5 hours) (Del Zoppo et al., 2009; Powers et al., 2019). Delays make the patient ineligible for therapies, because revascularization of necrotic tissue (which develops after 3 hours) increases the risk of cerebral edema and hemorrhage.

## Endovascular Therapy

It is now recommended that patients with acute ischemic stroke receive endovascular therapy and medical management with a stent retriever if they meet specific criteria (Powers et al., 2019). All of the following criteria need to be met:

- Prestroke status of no deficits
- Acute ischemic stroke receiving IV t-PA within 4.5 hours of onset according to guidelines from professional medical societies
- Causative occlusion of the internal carotid artery or middle cerebral artery segment
- Age  $\geq 18$  years
- National Institutes of Health Stroke Scale (NIHSS) score of  $\geq 6$  (see later discussion)
- An Alberta Stroke Program Evaluation of Computed Tomography (ASPECT) score of  $\geq 6$  (a radiologic assessment of the CT scan), and treatment can be initiated (groin puncture) within 6 hours of symptom onset

Patients eligible for t-PA should receive IV t-PA even if endovascular treatments are being considered (Powers et al., 2019). Thrombolytic therapy should not be delayed.

## Enhancing Prompt Diagnosis

After being notified by emergency medical service personnel, the ED contacts the appropriate staff (neurologist, neuroradiologist, radiology department, nursing staff, ECG, and laboratory technicians) and informs them of the patient's imminent arrival at the hospital. Many institutions have acute stroke teams that respond rapidly, ensuring that treatment occurs within the allotted period. This may be called a Code Stroke.

Initial management requires the definitive diagnosis of an ischemic stroke by brain imaging and a careful history to determine whether the patient meets the criteria for t-PA therapy (see [Chart 62-2](#)). The goal is that diagnostic results from imaging are completed within 25 minutes of the patient's arrival to the ED. Some of the contraindications for thrombolytic therapy include symptom onset greater than 3 hours before admission (expanded to 4.5 hours), a patient who is anticoagulated (with an INR above 1.7), or a patient who has recently had any type of

significant intracranial pathology (e.g., previous stroke, head injury, trauma) in the last 3 months.

Before receiving t-PA, the patient is assessed using the NIHSS, a standardized assessment tool that helps evaluate stroke severity (see [Table 62-4](#)). Total NIHSS scores range from 0 (normal) to 42 (severe stroke). Certification in the administration of the scale is recommended and is available for nurses and other health care professionals.

## Chart 62-2

## **Eligibility Criteria for Tissue Plasminogen Activator Administration**

- Age  $\geq$ 18 years
- Clinical diagnosis of ischemic stroke
- Systolic blood pressure  $\leq$ 185 mm Hg; diastolic  $\leq$ 110 mm Hg
- No minor (nondisabling) stroke
- Prothrombin time  $\leq$ 15 seconds or international normalized ratio  $\leq$ 1.7 (if taking an anticoagulant, the same guidance is used)
- Not received low-molecular weight heparin during the past 24 hours
- Platelet count  $\geq$ 100,000/mm<sup>3</sup>
- No symptoms consistent with infective endocarditis
- No prior intracranial hemorrhage
- No subarachnoid hemorrhage
- No stroke, serious head trauma, or intracranial surgery within 3 months
- No gastrointestinal bleeding within 21 days, or gastrointestinal malignancy

Some of these are relative contraindications (the provider administering the medication needs to weigh the risks and benefits of the therapy). There are more stringent t-PA administration guidelines for patients with stroke symptoms that began 3 to 4.5 hours ago and also for those whose symptoms began longer than 4.5 hours ago or for whom time of onset is unclear, coupled with specific MRI findings. Specific guidance also exists for patients taking thrombin inhibitors or factor Xa inhibitors.

Adapted from Powers, W. J., Rabinstein, A. A., Ackerson, T., et al. (2019). 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*, 50(12), e344–e418.

## **Dosage and Administration**

The patient is weighed to determine the dose of t-PA. Typically, two or more IV sites are established prior to administration of t-PA (one for the t-PA and the other for administration of IV fluids). The dosage for t-PA is 0.9 mg/kg, with a maximum dose of 90 mg. Ten percent of the calculated dose is given as an IV bolus over 1 minute. The remaining

dose (90%) is given IV over 1 hour via an infusion pump (Comerford & Durkin, 2020; Hickey & Strayer, 2020; Powers et al., 2019).

The patient is admitted to the intensive care unit or an acute stroke unit, where continuous cardiac monitoring and frequent neurologic assessments are conducted (Amatangelo & Thomas, 2019). Vital signs are obtained frequently, with particular attention to blood pressure (with the goal of lowering the risk of intracranial hemorrhage) and temperature. An example of a standard protocol would be to obtain vital signs every 15 minutes for the first 2 hours, every 30 minutes for the next 6 hours, then every hour until 24 hours after treatment. Blood pressure should be maintained with the systolic pressure less than 185 mm Hg and the diastolic pressure less than 110 mm Hg (Powers et al., 2019). Fever needs to be treated. Airway management is instituted based on the patient's clinical condition and arterial blood gas values.

### Side Effects

Once it is determined that the patient is a candidate for t-PA therapy, no anticoagulant or platelet inhibiting medications (e.g., aspirin, clopidogrel) are given for the next 24 hours. Bleeding is the most common side effect of t-PA administration, and the patient is closely monitored for any bleeding (IV insertion sites, urinary catheter site, endotracheal tube, nasogastric tube, urine, stool, emesis, other secretions). A 24-hour delay in placement of nasogastric tubes, urinary catheters, and intra-arterial pressure catheters is recommended. Intracranial bleeding is a major complication that occurred in approximately 6.4% of patients in the initial t-PA study (NINDS, 1995). A number of factors are associated with the occurrence of symptomatic intracranial bleeding: age greater than 70 years, baseline NIHSS score greater than 20, serum glucose concentration 300 mg/dL or higher, and edema or mass effect observed on the patient's initial CT scan (NINDS, 1995).

### Therapy for Patients with Ischemic Stroke Not Receiving Tissue Plasminogen Activator

Not all patients are candidates for t-PA therapy. In some centers, other treatments may include anticoagulant administration (IV heparin or low-molecular-weight heparin). Because of the risks associated with urgent anticoagulation, their general use is not recommended for patients with acute ischemic stroke (Powers et al., 2019).

Careful maintenance of cerebral hemodynamics to maintain cerebral perfusion is extremely important after a stroke. Increased intracranial pressure (ICP) from brain edema and associated complications may occur after a large ischemic stroke. Interventions during this period include measures to reduce ICP, such as administering an osmotic diuretic (e.g., mannitol) to those that are declining clinically. Other treatment measures include the following (Powers et al., 2019):

**TABLE 62-4** Summary of National Institutes of Health Stroke Scale  
(NIHSS)

Category	Description	Score
1a. LOC	Alert	0
	Arousalable by minor stimulation	1
	Obtunded, strong stimulation to attend	2
	Unresponsive, or reflexive responses only	3
1b. LOC questions (month, age)	Answers both correctly	0
	Answers one correctly	1
	Both incorrect	2
1c. LOC commands (open, close eyes; make fist, let go)	Obeys both correctly	0
	Obeys one correctly	1
	Both incorrect	2
2. Best gaze (eyes open—patient follows examiner's finger or face)	Normal	0
	Partial gaze palsy	1
	Forced deviation	2
3. Visual (introduce visual stimulus/threat to patient's visual field quadrants)	No visual loss	0
	Partial hemianopsia	1
	Complete hemianopsia	2
	Bilateral hemianopsia	3
4. Facial palsy (show teeth, raise eyebrows, and squeeze eyes shut)	Normal	0
	Minor	1
	Partial	2
	Complete	3
5a. Motor; arm—left (elevate extremity to 90 degrees and score drift/movement)	No drift	0
	Drift but maintains in air	1
	Unable to maintain in air	2
	No effort against gravity	3
	No movement	4
	Amputation, joint fusion (explain)	N/A
5b. Motor; arm—right (elevate extremity to 90 degrees and score drift/movement)	No drift	0
	Drift but maintains in air	1
	Unable to maintain in air	2
	No effort against gravity	3
	No movement	4
	Amputation, joint fusion (explain)	N/A
6a. Motor; leg—left (elevate extremity to 30 degrees and score drift/movement)	No drift	0
	Drift but maintains in air	1
	Unable to maintain in air	2
	No effort against gravity	3
	No movement	4
	Amputation, joint fusion (explain)	N/A
6b. Motor; leg—right (elevate	No drift	0

extremity to 30 degrees and score drift/movement)	Drift but maintains in air	1
	Unable to maintain in air	2
	No effort against gravity	3
	No movement	4
	Amputation, joint fusion (explain)	N/A
7. Limb ataxia (finger-to-nose and heel-to-shin testing)	Absent	0
	Present in one limb	1
	Present in two limbs	2
8. Sensory (pinprick to face, arm, trunk, and leg—compare side to side)	Normal	0
	Mild to moderate loss	1
	Severe to total loss	2
9. Best language (name items, describe a picture, and read sentences)	No aphasia	0
	Mild to moderate aphasia	1
	Severe aphasia	2
	Mute	3
10. Dysarthria (evaluate speech clarity by having patient repeat words)	Normal	0
	Mild to moderate dysarthria	1
	Severe dysarthria, mostly unintelligible or worse	2
	Intubated or other physical barrier	N/A
11. Extinction and inattention (use information from prior testing to score)	No abnormality	0
	Visual, tactile, auditory, or other extinction to bilateral simultaneous stimulation	1
	Profound hemiattention or extinction to more than one modality	2

Total score \_\_\_\_\_

LOC, level of consciousness; N/A, not applicable.

Adapted from the version available at the National Institute of Neurological Disorders and Stroke (NINDS). (n.d.). *NIH stroke scale*. Bethesda, MD: National Institutes of Health. Retrieved on 3/8/2020 at: [www.ninds.nih.gov/sites/default/files/NIH\\_Stroke\\_Scale\\_Booklet.pdf](http://www.ninds.nih.gov/sites/default/files/NIH_Stroke_Scale_Booklet.pdf). It is recommended that the full scale with all instructions be used.

- Providing supplemental oxygen if oxygen saturation is below 95%
- Elevation of the head of the bed to 30 degrees to assist the patient in handling oral secretions and decrease ICP
- Possible hemicraniectomy for increased ICP from brain edema in a very large stroke
- Intubation with an endotracheal tube to establish a patent airway, if necessary

- Continuous hemodynamic monitoring (the goals for blood pressure in the first 24 hours after a stroke remain controversial for a patient who has not received thrombolytic therapy; antihypertensive treatment may be given to lower the blood pressure by 15% if the systolic blood pressure exceeds 220 mm Hg or the diastolic blood pressure exceeds 120 mm Hg)
- Frequent neurologic assessments to determine if the stroke is evolving and if other acute complications are developing (such complications may include seizures, bleeding from anticoagulation, or medication-induced bradycardia, which can result in hypotension and subsequent decreases in cardiac output and cerebral perfusion pressure)
- Monitoring for the development of fever (elevated temperature in the first 24 hours after stroke has been associated with increased in-hospital mortality)
- Monitoring of blood glucose and management with sliding scale insulin to keep levels in the range of 140 to 180 mg/dL

## Managing Potential Complications

Adequate cerebral blood flow is essential for cerebral oxygenation. If cerebral blood flow is inadequate, the amount of oxygen supplied to the brain will decrease, and tissue ischemia will result. Adequate oxygenation begins with pulmonary care, maintenance of a patent airway, and administration of supplemental oxygen as needed. The importance of adequate gas exchange in these patients cannot be overemphasized, because many patients are at risk for aspiration pneumonia.

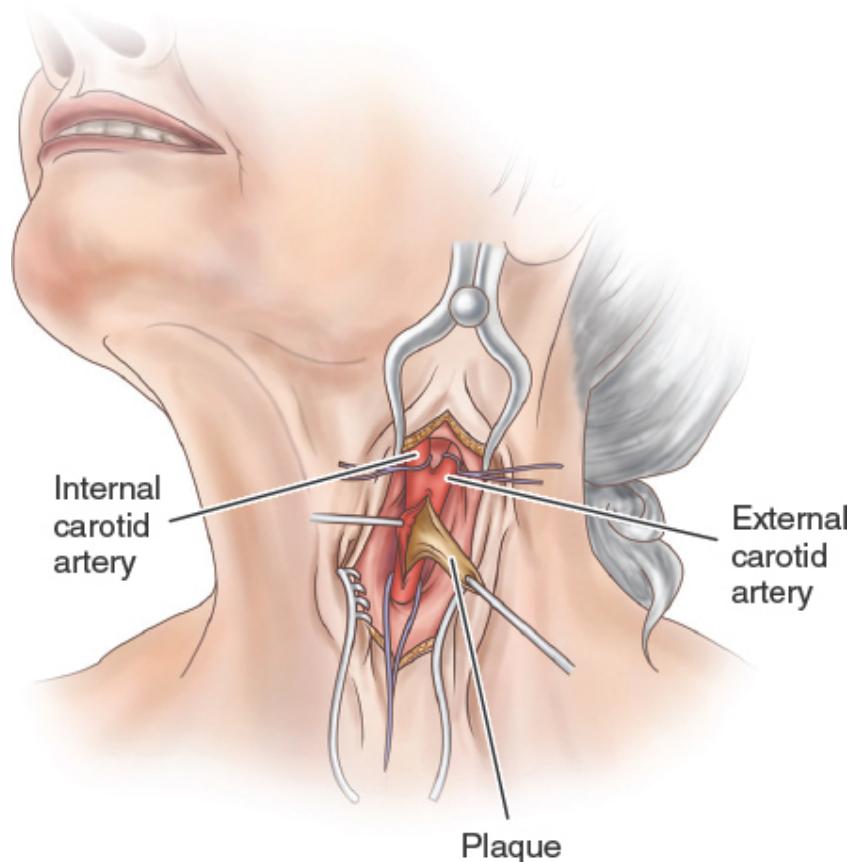
Other potential complications after a stroke include urinary tract infections, cardiac arrhythmias (ventricular ectopy, tachycardia, and heart blocks), and complications of immobility. Hyperglycemia has been associated with poor neurologic outcomes in acute stroke; therefore, blood glucose levels are monitored and hypoglycemia avoided as well (Powers et al., 2019).

## Surgical Prevention of Ischemic Stroke

One surgical procedure for select patients with TIAs and mild stroke is CEA. A CEA is the removal of an atherosclerotic plaque or thrombus from the carotid artery to prevent stroke in patients with occlusive disease of the extracranial cerebral arteries (see Fig. 62-2). This surgery

is indicated for patients with symptoms of TIA or mild stroke (or those without symptoms) who are found to have severe (70% to 99%) carotid artery stenosis or moderate (50% to 69%) stenosis with other significant risk factors.

Carotid artery stenting (CAS), with or without angioplasty, is a less invasive procedure that is used for treatment of carotid stenosis. This procedure has less discomfort for the patient and a shorter recovery time than CEA. Age can be considered when deciding which procedure will be best for the patient. For patients older than 70 years, CEA demonstrated improved outcomes in research studies; for those who were younger, outcomes between CAS and CEA were similar when comparing procedure complications (Kernan et al., 2014).



**Figure 62-2** • Plaque, a potential source of emboli in transient ischemic attack and stroke, is surgically removed from the carotid artery.



## Nursing Management

The main complications of CEA are stroke, cranial nerve injuries, infection or hematoma at the incision, and carotid artery disruption. It is important to maintain adequate blood pressure levels in the immediate postoperative period. Blood pressure instability is common, and lasts 12 to 24 hours after the procedure (Hickey & Strayer, 2020). Hypotension is avoided to prevent cerebral ischemia and thrombosis. Uncontrolled hypertension may precipitate cerebral hemorrhage, edema, hemorrhage at the surgical incision, or disruption of the arterial reconstruction. Medications are used to reduce the blood pressure to previous levels. Close cardiac monitoring is necessary, because these patients frequently have concomitant coronary artery disease.

After CEA, a neurologic observation record (see Chapter 61, [Fig. 61-6](#)) is used to monitor and document assessment parameters for all body systems, with particular attention to neurologic status. The primary provider is notified immediately if a neurologic deficit develops. Formation of a thrombus at the site of the endarterectomy is suspected if there is a sudden new onset of neurologic deficits, such as weakness on one side of the body. The patient should be prepared for repeat endarterectomy.

Difficulty in swallowing, hoarseness, or other signs of cranial nerve dysfunction must be assessed. Cranial nerve injury is the most common complication following CEA. The nurse focuses on assessment of the following cranial nerves: facial (VII), glossopharyngeal (IX), vagus (X), spinal accessory (XI), and hypoglossal (XII). Some edema in the neck after surgery is expected; however, extensive edema and hematoma formation can obstruct the airway. Emergency airway supplies, including those needed for a tracheostomy, must be available (Rich, Treat-Jacobson, DeVeaux, et al., 2017). [Table 62-5](#) provides more information about potential complications of carotid surgery.

Management after carotid stenting also requires monitoring of neurologic status and evaluation for hematoma formation (at the catheterization site). Cardiac monitoring is necessary with assessment for bilateral pulses distal to the catheterization site. Typically, patients are discharged the day after stenting if there are no complications (Rich et al., 2017).

**TABLE 62-5** Select Complications of Carotid Endarterectomy (CEA)  
and Nursing Interventions

Complication	Characteristics	Nursing Interventions
Incision hematoma	Large or rapidly expanding hematomas require emergency treatment. Risks include tracheal deviation and airway compromise. If the airway is obstructed by the hematoma, the incision may be opened at the bedside.	Monitor neck discomfort and wound expansion. Report swelling, subjective feelings of pressure in the neck, difficulty breathing.
Hypertension	Poorly controlled hypertension increases the risk of postoperative complications, including hematoma and hyperperfusion syndrome. There is an increased incidence of neurologic impairment and death due to intracerebral hemorrhage. May be related to surgically induced abnormalities (manipulation) of carotid baroreceptor sensitivity.	Keep in mind that risk is highest in the first 48 h after surgery. Check blood pressure frequently, and report deviations from baseline. Administer medications, as prescribed, to reduce hypertension. Observe for and report new onset of neurologic deficits.
Postoperative hypotension	Treated with fluids (if suspected to be related to hypovolemia) and low-dose phenylephrine infusion (if occurs in normovolemia). Usually resolves in 24–48 h. Patients with hypotension should have serial electrocardiograms to rule out myocardial infarction.	Monitor blood pressure and observe for signs and symptoms of hypotension.
Hyperperfusion syndrome	Occurs when cerebral vessel autoregulation fails. Arteries accustomed to diminished blood flow may be permanently dilated; increased blood flow after endarterectomy coupled with insufficient vasoconstriction leads to capillary bed damage, edema, and hemorrhage. Typically occurs within 2 wks of surgery.	Observe for severe unilateral headache improved by sitting upright or standing. Monitor for any changes in level of consciousness or confusion.
Intracerebral hemorrhage	Occurs infrequently but is often fatal or results in serious neurologic impairment. Can occur secondary to	Monitor neurologic status, and report any changes in mental status or

hyperperfusion syndrome. Increased risk with advanced age, hypertension, presence of high-grade stenosis, poor collateral flow, and slow flow in the region of the middle cerebral artery.

neurologic functioning immediately.

Adapted from Rich, K., Treat-Jacobson, D., DeVeaux, T., et al; Society for Vascular Nursing Practice and Research Committee. (2017). Society for Vascular Nursing-carotid endarterectomy (CEA) updated nursing clinical practice guideline. *Journal of Vascular Nursing*, 35(2), 90–111.

## NURSING PROCESS

### The Patient Recovering from an Ischemic Stroke



The acute phase of an ischemic stroke may last 1 to 3 days, but ongoing monitoring of all body systems is essential as long as the patient requires care. The patient who has had a stroke is at risk for multiple complications, including deconditioning and other musculoskeletal problems, swallowing difficulties, bowel and bladder dysfunction, inability to perform self-care, and skin breakdown. Nursing management focuses on the prompt initiation of rehabilitation for any deficits.

#### Assessment

During the acute phase, a neurologic flow sheet is maintained to provide data about the following important measures of the patient's clinical status:

- Decrease in level of consciousness or responsiveness as evidenced by movement, resistance to changes of position, and response to stimulation; orientation to time, place, and person
- Change in vital signs with particular attention to blood pressure and temperature; maintenance of both within desired parameters
- Presence or absence of voluntary or involuntary movements of the extremities, muscle tone and strength, body posture, and position of the head
- Eye opening, comparative size of pupils and pupillary reactions to light, and ocular position
- Color of the face and extremities; temperature and moisture of the skin
- Quality and rates of pulse and respiration; arterial blood gas values as indicated, body temperature, and arterial pressure
- Ability to speak
- Volume of fluids ingested or given; volume of urine excreted each 24 hours
- Presence of bleeding
- Monitoring of continuous oxygen saturation
- Monitoring blood glucose

After the acute phase, the nurse assesses mental status (memory, attention span, perception, orientation, affect, speech/language), sensation/perception (the patient may have decreased awareness of pain and temperature), motor control (upper and lower extremity movement), swallowing ability, nutritional and hydration status, skin integrity, activity tolerance, and bowel and bladder function. Ongoing nursing assessment continues to focus on any impairment of function in the patient's daily activities, because the quality of life after stroke is closely related to the patient's functional status.

## Diagnosis

### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired mobility associated with hemiparesis, loss of balance and coordination, spasticity, and brain injury
- Acute pain (painful shoulder) associated with hemiplegia and disuse
- Impaired ability to perform hygiene, impaired self-toileting, impaired ability to dress, impaired self-feeding associated with stroke sequelae
- Discomfort associated with altered sensory reception, transmission, or integration
- Impaired swallowing
- Impaired urination associated with flaccid bladder, detrusor instability, confusion, or difficulty in communicating
- Constipation associated with change in mental status or difficulty communicating
- Acute confusion associated with brain infarction
- Impaired verbal communication associated with brain damage
- Risk for impaired skin integrity associated with hemiparesis, hemiplegia, or decreased mobility
- Impaired family process associated with catastrophic illness and caregiving burdens
- Impaired sexual functioning associated with neurologic deficits or fear of failure

### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Decreased cerebral blood flow due to increased ICP
- Inadequate oxygen delivery to the brain
- Pneumonia
- Seizure

### **Planning and Goals**

Although rehabilitation begins on the day the patient has the stroke, the process is intensified during convalescence and requires a coordinated team effort. It is helpful for the team to know what the patient was like before the stroke: their illnesses, abilities, mental and emotional state, behavioral characteristics, and activities of daily living. It is also helpful for clinicians to be knowledgeable about the relative importance of predictors of stroke outcome (age, NIHSS score, and level of consciousness at the time of admission) in order to provide survivors of stroke and their families with realistic goals (Powers et al., 2019).

The major goals for the patient (and family) may include improved mobility, avoidance of shoulder pain, achievement of self-care, relief of discomfort, prevention of aspiration, continence of bowel and bladder, decreasing confusion, achieving a form of communication, maintaining skin integrity, restored family functioning, improved sexual function, and absence of complications.

### **Nursing Interventions**

Nursing care has a significant impact on the patient's recovery. Often, many body systems are impaired as a result of the stroke, and conscientious care and timely interventions can prevent debilitating complications. During and after the acute phase, nursing interventions focus on the whole person. In addition to providing physical care, the nurse encourages and fosters recovery by listening to the patient and asking questions to elicit the meaning of the stroke experience.

#### **IMPROVING MOBILITY AND PREVENTING JOINT DEFORMITIES**

A patient with hemiplegia has unilateral paralysis (paralysis on one side). When control of the voluntary muscles is lost, the strong flexor muscles exert control over the extensors. The arm tends to adduct (adductor muscles are stronger than abductors) and rotate internally. The elbow and the wrist tend to flex, the affected leg tends to rotate externally at the hip joint and flex at the knee, and the foot at the ankle joint supinates and tends toward plantar flexion.

Correct positioning is important to prevent contractures; measures are used to relieve pressure, assist in maintaining good body alignment, and prevent compressive neuropathies, especially of the ulnar and peroneal nerves. Because flexor muscles are stronger than extensor muscles, a splint applied at night to the affected extremity may prevent flexion and maintain correct positioning during sleep.

**Preventing Shoulder Adduction.** To prevent adduction of the affected shoulder while the patient is in bed, a pillow is placed in the axilla when there is limited external rotation; this keeps the arm away from the chest. A pillow is placed under the arm, and the arm is placed in a neutral (slightly flexed) position, with distal joints positioned higher than the more proximal joints (i.e., the elbow is positioned higher than the shoulder and the wrist higher than the elbow). This helps to prevent edema and the resultant joint fibrosis that will limit range of motion if the patient regains control of the arm (see [Fig. 62-3](#)).

**Positioning the Hand and Fingers.** The fingers are positioned so that they are barely flexed. The hand is placed in slight supination (palm faces upward), which is its most functional position. If the upper extremity is flaccid, a splint can be used to support the wrist and hand in a functional position. If the upper extremity is spastic, a hand roll is not used, because it stimulates the grasp reflex. In this instance, a dorsal wrist splint is useful in allowing the palm to be free of pressure. Every effort is made to prevent hand edema.



**Figure 62-3 •** Correct positioning to prevent shoulder adduction.

Spasticity, particularly in the hand, can be a disabling complication after stroke. Botulinum toxin type A injected intramuscularly into wrist and finger muscles has been shown to be effective in reducing this spasticity (although the effect is temporary, typically lasting 2 to 4 months). This treatment is also effective for treating lower limb spasticity (Sun, Chen, Fu, et al., 2019). Other treatments for spasticity may include stretching, splinting (in select patients), and oral medications such as baclofen and tizanidine (Teasell, Salbach, Foley, et al., 2020).

**Changing Positions.** The patient's position should be changed every 2 hours. To place a patient in a lateral (side-lying) position, a pillow is placed between the legs before the patient is turned. To promote venous return and prevent edema, the upper thigh should not be acutely flexed. The patient may be turned from side to side, but if sensation is impaired, the amount of time spent on the affected side should be limited.

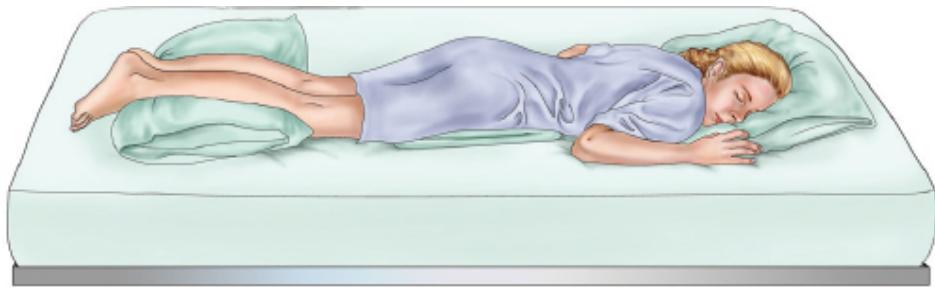
If possible, the patient is placed in a prone position for 15 to 30 minutes several times a day. A small pillow or a support is placed under the pelvis, extending from the level of the umbilicus to the upper third of the thigh (see Fig. 62-4). This position helps promote hyperextension of the hip joints, which is essential for normal gait and helps prevent knee and hip flexion contractures. The prone position also helps drain bronchial secretions and prevents contractual deformities of the shoulders and knees. During positioning, it is important to reduce pressure and change position frequently to prevent pressure injuries.

**Establishing an Exercise Program.** The affected extremities are exercised passively and put through a full range of motion four or five times a day to maintain joint mobility, regain motor control, prevent contractures in the paralyzed extremity, prevent further deterioration of the neuromuscular system, and enhance circulation. Exercise is helpful in preventing venous stasis, which may predispose the patient to venous thromboembolism (VTE). VTE includes deep vein thrombosis (DVT) and pulmonary embolism (PE).

Repetition of an activity forms new pathways in the CNS and therefore encourages new patterns of motion. At first, the extremities are usually flaccid. If tightness occurs in any area, the range-of-motion exercises should be performed more frequently.

The patient is observed for signs and symptoms that may indicate PE or excessive cardiac workload during exercise; these include

shortness of breath, chest pain, cyanosis, and increasing pulse rate with exercise. Frequent short periods of exercise always are preferable to longer periods at infrequent intervals. Regularity in exercise is most important. Improvement in muscle strength and maintenance of range of motion can be achieved only through daily exercise.



**Figure 62-4** • Prone position with pillow support helps prevent hip flexion.

The patient is encouraged and reminded to exercise the unaffected side at intervals throughout the day. It is helpful to develop a written schedule to remind the patient of the exercise activities. The nurse supervises and supports the patient during these activities. The patient can be instructed to put the unaffected leg under the affected one to assist in moving it when turning and exercising. Flexibility, strengthening, coordination, endurance, and balancing exercises prepare the patient for ambulation. Quadriceps muscle setting and gluteal setting exercises (see [Chapter 37, Chart 37-4](#)) are started early to improve the muscle strength needed for walking; these are performed at least five times daily for 10 minutes at a time.

**Preparing for Ambulation.** As soon as possible, the patient is assisted out of bed and an active rehabilitation program is started. The patient is first educated to maintain balance while sitting and then to learn to balance while standing. If the patient has difficulty in achieving standing balance, a tilt table, which slowly brings the patient to an upright position, can be used. Tilt tables are especially helpful for patients who have been on bed rest for prolonged periods and have orthostatic blood pressure changes.

If the patient needs a wheelchair, the folding type with hand brakes is the most practical because it allows the patient to manipulate the chair. The chair should be low enough to allow the patient to propel it with the uninvolved foot and narrow enough to permit it to be used at

home. When the patient is transferred from the wheelchair, the brakes must be applied and locked on both sides of the chair.

The patient is usually ready to walk as soon as standing balance is achieved. Parallel bars are useful in these first efforts. A chair or wheelchair should be readily available in case the patient suddenly becomes fatigued or feels dizzy.

The training periods for ambulation should be short and frequent. As the patient gains strength and confidence, an adjustable cane can be used for support. In general, a three- or four-pronged cane provides a stable support in the early phases of rehabilitation.

#### **PREVENTING SHOULDER PAIN**

The incidence of shoulder pain after stroke can vary widely, but has been measured to be as high as 84% (Zhou, Li, Lu, et al., 2018). That pain may prevent patients from learning new skills and affect their quality of life. Shoulder function is essential in achieving balance and performing transfers and self-care activities. Problems that can occur include rotator cuff disorders, spasticity of the shoulder muscles, painful shoulder, subluxation of the shoulder, and shoulder-hand syndrome. Development of a condition known as central pain syndrome may also contribute to the development of shoulder pain after a stroke.

A flaccid shoulder joint may be overstretched by the use of excessive force in turning the patient or from overstrenuous arm and shoulder movement. To prevent shoulder pain, the nurse should never lift the patient by the flaccid shoulder or pull on the affected arm or shoulder. Overhead pulleys should also be avoided. If the arm is paralyzed, subluxation (incomplete dislocation) at the shoulder can occur as a result of overstretching of the joint capsule and musculature by the force of gravity when the patient sits or stands in the early stages after a stroke. This results in severe pain. Shoulder-hand syndrome (painful shoulder and generalized swelling of the hand) can cause a frozen shoulder and ultimately atrophy of subcutaneous tissues. When a shoulder becomes stiff, it is usually painful.

Many shoulder problems can be prevented by proper patient movement and positioning. The flaccid arm is positioned on a table or with pillows while the patient is seated. A sling maybe worn on the flaccid arm when the patient first becomes ambulatory, to prevent the paralyzed upper extremity from dangling without support. Range-of-

motion exercises are important in preventing painful shoulder. Overstrenuous arm movements are avoided. The patient is instructed to interlace the fingers, place the palms together, and push the clasped hands slowly forward to bring the scapulae forward; the patient then raises both hands above the head. This is repeated throughout the day. The patient is instructed to flex the affected wrist at intervals and move all the joints of the affected fingers. The patient is encouraged to touch, stroke, rub, and look at both hands. Pushing the heel of the hand firmly down on a surface is useful. Elevation of the arm and hand is also important in preventing dependent edema of the hand. Patients with continuing pain after attempted movement and positioning may require the addition of analgesia to their treatment program. Other treatments may include injections to the shoulder joint with corticosteroid medications or botulinum toxin type A, shoulder taping/strapping, acupuncture, electrical stimulation, heat or ice, and soft tissue massage (Teasell et al., 2020; Treister, Hatch, Cramer, et al., 2017).

Medications are often helpful in the management of poststroke pain. The medications that are used include amitriptyline, gabapentin, lamotrigine, and pregabalin (Teasell et al., 2020; Treister et al., 2017).

#### **ENHANCING SELF-CARE**

As soon as the patient can sit up, they are encouraged to participate in personal hygiene activities. The patient is helped to set realistic goals; if feasible, a new task is added daily. The first step is to carry out all self-care activities on the unaffected side. Such activities as combing the hair, brushing the teeth, shaving with an electric razor, bathing, and eating can be carried out with one hand and should be encouraged. Although the patient may feel awkward at first, these motor skills can be learned by repetition, and the unaffected side will become stronger with use. The nurse must be sure that the patient does not neglect the affected side. Assistive devices will help make up for some of the patient's deficits (see [Chart 62-3](#)). A small towel is easier to control while drying after bathing, and boxed paper tissues are easier to use than a roll of toilet tissue.

Return of functional ability is important to the patient recovering after a stroke. An early baseline assessment of functional ability with an instrument such as the Functional Independence Measure (FIM<sup>TM</sup>) is important in team planning and goal setting for the patient. The FIM<sup>TM</sup> is a widely used instrument in stroke rehabilitation and

provides valuable information about motor, social, and cognitive function. The patient's morale may improve if ambulatory activities are carried out in street clothes. The family is instructed to bring in clothing that is preferably a size larger than that usually worn. Clothing fitted with front or side fasteners or Velcro closures is the most suitable. The patient has better balance if most of the dressing activities are carried out while seated.

### Chart 62-3

#### Assistive Devices to Enhance Self-Care After Stroke

##### Bathing and Grooming Devices

- Electric razors with head at 90 degrees to handle
- Grab bars, nonskid mats, handheld shower heads
- Long-handled bath sponge
- Shower and tub seats, stationary or on wheels

##### Dressing Aids

- Elastic shoelaces
- Long-handled shoehorn
- Velcro closures

##### Eating Devices

- Nonskid mats to stabilize plates
- Plate guards to prevent food from being pushed off plate
- Wide-grip utensils to accommodate a weak grasp

##### Mobility Aids

- Canes, walkers, wheelchairs
- Transfer devices such as transfer boards and belts

##### Toileting Aids

- Grab bars next to toilet
- Raised toilet seat

Perceptual problems may make it difficult for the patient to dress without assistance because of an inability to match the clothing to the body parts. To assist the patient, the nurse can take steps to keep the

environment organized and uncluttered, because the patient with a perceptual problem is easily distracted. The clothing is placed on the affected side in the order in which the garments are to be put on. The use of a large mirror while dressing promotes the patient's awareness of what they are putting on the affected side. The patient has to make many compensatory movements when dressing; these can produce fatigue and painful twisting of the intercostal muscles. Support and encouragement are provided to prevent the patient from becoming overly fatigued and discouraged. Even with intensive training, not all patients can achieve independence in dressing.

#### **ADJUSTING TO PHYSICAL CHANGES**

Patients with a decreased field of vision should be approached on the side where visual perception is intact. All visual stimuli (e.g., clock, calendar, television) should be placed on this side. The patient can be educated to turn the head in the direction of the defective visual field to compensate for this loss. The nurse should make eye contact with the patient and draw their attention to the affected side by encouraging the patient to move the head. The nurse may also want to stand at a position that encourages the patient to move or turn to visualize who is in the room. Increasing the natural or artificial lighting in the room and providing eyeglasses are important aids to increasing vision.

The patient with homonymous hemianopsia turns away from the affected side of the body and tends to neglect that side and the space on that side; this is known as amorphosynthesis. In such instances, the patient cannot see food on half of the tray, and only half of the room is visible. It is important for the nurse to constantly remind the patient of the other side of the body; to maintain alignment of the extremities; and, if possible, to place the extremities where the patient can see them.

#### **ASSISTING WITH NUTRITION**

Stroke can result in **dysphagia** (difficulty swallowing) due to impaired function of the mouth, tongue, palate, larynx, pharynx, or upper esophagus. Patients must be observed for paroxysms of coughing, food dribbling out of or pooling in one side of the mouth, food retained for long periods in the mouth, or nasal regurgitation when swallowing liquids. Swallowing difficulties place the patient at risk for aspiration, pneumonia, dehydration, and malnutrition.

A swallow assessment should be performed as soon as possible after the patient's arrival to the ED (preferably within 4 to 24 hours). This is done before allowing any oral intake. A speech therapist will evaluate the patient's swallowing ability, but an assessment may also be done by the nurse using a validated and reliable assessment tool (Stroke Foundation, 2019).

If swallowing function is partially impaired, it may return over time, or the patient may be educated in alternative swallowing techniques, advised to take smaller boluses of food, and educated about types of foods that are easier to swallow. The patient may be started on a thick liquid or pureed diet, because these foods are easier to swallow than thin liquids. Having the patient sit upright, preferably out of bed in a chair, and instructing them to tuck the chin toward the chest as they swallow will help prevent aspiration. The diet may be advanced as the patient becomes more proficient at swallowing. If the patient cannot resume oral intake, a gastrointestinal feeding tube is placed for ongoing tube feedings and medication administration.

Enteral tubes can be either nasogastric (placed in the stomach) or nasoenteral (placed in the duodenum) to reduce the risk of aspiration. Nursing responsibilities in feeding include elevating the head of the bed at least 30 degrees to prevent aspiration, checking the position of the tube before feeding, ensuring that the cuff of the tracheostomy tube (if in place) is inflated, and giving the tube feeding slowly. The feeding tube is aspirated periodically to ensure that the feedings are passing through the gastrointestinal tract. Retained or residual feedings increase the risk of aspiration. Patients with retained feedings may benefit from the placement of a gastrostomy tube or a percutaneous endoscopic gastrostomy (PEG) tube. In a patient with a feeding tube, the tube should be placed in the duodenum to reduce the risk of aspiration. For long-term feedings, a gastrostomy tube is preferred (see [Chapter 39](#)).

#### **ATTAINING BLADDER AND BOWEL CONTROL**

After a stroke, the patient may have transient urinary incontinence due to confusion, inability to communicate needs, and inability to use the urinal or bedpan because of impaired motor and postural control. Occasionally after a stroke, the bladder becomes atonic, with impaired sensation in response to bladder filling. Sometimes, control of the external urinary sphincter is lost or diminished. During this period, intermittent catheterization with sterile technique is carried

out. After muscle tone increases and deep tendon reflexes return, bladder tone increases and spasticity of the bladder may develop. Because the patient's sense of awareness is clouded, persistent urinary incontinence or urinary retention may be symptomatic of bilateral brain damage. The voiding pattern is analyzed, and the urinal or bedpan is offered on this pattern or schedule. The upright posture and standing position are helpful for male patients during this aspect of rehabilitation.

Patients may have problems with bowel control, particularly constipation. Unless contraindicated, a high-fiber diet and adequate fluid intake (2 to 3 L/day) should be provided, and a regular time (usually after breakfast) should be established for toileting.

#### **IMPROVING THOUGHT PROCESSES**

After a stroke, the patient may have problems with cognitive, behavioral, and emotional deficits related to brain damage. However, in many instances, a considerable degree of function can be recovered, because not all areas of the brain are equally damaged; some remain more intact and functional than others.

After assessment that delineates the patient's deficits, the neuropsychologist, in collaboration with the primary provider, psychiatrist, nurse, and other professionals, structures a training program using cognitive-perceptual retraining, visual imagery, reality orientation, and cueing procedures to compensate for losses. Specific techniques used maybe conventional, computer-assisted, or virtual reality based.

The role of the nurse is supportive. The nurse reviews the results of neuropsychological testing; observes the patient's performance and progress; gives positive feedback; and, most importantly, conveys an attitude of confidence and hope. Interventions capitalize on the patient's strengths and remaining abilities while attempting to improve performance of affected functions. Other interventions are similar to those for improving cognitive functioning after a head injury (see [Chapter 63](#)).

#### **IMPROVING COMMUNICATION**

Aphasia, which impairs the ability to express oneself and to understand what is being said, may become apparent in various ways. The cortical area that is responsible for integrating the myriad pathways required for the comprehension and formulation of language is called *Broca area*. It is located in a convolution adjoining

the middle cerebral artery. This area is responsible for control of the combinations of muscular movements needed to speak each word. Broca area is so close to the left motor area that a disturbance in the motor area often affects the speech area. This is why so many patients who are paralyzed on the right side (due to damage or injury to the left side of the brain) cannot speak, whereas those paralyzed on the left side are less likely to have speech disturbances.

The speech therapist assesses the communication needs of the patient who has had a stroke, describes the precise deficit, and suggests the best overall method of communication. Most language intervention strategies can be tailored for the individual patient. The patient is expected to take an active part in establishing goals.

A person with aphasia may become depressed. The inability to talk on the telephone, text, answer a question, or participate in conversation often causes anger, frustration, fear of the future, and hopelessness. Nursing interventions include strategies to make the atmosphere conducive to communication. This includes being sensitive to the patient's reactions and needs and responding to them in an appropriate manner while always treating the patient as an adult. The nurse provides strong emotional support and understanding to allay anxiety and frustration.

A common pitfall is for the nurse or other health care team member to complete the thoughts or sentences of the patient. This should be avoided, because it causes the patient to become more frustrated at not being allowed to speak and may deter efforts to practice putting thoughts together and completing sentences. A consistent schedule, routines, and repetition help the patient to function despite significant deficits. A written copy of the daily schedule, a folder of personal information (birth date, address, names of relatives), checklists, and recorded lists help improve the patient's memory and concentration. The patient may also benefit from a communication board (electronic or written), which has pictures of common needs and phrases. The board may be translated into any language.

When talking with the patient, it is important for the nurse to gain the patient's attention, speak slowly, and keep the language of instruction consistent. One instruction is given at a time, and time is allowed for the patient to process what has been said. The use of gestures may enhance comprehension. Speaking is thinking out loud, and the emphasis is on thinking. Listening and sorting out incoming

messages require mental effort; the patient must struggle against mental inertia and needs time to organize a response.

In working with the patient with aphasia, the nurse must remember to talk to the patient during care activities. This provides social contact for the patient. [Chart 62-4](#) describes points to keep in mind when communicating with the patient with aphasia.

#### **MAINTAINING SKIN INTEGRITY**

The patient who has had a stroke may be at risk for skin and tissue breakdown because of altered sensation and inability to respond to pressure and discomfort by turning and moving. Preventing skin and tissue breakdown requires frequent assessment of the skin, with emphasis on bony areas and dependent parts of the body. During the acute phase, a specialty bed (e.g., low air-loss bed) may be used until the patient can move independently or assist in moving.

#### **Chart 62-4**

##### **Communicating with the Patient with Aphasia**

- Face the patient and establish eye contact.
- Speak in a clear, unhurried manner, and normal tone of voice.
- Use short phrases, and pause between phrases to allow the patient time to understand what is being said.
- Limit conversation to practical and concrete matters.
- Use gestures, pictures, objects, and writing.
- As the patient uses and handles an object, say what the object is. It helps to match the words with the object or action.
- Be consistent in using the same words and gestures each time you give instructions or ask a question.
- Keep extraneous noises and sounds to a minimum. Too much background noise can distract the patient or make it difficult to sort out the message being spoken.

A regular turning schedule (e.g., every 2 hours) is adhered to, even if pressure-relieving devices are used to prevent tissue and skin breakdown. When the patient is positioned or turned, care must be used to minimize shear and friction forces, which cause damage to tissues and predispose the skin to breakdown.

The patient's skin must be kept clean and dry; gentle massage of healthy (nonreddened) skin and adequate nutrition are other factors that help to maintain skin and tissue integrity.

### **IMPROVING FAMILY COPING**

Family members play an important role in the patient's recovery. Family members are encouraged to participate in counseling and to use support systems that will help with the emotional and physical stress of caring for the patient. Involving others in the patient's care and providing education about stress management techniques and methods for maintaining personal health also facilitate family coping.

The family may have difficulty accepting the patient's disability and may be unrealistic in their expectations. They are given information about the expected outcomes and are counseled to avoid doing activities that the patient is able to do. They are assured that their love and interest are part of the patient's therapy.

The family needs to be informed that the rehabilitation of the patient with hemiplegia requires many months and that progress may be slow. The gains made by the patient in the hospital or rehabilitation unit must be maintained. All caregivers should approach the patient with a supportive and optimistic attitude, focusing on the patient's remaining abilities. The rehabilitation team, the medical and nursing team, the patient, and the family must all be involved in developing attainable goals for the patient at home.

Most relatives of patients with stroke handle the physical changes better than the emotional aspects of care. The family should be prepared to expect occasional episodes of emotional lability. The patient may laugh or cry easily or without cause (pseudobulbar affect) and may be irritable and demanding or depressed and confused. The nurse can explain to the family that the patient's laughter does not necessarily connote happiness, nor does crying reflect sadness, and that emotional lability usually improves with time.

Family-centered care involves seeing patients and family caregivers as a one unit. Nurses can assess caregivers' strengths and ability to provide care. This assessment should be a continual process because needs change throughout the hospitalization period and rehabilitation stay. Providing information about community resources, respite care and adult day care, and mental health issues (for the patient who has had a stroke and for caregivers) will help with the transition to home.

### **HELPING THE PATIENT COPE WITH SEXUAL DYSFUNCTION**

Sexual functioning can be profoundly altered by stroke. Although research in this area of stroke management is limited, it appears that patients who have had a stroke consider sexual function important, and many have sexual dysfunction. Sexual dysfunction after stroke is multifactorial. There may be medical reasons for the dysfunction (neurologic and cognitive deficits, previous diseases, medications) as well as various psychosocial factors, including depression. A stroke is such a catastrophic illness that the patient experiences loss of self-esteem and value as a sexual being. These psychosocial factors play an important role in determining sexual drive, activity, and satisfaction after a stroke.

Nurses in the rehabilitation setting play a crucial role in beginning a dialogue between the patient and their partner about sexuality after a stroke. In-depth assessments to determine sexual history before and after the stroke should be followed by appropriate interventions. Interventions for the patient and partner focus on providing relevant information, education, reassurance, adjustment of medications, counseling regarding coping skills, suggestions for alternative sexual positions, and a means of sexual expression and satisfaction.

### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Decreased cerebral blood flow due to increased ICP, leading to inadequate oxygen delivery to the brain, seizures, and pneumonia are potential complications in any patient who has had an ischemic stroke. The more severe the stroke (i.e., the higher the NIHSS), the greater the risk of complications.

During the acute phase of care, a neurologic flow sheet (see [Chapter 61, Fig. 61-6](#)) is used to monitor and document assessment parameters. Changes in blood pressure, pulse, and respiration are important clinically because they suggest increased ICP and are reported immediately. If signs and symptoms of pneumonia develop, cultures are obtained to identify the organism so that appropriate antibiotic agents can be given.

### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



**Educating Patients About Self-Care.** Patient and family education is a fundamental component of stroke recovery. The nurse provides education about stroke, its causes and prevention, and the

rehabilitation process. In both acute care and rehabilitation facilities, the focus is on educating the patient to resume as much self-care as possible. This may entail using assistive devices or modifying the home environment to help the patient live with a disability.

An occupational therapist may be helpful in assessing the home environment and recommending modifications to help the patient become more independent. For example, a shower is more convenient than a tub for the patient with hemiplegia because most patients do not gain sufficient strength to get up and down from a tub. Sitting on a stool of medium height with rubber suction tips allows the patient to wash with greater ease. A long-handled bath brush with a soap container is helpful to the patient who has only one functional hand. If a shower is not available, a stool may be placed in the tub and a portable shower hose attached to the faucet. Hand rails may be attached alongside the bathtub and the toilet. Other assistive devices include special utensils for eating, grooming, dressing, and writing (see [Chart 62-3](#)).

A program of physical therapy can be beneficial, whether it takes place in the home or in an outpatient program. Constraint-induced movement therapy has been used in stroke rehabilitation and involves constraint of the less affected upper limb, and intensely training the more affected limb. Robotic-assisted therapy uses sensorimotor training of the upper limb. This method allows patients to train without the presence of a therapist. Other techniques may include using virtual reality and video game applications, functional/neuromuscular/transcutaneous electrical nerve stimulation, transcranial magnetic stimulation, and ambulation with body weight support and treadmill training to assist with recovery (Veteran's Administration/Department of Defense, *The Management of Stroke Rehabilitation Work Group*, 2019).

#### **CONTINUING AND TRANSITIONAL CARE**

A variety of transitional care models are being used in patients with stroke. Some evidence supports positive outcomes using transitional care but more research and standardization of interventions is needed for confirmation. One model currently being investigated is a person-centered model. This model includes a telephone call at 2, 30, and 60 days after discharge. Calls are conducted either by a nurse or an advanced practice provider. Patients are also seen in the outpatient clinic within 2 weeks after discharge (Bushnell, Duncan, Lycan, et al.,

2018). Depending on the specific neurologic deficits resulting from the stroke, the patient at home may require the services of a number of health care professionals. The nurse often coordinates the care of the patient at home and considers the many educational needs of caregivers and patients. The family (often the spouse) requires education as well as assistance in planning and providing care.

The family is advised that the patient may tire easily, may become irritable and upset by small events, and may be less interested in events than expected. Emotional problems associated with stroke are often related to speech dysfunction and the frustrations of being unable to communicate. A speech therapist allows the family to be involved and gives the family practical instructions to help the patient between therapy sessions.

Depression is a common and serious problem (increases mortality) in the patient who has had a stroke. Approximately one third of patients who have had a stroke will suffer from depression (Virani et al., 2020). Risk factors include gender (more prevalent in women), a history of depression, cognitive or physical impairment, anxiety, aphasia and stroke severity (Villa, Ferrari, & Moretti, 2018). Because the length of hospital stays has shortened, depression may not be identified in the acute setting. Nurses in all care settings should identify patients who may be at risk for depression or who show depressive symptoms. In the home or in the rehabilitation setting, nurses may be involved in coordinating care and referring patients and family to appropriate resources. The family can help by continuing to support the patient and by giving positive reinforcement for the progress that is being made. Antidepressant therapy may be prescribed, and may help with recovery from stroke (Elzib, Pawloski, Ding, et al., 2019).

Community-based stroke support groups may allow the patient and family to learn from others with similar problems and to share their experiences. Support groups take the form of in-person meetings as well as Internet-based support programs. The patient is encouraged to continue hobbies and recreational and leisure interests and to maintain contact with friends to prevent social isolation. All nurses coming in contact with the patient should encourage the patient to keep active, adhere to the exercise program, and remain as self-sufficient as possible.

**Chart 62-5**



## **NURSING RESEARCH PROFILE**

## Early Identification of Depression in Caregivers of Stroke Survivors

Byun, E., Evans, L., Sommers, M., et al. (2019). Depressive symptoms in caregivers immediately after stroke. *Topics in Stroke Rehabilitation*, 26(3), 187–194.

### Purpose

In the early weeks post stroke, caregivers face many challenges and increased stress related to caring for a stroke survivor. Recognizing those caregivers most at risk for developing depression early after hospital discharge may lead to decreased symptoms and improved health and quality of life for caregivers. The long-term health of caregivers of stroke survivors is significant; poor health can impact their ability to serve in the capacity of caregiver. The purpose of this study was to identify characteristics of caregivers and stroke survivors associated with caregiver depressive symptoms in the early weeks following a family member's stroke.

### Design

This study used a prospective, longitudinal exploratory design. The participants were a convenience sample of 63 caregivers of older adult stroke survivors who had been diagnosed with a new or recurrent ischemic or hemorrhagic stroke within the past 2 weeks. They were recruited from urban acute care settings. Caregivers were enrolled in the study by 2 weeks post stroke (T1) and then revisited 4 weeks later (T2). Symptoms of depression were measured using the Patient Health Questionnaire (PHQ-9). The PHQ-9 is a 9-item scale used as a screening tool for major and minor depression. Uncertainty, stress, coping capacity, social support, chronic illness, and sociodemographic information were measured. Perceived stress was measured with the Perceived Stress Scale. Physiologic stress was measured by salivary cortisol levels measured upon waking and in the evening. The stroke survivor's functional status was measured using the Barthel Index, a 10-item scale measuring functional status. Sociodemographic information and clinical characteristics (severity of stroke, description of stroke, presence of communication disability, and days post stroke) of the stroke survivors were also collected.

### Findings

This study began with 63 caregivers enrolled. By the second time point evaluation (T2), 13 stroke survivors had died, 3 caregivers

were lost to follow-up. A total of 40 caregivers completed the second time point evaluation (T2). Ages of caregivers ranged from 30 to 89 years (mean 56.92 years). Ages of stroke survivors ranged from 65 to 95 years (mean 75.92 years). More than half (57%) of caregivers had at least mild depressive symptoms in the early weeks of caregiving. Six weeks after the stroke, 40% continued to have depressive symptoms. About 30% had at least moderate depressive symptoms at both time points (T1 and T2). Greater depressive symptoms were correlated with elevated salivary cortisol levels in the evening but not with levels upon waking. Characteristics associated with more depressive symptoms across the first 6 weeks post stroke included caregiver uncertainty, perceived stress, coping, social support, race, income, time spent in caregiving, and stroke survivor race and functional status.

### Nursing Implications

This study found that depressive symptoms in this sample of caregivers were common in the early weeks of caregiving. It is important for nurses to understand the caregiving experience and the risk in both patients and caregivers for developing depression. Clearly identifying characteristics that are associated with caregivers developing depression can help with earlier recognition of those at risk. If these caregivers receive appropriate interventions and additional support soon after hospital discharge, they may experience decreased depressive symptoms and improved quality of life. Nurses should be involved in the discharge process to home and can help caregivers adjust and be prepared for their new role and challenges.

The nurse should recognize the potential effects of caregiving on the family. Not all families have the adaptive coping skills and adequate psychological functioning necessary for the long-term care of another person. The patient's spouse may be older, with their own health concerns; in some instances, the patient may have been the provider of care to the spouse. A spouse may have to take on new roles and responsibilities in the relationship and around the home. Spouses may also feel a sense of loss (of freedom and leisure time as well as of the marital relationship) and may experience social isolation and financial burdens. Depression is common in caregivers of patients who have survived a stroke, with rates globally documented as approximately 40% (Loh, Tan, Zhang, et al., 2017).

Nurses should assess caregivers for signs and symptoms of depression (Byun, Evans, Sommers, et al., 2019). See the Nursing Research Profile in [Chart 62-5](#).

Caregivers may require reminders to attend to their own health concerns and well-being. Even healthy caregivers may find it difficult to maintain a schedule that includes being available around the clock. The nurse encourages the family to arrange for respite care services (planned short-term care to relieve the family from having to provide continuous 24-hour care), which may be available from an adult day care center. Some hospitals also offer weekend respite care that can provide caregivers with needed time for themselves. The nurse involved in home and continuing care also needs to remind the patient and family of the need for respite care as well as continuing health promotion and screening practices.

### Evaluation

Expected patient outcomes may include:

1. Achieves improved mobility
  - a. Avoids deformities (contractures and footdrop)
  - b. Participates in prescribed exercise program
  - c. Achieves sitting balance
  - d. Uses unaffected side to compensate for loss of function of hemiplegic side
2. Reports absence of shoulder pain
  - a. Demonstrates shoulder mobility; exercises shoulder
  - b. Elevates arm and hand at intervals
3. Achieves self-care; performs hygiene care; uses adaptive equipment
4. Demonstrates techniques to compensate for the discomfort of sensory deficits, such as turning the head to see people or objects
5. Demonstrates safe swallowing
6. Achieves usual pattern of bowel and bladder elimination
7. Participates in cognitive improvement program
8. Demonstrates improved communication
9. Maintains intact skin without breakdown

- a. Demonstrates skin turgor within normal limits
  - b. Participates in turning and positioning activities
10. Family members demonstrate a positive attitude and coping mechanisms
- a. Encourage patient in exercise program
  - b. Take an active part in rehabilitation process
  - c. Contact respite care programs or arrange for other family members to assume some responsibilities for care
11. Develops alternative approaches to sexual expression
12. Absence of complications
- a. Has ICP values that remain within normal limits
  - b. Has no signs and symptoms of pneumonia



## Hemorrhagic Stroke

Hemorrhagic strokes are primarily caused by intracerebral (10%) and subarachnoid hemorrhage (3%) and are caused by bleeding into the brain tissue, the ventricles, or the subarachnoid space (Norris, 2019). Primary intracerebral hemorrhage from a spontaneous rupture of small vessels accounts for approximately 80% of hemorrhagic strokes and is caused chiefly by uncontrolled hypertension. Subarachnoid hemorrhage results from a ruptured intracranial aneurysm (discussed later in this chapter) (Hickey & Strayer, 2020; Virani et al., 2020).

A common cause of primary intracerebral hemorrhage in the older adult is cerebral amyloid angiopathy, which involves damage caused by the deposit of beta-amyloid protein in the small- and medium-sized blood vessels of the brain. Cerebral amyloid angiopathy makes these blood vessels fragile and prone to bleeding. Secondary intracerebral hemorrhage is associated with arteriovenous malformations (AVMs), trauma, intracranial neoplasms, or certain medications (e.g., anticoagulants, cocaine, or amphetamines). The mortality rate has been reported to be as high as 50% after an intracranial hemorrhage (Hickey & Strayer, 2020; Norris, 2019). Patients who survive the acute phase of

care usually have more severe deficits and a longer recovery phase compared to those with ischemic stroke.

## Pathophysiology



The pathophysiology of hemorrhagic stroke depends on the cause and underlying type of cerebrovascular disorder. Symptoms are produced when a primary hemorrhage, aneurysm, or AVM presses on nearby cranial nerves or brain tissue or, more dramatically, when an aneurysm or AVM ruptures, causing subarachnoid hemorrhage (hemorrhage into the cranial subarachnoid space). Normal brain metabolism is disrupted by the brain's exposure to blood; by an increase in ICP resulting from the sudden entry of blood into the subarachnoid space, which compresses and injures brain tissue; or by secondary ischemia of the brain resulting from the reduced perfusion pressure and vasospasm that frequently accompany subarachnoid hemorrhage.

### Intracerebral Hemorrhage

An intracerebral hemorrhage, or bleeding into the brain tissue, is most common in patients with hypertension and cerebral atherosclerosis, because degenerative changes from these diseases cause rupture of the blood vessel. An intracerebral hemorrhage may also result from certain types of arterial pathology, trauma, brain tumors, and the use of medications (e.g., anticoagulants, amphetamines, and cocaine).

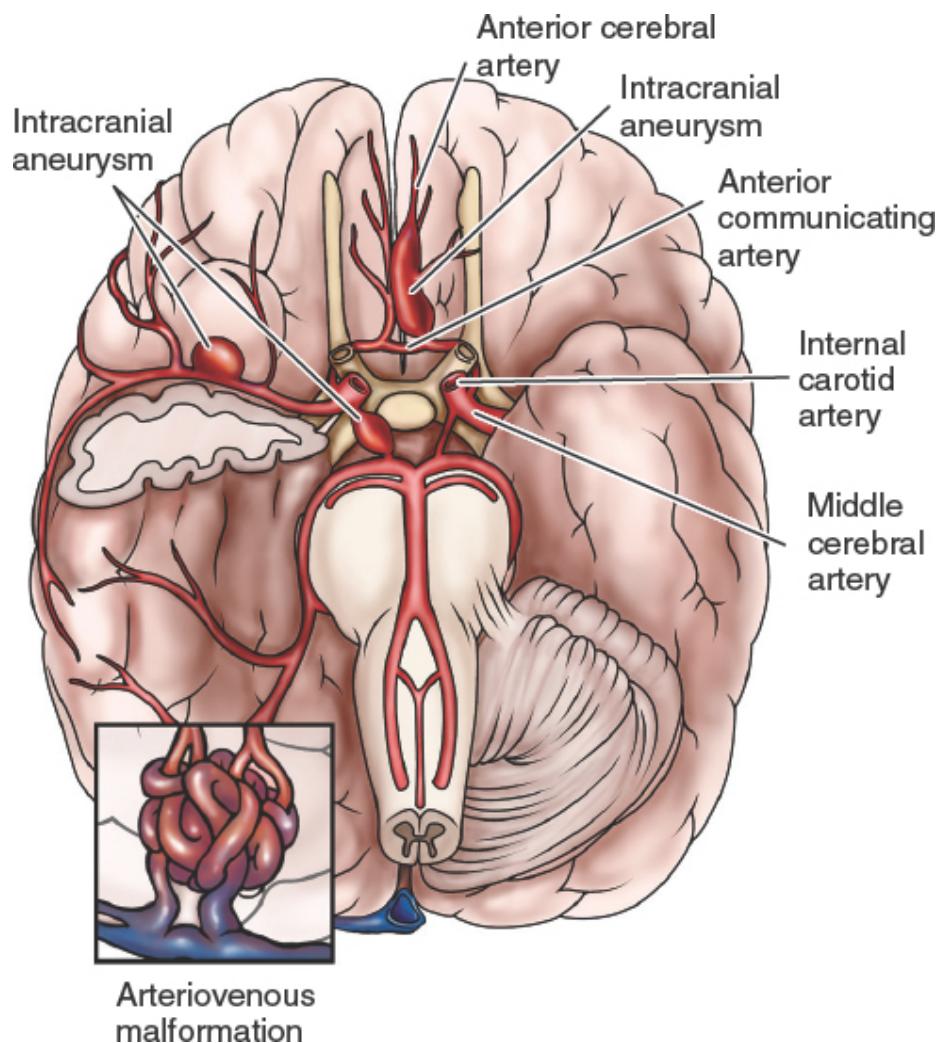
Bleeding related to hypertension occurs most commonly in the deeper structures of the brain (basal ganglia and thalamus); it occurs less frequently in the brainstem (mostly the pons) and cerebellum (Hickey & Strayer, 2020). Bleeding in the outer cerebral lobes (lobar hemorrhages) in those 75 or older can be related to cerebral amyloid angiopathy and is frequently in the frontal and parietal lobes. Occasionally, the bleeding ruptures the wall of the lateral ventricle and causes intraventricular hemorrhage, which is associated with poor outcomes and death (Hickey & Strayer, 2020)).

### Intracranial (Cerebral) Aneurysm

An intracranial (cerebral) **aneurysm** is a dilation of the walls of a cerebral artery that develops as a result of weakness in the arterial wall. The cause of aneurysms is unknown, although research is ongoing. An

aneurysm may be due to atherosclerosis, which results in a defect in the vessel wall with subsequent weakness of the wall; a congenital defect of the vessel wall; hypertensive vascular disease; head trauma; or advancing age.

Any artery within the brain can be the site of a cerebral aneurysm, but these lesions usually occur at the bifurcations of the large arteries at the circle of Willis (see Fig. 62-5). The cerebral arteries most commonly affected by an aneurysm are the internal carotid artery, anterior cerebral artery, anterior communicating artery, posterior communicating artery, posterior cerebral artery, and middle cerebral artery. Multiple cerebral aneurysms are not uncommon.



**Figure 62-5 •** Common sites of intracranial aneurysms and an arteriovenous malformation.

## **Arteriovenous Malformations**

Most AVMs are caused by an abnormality in embryonal development that leads to a tangle of arteries and veins in the brain that lacks a capillary bed (see Fig. 62-5). The absence of a capillary bed leads to dilation of the arteries and veins and eventual rupture. AVM is a common cause of hemorrhagic stroke in young people (Hickey & Strayer, 2020).

## **Subarachnoid Hemorrhage**

A subarachnoid hemorrhage (hemorrhage into the subarachnoid space) may occur as a result of an AVM, intracranial aneurysm, trauma, or hypertension. The most common causes are a leaking aneurysm in the area of the circle of Willis and a congenital AVM of the brain (Hickey & Strayer, 2020).

## **Clinical Manifestations**

The patient with a hemorrhagic stroke can present with a wide variety of neurologic deficits, similar to the patient with ischemic stroke. The conscious patient most commonly reports a severe headache. A comprehensive assessment reveals the extent of the neurologic deficits. Many of the same motor, sensory, cranial nerve, cognitive, and other functions that are disrupted after ischemic stroke are also altered after a hemorrhagic stroke. Table 62-2 reviews the neurologic deficits frequently seen in patients who have had a stroke. Table 62-3 compares the symptoms seen in right hemispheric stroke with those seen in left hemispheric stroke. Other symptoms that may be observed more frequently in patients with acute intracerebral hemorrhage (compared with ischemic stroke) are nausea or vomiting, headache, an early sudden change in level of consciousness (confusion to coma), and possibly seizures.

In addition to the neurologic deficits (similar to those of ischemic stroke), the patient with an intracranial aneurysm or AVM may have some unique clinical manifestations. Rupture of an aneurysm or AVM usually produces a sudden, unusually severe headache and often loss of consciousness for a variable period of time. There may be pain and rigidity of the back of the neck (nuchal rigidity) and spine due to meningeal irritation. Visual disturbances (visual loss, diplopia, ptosis) occur if the aneurysm is adjacent to the oculomotor nerve. Tinnitus,

dizziness, photophobia (visual intolerance of light), nausea or vomiting, and hemiparesis may also occur.

At times, an aneurysm or AVM leaks blood, leading to the formation of a clot that seals the site of rupture. In this instance, the patient may show little neurologic deficit. In other cases, severe bleeding occurs, resulting in cerebral damage, followed rapidly by coma and death.

Prognosis depends on the neurologic condition of the patient, the patient's age, associated diseases, and the extent and location of the hemorrhage or intracranial aneurysm. Subarachnoid hemorrhage from an aneurysm is a catastrophic event with significant morbidity and mortality.

## Assessment and Diagnostic Findings

Any patient with suspected stroke should undergo a CT scan or MRI scan to determine the type of stroke, the size and location of the hematoma, and the presence or absence of ventricular blood and hydrocephalus. Because hemorrhagic stroke is an emergency, CT scan is usually obtained first because it can be done rapidly. Cerebral angiography using the conventional method or a CT angiography confirms the diagnosis of an intracranial aneurysm or AVM. These tests show the location and size of the lesion and provide information about the affected arteries, veins, adjoining vessels, and vascular branches. Lumbar puncture may be performed if there is no evidence of increased ICP, the CT scan results are negative, and subarachnoid hemorrhage must be confirmed. Lumbar puncture in the presence of increased ICP could result in brainstem herniation or rebleeding.



For the procedural guidelines for assisting with a lumbar puncture, go to [thepoint.lww.com/Brunner15e](http://thepoint.lww.com/Brunner15e).

When diagnosing a hemorrhagic stroke in a patient younger than 40 years, some clinicians obtain a toxicology screen for illicit drug use.

## Prevention

Primary prevention of hemorrhagic stroke is the best approach and includes managing hypertension and ameliorating other significant risk factors. Control of hypertension can reduce the risk of hemorrhagic stroke. Additional risk factors are increased age, male gender, certain ethnicities (Latino, African American, and Japanese) and moderate or excessive alcohol intake (Hickey & Strayer, 2020). Stroke risk screenings provide an ideal opportunity to lower hemorrhagic stroke risk by identifying individuals or groups at high risk and educating patients and the community about recognition and prevention.

## Complications

Potential complications of hemorrhagic stroke include rebleeding or hematoma expansion; cerebral vasospasm resulting in cerebral ischemia; acute hydrocephalus, which results when free blood obstructs the reabsorption of cerebrospinal fluid (CSF) by the arachnoid villi; and seizures.

### Cerebral Hypoxia and Decreased Blood Flow

Immediate complications of a hemorrhagic stroke include cerebral hypoxia, decreased cerebral blood flow, and extension of the area of injury. Providing adequate oxygenation of blood to the brain minimizes cerebral hypoxia. Brain function depends on delivery of oxygen to the tissues. Administering supplemental oxygen and maintaining the hemoglobin and hematocrit at acceptable levels will assist in maintaining tissue oxygenation.

Cerebral blood flow depends on the blood pressure, cardiac output, and integrity of cerebral blood vessels. Adequate hydration (IV fluids) must be ensured to reduce blood viscosity and improve cerebral blood flow. Extremes of hypertension or hypotension need to be avoided to prevent changes in cerebral blood flow and the potential for extending the area of injury.

A seizure can also compromise cerebral blood flow, resulting in further injury to the brain. Observing for seizure activity and initiating appropriate treatment are important components of care after a hemorrhagic stroke.

### Vasospasm

The development of cerebral vasospasm (narrowing of the lumen of the involved cranial blood vessel) is a serious complication of subarachnoid hemorrhage and is a leading cause of morbidity and mortality in those who survive the initial subarachnoid hemorrhage. The mechanism responsible for vasospasm is not clear, but it is associated with increasing amounts of blood in the subarachnoid cisterns and cerebral fissures, as visualized by CT scan. Monitoring for vasospasm may be performed through the use of bedside transcranial Doppler ultrasonography or follow-up cerebral angiography (Connolly, Rabinstein, Carhuapoma, et al., 2012; Wilson, Ashcroft, & Troiani, 2019).

Vasospasm most frequently occurs 7 to 8 days after initial hemorrhage (American Association of Neuroscience Nurses [AANN], 2018), when the clot undergoes lysis (dissolution), and the chance of rebleeding is increased. It leads to increased vascular resistance, which impedes cerebral blood flow and causes brain ischemia (delayed cerebral ischemia) and infarction. Vasospasm can also occur 3 to 14 days after subarachnoid hemorrhage (Hickey & Strayer, 2020). Vasospasm is likely not the only factor that plays a role in the development of delayed cerebral ischemia. The signs and symptoms reflect the areas of the brain involved. Vasospasm is often heralded by a worsening headache, a decrease in level of consciousness (confusion, lethargy, and disorientation), or a new focal neurologic deficit (aphasia, hemiparesis).

Management of vasospasm remains difficult and controversial. It is believed that early surgery to clip the aneurysm prevents rebleeding and that removal of blood from the basal cisterns around the major cerebral arteries may prevent vasospasm.

Medication may be effective in the treatment and prevention of vasospasm. Based on the theory that vasospasm is caused by an increased influx of calcium into the cell, medication therapy may be used either to block or antagonize this action, or to prevent or reverse the action of vasospasm if already present. Nimodipine is the most studied calcium channel blocker for prevention of vasospasm in subarachnoid hemorrhage. Current guidelines recommend that nimodipine be prescribed for all patients with subarachnoid hemorrhage (Connolly et al., 2012). This is currently the only drug approved by the FDA for the prevention and treatment of vasospasm in subarachnoid hemorrhage.

Another therapy for vasospasm and the resulting delayed cerebral ischemia, referred to as triple-H therapy, is aimed at minimizing the deleterious effects of the associated cerebral ischemia and includes (1)

fluid volume expanders (hypervolemia), (2) induced arterial hypertension, and (3) hemodilution. However, current research and guidelines now endorse euvolemia to prevent delayed cerebral ischemia and induced arterial hypertension for treatment of delayed cerebral ischemia (Connolly et al., 2012).

### **Increased Intracranial Pressure**

An increase in ICP can occur after either an ischemic or a hemorrhagic stroke but almost always follows a subarachnoid hemorrhage, usually because of disturbed circulation of CSF caused by blood in the basal cisterns. Neurologic assessments are performed frequently, and if there is evidence of deterioration from increased ICP (due to cerebral edema, herniation, hydrocephalus, or vasospasm), CSF drainage may be instituted by ventricular catheter drainage (Hemphill, Greenberg, Anderson, et al., 2015). Mannitol may be administered to reduce ICP. When mannitol is used as a long-term measure to control ICP, dehydration and disturbances in electrolyte balance (hyponatremia or hypernatremia; hypokalemia or hyperkalemia) may occur. Mannitol pulls water out of the brain tissue by osmosis and reduces total body water through diuresis. The patient's fluid balance is monitored continuously and is assessed for signs of dehydration and for rebound elevation of ICP. Other interventions may include elevating the head of the bed to 30 to 45 degrees, avoidance of hyperglycemia and hypoglycemia, sedation, and use of hypertonic saline in a variety of concentrations (e.g., 3%, 7.5%, or 23%) (Hickey & Strayer, 2020).

### **Hypertension**

Hypertension is the most common cause of intracerebral hemorrhage, and its treatment is critical. Specific goals for blood pressure management, which are individualized for each patient, remain controversial. Blood pressure goals may depend on the presence of increased ICP. Guidelines for management of intracerebral hemorrhage recommend early blood pressure lowering (if the systolic blood pressure is between 150 and 220 mm Hg) to a goal systolic of 140 mm Hg, and report that lowering blood pressure can be effective for improving patient outcomes. If systolic blood pressure is above 220 mm Hg, IV continuous infusions of antihypertensive agents may be prescribed (Hemphill et al., 2015). Nicardipine is one agent that may be used as a continuous IV infusion. Labetalol and hydralazine are other examples of

medications that may be given as an IV bolus. During the administration of antihypertensive agents, hemodynamic monitoring is important to detect and avoid a precipitous drop in blood pressure, which can produce brain ischemia. Stool softeners are used to prevent straining, which can elevate the blood pressure.

## **Medical Management**

The goals of medical treatment for hemorrhagic stroke are to allow the brain to recover from the initial insult (bleeding), to prevent or minimize the risk of rebleeding, and to prevent or treat complications. Management may consist of bed rest with sedation to prevent agitation and stress, management of vasospasm, and surgical or medical treatment to prevent rebleeding. If the bleeding is caused by anticoagulation with warfarin, the INR may be corrected with fresh-frozen plasma and vitamin K or prothrombin complex concentrations. Reversing the anticoagulation effect of DOACs requires use of an antidote. Idarucizumab is a medication that was approved for reversing dabigatran and andexanet alfa; it is an antidote for patients treated with rivaroxaban and apixaban; both are factor Xa inhibitors (Cordonnier, Demchuk, Ziai, et al., 2018). If seizures occur, they are treated with anti-epileptic drugs such as levetiracetam or phenytoin. Hyperglycemia should also be treated, and hypoglycemia is avoided. Intermittent pneumatic compression devices should be used starting on the first day of the hospital admission to prevent DVT. If the patient is not mobile after 1 to 4 days from the onset of the hemorrhage and there is documentation of the bleeding ceasing, then DVT prevention medications (low-molecular-weight heparin or unfractionated heparin) may be prescribed (Hemphill et al., 2015). Analgesic agents may be prescribed for head and neck pain. Fever should be treated with acetaminophen and devices such as cooling blankets. After discharge, most patients will require antihypertensive medications to decrease their risk of another intracerebral hemorrhage.

## **Surgical Management**

In many cases, a primary intracerebral hemorrhage is not treated surgically. However, if the patient is showing signs of worsening neurologic examination, increased ICP, or signs of brainstem compression, then surgical evacuation is recommended for the patient with a cerebellar hemorrhage (Hemphill et al., 2015). Surgical evacuation is most frequently accomplished via a craniotomy (see

[Chapter 61](#)). Minimally invasive surgical techniques have also been investigated.

The patient with an intracranial aneurysm is prepared for surgical intervention as soon as their condition is considered stable. Surgical treatment of the patient with an unruptured aneurysm is an option. The goal of surgery is to prevent bleeding in an unruptured aneurysm or further bleeding in an already ruptured aneurysm. This objective is accomplished by isolating the aneurysm from its circulation or by strengthening the arterial wall. An aneurysm may be excluded from the cerebral circulation by means of a ligature or a clip across its neck. If this is not anatomically possible, the aneurysm can be reinforced by wrapping it with some substance to provide support and induce scarring.

Advances in technology have led to the introduction of interventional neuroradiology for the treatment of aneurysms. These techniques are now being used more frequently. Endovascular techniques may be used in select patients to occlude the blood flow from the artery that feeds the aneurysm with coils, liquid embolic agents, or other techniques to occlude the aneurysm itself. If the aneurysm is very large or very wide at its neck, a stent-like device made of a very fine mesh may be used to divert the blood flow away from the aneurysm. The determination of which technique should be used is based on many factors (characteristics of the patient and aneurysm) and is made by experienced endovascular specialists (Connolly et al., 2012; Thompson, Brown, Amin-Hanjani, et al., 2015).

Postoperative complications are rare but can occur. Potential complications include psychological symptoms (disorientation, amnesia, Korsakoff syndrome), intraoperative embolization or artery rupture, postoperative artery occlusion, fluid and electrolyte disturbances (from dysfunction of the neurohypophyseal system), and gastrointestinal bleeding. Complications post procedurally may also include bleeding, hematoma, vascular complications, allergic reactions, and stroke.

## NURSING PROCESS

### The Patient with a Hemorrhagic Stroke



#### Assessment

A complete neurologic assessment is performed initially and includes evaluation for the following:

- Altered level of consciousness
- Sluggish pupillary reaction
- Motor and sensory dysfunction
- Cranial nerve deficits (extraocular eye movements, facial droop, presence of ptosis)
- Speech difficulties and visual disturbance
- Headache and nuchal rigidity or other neurologic deficits

All patients should be monitored in the intensive care unit after an intracerebral or subarachnoid hemorrhage. Neurologic assessment findings are documented and reported as indicated. The frequency of these assessments varies depending on the patient's condition. Any changes in the patient's condition require reassessment and thorough documentation; changes should be reported immediately.



#### Concept Mastery Alert

*Alteration in level of consciousness often is the earliest sign of deterioration in a patient with a hemorrhagic stroke. Because nurses have the most frequent contact with patients, they are in the best position to detect subtle changes. Drowsiness and slight slurring of speech may be early signs that the level of consciousness is deteriorating.*

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Risk for ineffective tissue perfusion associated with bleeding or vasospasm
- Anxiety associated with illness or medically imposed restrictions (aneurysm precautions)

### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Vasospasm
- Seizures
- Hydrocephalus
- Rebleeding
- Hyponatremia

### **Planning and Goals**

The goals for the patient may include improved cerebral tissue perfusion, relief of anxiety, and the absence of complications.

### **Nursing Interventions**

#### **OPTIMIZING CEREBRAL TISSUE PERFUSION**

The patient is closely monitored for neurologic deterioration resulting from recurrent bleeding, increasing ICP, or vasospasm. A neurologic flow record is maintained. The blood pressure, pulse, level of consciousness (an indicator of cerebral perfusion), pupillary responses, and motor function are checked hourly. Respiratory status is monitored, because a reduction in oxygen in areas of the brain with impaired autoregulation increases the chances of a cerebral infarction. Any changes are reported immediately.

**Implementing Aneurysm Precautions.** Cerebral aneurysm precautions are implemented for the patient with a diagnosis of aneurysm (prior to any intervention) to provide a nonstimulating environment, prevent increases in ICP, and prevent further bleeding. The patient is placed on bed rest in a quiet, nonstressful environment, because activity, pain, and anxiety are thought to elevate the blood pressure, which may increase the risk for bleeding. Visitors may be restricted (AANN, 2018).

The head of the bed is elevated 30 to 45 degrees to promote venous drainage and decrease ICP. Any activity that suddenly increases the blood pressure or obstructs venous return is avoided. This includes the Valsalva maneuver, straining, forceful sneezing, pushing oneself up in bed, and acute flexion or rotation of the head and neck (which compromises the jugular veins). Stool softeners and mild laxatives are prescribed. Both prevent constipation, which can cause an increase in ICP. Dim lighting is helpful, because photophobia is common. The purpose of aneurysm precautions should be thoroughly explained to both the patient (if possible) and

family. Intermittent pneumatic compression devices and unfractionated heparin or low-molecular weight heparin are prescribed to decrease the incidence of DVT resulting from immobility. The legs are observed for signs and symptoms of DVT (tenderness, redness, swelling, warmth, and edema), and abnormal findings are reported.

### **RELIEVING ANXIETY**

Sensory stimulation is kept to a minimum for patients on aneurysm precautions. For patients who are awake, alert, and oriented, an explanation of the restrictions helps reduce the patient's sense of isolation. Reality orientation is provided to help maintain orientation.

Keeping the patient well informed of the plan of care provides reassurance and helps minimize anxiety. Appropriate reassurance also helps relieve the patient's fears and anxiety. The family also requires information and support.

### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

**Vasospasm.** The patient is assessed for signs of possible vasospasm: intensified headaches, a decrease in level of responsiveness (confusion, disorientation, lethargy), or evidence of aphasia or partial paralysis. These signs may develop several days after surgery or on the initiation of treatment and must be reported immediately. The calcium channel blocker nimodipine should be given for prevention of vasospasm, and fluid volume expanders in the form of triple-H therapy may be prescribed as well (Connolly et al., 2012).

**Seizures.** Seizure precautions are maintained for every patient who may be at risk for seizure activity. Should a seizure occur, maintaining the airway and preventing injury are the primary goals. Medication therapy is initiated at this time (see [Chapter 61](#)).

**Hydrocephalus.** Blood in the subarachnoid space or ventricles impedes the circulation of CSF, resulting in hydrocephalus. A CT scan that indicates dilated ventricles confirms the diagnosis. Hydrocephalus can occur within the first 24 hours (acute) after subarachnoid hemorrhage or several days (subacute) to several weeks (delayed) later. Symptoms vary according to the time of onset and may be nonspecific. Acute hydrocephalus is characterized by sudden onset of stupor or coma and is managed with a ventriculostomy drain to decrease ICP. Symptoms of subacute and delayed hydrocephalus include gradual onset of drowsiness, behavioral changes, and ataxic

gait. A ventriculoperitoneal shunt is surgically placed to treat chronic hydrocephalus. Changes in patient responsiveness are reported immediately.

**Rebleeding.** The rate of recurrent hemorrhage is approximately 1% to 5% per patient per year after intracerebral hemorrhage (Hemphill et al., 2015). Hypertension is the most serious and modifiable risk factor, which shows the importance of appropriate antihypertensive treatment.

Aneurysm rebleeding is the highest during the first 2 to 12 hours after the initial hemorrhage (Connolly et al., 2012) and is considered a major complication. Symptoms of rebleeding include sudden severe headache, nausea, vomiting, decreased level of consciousness, and neurologic deficit. Rebleeding is confirmed by CT scan. Blood pressure is carefully maintained with medications. The most effective preventive treatment is to secure the aneurysm if the patient is a candidate for surgery or endovascular treatment.

**Hyponatremia.** After subarachnoid hemorrhage, hyponatremia is found in 30% to 50% of patients (Wilson et al., 2019). Hyponatremia has been found to be associated with the onset of vasospasm (Connolly et al., 2012). Laboratory data must be checked frequently, and hyponatremia (defined as a serum sodium concentration less than 135 mEq/L) must be identified as early as possible. The patient's primary provider needs to be notified of a low serum sodium level that has persisted for 24 hours or longer. The patient is then evaluated for syndrome of inappropriate antidiuretic hormone (SIADH) or cerebral salt-wasting syndrome. SIADH is described in [Chapter 10](#). Cerebral salt-wasting syndrome occurs when the kidneys are unable to conserve sodium and volume depletion results. The treatment most often is the use of IV hypertonic 3% saline.

#### PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



Educating Patients About Self-Care. The patient and family are provided with education that will enable them to cooperate with the care and restrictions required during the acute phase of hemorrhagic stroke and to prepare them to return home. Patient and family education includes information about the causes of hemorrhagic stroke and its possible consequences. In addition, the patient and family are informed about the medical treatments that are implemented, including surgical intervention if warranted, and the

importance of interventions taken to prevent and detect complications (i.e., aneurysm precautions, close monitoring of the patient). Depending on the presence and severity of neurologic impairment and other complications resulting from the stroke, the patient may be transferred to a rehabilitation unit or center for additional patient and family education about strategies to regain self-care ability. Education addresses the use of assistive devices or modification of the home environment to help the patient live with the disability. Modifications of the home may be required to provide a safe environment.

**Chart 62-6**



## **HOME CARE CHECKLIST**

## The Patient Recovering from a Stroke

### **At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of the stroke on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State names, dose, side effects, frequency and schedule for all medications.
- State how to contact all members of the treatment team (e.g., health care providers, home care professionals, rehabilitation team, and durable medical equipment and supply vendor).
- State changes in lifestyle (e.g., diet, ADLs, IADLs, activity) necessary for recovery and health maintenance as applicable.
  - Demonstrate environmental modifications and adaptive techniques for accomplishing activities of daily living.
  - Demonstrate home exercises, the use of splints or orthotics, proper positioning, and frequent repositioning.
  - Identify safety measures to prevent falls.
  - Identify holistic interventions for pain management (e.g., positioning, distraction).
  - Describe procedures for maintaining skin integrity.
  - Demonstrate indwelling catheter care, if applicable. Describe a bowel and bladder elimination program as appropriate.
  - Verbalize dietary adjustments (e.g., thickened liquids, pureed diet, small frequent meals) during recovery.
  - Demonstrate swallowing techniques or care of enteral feeding tube.
- Identify psychosocial consequences of stroke (e.g., depression, emotional lability, frustration, fatigue) and appropriate interventions.
- Discuss measures to prevent subsequent strokes.
- Identify potential complications and discuss measures to prevent them (blood clots, aspiration, pneumonia, urinary tract infection, fecal impaction, skin breakdown, contracture).
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.

- Identify resources and other sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families.
- Identify the need for health promotion, disease prevention, and screening activities.
- Identify appropriate recreational or diversional activities.

## **Resources**

See [Chapter 2, Chart 2-6](#) for additional information about durable medical equipment and [Chapter 7, Chart 7-6: Home Care Checklist: Managing Chronic Illness and Disability at Home](#).

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

**Continuing and Transitional Care.** The acute and rehabilitation phase of care focuses on obvious needs, issues, and deficits for the patient with a hemorrhagic stroke. The patient and family are reminded of the importance of following recommendations to prevent further hemorrhagic stroke and keeping follow-up appointments with health care providers for monitoring of risk factors. Referral for home, community-based, or transitional care may be warranted to assess the home environment and the ability of the patient and to ensure that the patient and family are able to manage at home. Home visits provide opportunities to monitor the physical and psychological status of the patient and the ability of the family to cope with any alterations in the patient's status. In addition, the home health nurse reminds the patient and family of the importance of continuing health promotion and screening practices. Chart 62-6 lists education for the patient recovering from a stroke.

## **Evaluation**

Expected patient outcomes may include:

1. Demonstrates stable neurologic status and vital signs and respiratory patterns within normal limits
  - a. Is alert and oriented to time, place, and person
  - b. Demonstrates understandable speech patterns and stable cognitive processes
  - c. Demonstrates usual and equal strength, movement, and sensation of all four extremities

- d. Exhibits deep tendon reflexes and pupillary responses within normal limits
- 2. Exhibits reduced anxiety level
  - a. States rationale for aneurysm precautions
  - b. Exhibits clear thought processes
  - c. Is less restless
  - d. Exhibits absence of physiologic indicators of anxiety (e.g., has vital signs within normal limits; usual respiratory rate; absence of excessive, fast speech)
- 3. Is free of complications
  - a. Exhibits absence of vasospasm
  - b. Exhibits vital signs within normal limits and is without seizures
  - c. Verbalizes understanding of seizure precautions
  - d. Exhibits intact mental, motor, and sensory status
  - e. Reports no visual changes



## Veterans Considerations

Each year, an estimated 15,000 veterans are hospitalized with a stroke-related diagnosis; of these, between 15% and 30% have severe impairment while 40% have some type of functional limitation (Veteran's Administration/Department of Defense, The Management of Stroke Rehabilitation Work Group, 2019). Care for the veteran who has had a stroke takes place in a variety of settings. There are 33 primary stroke centers, 32 limited hours stroke centers, 43 supporting stroke facilities, and 45 acute rehabilitation facilities within the Department of Veterans Affairs. Nurses who work with patients who have had strokes should be aware of these systems as they may need to assist the veteran and their family to facilitate transfer or referral to facilities that can best meet their health care needs.

### CRITICAL THINKING EXERCISES

**1 pq** A 61-year-old woman arrived in the ED and is being evaluated for suspected stroke. Her family reports that they last saw her normal 2 hours ago. What steps can you take to ensure rapid evaluation and treatment? What are your priorities for her care? If she is found to be eligible for t-PA and/or intra-arterial thrombolysis, how will your priorities change?

**2 ipc** You are caring for a patient who experienced an ischemic stroke and now has visual loss (homonymous hemianopsia). What nursing interventions can be implemented at the bedside to assist the patient with this visual deficit? What education can be provided to the patient when they are ready for discharge, and what other disciplines would you collaborate with to ensure that the patient is safely discharged to home? What are the specific roles and responsibilities of those other professionals?

**3 ebp** A 74-year-old male patient has experienced a large left hemispheric ischemic stroke. He has just arrived to the nursing unit from the ED and you are his nurse. Your initial assessment reveals that he has a fever, and a finger stick is performed and shows an elevated blood glucose. Evaluate the strength of the evidence surrounding elevated temperature and elevated blood glucose in acute stroke. Is there any evidence supporting the use of hypothermia in acute stroke?

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference.

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## Resources

- American Stroke Association, a Division of the American Heart Association,  
[www.stroke.org](http://www.stroke.org)
- Brain Attack Coalition, [www.brainattackcoalition.org](http://www.brainattackcoalition.org)
- National Aphasia Association, [www.aphasia.org](http://www.aphasia.org)
- National Institute of Neurological Disorders and Stroke, [www.ninds.nih.gov](http://www.ninds.nih.gov)

# 63 Management of Patients with Neurologic Trauma

## LEARNING OUTCOMES

*On completion of this chapter, the learner will be able to:*

1. Describe the mechanisms of injury, clinical signs and symptoms, diagnostic testing, and treatment options for patients with traumatic brain and spinal cord injury.
2. Use the nursing process as a framework for care of the patient with traumatic brain injury.
3. Identify the population at risk for spinal cord injury and explain the clinical features and management of the patient with neurogenic shock.
4. Discuss the pathophysiology of autonomic dysreflexia and describe the appropriate nursing interventions.
5. Apply the nursing process as a framework for care of the patient with spinal cord injury and the patient with tetraplegia or paraplegia.

## NURSING CONCEPTS

Family  
Fluids and Electrolytes  
Functional Ability  
Infection  
Intracranial Regulation  
Medical Emergencies  
Nutrition  
Sensory Perception

## GLOSSARY

**autonomic dysreflexia:** a life-threatening emergency in patients with spinal cord injury that causes a hypertensive emergency (*synonym: autonomic hyperreflexia*)

**complete spinal cord lesion:** a condition that involves total loss of sensation and voluntary muscle control below the lesion

**concussion:** a temporary loss of neurologic function with no apparent structural damage to the brain

**contusion:** bruising of the brain surface

**incomplete spinal cord lesion:** a condition in which there is preservation of the sensory or motor fibers, or both, below the lesion

**neurogenic bladder:** bladder dysfunction that results from a disorder or dysfunction of the nervous system; may result in either urinary retention or bladder overactivity

**paraplegia:** paralysis of the lower extremities with dysfunction of the bowel and bladder from a lesion in the thoracic, lumbar, or sacral region of the spinal cord

**primary injury:** initial damage to the brain that results from the traumatic event

**secondary injury:** an insult to the brain subsequent to the original traumatic event

**spinal cord injury (SCI):** an injury to the spinal cord, vertebral column, supporting soft tissue, or intervertebral discs caused by trauma

**tetraplegia:** varying degrees of paralysis of both arms and legs, with dysfunction of bowel and bladder from a lesion of the cervical segments of the spinal cord; formerly called *quadriplegia*

**transection:** severing of the spinal cord; transection can be complete (all the way through the cord) or incomplete (partially through)

**traumatic brain injury:** an injury to the skull or brain that is severe enough to interfere with normal functioning (*synonym: craniocerebral trauma*)

**traumatic brain injury, closed (blunt):** occurs when the head accelerates and then rapidly decelerates or collides with another object and brain tissue is damaged, but there is no opening through the skull and dura

**traumatic brain injury, open (penetrating):** occurs when an object penetrates the skull, enters the brain, and damages the soft brain tissue in its path (penetrating injury), or when blunt trauma to the head is so severe that it opens the scalp, skull, and dura to expose the brain

Trauma involving the central nervous system can be life-threatening. Even if not life-threatening, brain and spinal cord injury (SCI) may result in major physical and psychological dysfunction and can alter the patient's life completely. Neurologic trauma affects the patient, the family, the health care system, and the society as a whole because of its major sequelae and the costs of acute and long-term care of patients with trauma to the brain and spinal cord.

## Head Injuries

Head injury is a broad classification that encompasses any damage to the head as a result of trauma. A head injury does not necessarily mean a brain injury is present. **Traumatic brain injury** (TBI) or craniocerebral trauma describes an injury that is the result of an external force and is of sufficient magnitude to interfere with daily life and prompts the seeking of treatment.

The Centers for Disease Control and Prevention (CDC) estimates that there are 2.9 million emergency department (ED) visits in the United States each year, the majority of which are for a mild TBI (CDC, 2019). As a result of TBI, approximately 56,800 people die (contributing to about 30% of all injury-related deaths), 288,000 are hospitalized, and 80,000 to 90,000 will have long-term disability (CDC, 2019; Hickey & Strayer, 2020). Approximately 78% of patients are treated in the ED and released (Williamson & Rajajee, 2018). The most common causes of TBIs are falls (48%), motor vehicle crashes (14%), being struck by objects (15%), and assaults (10%). Children up to 4 years of age, adolescents 15 to 19 years, and adults 65 years and older are most likely to sustain a TBI. In every age group, TBI rates are higher for males than for females (Hickey & Strayer, 2020). An estimated 5.3 million people are living with a TBI-related disability, producing an annual economic impact of approximately \$76.5 billion due to medical expenses and the cost of lost productivity (CDC, 2019). The best approach to head injury is prevention (see [Chart 63-1](#)).

Chart 63-1



HEALTH PROMOTION

## Preventing Head and Spinal Cord Injuries

- Advise drivers to obey traffic laws and to avoid speeding or driving when under the influence of drugs or alcohol.
- Advise all drivers and passengers to wear seat belts and shoulder harnesses. Children younger than 12 years should use an age/size-appropriate system in the back seat.
- Caution passengers against riding in the back of pickup trucks.
- Advise motorcyclists, scooter riders, bicyclists, skateboarders, and roller skaters to wear helmets.
- Promote educational programs that are directed toward violence and suicide prevention in the community.
- Provide water safety instruction.
- Educate patients about steps that can be taken to prevent falls, particularly in older adults.
- Advise athletes to use protective devices. Recommend that coaches be educated in proper coaching techniques.
- Advise owners of firearms to keep them locked in a secure area where children cannot access them.

## Pathophysiology

Damage to the brain from traumatic injury takes two forms: primary injury and secondary injury. **Primary injury** is defined as the consequence of direct contact to the head/brain during the instant of initial injury, causing extracranial focal injuries (e.g., contusions, lacerations, external hematomas, and skull fractures), as well as possible focal brain injuries from sudden movement of the brain within the cranial vault (e.g., subdural hematomas [SDHs], concussion, diffuse axonal injury [DAI]). The greatest opportunity for decreasing TBI is the implementation of prevention strategies (see [Chart 63-1](#)).

**Secondary injury** evolves over the ensuing hours and days after the initial injury and results from inadequate delivery of glucose and oxygen to the cells. Identification, prevention, and treatment of secondary injury are the main foci of early management of severe TBI. Contributors to this process include intracranial pathologic processes such as intracranial hemorrhage, cerebral edema, intracranial hypertension, hyperemia, seizures, and vasospasm (Hickey & Strayer, 2020; Kaur & Sharma, 2018). Systemic effects from hypotension, hyperthermia, hypoxia, hypercarbia, infection, electrolyte imbalances, and anemia can also be factors which add to the complex biochemical, metabolic, and inflammatory changes that further compromise an injured brain (Hickey & Strayer, 2020).

The Monro–Kellie hypothesis, also known as the Monro–Kellie doctrine, explains the dynamic equilibrium of cranial contents. The cranial vault contains three main components: brain, blood, and cerebrospinal fluid (CSF). According to

the Monro–Kellie hypothesis, the cranial vault is a closed system, and if one of the three components increases in volume, at least one of the other two must decrease in volume or the pressure will increase. Any bleeding or swelling within the skull increases the volume of contents within the skull and therefore causes increased intracranial pressure (ICP) (see [Chapter 61](#)). If the pressure increases enough, it can cause displacement of the brain through or against the rigid structures of the skull. This causes restriction of blood flow to the brain, decreasing oxygen delivery and waste removal. Cells within the brain become anoxic and cannot metabolize properly, producing ischemia, infarction, irreversible brain damage, and eventually brain death (see [Fig. 63-1](#)).

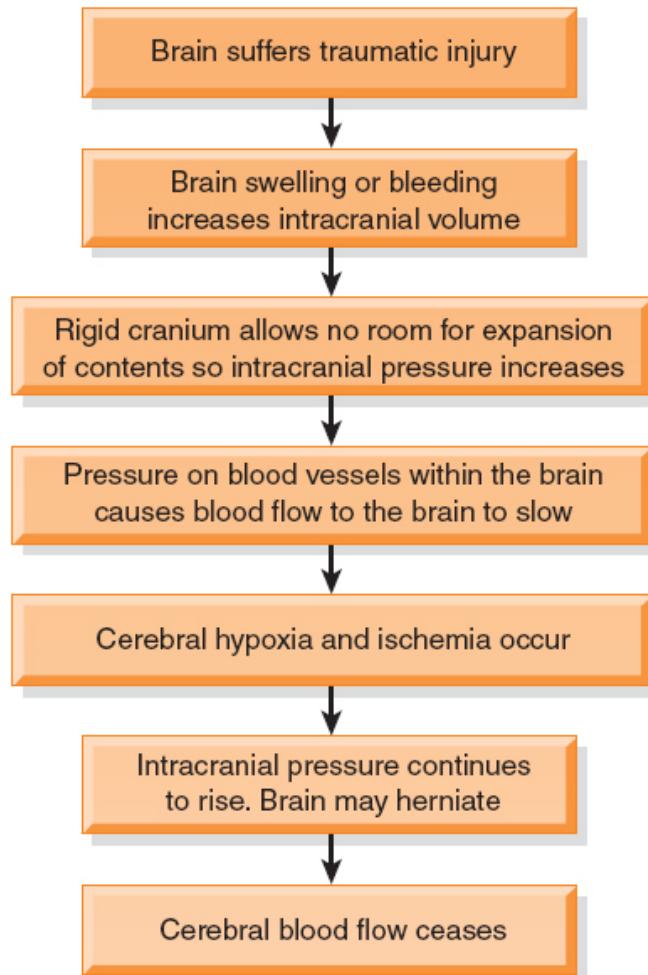
## Scalp Injury

Isolated scalp trauma is generally classified as a minor injury. Because its many blood vessels constrict poorly, the scalp bleeds profusely when injured. Trauma may result in an abrasion (brush wound), contusion, laceration, or subgaleal hematoma (hematoma beneath the layers of tissue of the scalp) (Hickey & Strayer, 2020). A large avulsion (tearing away) of the scalp may be potentially life-threatening and is a true emergency. Diagnosis of a scalp injury is based on physical examination, inspection, and palpation. Scalp wounds are potential portals of entry for organisms that cause intracranial infections. Therefore, the area is irrigated before the laceration is sutured to remove foreign material and to reduce the risk for infection (Hollander, 2019). Subgaleal hematomas usually reabsorb and do not require any specific treatment.

## Skull Fractures

A skull fracture is a break in the continuity of the skull caused by forceful trauma. It may occur with or without damage to the brain. Skull fractures are classified by type and location. Types include linear, comminuted, and depressed skull fractures, whereas location fractures include frontal, temporal, and basal skull fractures. A simple (linear) fracture is a break in the continuity of the bone. A comminuted skull fracture refers to a splintered or multiple fracture line. Depressed skull fractures occur when the bones of the skull are forcefully displaced downward and can vary from a slight depression to bones of the skull being splintered and embedded within brain tissue. A fracture of the base of the skull is referred to as a basal skull fracture (Hickey & Strayer, 2020). A fracture may be open, indicating a scalp laceration or tear in the dura (e.g., from a bullet or an ice pick), or closed, in which case the dura is intact.

## Physiology/Pathophysiology



**Figure 63-1 •** Pathophysiology of traumatic brain injury.

## Clinical Manifestations

Symptoms, apart from those of the local injury, depend on the severity and the anatomic location of the underlying brain injury. Persistent, localized pain usually suggests that a fracture is present. Fractures of the cranial vault may or may not produce swelling in the region of the fracture. Fractures of the base of the skull tend to traverse the paranasal sinus of the frontal bone or the middle ear located in the temporal bone. Therefore, they frequently produce hemorrhage from the nose, pharynx, or ears, and blood may appear under the conjunctiva. An area of ecchymosis (bruising) may be seen over the mastoid (Battle sign). Basal skull fractures are suspected when CSF escapes from the ears (CSF otorrhea) and the nose (CSF rhinorrhea). Drainage of CSF is a serious problem, because meningeal infection can occur if organisms gain access to the cranial contents via the nose, ear, or sinus through a tear in the dura.



## Veterans Considerations

Military personnel in combat roles are at increased risk for TBI, with a prevalence rate ranging from 15% to 23% (Turgoose & Murphy, 2018). A common cause of TBI in a veteran is a combat-related blast injury from various forms of improvised explosive devices. Unlike TBI in the civilian population, which causes a primary and secondary injury, TBI in the military population causes four levels of injury (Chapman & Diaz-Arrastia, 2014). The primary injury is due to the atmospheric overpressure followed by underpressure or vacuum. The secondary injury occurs when objects are placed in motion (shrapnel) by the blast, hitting the service member. Tertiary injury occurs when the service member is thrown by the blast and hits their head against the ground, a wall, or other solid surface. Quaternary injury involves other injuries from the blast, such as burns and crush injuries. At this time, there is no evidence to suggest significant differences between a blast injury and a blunt brain injury. Magnetic resonance imaging (MRI) studies do not indicate any microstructural differences. It appears there are no cognitive differences either. Treatment options for a veteran with TBI are the same as those for a civilian with TBI; however, veterans can have complex needs, especially those with multiple injuries.

## Assessment and Diagnostic Findings

A computed tomography (CT) scan can be used to diagnose a skull fracture. The ease with which a diagnosis of skull fracture is made depends on the site of the fracture. If a fracture is found on CT scan, there is always the question of associated brain injury, and an MRI scan provides better resolution and clearer pictures of the injured area (Hickey & Strayer, 2020).



## Gerontologic Considerations

Older patients with head injuries differ from those who are younger in terms of etiology of injury, higher mortality rates, longer lengths of hospital stay, and worse functional outcomes (Thompson, Rivara, & Wang, 2020) (see the Nursing Research Profile in [Chart 63-2](#)). Neurologic assessment can be challenging, as the older adult patient with a TBI can have hearing or visual deficits or preexisting dementia or cognitive issues, making establishment of a neurologic baseline difficult. The most common causes of injury in older adult patients are falls and motor vehicle crashes. Approximately 81% of all TBIs among adults aged 65 years and older result from falls (CDC, 2019). Physiologic changes related to aging may place the older adult at increased risk for injury, alter the type and severity of injury that occurs, or lead to complications.

Several factors place older adults at increased risk for hematomas. Brain weight decreases, the dura becomes more adherent to the skull, and reaction times slow with increasing age (Battaglini, Gentile, Luchetti, et al., 2019). Also, many

older adults take aspirin and anticoagulant agents as part of routine management of chronic conditions.

## **Medical Management**

Nondepressed skull fractures generally do not require surgical treatment; however, close observation of the patient is essential. Nursing personnel may observe the patient in the hospital, but if no underlying brain injury is present, the patient may be allowed to return home. If the patient is discharged home, specific instructions must be given to the family (see later discussion of concussion).

**Chart 63-2**



### **NURSING RESEARCH PROFILE**

## **Head Injury in Older Adults**

Thompson, H. J., Rivara, F. P., & Wang, J. (2020). Symptoms, function, and outcomes in the first year after mild-moderate traumatic brain injury. *Journal of Neuroscience Nursing*, 52(2), 46–52.

### **Purpose**

Older adults have higher rates of emergency department (ED) visits, hospitalization, and death for traumatic brain injury (TBI) compared to younger adults. The purpose of this study was to describe and compare the injury trajectory at 1 year for older and younger adults who had a TBI.

### **Design**

This was a prospective longitudinal cohort study of 33 adults who had a mild to moderate TBI. Participants were recruited in the ED and followed for 1 year. Data were collected on symptoms, function using the Glasgow Outcome Scale-Extended Functional Status Examination, and health-related quality of life (HRQOL) for 1 week, then at 1, 3, 6, and 12 months after the injury.

### **Findings**

The total number of symptoms did not differ when younger and older adults were compared, but the symptoms clusters were different. Older adults reported more physical symptoms such as fatigue, balance and coordination problems, and being bothered by noise. Younger adults reported more psychological symptoms such as anxiety. Function measured on the Glasgow Outcome Scale-Extended Functional Status Examination was lower in older adults at 1 year post injury compared to younger adults. Physical HRQOL was lower in older adults consistently over the year compared to younger adults. In contrast, mental HRQOL was higher in older adults.

### **Nursing Implications**

Nurses working with older adults, who have had a TBI should know that they report different symptoms clusters compared to younger adults. Nursing interventions with older adults who have had a TBI should focus on balance, coordination, as well as energy conservation measures to minimize fatigue and other measures to reduce environmental noises.

Depressed skull fractures usually require surgery with elevation of the skull and débridement, usually within 24 hours of injury. Skull fractures can be a combination of open, compound, closed, or simple. Associated injuries include concurrent scalp laceration, dural tears, and brain injury directly below the fracture from compression of the tissue below the bony injury and from lacerations produced by the bony fragments (Hickey & Strayer, 2020).

## **Brain Injury**

The most important consideration in any head injury is whether the brain is injured. Even seemingly minor injury can cause significant brain damage secondary to obstructed blood flow and decreased tissue perfusion. The brain cannot store oxygen or glucose to any significant degree. Because the cerebral cells need an uninterrupted blood supply to obtain these nutrients, irreversible brain damage and cell death occur if the blood supply is interrupted for even a few minutes. A **traumatic brain injury, closed (blunt)** occurs when the head accelerates and then rapidly decelerates or collides with another object (e.g., a wall, the dashboard of a car) and brain tissue is damaged but there is no opening through the skull and dura. A **traumatic brain injury, open (penetrating)** occurs when an object penetrates the skull, enters the brain, and damages the soft brain tissue in its path or when blunt trauma to the head is so severe that it opens the scalp, skull, and dura to expose the brain.

## Types of Brain Injury

Injuries to the brain can be focal or diffuse. Focal injuries include contusions and several types of hematomas. Concussions and DAI are the major diffuse injuries (Hickey & Strayer, 2020).

### Contusion

In cerebral **contusion**, the brain is bruised and damaged in a specific area because of severe acceleration-deceleration force or blunt trauma. The impact of the brain against the skull leads to a contusion. Clinical manifestations of a contusion are dependent upon size, location, and the extent of surrounding cerebral edema. Although a contusion may occur in any area of the brain, most are usually located in the anterior portions of the frontal and temporal lobes, around the sylvian fissure, and at the orbital areas; less commonly, contusions are located at the parietal and occipital areas.

Contusions can be characterized by loss of consciousness associated with stupor and confusion. The effects of injury, particularly hemorrhage and edema, peak after about 18 to 36 hours. These effects, which can cause secondary effects resulting in an increased ICP and possible herniation syndromes, are most pronounced in temporal lobe contusions. Patients are most often managed medically with interventions directed toward prevention of additional insults. Deep contusions are more often associated with hemorrhage and destruction of the reticular activating fibers, altering arousal (Hickey & Strayer, 2020).

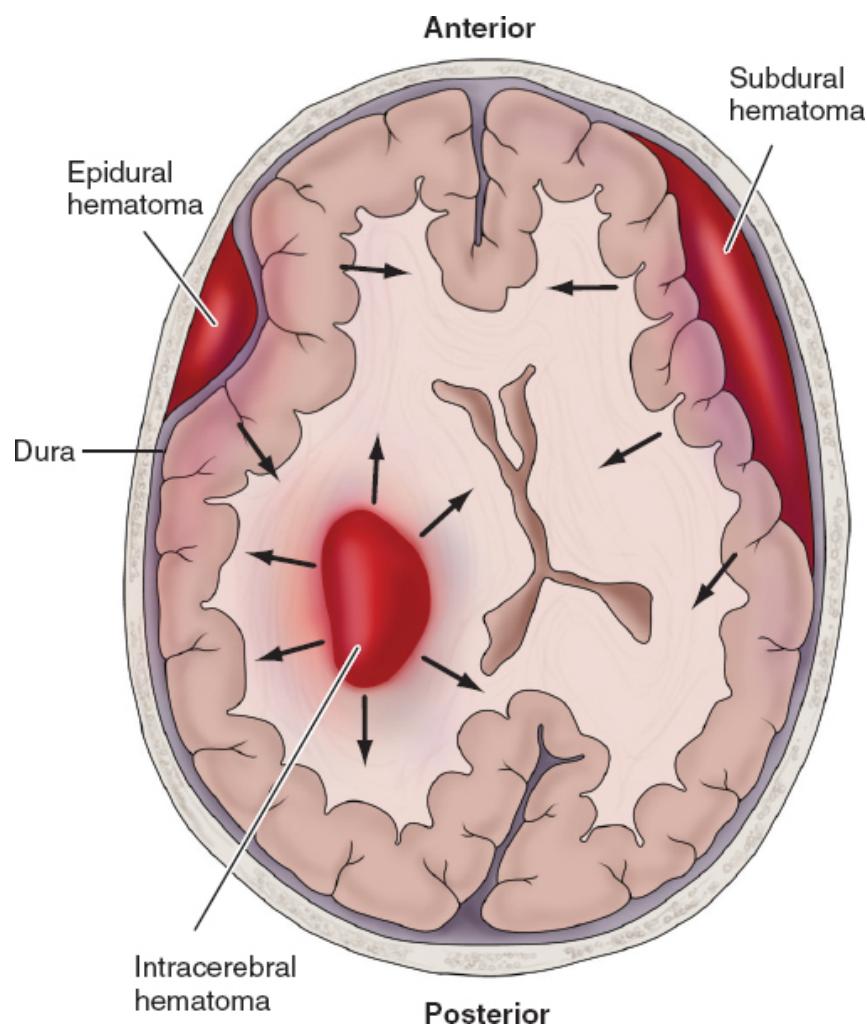
### Intracranial Hemorrhage

Hematomas are collections of blood in the brain that may be epidural (above the dura), subdural (below the dura), or intracerebral (within the brain) (see Fig. 63-2). Major symptoms are frequently delayed until the hematoma is large enough to cause distortion of the brain and increased ICP. The signs and symptoms of cerebral ischemia resulting from compression by a hematoma are variable and

depend on the speed with which vital areas are affected and the area that is injured. A rapidly developing hematoma, even if small, may be fatal, whereas a larger but slowly developing one may allow compensation for increases in ICP.

### Epidural Hematoma

After a head injury, blood may collect in the epidural (extradural) space between the skull and the dura mater. This can result from a skull fracture that causes a rupture or laceration of the middle meningeal artery, the artery that runs between the dura and the skull inferior to a thin portion of temporal bone. Hemorrhage from this artery causes rapid pressure in the brain. Epidural hematomas (EDHs) account for approximately 2.7% to 4% of traumatic head injuries (Hickey & Strayer, 2020).



**Figure 63-2 •** Location of epidural, subdural, and intracerebral hematomas.

Symptoms are caused by the expanding hematoma. EDHs are often characterized by a brief loss of consciousness, followed by a lucid interval in

which the patient is awake and conversant. During this lucid interval, compensation for the expanding hematoma takes place by rapid absorption of CSF and decreased intravascular volume, both of which help to maintain the ICP within normal limits. When these mechanisms can no longer compensate, even a small increase in the volume of the blood clot produces a marked elevation in ICP. The patient then becomes increasingly restless, agitated, and confused as the condition progresses to coma. Then, often suddenly, signs of herniation appear (usually deterioration of consciousness and signs of focal neurologic deficits, such as dilation and fixation of a pupil or paralysis of an extremity), and the patient's condition deteriorates rapidly. The most common type of herniation syndrome associated with an EDH is uncal herniation causing pressure on the midbrain (Hickey & Strayer, 2020).

An EDH is considered an extreme emergency; marked neurologic deficit or even respiratory arrest can occur within minutes. Treatment consists of making openings through the skull (burr holes; see [Chapter 61, Fig. 61-8](#)) to decrease ICP emergently, remove the clot, and control the bleeding. A craniotomy may be required to remove the clot and control the bleeding. A drain is usually inserted after creation of burr holes or a craniotomy to prevent reaccumulation of blood.

### **Subdural Hematoma (SDH)**

An SDH is a collection of blood between the dura and the brain, a space normally occupied by a thin cushion of fluid. The most common cause is trauma, but it can also occur as a result of coagulopathies or rupture of an aneurysm. An SDH is more frequently venous in origin and is caused by the rupture of small vessels that bridge the subdural space (Vacca & Argento, 2018). SDHs may be acute or chronic depending on the size of the involved vessel and the amount of bleeding in the CT scan.

#### **Acute SDH**

An acute SDH is usually caused by some kind of head injury, typically a fall. Signs and symptoms include changes in the level of consciousness (LOC), pupillary signs, and hemiparesis. There may be minor or even no symptoms with small collections of blood. Coma, increasing blood pressure, decreasing heart rate, and slowing respiratory rate are all signs of a rapidly expanding mass requiring immediate intervention.

If the patient can be transported rapidly to the hospital, an immediate craniotomy is performed to open the dura, allowing the subdural clot to be evacuated. Successful outcome also depends on the control of ICP and careful monitoring of respiratory function (see [Chapter 61](#)). The mortality rate for patients with acute SDH is high because of associated brain damage (Hickey & Strayer, 2020).

#### **Chronic SDH**

A chronic SDH can develop from seemingly minor head injuries and is seen most frequently in older adults who are prone to this type of head injury due to brain atrophy, which is a consequence of the aging process (Vacca & Argento, 2018).

Seemingly minor head trauma may produce enough impact to shift the brain contents abnormally. The time between injury and onset of symptoms can be lengthy (e.g., 3 weeks to months), so the actual injury may be forgotten.

A chronic SDH can resemble other conditions—for example, it may be mistaken for a stroke. The bleeding is less profuse, but compression of the intracranial contents still occurs. The blood within the brain changes in character in 2 to 4 days, becoming thicker and darker. In a few weeks, the clot breaks down and has the color and consistency of motor oil. Eventually, calcification or ossification of the clot takes place. The brain adapts to this foreign body invasion, and the clinical signs and symptoms fluctuate. Symptoms include severe headache, which tends to come and go; alternating focal neurologic signs; personality changes; mental deterioration; and focal seizures (Vacca & Argento, 2018).

The treatment for a chronic SDH consists of surgical evaluation for evacuation of the clot. Consideration must be made for reversal of coagulopathies and iatrogenic anticoagulation (Vacca & Argento, 2018). The operative procedure may be carried out through multiple burr holes, or a craniotomy may be performed for a sizable subdural mass that cannot be suctioned or drained through burr holes.

### Intracerebral Hemorrhage and Hematoma

Intracerebral hemorrhage is bleeding into the parenchyma of the brain. It is commonly seen in head injuries when force is exerted to the head over a small area (e.g., missile injuries, bullet wounds, stab injuries). These hemorrhages within the brain may also result from the following:

- Systemic hypertension, which causes degeneration and rupture of a vessel
- Rupture of an aneurysm
- Vascular anomalies
- Intracranial tumors
- Bleeding disorders such as leukemia, hemophilia, aplastic anemia, and thrombocytopenia
- Complications of anticoagulant therapy

Nontraumatic causes of intracerebral hemorrhage are discussed in [Chapter 62](#).

The onset may be insidious, beginning with the development of neurologic deficits, followed by headache. Management includes supportive care; control of ICP; and careful administration of fluids, electrolytes, and antihypertensive medications. Surgical intervention by craniotomy or craniectomy permits removal of the blood clot and control of hemorrhage but may not be possible because of the inaccessible location of the bleeding or the lack of a clearly circumscribed area of blood that can be removed.

### Concussion

**A concussion** is a temporary loss of neurologic function with no apparent structural damage to the brain. Of the 1.7 million TBIs that occur in the United States each year, it is estimated that approximately 80% of them are concussions, also referred to as “mild TBI” (CDC, 2019). The mechanism of injury is usually blunt trauma from an acceleration–deceleration force, a direct blow, or a blast injury. If brain tissue in the frontal lobe is affected, the patient may exhibit bizarre irrational behavior, whereas involvement of the temporal lobe can produce temporary amnesia or disorientation.

The duration of mental status abnormalities is an indicator of the grade of the concussion. The patient is discharged from the hospital or ED once they return to baseline after a concussion. Monitoring includes observing the patient for a decrease in LOC, worsening headache, dizziness, seizures, abnormal pupil response, vomiting, irritability, slurred speech, and numbness or weakness in the arms or legs (Silverberg, Iaccarino, Panenka, et al., 2020). The occurrence of these symptoms is a red flag indicating the need for further intervention. Recovery may appear complete, but long-term sequelae are possible and repeat injuries are common.

Repeated concussive incidents can lead to a syndrome known as chronic traumatic encephalopathy. This syndrome has been recognized in those participating in contact sports such as football and boxing. The presentation is similar to Alzheimer’s disease, characterized by personality changes, memory impairment, and speech and gait disturbances. Imaging findings show gross cerebral, particularly temporal lobe, atrophy (Hickey & Strayer, 2020; Turk & Budson, 2019).

### **Diffuse Axonal Injury (DAI)**

DAI results from widespread shearing and rotational forces that produce damage throughout the brain—to axons in the cerebral hemispheres, corpus callosum, and brain stem. The injured area may be diffuse with no identifiable focal lesion. DAI is associated with prolonged traumatic coma; it is more serious and is associated with a poorer prognosis than a focal lesion. The patient with DAI in severe head trauma experiences no lucid interval, immediate coma, decorticate and decerebrate posturing (see [Chapter 61, Fig. 61-1](#)), and global cerebral edema. Diagnosis is made by clinical signs in conjunction with a CT or MRI scan (Schweitzer, Niogi, Whitlow, et al., 2019). Recovery depends on the severity of the axonal injury.

## **Medical Management**

Assessment and diagnosis of the extent of injury are accomplished by the initial physical and neurologic examinations. CT and MRI scans are the main neuroimaging diagnostic tools and are useful in evaluating the brain structure (Schweitzer et al., 2019). Positron emission tomography (PET) is available in some trauma centers for assessing brain function.

Any patient with a head injury is presumed to have a cervical spine injury until proven otherwise. The patient is transported from the scene of the injury on a board with the head and neck maintained in alignment with the axis of the body. A cervical collar should be applied and maintained until cervical spine x-rays have been obtained and the absence of cervical SCI documented.

All therapy is directed toward preserving brain homeostasis and preventing secondary brain injury, which is injury to the brain that occurs after the original traumatic event. Common causes of secondary injury are cerebral edema, hypotension, and respiratory depression that may lead to hypoxemia and electrolyte imbalance. Treatments to prevent secondary injury include stabilization of cardiovascular and respiratory function to maintain adequate cerebral perfusion, control of hemorrhage and hypovolemia, and maintenance of optimal blood gas values.

Acute TBI treatment guidelines have been developed by the Brain Trauma Foundation. Adherence to these treatment guidelines may subsequently improve patient care and outcomes (Saherwala, Bader, Stutzman, et al., 2018).



### Treatment of Increased Intracranial Pressure

As the damaged brain swells with edema or as blood collects within the brain, an increase in ICP occurs; this requires aggressive treatment (see [Chapter 61](#) for a discussion on the relationship between ICP and cerebral perfusion pressure [CPP]). If the ICP remains elevated, it can decrease the CPP. Initial management is based on preventing secondary injury and maintaining adequate cerebral oxygenation (Sacco & Delibert, 2018).

Surgery is required for evacuation of blood clots, débridement and elevation of depressed fractures of the skull, and suture of severe scalp lacerations. ICP is monitored closely; if increased, it is managed by maintaining adequate oxygenation, elevating the head of the bed, and maintaining normal blood volume (McCafferty, Neal, Marshall, et al., 2018). Devices to monitor ICP or drain CSF can be inserted during surgery or at the bedside using aseptic technique. The patient is cared for in the intensive care unit (ICU), where expert nursing care and medical treatment are readily available.

### Supportive Measures

Treatment also includes ventilatory support, seizure prevention, fluid and electrolyte maintenance, nutritional support, and management of pain and anxiety. Patients who are comatose are intubated and mechanically ventilated to ensure adequate oxygenation and protect the airway.

Because seizures can occur after head injury and can cause secondary brain damage from hypoxia, anticonvulsant agents may be given. If the patient is very agitated, benzodiazepines are the most commonly used sedative agents which do not affect cerebral blood flow or ICP. Lorazepam and midazolam are frequently

used but have active metabolites that may cause prolonged sedation, making it difficult to conduct a neurologic assessment. Propofol, a sedative-hypnotic agent that is supplied in an intralipid emulsion for intravenous (IV) use, is the sedative of choice. It is an ultrashort-acting, rapid-onset drug with elimination half-life of less than an hour. It has a major advantage of being titratable to its desired clinical effect but still provides the opportunity for an accurate neurologic assessment (Hickey & Strayer, 2020). A nasogastric tube may be inserted, because reduced gastric motility and reverse peristalsis are associated with head injury, making regurgitation and aspiration common in the first few hours.

Chart 63-3



### ETHICAL DILEMMA

## How Can Advocacy Be Assured for a Patient Who Is Undocumented and Incapacitated?

### Case Scenario

J.S. is a 24-year-old man who is undocumented and undomiciled. He was brought to the hospital by ambulance after falling from a 20-foot-high scaffold at a construction site 2 days ago. You are J.S.'s nurse in the intensive care unit (ICU). According to J.S.'s assigned medical social worker, a coworker on the work site where J.S. fell followed him to the hospital post injury. Information gathered from the coworker included that J.S. was working as a day laborer and was sending money to his parents in Honduras. J.S. reportedly speaks no English and only some Spanish; his primary language is native Honduran Moskitu. Despite aggressive interventions, including emergent craniotomy, ventriculostomy placement, and endotracheal intubation and mechanical ventilation, J.S. has remained unresponsive to all stimuli since admission to the ICU, with a current Glasgow Coma Scale score of 3. The consulting neurologist has opined that J.S. will not recover from his traumatic brain injury, and that he will either succumb to his injuries or remain in a persistent vegetative state (see [Chapter 61](#)). The state where you work has a provision that two physicians may decide to withdraw life-sustaining therapy for patients who are unbefriended (i.e., without next of kin/surrogates) and decisionally incapacitated such as J.S. The attending physician intensivist assigned to J.S. tells you during her rounds that she intends to consult with another intensivist colleague so that J.S. is extubated, and wants you to prepare to assist them. You ask the intensivist if attempts should be made to find J.S.'s parents to get their permission to withdraw treatment. The intensivist says to you "How are we supposed to find them? And even if we do locate them, they do not speak English, and maybe they do not understand Spanish, either. How can we tell them what has happened and get them on board? For heaven's sake, we are not doing this man any favors by letting him linger like this!"

### Discussion

People from socioeconomically disadvantaged groups and those who are racial and ethnic minorities are at greater risk for morbidity and mortality than those who are socioeconomically secure and White. No group is at greater risk for suffering health disparities than those who are undocumented and undomiciled. Patients who are undocumented will typically forego seeking treatment for anything other than a dire health emergency, as they typically are uninsured, not eligible for assistance programs, and risk deportation by seeing a health care provider.

### Analysis

- Describe the ethical principles that are in conflict in this case (see [Chapter 1, Chart 1-7](#)). Assume that the intensivist believes she is advocating for what is best for J.S. by withdrawing life support. What dangers might be inherent in this type of paternalistic attitude (i.e., *I know what is best for the patient*)?

- How might it be possible to ascertain what J.S.'s wishes would be if he were not incapacitated? Is trying to determine this futile and needlessly prolonging his suffering?
- What resources might you mobilize to be of assistance to you, to J.S., and to J.S.'s parents in Honduras? Do J.S.'s parents have the right to know what has happened to their son? What if his parents are found, and want J.S. to continue life-sustaining treatments? Can they legitimately make that decision on his behalf?

## References

- Fins, J. J., & Real de Asúa, D. (2019). North of home: Obligations to families of undocumented patients. *Hastings Center Report*, 49(1), 12–14.
- Radtke, K. & Matzo, M. (2017). Liberty and justice for all: When an unauthorized immigrant suffers a brain injury, who decides when treatment is withdrawn? *American Journal of Nursing*, 117(11), 52–56.

## Resources

See [Chapter 1, Chart 1-10](#) for Steps of an Ethical Analysis and Ethics Resources.

## Brain Death

When a patient has sustained a severe head injury incompatible with life, the patient is a potential organ donor. The nurse may assist in the clinical examination for determination of brain death and in the process of organ procurement. The three cardinal signs of brain death on clinical examination are coma, the absence of brain stem reflexes, and apnea. Adjunctive tests, such as cerebral blood flow studies, electroencephalogram (EEG), transcranial Doppler, and brain stem auditory-evoked potential, are often used to confirm brain death (Hickey & Strayer, 2020). The health care team provides information to the family and assists them with the decision-making process about end-of-life care (see the section Supporting Family Coping and [Chart 63-3](#)).

## NURSING PROCESS

### The Patient with a Traumatic Brain Injury

#### Assessment

Depending on the patient's neurologic status, the nurse may elicit information from the patient, from the family, or from witnesses or emergency rescue personnel. Although all usual baseline data may not be collected initially, the immediate health history should include the following questions:

- When did the injury occur?
- What caused the injury? A high-velocity missile? An object striking the head? A fall?
- What was the direction and force of the blow?

A history of unconsciousness or amnesia after a head injury indicates a significant degree of brain damage, and changes that occur minutes to hours after the initial injury can reflect recovery or indicate the development of secondary brain damage. The nurse should determine if there was a loss of consciousness, the duration of the unconscious period, and if the patient could be aroused.

In addition to asking questions that establish the nature of the injury and the patient's condition immediately after the injury, the nurse examines the patient thoroughly. This assessment includes determining the patient's LOC using the Glasgow Coma Scale (GCS) and assessing the patient's response to tactile stimuli (if unconscious), pupillary response to light, corneal and gag reflexes, and motor function (Teasdale & Jennett, 1974). The GCS (see [Chart 63-4](#)) is based on the three criteria of eye opening, verbal responses, and motor responses to verbal commands or painful stimuli. It is particularly useful for monitoring changes during the acute phase, the first few days after a head injury. It does not take the place of an indepth neurologic assessment.

Detailed assessments are made initially and at frequent intervals throughout the acute phase of care (Hickey & Strayer, 2020). Monitoring of ICP is crucial to decision making for patients with neurologic injuries, yet research findings indicate that proper training and a standard guideline is necessary to correctly document ICP (Liu, Griffith, Jang, et al., 2020) (see the Nursing Research Profile in [Chapter 61](#), [Chart 61-2](#)). Baseline and ongoing assessments are critical in nursing assessment of the patient with brain injury, whose condition can worsen dramatically and irrevocably if subtle signs are overlooked (Sacco & Davis, 2019; Urden, Stacy, & Lough, 2018). More information on assessment is provided in the following sections and in Figure 63-3 and [Table 63-1](#).

Chart 63-4



ASSESSMENT

## Glasgow Coma Scale

The Glasgow Coma Scale is a tool for assessing a patient's response to stimuli. Scores range from 3 (deep coma) to 15 (normal).

Eye-opening response	Spontaneous	4
	To voice	3
	To pain	2
	None	1
Best verbal response	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Best motor response	Obeys command	6
	Localizes pain	5
	Withdraws	4
	Flexion	3
	Extension	2
	None	1
Total		3 to 15

Adapted from Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet*, 2(7872), 81–84. Used with permission.



**Figure 63-3 •** Assessment parameters for the patient with a head injury include eye opening and responsiveness (**A**), vital signs (**B**), and motor response reflected in hand strength or response to painful stimulus (**C**, **D**). Photos by B. Proud.

**TABLE 63-1**



Multisystem Assessment Measures for the Patient  
with Traumatic Brain Injury

System-Specific Considerations	Assessment Data
<b>Neurologic System</b>	
<ul style="list-style-type: none"> <li>• Severe TBI results in unconsciousness and alters many neurologic functions.</li> <li>• All body functions must be supported.</li> <li>• Increased ICP and herniation syndromes are life-threatening.</li> <li>• Measures are instituted to control elevated ICP.</li> </ul>	<ul style="list-style-type: none"> <li>• Assessment of neurologic status</li> <li>• Assessment for signs and symptoms of ICP elevation</li> <li>• Calculation of cerebral perfusion pressure if ICP monitor is in place</li> <li>• Monitoring of anticonvulsant medication blood levels</li> </ul>
<b>Respiratory System</b>	
<ul style="list-style-type: none"> <li>• Complete or partial airway obstruction will compromise the oxygen supply to the brain.</li> <li>• An altered respiratory pattern can result in cerebral hypoxia.</li> <li>• A short period of apnea at the moment of impact can result in spotty atelectasis.</li> <li>• Systemic disturbances from head injury can cause hypoxemia.</li> <li>• Brain injury can alter brain stem respiratory function.</li> <li>• Shunting of blood to the lungs as a result of a sympathetic discharge at the time of injury can cause neurogenic pulmonary edema.</li> </ul>	<ul style="list-style-type: none"> <li>• Assessment of respiratory function:</li> <li>• Auscultate chest for breath sounds</li> <li>• Note the respiratory pattern, if possible (not possible if a ventilator is being used)</li> <li>• Note the respiratory rate</li> <li>• Note whether the cough reflex is intact</li> <li>• Arterial blood gas levels</li> <li>• Complete blood count</li> <li>• Chest x-ray studies</li> <li>• Sputum cultures</li> <li>• Oxygen saturation using pulse oximetry</li> </ul>
<b>Cardiovascular System</b>	
<ul style="list-style-type: none"> <li>• The patient may develop cardiac arrhythmias, tachycardia, or bradycardia.</li> <li>• The patient may develop hypotension or hypertension.</li> <li>• Because of immobility and unconsciousness, the patient is at high risk for DVT and PE.</li> <li>• Fluid and electrolyte imbalance can be related to several problems including alterations in antidiuretic hormone secretion, the stress response, or fluid restriction.</li> <li>• Specific conditions may occur:</li> <li>• Diabetes insipidus</li> <li>• Syndrome of inappropriate secretion of antidiuretic hormone</li> <li>• Electrolyte imbalance</li> <li>• Hyperglycemic hyperosmolar syndrome.</li> </ul>	<ul style="list-style-type: none"> <li>• Assessment of vital signs</li> <li>• Monitoring for cardiac arrhythmias</li> <li>• Assessment for venous thromboembolism including PE and DVT</li> <li>• Electrocardiogram</li> <li>• Electrolyte studies</li> <li>• Blood coagulation studies</li> <li>• Blood glucose level</li> <li>• Blood acetone level</li> <li>• Blood osmolality</li> <li>• Urine-specific gravity</li> </ul>
<b>Gastrointestinal System</b>	

- Injury to the GI tract can result in paralytic ileus.
- Constipation can result from bed rest, NPO status, fluid restriction, and opioids given for pain control.
- Bowel incontinence is related to the patient's unconscious state or altered mental state.
- Assessment of abdomen for bowel sounds and distention
- Monitoring for decreased hemoglobin

### **Metabolic (Nutritional) System**

- The patient receives all fluids IV for the first few days until the GI tract is functioning.
- A nutritional consultation is initiated within the first 24–48 h; parenteral or enteral nutrition may be started.
- Assessment of fluid and electrolyte balance
- Recording of weight, if possible
- Hematocrit
- Electrolyte studies

### **Genitourinary System**

- Fluid restriction or the use of diuretic agents can alter the amount of urinary output.
- Urinary incontinence is related to the patient's unconscious state.
- Intake and output record

### **Musculoskeletal System**

- Immobility contributes to musculoskeletal changes.
- Decerebrate or decorticate posturing makes proper positioning difficult (see [Chapter 61](#), Fig. 61-1).
- Assessment of range of motion of joints and development of deformities or spasticity

### **Integumentary System (Skin and Mucous Membranes)**

- Immobility secondary to TBI and unconsciousness contributes to the development of pressure areas and skin breakdown.
- Intubation causes irritation of the mucous membrane and deterioration of oral health.
- Assessment of skin integrity and character
- Assessment of oral mucous membrane and oral health of the skin

### **Psychological/Emotional Response**

- The patient with TBI is unconscious.
- The family needs emotional support to deal with the crisis.
- Alternative methods of assessment for pain are indicated in the patient who is unconscious
- Collection of information about the family and the role within the family of the person with head injury
- Assessment of the family to determine how functional it was before the injury occurred

DVT, deep vein thrombosis; GI, gastrointestinal; ICP, intracranial pressure; IV, intravenous; NPO, nothing by mouth; PE, pulmonary embolism; TBI, traumatic brain injury.

Adapted from Hickey, J. V., & Strayer, A. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.

## Diagnosis

### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired airway clearance and impaired gas exchange associated with brain injury
- Risk for ineffective tissue perfusion associated with increased ICP, decreased CPP, and possible seizures
- Hypovolemia associated with decreased LOC and hormonal dysfunction
- Impaired nutritional status associated with increased metabolic demands, fluid restriction, and inadequate intake
- Risk for injury (self-directed and directed at others) associated with seizures, disorientation, restlessness, or brain damage
- Risk for impaired thermoregulation associated with damaged temperature-regulating mechanisms in the brain
- Risk for impaired skin integrity associated with bed rest, hemiparesis, hemiplegia, immobility, or restlessness
- Difficulty coping associated with brain injury
- Impaired sleep associated with brain injury and frequent neurologic checks
- Risk for impaired family coping associated with unresponsiveness of patient, unpredictability of outcome, prolonged recovery period, and the patient's residual physical disability and emotional deficit
- Lack of knowledge about brain injury, recovery, and the rehabilitation process

The nursing diagnoses for the patient who is unconscious and the patient with increased ICP also apply (see [Chapter 61](#)).

### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Decreased cerebral perfusion
- Cerebral edema and herniation
- Impaired oxygenation and ventilation
- Impaired fluid, electrolyte, and nutritional balance
- Risk for posttraumatic seizures

## Planning and Goals

The goals for the patient may include maintenance of a patent airway, adequate CPP, fluid and electrolyte balance, adequate nutritional status, prevention of secondary injury, maintenance of body temperature within normal limits, maintenance of skin integrity, improvement of coping, prevention of sleep deprivation, effective family coping, increased knowledge about the rehabilitation process, and absence of complications.

### **Nursing Interventions**

The nursing interventions for the patient with a TBI are extensive and diverse. They include making nursing assessments, setting priorities for nursing interventions, anticipating needs and complications, and initiating rehabilitation.

#### **MAINTAINING THE AIRWAY**

One of the most important nursing goals in the management of head injury is to establish and maintain an adequate airway. The brain is extremely sensitive to hypoxia, and a neurologic deficit can worsen if the patient is hypoxic. Therapy is directed toward maintaining optimal oxygenation to preserve cerebral function. An obstructed airway causes carbon dioxide retention and hypoventilation, which can produce cerebral vessel dilation and increased ICP (Urden et al., 2018).

Interventions to ensure an adequate exchange of air are discussed in [Chapter 61](#) and include the following (Hickey & Strayer, 2020):

- Maintaining the patient who is unconscious in a position that facilitates drainage of oral secretions, with the head of the bed elevated about 30 degrees to decrease intracranial venous pressure
- Establishing effective suctioning procedures (pulmonary secretions produce coughing and straining, which increase ICP)
- Guarding against aspiration and respiratory insufficiency
- Closely monitoring arterial blood gas values to assess the adequacy of ventilation. The goal is to keep blood gas values within normal limits to ensure adequate cerebral blood flow
- Monitoring the patient who is receiving mechanical ventilation for pulmonary complications such as acute respiratory distress syndrome and pneumonia

The patient who is intubated is at high risk for ventilator-associated pneumonia and providing good oral hygiene can help prevent this complication (Gallagher, 2017).

#### **MONITORING NEUROLOGIC FUNCTION**

Patients with severe TBI are admitted to the ICU for close assessment and monitoring (cardiac monitoring, pulse oximetry, invasive arterial blood pressure monitoring, end-tidal CO<sub>2</sub>, and temperature monitoring). Parameters are assessed initially and as frequently as the patient's condition requires. As soon as the initial assessment is made, the use of a neurologic observational

flow record is started and maintained. The importance of ongoing assessment and monitoring of the patient with brain injury cannot be overstated.

**Level of Consciousness.** The GCS is used to assess LOC at regular intervals, because changes in the LOC precede all other changes in vital and neurologic signs. The patient's best responses to predetermined stimuli are recorded (see [Chart 63-4](#)). Each response is scored (the greater the number, the better the functioning), and the sum of these scores gives an indication of the severity of coma and a prediction of possible outcome. The lowest score is 3 (least responsive); the highest is 15 (most responsive). A GCS score between 3 and 8 is generally accepted as indicating a severe head injury (Hickey & Strayer, 2020).



### Concept Mastery Alert

*The GCS is considered the most sensitive indicator of a lapse in neurologic functioning in patients with TBI and is often the earliest sign of acute change in ICP.*

**Vital Signs.** Although a change in LOC is the most sensitive neurologic indication of deterioration of the patient's condition, vital signs are also monitored at frequent intervals to assess the intracranial status. [Table 63-1](#) depicts the general assessment parameters for the patient with a head injury.

Signs of increasing ICP include bradycardia (slowing of the heart rate), increasing systolic blood pressure, and widening pulse pressure (Cushing's reflex). As brain compression increases, respirations become rapid, the blood pressure may decrease, and the pulse slows further. This is an ominous development, as is a rapid fluctuation of vital signs (Hickey & Strayer, 2020). The temperature is maintained at less than 38°C (100.4°F) (Young & Prescott, 2019). Tachycardia and arterial hypotension may indicate that bleeding is occurring elsewhere in the body.



### Concept Mastery Alert

*In a patient with a head injury, a rapid increase in body temperature is regarded as unfavorable because hyperthermia increases the metabolic demands of the brain and may indicate brain stem damage—a poor prognostic sign.*

**Motor Function.** Motor function is assessed frequently by observing spontaneous movements, asking the patient to raise and lower the extremities, and comparing the strength and equality of the upper and lower extremities at periodic intervals. To assess upper extremity strength, the nurse instructs the patient to squeeze the examiner's fingers tightly. The nurse assesses lower extremity motor strength by placing the hands on the soles of the patient's feet and asking the patient to push down against the examiner's hands. Examination of the motor system is discussed in more detail in [Chapter 60](#).

The presence or absence of spontaneous movement of each extremity is also noted, and speech and eye signs are assessed.

If the patient does not demonstrate spontaneous movement, responses to painful stimuli are assessed (Hickey & Strayer, 2020). Motor response to pain is assessed by applying a central stimulus, such as pinching the pectoralis major muscle, to determine the patient's best response. Peripheral stimulation may provide inaccurate assessment data because it may result in a reflex movement rather than a voluntary motor response. Abnormal responses (lack of motor response; extension responses) are associated with a poorer prognosis.

**Other Neurologic Signs.** The size and equality of the pupils and their reaction to light need to be continuously assessed. A unilaterally dilated and poorly responding pupil may indicate a developing hematoma, with subsequent pressure on the third cranial nerve due to shifting of the brain. If both pupils become fixed and dilated, this indicates acute injury and intrinsic damage to the upper brain stem and is a poor prognostic sign (Hickey & Strayer, 2020).

The patient with a head injury may develop deficits such as anosmia (lack of sense of smell), eye movement abnormalities, aphasia, memory deficits, and posttraumatic seizures or epilepsy. Patients may be left with residual psychological deficits (impulsiveness; emotional lability; or uninhibited, aggressive behaviors) and, as a consequence of the impairment, may lack insight into their emotional responses.

#### **MONITORING FLUID AND ELECTROLYTE BALANCE**

Brain damage can produce metabolic and hormonal dysfunctions. The monitoring of serum electrolyte levels is important, especially in patients receiving osmotic diuretics, those with syndrome of inappropriate antidiuretic hormone (SIADH) secretion, and those with posttraumatic diabetes insipidus.

Serial studies of blood and urine electrolytes and osmolality are carried out because head injuries may be accompanied by disorders of sodium regulation. Hyponatremia is common after head injury due to shifts in extracellular fluid, electrolytes, and volume. Hyperglycemia, for example, can cause an increase in extracellular fluid that lowers sodium. Hypernatremia may also occur as a result of sodium retention that may last several days, followed by sodium diuresis. Increasing lethargy, confusion, and seizures may be the result of electrolyte imbalance.

Endocrine function is evaluated by monitoring serum electrolytes, blood glucose values, and intake and output. Urine is tested regularly for acetone. A record of daily weights is maintained, especially if the patient has hypothalamic involvement and is at risk for the development of diabetes insipidus.

#### **PROMOTING ADEQUATE NUTRITION**

Head injury results in metabolic changes that increase calorie consumption and nitrogen excretion. Protein demand increases. Early initiation of nutritional

therapy has been shown to improve outcomes in patients with head injury. Patients with brain injury are assumed to be catabolic, and nutritional support consultation should be considered as soon as the patient is admitted. Parenteral nutrition via a central line or enteral feedings given via a nasogastric or nasojejunal feeding tube should be considered, though enteral is the preferred route (Hickey & Strayer, 2020). If CSF rhinorrhea occurs or if there is any suspicion of disruption to the skull base, an oral feeding tube should be inserted instead of a nasal tube.

Laboratory values should be monitored closely in patients receiving parenteral nutrition. Elevating the head of the bed can help prevent distention, regurgitation, and aspiration. A continuous-drip infusion or pump may be used to regulate the feeding. Enteral or parenteral feedings are usually continued until the swallowing reflex returns and the patient can meet caloric requirements orally. See [Chapter 39](#) for the principles and technique of enteral feedings.

#### **PREVENTING INJURY**

Often, as the patient emerges from coma, a period of lethargy and stupor is followed by a period of agitation. Each phase is variable and depends on the person, the location of the injury, the depth and duration of coma, and the patient's age. Restlessness may be caused by hypoxia, fever, pain, or a full bladder. It may indicate injury to the brain but may also be a sign that the patient is regaining consciousness. (Some restlessness may be beneficial because the lungs and extremities are exercised.) Agitation may also be the result of discomfort from catheters, IV lines, restraints, and repeated neurologic checks. Alternatives to restraints must be used whenever possible.

Strategies to prevent injury include the following:

- Assessing the patient to ensure that oxygenation is adequate, and the bladder is not distended. Dressings and casts are checked for constriction
- Using padded side rails or wrapping the patient's hands in mitts to protect the patient from self-injury and dislodging of tubes. Restraints are used judiciously because straining against them can increase ICP or cause other injury. Enclosed or floor-level specialty beds may be indicated
- Avoiding opioids as a means of controlling restlessness, because they depress respiration, constrict the pupils, and alter responsiveness
- Reducing environmental stimuli by keeping the room quiet, limiting visitors, speaking calmly, and providing frequent orientation information (e.g., explaining where the patient is and what is being done)
- Providing adequate lighting to prevent visual hallucinations
- Minimizing disruption of the patient's sleep-wake cycles
- Lubricating the patient's skin with oil or emollient lotion to prevent irritation due to rubbing against the sheet

- Using an external sheath catheter for a male patient if incontinence occurs. Because prolonged use of an indwelling catheter inevitably produces infection, the patient may be placed on an intermittent catheterization schedule.

#### **MAINTAINING THERMOREGULATION**

Fever in the patient with a TBI can be the result of damage to the hypothalamus, cerebral irritation from hemorrhage, or infection. The nurse monitors the patient's temperature every 2 to 4 hours. If the temperature increases, efforts are made to identify the cause and to control it using acetaminophen and cooling devices to maintain normothermia. Cooling devices should be used with caution so as not to induce shivering, which increases ICP. If infection is suspected, potential sites of infection are cultured, and antibiotic agents are prescribed and administered. Research about therapeutic hypothermia for patients with TBI suggests there is no clear evidence to guide therapy (Watson, Shepherd, Rhodes, et al., 2018; Weng, Yang, Huang, et al., 2018).

#### **MAINTAINING SKIN INTEGRITY**

Patients with TBI often require assistance in turning and positioning because of immobility or unconsciousness. Prolonged pressure on the tissues decreases circulation and leads to tissue necrosis. Potential areas of breakdown need to be identified early to avoid the development of pressure injuries. Specific nursing measures include the following:

- Assessing all body surfaces and documenting skin integrity every 8 hours
- Turning and repositioning the patient every 2 hours
- Providing skin care every 4 hours
- Assisting the patient to get out of bed to a chair three times a day

#### **IMPROVING COPING**

Although many patients with head injury survive because of resuscitative and supportive technology, they frequently have ineffective coping due to cognitive sequelae. Cognitive impairment includes memory deficits; decreased ability to focus and sustain attention to a task (distractibility); impulsivity; egocentricity; and slowness in thinking, perceiving, communicating, reading, and writing. Psychiatric, emotional, and relationship problems develop in many patients after head injury. Resulting psychosocial, behavioral, emotional, and cognitive impairments are devastating to the family as well as to the patient (Oyesanya, Arulselvam, Thompson, et al., 2019).

These problems require collaboration among many disciplines. A neuropsychologist (specialist in evaluating and treating cognitive problems) plans a program and initiates therapy or counseling to help the patient reach maximal potential. Cognitive rehabilitation activities help the patient devise new problem-solving strategies. The retraining is carried out over an extended period and may include the use of sensory stimulation and reinforcement,

behavior modification, reality orientation, computer training programs, and video games. Assistance from many disciplines is necessary during this phase of recovery. Even if intellectual ability does not improve, social and behavioral abilities may improve.

The patient recovering from a TBI may experience fluctuations in the level of cognitive function, with orientation, attention, and memory frequently affected. Many types of sensory stimulation programs have been tried, and research on these programs is ongoing (Hickey & Strayer, 2020). When pushed to a level greater than the impaired cortical functioning allows, the patient may show symptoms of fatigue, anger, and stress (headache, dizziness). The Rancho Los Amigos Scale: Levels of Cognitive Functioning is frequently used to assess cognitive function and evaluate ongoing recovery from head injury. Progress through the levels of cognitive function can vary widely for individual patients (Hagen, Malkmus, & Durham, 1972). Nursing management and a description of each level are included in [Table 63-2](#).

#### **PREVENTING SLEEP PATTERN DISTURBANCE**

Patients who require frequent monitoring of neurologic status may experience sleep deprivation as they are awakened hourly for assessment of LOC. To allow the patient longer times of uninterrupted sleep and rest, the nurse can group nursing care activities so that the patient is disturbed less frequently. Environmental noise is decreased, and the room lights are dimmed. Measures to increase comfort may promote sleep and rest (Giusti, Tuteri, & Mirella, 2016).

#### **SUPPORTING FAMILY COPING**

Having a loved one sustain a TBI produces a great deal of stress in the family. This stress can result from the patient's physical and emotional deficits, the unpredictable outcome, and altered family relationships. Families report difficulties in coping with changes in the patient's temperament, behavior, and personality (Oyesanya et al., 2019). Such changes are associated with disruption in family cohesion, loss of leisure pursuits, and loss of work capacity, as well as social isolation of the caretaker. The family may experience marital disruption, anger, grief, guilt, and denial in recurring cycles.

To promote effective coping, the nurse can ask the family how the patient is different now, what has been lost, and what is most difficult about coping with this situation. Helpful interventions include providing family members with accurate and honest information and encouraging them to continue to set well-defined short-term goals. Family counseling helps address the family members' acute feelings of loss and helplessness and gives them guidance for the management of inappropriate behaviors. Support groups help the family members share problems, develop insight, gain information, network, and gain assistance in maintaining realistic expectations, hope, and a good quality of life (Oyesanya et al., 2019).

The Brain Injury Association of America (see the Resources section) serves as a clearinghouse for information and resources for patients with head injuries

and their families, including specific information on coma, rehabilitation, behavioral consequences of head injury, and family issues. This organization can provide names of facilities and professionals who work with patients with head injuries and can assist families in organizing local support groups.

**TABLE 63-2** Rancho Los Amigos Scale: Levels of Cognitive Function

Cognitive Level	Description	Nursing Management
<b>For levels I–III, the key approach is to provide stimulation.</b>		
I: No response	Completely unresponsive to all stimuli, including painful stimuli	Multiple modalities of sensory input should be used. Examples are listed here, but management should be individualized and expanded based on available materials and patient preferences (determined by obtaining information from the family).
II: Generalized response	Nonpurposeful response; responds to pain but in a nonpurposeful manner	<i>Olfactory:</i> Perfumes, flowers, shaving lotion. <i>Visual:</i> Family pictures, card, personal items.
III: Localized response	Responses more focused—withdraws to pain; turns toward sound; follows moving objects that pass within the visual field; pulls on sources of discomfort (e.g., tubes, restraints); may follow simple commands but inconsistently and in a delayed manner	<i>Auditory:</i> Radio, television, recordings of family voices or favorite recordings, talking to patient (nurse, family members). The nurse should tell patient what is going to be done, discuss the environment, provide encouragement. <i>Tactile:</i> Touching of skin, rubbing various textures on skin. <i>Movement:</i> Range-of-motion exercises, turning, repositioning, the use of water mattress.
<b>For levels IV–VI, the key approach is to provide structure.</b>		
IV: Confused, agitated response	Alert, hyperactive state in which patient responds to internal confusion/agitation; behavior nonpurposeful in relation to the environment; aggressive, bizarre behavior common	For level IV, which lasts 2–4 wks, interventions are directed at decreasing agitation, increasing environmental awareness, and promoting safety.

		<ul style="list-style-type: none"> <li>• Approach patient in a calm manner, and use a soft voice.</li> <li>• Screen patient from environmental stimuli (e.g., sounds, sights); provide a quiet, controlled environment.</li> <li>• Remove devices that contribute to agitation (e.g., tubes), if possible.</li> <li>• Functional goals cannot be set because the patient is unable to cooperate.</li> </ul>
V: Confused, inappropriate response	When agitation occurs, it is the result of external rather than internal stimuli; focused attention is difficult; memory is severely impaired; responses are fragmented and inappropriate to the situation; there is no carryover of learning from one situation to the other	<p>For levels V and VI, interventions are directed at decreasing confusion, improving cognitive function, and improving independence in performing ADLs.</p> <ul style="list-style-type: none"> <li>• Provide supervision.</li> <li>• Use repetition and cues to educate about ADLs. Focus the patient's attention, and help to increase their concentration.</li> <li>• Help the patient organize activities.</li> <li>• Clarify misinformation and reorient when confused.</li> <li>• Provide a consistent, predictable schedule (e.g., post daily</li> </ul>

		schedule on large poster board).
VI: Confused, appropriate response	Follows simple directions consistently but is inconsistently oriented to time and place; short-term memory worse than long-term memory; can perform some ADLs	
<b>For levels VII–X, the key approach is <i>integration into the community</i>.</b>		
VII: Automatic, appropriate response	Appropriately responsive and oriented within the hospital setting; needs little supervision in ADLs; some carryover of learning; patient has superficial insight into disability; has decreased judgment and problem-solving abilities; lacks realistic planning for future	For levels VII–X, interventions are directed at increasing the patient's ability to function with minimal or no supervision in the community. <ul style="list-style-type: none"> <li>• Reduce environmental structure.</li> <li>• Help the patient plan for adapting ADLs for self into the home environment.</li> <li>• Discuss and adapt home-living skills (e.g., cleaning, cooking) to patient's ability.</li> <li>• Provide standby assistance, as needed, for ADLs and home-living skills.</li> </ul>
VIII: Purposeful, appropriate	Alert, oriented, intact memory; has realistic goals for the future. Able to complete familiar tasks for 1 h in a distracting environment; overestimates or underestimates abilities, argumentative, easily frustrated, self-centered; uncharacteristically dependent/independent	
IX: Purposeful, appropriate	Independently shifts back and forth between tasks and completes them accurately for at least 2 consecutive hours; uses assistive memory devices to recall schedule and activities; aware of and acknowledges impairments and disability when they interfere with task completion; depression may continue; may be easily irritable and have a low frustration tolerance	<ul style="list-style-type: none"> <li>• Provide assistance on request for adapting ADLs and home-living skills.</li> </ul>
X: Purposeful,	Able to handle multiple tasks simultaneously	<ul style="list-style-type: none"> <li>• Monitor for signs</li> </ul>

appropriate	<p>in all environments but may require periodic breaks; independently initiates and carries out familiar and unfamiliar tasks but may require more than usual amount of time or compensatory strategies to complete them; accurately estimates abilities and independently adjusts to task demands; periodic periods of depression may occur; irritability and low frustration tolerance when sick, fatigued, or under stress</p>	<p>and symptoms of depression.</p> <ul style="list-style-type: none"> <li>Help the patient plan, anticipate concerns, and solve problems.</li> </ul>
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ADLs, activities of daily living.

Adapted from Los Amigos Research and Education Institute, Inc., & Downey, C. A. (2002). Used with permission.

Many patients with severe head injury die of their injuries, and many of those who survive experience long-term disability that prevents them from resuming their previous roles and functions. During the most acute phase of injury, family members need factual information and support from the health care team.

Many patients with severe head injuries that result in brain death are young and otherwise healthy and are therefore considered for organ donation. Family members of patients with such injuries need support during this extremely stressful time and assistance in making decisions to end life support and permit donation of organs. They need to know that the patient who is brain dead and whose respiratory and cardiovascular systems are maintained through life support is not going to survive and that the severe head injury, not the removal of the patient's organs or the removal of life support, is the cause of the patient's death. Bereavement counselors and members of the organ procurement team are often immensely helpful to family members in making decisions about organ donation and in helping them cope with stress.

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

**Decreased Cerebral Perfusion Pressure.** Maintenance of adequate CPP is important to prevent serious complications of head injury due to decreased cerebral perfusion. Adequate CPP is greater than 50 mm Hg. If CPP falls below a patient's threshold, a vasodilating cascade occurs, causing the volume of blood to increase inside the brain, which causes ICP to increase. Measures to maintain adequate CPP are essential because a decrease in CPP can impair cerebral perfusion and cause brain hypoxia and ischemia, leading to permanent brain damage. Once the threshold CPP is reached, vasoconstriction of the cerebral blood vessels occurs, causing ICP to decrease. Therapy (e.g., elevation of the head of the bed, increased IV fluids, CSF drainage) is directed toward decreasing cerebral edema and increasing venous outflow from the brain. Systemic hypotension, which causes vasoconstriction and a significant decrease in CPP, is treated with increased IV fluids or vasopressors (Livesay, McNett, Keller, et al., 2017).

**Cerebral Edema and Herniation.** The patient with a head injury is at risk for additional complications such as increased ICP and brain stem herniation. Cerebral edema is the most common cause of increased ICP in the patient with a head injury, with the swelling peaking approximately 48 to 72 hours after injury. Bleeding also may increase the volume of contents within the rigid, closed compartment of the skull, causing increased ICP and herniation of the brain stem and resulting in irreversible brain anoxia and brain death (Hickey & Strayer, 2020; Vijay & Jaison, 2019). ICP is measured continuously and nursing interventions such as turning and suctioning have been associated with variation in the ICP (Olson, Parcon, Santos, et al., 2017). Measures to control ICP are listed in [Chart 63-5](#) and discussed in [Chapter 61](#).

**Impaired Oxygenation and Ventilation.** Impaired oxygenation and ventilation may require mechanical ventilatory support. The patient must be monitored for a patent airway, altered breathing patterns, and hypoxemia and pneumonia. Interventions may include endotracheal intubation, mechanical ventilation, and positive end-expiratory pressure. See [Chapters 19](#) and [61](#) for a detailed discussion on these topics.

### Chart 63-5

#### Controlling ICP in Patients with Severe Brain Injury

- Elevate the head of the bed as prescribed.
- Maintain the patient's head and neck in neutral alignment (no twisting or flexing the neck).
- Initiate measures to prevent the Valsalva maneuver (e.g., stool softeners).
- Maintain body temperature within normal limits.
- Administer oxygen ( $O_2$ ) to maintain partial pressure of arterial oxygen ( $PaO_2$ )  $>90$  mm Hg.
- Maintain fluid balance with normal saline solution.
- Avoid noxious stimuli (e.g., excessive suctioning, painful procedures).
- Administer sedation to reduce agitation.
- Maintain cerebral perfusion pressure of 60–70 mm Hg.

Adapted from Hickey, J. V., & Strayer, A. (2020). *The clinical practice of neurological & neurosurgical nursing*. (8th ed.). Philadelphia, PA: Wolters Kluwer.

**Impaired Fluid, Electrolyte, and Nutritional Balance.** Fluid, electrolyte, and nutritional imbalances are common in the patient with a head injury. Common imbalances include hyponatremia, which is often associated with SIADH (see [Chapters 10](#) and [45](#)), hypokalemia, and hyperglycemia. Modifications in fluid intake with tube feedings or IV fluids, including hypertonic saline, may be necessary to treat these imbalances (Hickey & Strayer, 2020). Insulin

administration may be prescribed to treat hyperglycemia; blood glucose levels are maintained between 80 and 160 mg/dL (Vijay & Jaison, 2019).

Undernutrition is also a common problem in response to the increased metabolic needs associated with severe head injury. Decisions about early feeding should be individualized; options include IV hyperalimentation or placement of a feeding tube (jejunal or gastric). Caloric expenditure can increase up to 120% to 140% with TBI, requiring close monitoring of nutritional status, with a higher concentration of protein if tolerated (Quintard & Ichai, 2018).

**Posttraumatic Seizures.** Patients with head injury are at an increased risk for posttraumatic seizures. Posttraumatic seizures are classified as immediate (within 24 hours after injury), early (within 1 to 7 days after injury), or late (more than 7 days after injury) (Hickey & Strayer, 2020). Seizure prophylaxis is the practice of administering anticonvulsant medications to patients with head injury to prevent seizures. It is important to prevent posttraumatic seizures, especially in the immediate and early phases of recovery, because seizures may increase ICP and decrease oxygenation (Chartrain, Yaeger, Feng, et al., 2017; Zaman, Dubiel, Driver, et al., 2017). However, many anticonvulsant medications impair cognitive performance and can prolong the duration of rehabilitation. Therefore, the overall benefits of these medications must be weighed against their side effects. Research evidence supports the use of prophylactic anticonvulsant agents to prevent immediate and early seizures after head injury, but not for prevention of late seizures (Chartrain et al., 2017; Zaman et al., 2017). See [Chapter 61](#) for the nursing management of seizures.

Chart 63-6



## HOME CARE CHECKLIST

## The Patient with a TBI

**At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of TBI and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the purpose, dose, route, schedule, side effects, and precautions for prescribed medications.
- State how to contact all members of the treatment team (e.g., health care providers, home care professionals, rehabilitation team, and durable medical equipment and supply vendor).
- State changes in lifestyle (e.g., ADLs, IADLs, activity) necessary for recovery and health maintenance, as applicable.
  - Demonstrate safe techniques to assist patient with self-care, hygiene, and ambulation.
  - Demonstrate safe techniques for eating, feeding patient, or assisting patient with eating.
  - Identify the need for close monitoring of behavior due to changes in cognitive functioning.
  - Describe strategies for reinforcing positive behaviors.
  - Describe household modifications needed to ensure safe environment for the patient.
- Explain the need for monitoring for changes in neurologic status and for complications.
- Identify changes in neurologic status and signs and symptoms of complications (e.g., pneumonia, urinary tract infection, meningitis) that should be reported to the neurosurgeon or nurse.
- Relate how to reach primary provider with questions or complications.
- State the importance of continuing follow-up by health care team.
- State time and date of follow-up medical appointments, therapy, and testing.
- Identify sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families.
- Identify the need for health-promotion, disease prevention, and screening activities.

### Resources

See [Chapter 7](#), Chart 7-6 Home Care Checklist: Managing Chronic Illness and Disability at Home.

ADL, activities of daily living; IADL, instrumental activities of daily living; TBI, traumatic brain injury.



### Educating Patients About Self-Care.

Education early in the course of head injury often focuses on reinforcing information given to the family about the patient's condition and prognosis. As the patient's status and expected outcome change over time, family education may focus on interpretation and explanation of changes in the patient's physical and psychological responses.

Once the patient's physical status allows discharge to home, a rehabilitation center, or a subacute care facility, the patient and family are educated about limitations that can be expected and complications that may occur. The nurse explains to the patient and family, verbally and in writing, how to monitor for complications that merit contacting the primary provider. Depending on the patient's prognosis and physical and cognitive status, the patient may be included in education about self-care management strategies.

If the patient is at risk for late posttraumatic seizures, anticonvulsant medications may be prescribed at discharge. The patient and family require education about the side effects of these medications and the importance of continuing to take them as prescribed.

**Continuing and Transitional Care.** The rehabilitation phase of care for the patient with a TBI begins at hospital admission. Admission to the rehabilitation unit is a milestone in a patient's recovery and requires intense work by the patient to complete the daily schedule of therapies. The goals of rehabilitation are to maximize the patient's ability to return to their highest level of functioning and to their home and the community, address concerns before discharge for a smooth transition to home or rehabilitation, and promote independence with adaptation to deficits. The patient is encouraged to continue the rehabilitation program after discharge, because improvement in status may continue 3 or more years after injury. Changes in the patient with a TBI and the effects of long-term rehabilitation on the family and their coping abilities need ongoing assessment. Continued education and support of the patient and family are essential as their needs and the patient's status change. Education to address with the family of the patient who is about to return home is described in [Chart 63-6](#).

Depending on status, the patient is encouraged to return to usual activities gradually. Referral to support groups and to the Brain Injury Association of America may be warranted (see the Resources section).

During the acute and rehabilitation phases of care, the focus of education is on obvious needs, issues, deficits, and complications. Complications after TBI include infections (e.g., pneumonia, urinary tract infection [UTI], sepsis, wound infection, osteomyelitis, meningitis, ventriculitis, brain abscess) and heterotopic ossification (painful bone overgrowth in weight-bearing joints).

The nurse reminds the patient and family of the need for continuing health promotion and screening practices after the initial phase of care. Patients who

have not been involved in these practices in the past are educated about their importance and are referred to appropriate health care providers.

### Evaluation

Expected patient outcomes may include:

1. Attains or maintains effective airway clearance, ventilation, and brain oxygenation
  - a. Achieves blood gas values within normal limits and has breath sounds clear on auscultation
  - b. Mobilizes and clears secretions
2. Achieves satisfactory fluid and electrolyte balance
  - a. Demonstrates serum electrolytes within normal limits
  - b. Has no clinical signs of dehydration or overhydration
3. Attains adequate nutritional status
  - a. Is free of gastric distention and vomiting
  - b. Shows minimal weight loss
4. Avoids injury
  - a. Shows lessening agitation and restlessness
  - b. Is oriented to person, place, and time
5. Maintains body temperature within normal limits
  - a. Absence of fever
  - b. Absence of hypothermia
6. Demonstrates intact skin integrity
  - a. Exhibits no redness or breaks in skin integrity
  - b. Exhibits no pressure injuries
7. Shows improvement in coping
8. Demonstrates usual sleep-wake cycle
9. Family demonstrates adaptive family processes
  - a. Joins support group
  - b. Shares feelings with appropriate health care personnel
  - c. Makes end-of-life decisions, if needed
10. Demonstrates absence of complications
  - a. Demonstrates ICP within normal limits
  - b. Exhibits vital signs and body temperature within normal limits and increases orientation to person, place, and time
11. Experiences no posttraumatic seizures
  - a. Takes anticonvulsant medications as prescribed
  - b. Identifies side effects/adverse effects of anticonvulsant medications

12. Participates in rehabilitation process as indicated for patient and family members
- a. Takes active role in identifying rehabilitation goals and participating in recommended patient care activities
  - b. Prepares for discharge

## Spinal Cord Injury (SCI)

**Spinal cord injury (SCI)**, an injury to the spinal cord, vertebral column, supporting soft tissue, or intervertebral discs caused by trauma is a major health disorder. In the United States, approximately 294,000 persons are living with an SCI. An estimated 17,810 new cases occur annually; common causes are motor vehicle crashes, falls, violence (predominantly gunshot wounds), and sports-related injuries (National Spinal Cord Injury Statistical Center [NSCISC], 2020). Males account for 78% of patients with SCI. The average age of injury is 43 years of age (NSCISC, 2020). The indirect cost for the care of patients with SCI averages about \$77,701 per patient per year in 2019 dollars (NSCISC, 2020).

The predominant risk factors for SCI include younger age, male gender, and alcohol and illicit drug abuse. The frequency with which these risk factors are associated with SCI serves to emphasize the importance of primary prevention. The same interventions suggested earlier in this chapter for head injury prevention help decrease the incidence of SCI (see [Chart 63-1](#)). Life expectancy continues to increase for people with SCI because of improved health care but remains slightly lower than for those without SCI. The major causes of death are pneumonia, pulmonary embolism (PE), and sepsis (Hickey & Strayer, 2020).

**Paraplegia** (paralysis of the lower body) and **tetraplegia** (paralysis of all four extremities; formerly called *quadriplegia*) can occur, with incomplete tetraplegia being the most frequently occurring injury, followed by complete paraplegia, complete tetraplegia, and incomplete paraplegia.

## Pathophysiology

Damage in SCI ranges from transient concussion (from which the patient fully recovers) to contusion, laceration, and compression of the spinal cord tissue (either alone or in combination), to complete **transection** (severing) of the spinal cord (which renders the patient paralyzed below the level of the injury). The vertebrae most frequently involved are the fifth, sixth, and seventh cervical vertebrae (C5–C7), the 12th thoracic vertebra (T12), and the first lumbar vertebra (L1). These vertebrae are most susceptible because there is a greater range of mobility in the vertebral column in these areas (Hickey & Strayer, 2020).

SCI can be separated into two categories: primary injuries and secondary injuries. Primary injuries are the result of the initial insult or trauma and are usually permanent. Secondary injuries resulting from SCI include edema and

hemorrhage (Venkatesh, Ghosh, Mullick, et al., 2019). The secondary injury is a major concern for critical-care nurses. Early treatment is essential to prevent partial damage from becoming total and permanent.



## Veterans Considerations

Veterans account for a large proportion of those living with SCI (Gary, Cao, Burns, et al., 2020). Veterans who have suffered an SCI are significantly older and predominantly male compared to civilians with SCI (Furlan, Kurban, & Craven, 2019). However, the level of injury, severity, mechanisms of injury, and need for mechanical ventilation after the SCI are all similar to civilians (Furlan et al., 2019). Veterans with war-related SCIs are predominantly young, White, and male; they have commonly sustained thoracic, severe, SCI caused by gunshot or explosion; and they often have at least one other bodily injury in addition to SCI (Furlan, Gulasingam, & Craven, 2017). Differences between veterans and civilians with SCI may influence adjustment and functional outcomes. For example, factors such as the high rates of posttraumatic stress syndrome, the need to accommodate to civilian life, and the burden of not being able to serve increase the risk for poorer health and unhealthy behaviors, mental health problems, and substance use disorder (Gary et al., 2020). Veterans with SCI have higher cognitive function, social integration, self-perceived independence, and social support, as well as less pain and fewer secondary impairments than civilians with SCI. They also have better physical independence and mobility (Gary et al., 2020). Among civilians, there is a greater likelihood of employment post SCI for non-Hispanic White men with a college education. However, veterans with SCI lesions higher in the spinal cord are less likely to be employed.

## Clinical Manifestations

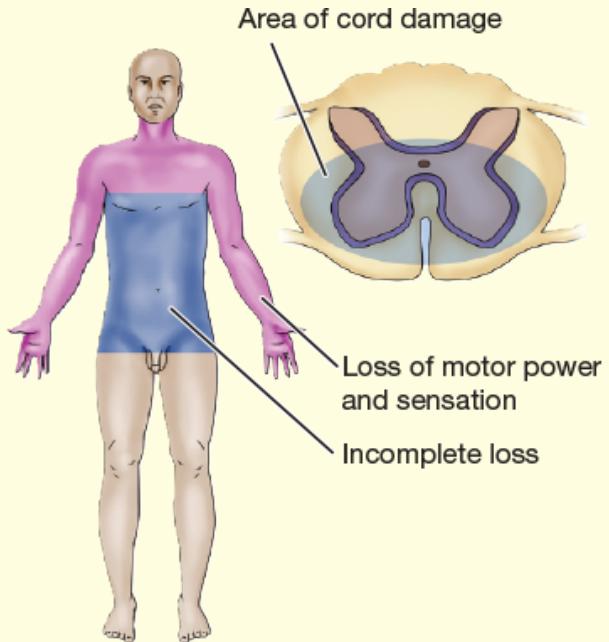
Manifestations of SCI depend on the type and level of injury (see [Chart 63-7](#)). The type of injury refers to the extent of injury to the spinal cord itself. A **complete spinal cord lesion** signifies loss of both sensory and voluntary motor communication from the brain to the periphery, resulting in paraplegia or tetraplegia. **Incomplete spinal cord lesion** denotes that the ability of the spinal cord to relay messages to and from the brain is not completely absent. Sensory and/or motor fibers are preserved below the lesion. Injuries are classified according to the area of spinal cord damage: central, lateral, anterior, or peripheral (see [Chart 63-7](#)).

### Chart 63-7

## Effects of Spinal Cord Injuries

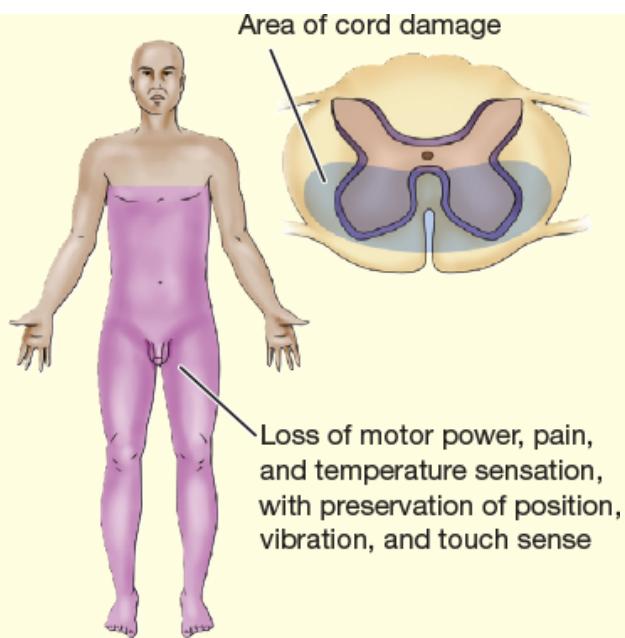
### Central Cord Syndrome

- **Characteristics:** Motor deficits (in the upper extremities compared to the lower extremities; sensory loss varies but is more pronounced in the upper extremities); bowel/bladder dysfunction is variable, or function may be completely preserved.
- **Cause:** Injury or edema of the central cord, usually of the cervical area. May be caused by hyperextension injuries.



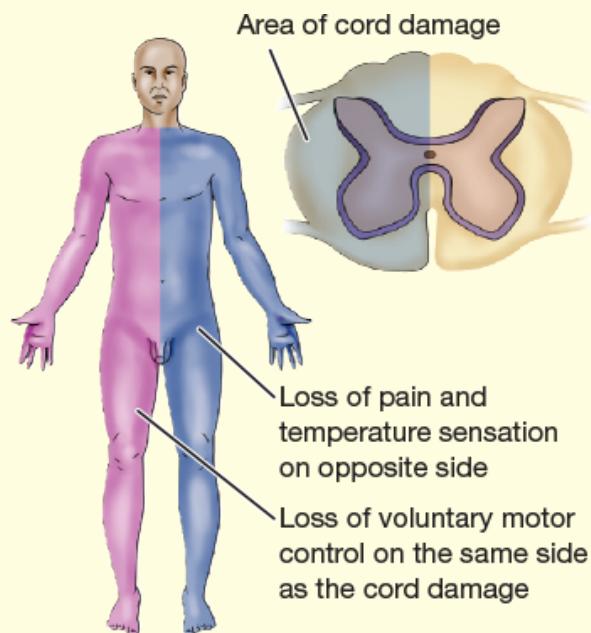
### Anterior Cord Syndrome

- **Characteristics:** Loss of pain, temperature, and motor function is noted below the level of the lesion; light touch, position, and vibration sensation remain intact.
- **Cause:** The syndrome may be caused by acute disc herniation or hyperflexion injuries associated with fracture/dislocation of vertebra. It may also occur as a result of injury to the anterior spinal artery, which supplies the anterior two thirds of the spinal cord.



### Lateral Cord Syndrome (Brown-Séquard Syndrome)

- **Characteristics:** Ipsilateral paralysis or paresis is noted, together with ipsilateral loss of touch, pressure, and vibration and contralateral loss of pain and temperature.
- **Cause:** The lesion is caused by a transverse hemisection of the cord (half of the cord is transected from north to south), usually as a result of a knife or missile injury, fracture/dislocation of a unilateral articular process, or possibly an acute ruptured disc.



Adapted from Hickey, J. V., & Strayer, A. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.

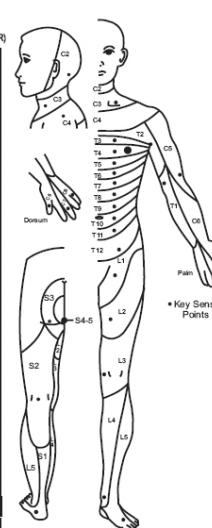
The American Spinal Injury Association (ASIA) provides classification of SCI according to the degree of sensory and motor function present after injury (ASIA, 2019; see Fig. 63-4). The neurologic level refers to the lowest level at which sensory and motor functions are intact. Below the neurologic level, there may be total or partial, sensory and/or motor paralysis (dependent upon affected tracts), loss of bladder and bowel control (usually with urinary retention and bladder distention), loss of sweating and vasomotor tone, and marked reduction of blood pressure from loss of peripheral vascular resistance.

If conscious, the patient usually complains of acute pain in the back or neck, which may radiate along the involved nerve. However, absence of pain does not rule out spinal injury, and a careful assessment of the spine should be conducted if there has been a significant force and mechanism of injury (i.e., concomitant head injury).

Respiratory dysfunction is related to the level of injury. The muscles contributing to respiration are the diaphragm (C4), intercostals (T1–T6), and abdominals (T6–T12). Injuries at C4 or above (causing paralysis of the diaphragm) often will require ventilator support, since acute respiratory failure is a leading cause of death (Hickey & Strayer, 2020). Injuries of T12 and above will have impact on respiratory function. Functional abilities by level of injury are described in Table 63-3.

## Assessment and Diagnostic Findings

A detailed neurologic examination is performed. Diagnostic x-rays (lateral cervical spine x-rays) and CT scanning are usually performed initially. An MRI scan may be ordered as a further workup if a ligamentous injury is suspected, because significant spinal cord damage may exist even in the absence of bony injury (Hickey & Strayer, 2020). If an MRI scan is contraindicated, a myelogram may be used to visualize the spinal axis. An assessment is made for other injuries because spinal trauma often is accompanied by concomitant injuries, commonly to the head and chest. Continuous electrocardiographic monitoring may be indicated if an SCI is suspected, because bradycardia (slow heart rate) and asystole (cardiac standstill) are common in patients with acute spinal cord injuries.

ASIA INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY (ISNCSCI)		Patient Name _____	Date/Time of Exam _____
		Examiner Name _____	Signature _____
<b>RIGHT</b> <b>MOTOR KEY MUSCLES</b> C2 C3 C4  <b>UEL</b> (Upper Extremity Right) Elbow flexors C5 Wrist extensors C6 Elbow extensors C7 Finger flexors C8 Finger abductors (little finger) T1  <b>Comments (Non-key Muscle? Reason for NT? Pain?)</b> Non-SCI condition?		<b>SENSORY KEY SENSORY POINTS</b> Light Touch (LTR) Pin Prick (PPR) C2 C3 C4  T2 T3 T4 T5 T6 T7 T8 T9 T10 T11 T12 L1  L2 L3 L4 L5  S1  (VAC) Voluntary Anal Contraction (Yes/No) <input type="checkbox"/> RIGHT TOTALS (MAXIMUM) (50)	 <b>SENSORY KEY SENSORY POINTS</b> Light Touch (LTL) Pin Prick (PPL) C2 C3 C4  T2 T3 T4 T5 T6 T7 T8 T9 T10 T11 T12 L1  L2 L3 L4 L5  S2 S3 S4-5  (DAP) Deep Anal Pressure (Yes/No) LEFT TOTALS (MAXIMUM) (50)
<b>MOTOR SUBSCORES</b> UER <input type="checkbox"/> + UEL <input type="checkbox"/> = UEMS TOTAL <input type="checkbox"/> (50) MAX (25) (25)  LER <input type="checkbox"/> + LEL <input type="checkbox"/> = LEMS TOTAL <input type="checkbox"/> (50) MAX (25) (25)		<b>SENSORY SUBSCORES</b> LTR <input type="checkbox"/> + LTL <input type="checkbox"/> = LT TOTAL <input type="checkbox"/> (56) MAX (56) (56)	<b>MOTOR KEY MUSCLES</b> C2 C3 C4  C5 Elbow flexors C6 Wrist extensors C7 Elbow extensors C8 Finger flexors T1 Finger abductors (little finger)  <b>UEL</b> (Upper Extremity Left)
R L 1. SENSORY <input type="checkbox"/> <input type="checkbox"/> 2. MOTOR <input type="checkbox"/> <input type="checkbox"/> Steps 1-6 for Classification as on reverse		4. COMPLETE OR INCOMPLETE? <input type="checkbox"/> Injuries with absent motor or sensory function in S4-5 <input type="checkbox"/> Incomplete: Any sensory or motor function in S4-5 <input type="checkbox"/> 5. ASIA IMPAIRMENT SCALE (AIS) Most complete level with any function	R L 6. ZONE OF PARTIAL SENSORY PRESERVATION Most complete level with any function
Page 1/2 This form may be copied freely but should not be altered without permission from the American Spinal Injury Association. REV 0412			

<b>Muscle Function Grading</b>																																					
0 = Total paralysis 1 = Palpable or visible contraction 2 = Active movement, full range of motion (ROM) with gravity eliminated 3 = Active movement, full ROM against gravity 4 = Active movement, full ROM against gravity and moderate resistance in a muscle specific position 5 = (Normal) active movement, full ROM against gravity and full resistance in a functional muscle position expected from an otherwise unimpaired person NT = Not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of > 50% of the normal ROM) 0*, 1*, 2*, 3*, 4*, NT = Non-SCI condition present*																																					
<b>Sensory Grading.</b>																																					
0 = Absent 1 = Altered, either decreased/impaired sensation or hypersensitivity 2 = Normal NT = Not testable 0*, 1*, NT* = Non-SCI condition present *																																					
<p>*Note: Abnormal motor and sensory score should be tagged with a '*' to indicate an impairment due to a non-SCI condition. The non-SCI condition should be explained in the comments box together with information about how the score is rated for classification purposes (at least normal / not normal for classification).</p> <p><b>When to Test Non-Key Muscles:</b></p> <p>In a patient with an apparent AIS B classification, non-key muscle functions more than 3 levels below the motor level on each side should be tested to most accurately classify the injury (differentiate between AIS B and C).</p> <table border="1"> <thead> <tr> <th>Movement</th> <th>Root level</th> </tr> </thead> <tbody> <tr> <td>Shoulder: Flexion, extension, abduction, adduction, internal and external rotation</td> <td>C5</td> </tr> <tr> <td>Elbow: Supination</td> <td>C6</td> </tr> <tr> <td>Elbow: Pronation</td> <td>C7</td> </tr> <tr> <td>Finger: Flexion at proximal joint, extension</td> <td></td> </tr> <tr> <td>Thumb: Flexion, extension and abduction in plane of thumb</td> <td>C7</td> </tr> <tr> <td>Finger: Flexion at MCP joint</td> <td></td> </tr> <tr> <td>Thumb: Opposition, adduction and abduction</td> <td>C8</td> </tr> <tr> <td>perpendicular to palm</td> <td></td> </tr> <tr> <td>Finger: Abduction of the index finger</td> <td>T1</td> </tr> <tr> <td>Hip: Adduction</td> <td>L2</td> </tr> <tr> <td>Hip: External rotation</td> <td>L3</td> </tr> <tr> <td>Hip: Extension, abduction, internal rotation</td> <td></td> </tr> <tr> <td>Knee: Flexion</td> <td>L4</td> </tr> <tr> <td>Ankle: Inversion and eversion</td> <td></td> </tr> <tr> <td>Toe: MP and IP extension</td> <td></td> </tr> <tr> <td>Hallux: DIP and PIP flexion and abduction</td> <td>L5</td> </tr> <tr> <td>Hallux: Adduction</td> <td>S1</td> </tr> </tbody> </table>		Movement	Root level	Shoulder: Flexion, extension, abduction, adduction, internal and external rotation	C5	Elbow: Supination	C6	Elbow: Pronation	C7	Finger: Flexion at proximal joint, extension		Thumb: Flexion, extension and abduction in plane of thumb	C7	Finger: Flexion at MCP joint		Thumb: Opposition, adduction and abduction	C8	perpendicular to palm		Finger: Abduction of the index finger	T1	Hip: Adduction	L2	Hip: External rotation	L3	Hip: Extension, abduction, internal rotation		Knee: Flexion	L4	Ankle: Inversion and eversion		Toe: MP and IP extension		Hallux: DIP and PIP flexion and abduction	L5	Hallux: Adduction	S1
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<b>ASIA Impairment Scale (AIS)</b>	
<b>A = Complete.</b> No sensory or motor function is preserved in the sacral segments S4-5. <b>B = Sensory Incomplete.</b> Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-5 (light touch or pin prick at S4-5 or deep anal pressure) AND motor function is preserved more than three levels below the motor level on either side of the body. <b>C = Motor Incomplete.</b> Motor function is preserved at the most caudal sacral segments for voluntary and contraction (VAC) OR the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments S4-5 by LT, PP or DAP), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body. (This includes key or non-key muscle functions to determine motor incomplete status.) For AIS C – less than half of key muscle functions below the single NLI have a muscle grade ≥ 3. <b>D = Motor Incomplete.</b> Motor incomplete status is defined above, with at least half (half or more) of key muscle functions below the single NLI having a muscle grade ≥ 3. <b>E = Normal.</b> If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade. <b>Using ND:</b> To document the sensory, motor and NLI levels, the ASIA Impairment Scale grade, and/or the zone of partial preservation (ZPP) when they are unable to be determined based on the examination results.	

<b>Steps in Classification</b>	
<p>The following order is recommended for determining the classification of individuals with SCI.</p> <ol style="list-style-type: none"> <li><b>Determine sensory levels for right and left sides.</b> The sensory level is the most caudal, intact dermatome for both pin prick and light touch sensation.</li> <li><b>Determine motor levels for right and left sides.</b> Determine by the lowest key muscle function that has a grade of at least 3 (on supine testing), provided the key muscle functions represented by segments above that level are judged to be intact (graded as a 6). Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.</li> <li><b>Determine the neurological level of injury (NLI).</b> This refers to the most caudal segment of the spine with intact sensation and antigravity (3 or more) muscle function strength, provided that there is normal (intact) sensory and motor function rostrally respectively. The NLI is the most cephalic of the sensory and motor levels determined in steps 1 and 2.</li> <li><b>Determine whether the injury is Complete or Incomplete.</b> (i.e. absence or presence of social sparing) If voluntary anal contraction = No AND all S4-5 sensory scores = 0 AND deep anal pressure = No, then injury is Complete. Otherwise, injury is Incomplete.</li> <li><b>Determine ASIA Impairment Scale (AIS) Grade.</b></li> </ol>	
<p><b>Is injury Complete?</b> If YES, AIS=A            NO ↓</p> <p><b>Is injury Motor Complete?</b> If YES, AIS=B            NO ↓ (Non=voluntary anal contraction OR motor function more than three levels below the <b>motor level</b> on a given side, if the patient has sensory incomplete classification)</p> <p><b>Are at least half (half or more) of the key muscles below the neurological level of injury graded 3 or better?</b></p> <p>NO ↓ YES ↓</p> <p><b>AIS=C AIS=D</b></p>	
<p>If sensation and motor function is normal in all segments, AIS=E            Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact and the ASIA Impairment Scale does not apply.</p>	
<p><b>6. Determine the zone of partial preservation (ZPP).</b>            The ZPP is used only in injuries with absent motor (no VAC) OR sensory function (no DAP, no LT and no PP sensation) in the lowest sacral segments S4-5, and refers to those dermatomes and myotomes caudal to the sensory and motor levels which remain partially innervated. With normal sparing of sensation in this zone, ZPP is not applicable and therefore "NA" is recorded in the block of the worksheet. Accordingly, if VAC is present, the motor ZPP is not applicable and is noted as "NA".</p>	

**Figure 63-4 • Worksheet for the classification of SCI. From the American Spinal Injury Association International Standards Committee: International Standards for Neurological Classification of Spinal Cord Injury. Retrieved on 3/03/2021 at: [https://asia-spinalinjury.org/wp-content/uploads/2019/10/ASIA-ISCOS-Worksheet\\_10.2019\\_PRINT-Page-1-2.pdf](https://asia-spinalinjury.org/wp-content/uploads/2019/10/ASIA-ISCOS-Worksheet_10.2019_PRINT-Page-1-2.pdf). © 2021 American Spinal Injury Association. Reprinted with permission.**

## Emergency Management

The immediate management at the scene of the injury is critical because improper handling of the patient can cause further damage and loss of neurologic function. Any patient who is involved in a motor vehicle crash, a diving or contact sports injury, a fall, or any direct trauma to the head and neck must be considered to have SCI until such an injury is ruled out. Initial care must include a rapid assessment, immobilization, extrication, and stabilization or control of life-threatening injuries, and transportation to the most appropriate medical facility. Immediate transportation to a trauma center with the capacity to manage major neurologic trauma is then necessary (Hickey & Strayer, 2020).

At the scene of the injury, the patient must be immobilized on a spinal (back) board, with the head and neck maintained in a neutral position, to prevent an incomplete injury from becoming complete. One member of the team must assume control of the patient's head to prevent flexion, rotation, or extension; this is done by placing the hands on both sides of the patient's head at about ear level to limit movement and maintain alignment while a spinal board and cervical

immobilizing device is applied. If possible, at least four people should slide the patient carefully onto a board for transfer to the hospital. Head blocks should also be considered, as they will further limit any neck movement. Any twisting movement may irreversibly damage the spinal cord by causing bony fragment or disc movement or exacerbating ligamentous injury, causing further instability.

The patient is referred to a regional spinal injury or trauma center because of the multidisciplinary personnel and support services required to counteract the destructive changes that occur in the first 24 hours after injury. During treatment in the emergency and x-ray departments, the patient is kept on the transfer board. The patient must always be maintained in an extended position. No part of the body should be twisted or turned, and the patient is not allowed to sit up. Once the extent of the injury has been determined, the patient may be placed on a rotating specialty bed or in a cervical collar (see Fig. 63-5). Later, if SCI and bone instability have been ruled out, the patient may be moved to a conventional bed or the collar may be removed without harm. If a specialty bed is needed but not available, the patient should be placed in a cervical collar and on a firm mattress.

**TABLE 63-3** Functional Abilities by Level of Spinal Cord Injury

Injury Level	Segmental Sensorimotor Function	Dressing, Eating	Elimination	Mobility <sup>a</sup>
C1	Little or no sensation or control of head and neck; no diaphragm control; requires continuous ventilation	Dependent	Dependent	Limited. Voice or sip-n-puff controlled electric wheelchair
C2–C3	Head and neck sensation; some neck control; independent of mechanical ventilation for short periods	Dependent	Dependent	Same as for C1
C4	Good head and neck sensation and motor control; some shoulder elevation; diaphragm movement	Dependent; may be able to eat with adaptive sling	Dependent	Limited to voice, mouth, head, chin, or shoulder-controlled electric wheelchair
C5	Full head and neck control; shoulder strength; elbow flexion	Independent with assistance	Maximal assistance	Electric or modified manual wheelchair, needs transfer assistance
C6	Fully innervated shoulder; wrist extension or dorsiflexion	Independent or with minimal assistance	Independent or with minimal assistance	Independent in transfers and wheelchair
C7–C8	Full elbow extension; wrist plantar flexion; some finger control	Independent	Independent	Independent; manual wheelchair
T1–T5	Full hand and finger control; use of intercostal and thoracic muscles	Independent	Independent	Independent; manual wheelchair
T6–T10	Abdominal muscle control, partial to good balance with trunk muscles	Independent	Independent	Independent; manual wheelchair
T11–L5	Hip flexors, hip abductors (L1–L3); knee extension (L2–L4); knee flexion; and ankle dorsiflexion (L4–L5)	Independent	Independent	Short distance to full ambulation with assistance
S1–S5	Full leg, foot, and ankle control; innervation of perineal muscles for bowel, bladder, and sexual function (S2–S4)	Independent	Normal to impaired bowel and bladder function	Ambulate independently with or without assistance

<sup>a</sup>Assistance refers to adaptive equipment, setup, or physical assistance.

Adapted from Hickey, J. V., & Strayer, A. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.



**Figure 63-5 •** Cervical collar. Used with permission from Aspen Medical Products.



## Medical Management (Acute Phase)

The goals of management are to prevent secondary injury, to observe for symptoms of progressive neurologic deficits, and to prevent complications. The patient is resuscitated as necessary, and oxygenation and cardiovascular stability are maintained. SCI is a devastating event; new treatment methods and medications are continually being investigated for the acute and chronic phases of care (Venkatesh et al., 2019).

### Pharmacologic Therapy

Administration of high-dose IV corticosteroids (methylprednisolone sodium succinate) in the first 24 or 48 hours is controversial. The validity of studies has been questioned based on critical analysis of the original and additional data. As a result, there is now a consensus that corticosteroids may offer only a slight benefit. Corticosteroids are no longer considered the standard of care for acute SCI, although some centers continue to use corticosteroid protocols (Hickey & Strayer, 2020).

### Respiratory Therapy

Oxygen is given to maintain a high partial pressure of arterial oxygen ( $\text{PaO}_2$ ), because hypoxemia can create or worsen a neurologic deficit of the spinal cord. If endotracheal intubation is necessary, extreme care is taken to avoid flexing or extending the patient's neck, which can result in extension of a cervical injury.

In high cervical spine injuries, spinal cord innervation to the phrenic nerve, which stimulates the diaphragm, is lost. Diaphragmatic pacing (electrical stimulation of the phrenic nerve) attempts to stimulate the diaphragm to help the patient breathe. Intramuscular diaphragmatic pacing is currently in the clinical trial phase for the patient with a high cervical injury. This is implanted via laparoscopic surgery, usually after the acute phase.

### Skeletal Fracture Reduction and Traction

Management of SCI requires immobilization and reduction of dislocations (restoration of preinjury position) and stabilization of the vertebral column. This can be accomplished by surgical or nonsurgical interventions; both aim to prevent new or worsening neurologic damage.

Cervical fractures can be reduced, and the cervical spine aligned with some form of skeletal traction, such as with skeletal tongs or with the use of the halo device. Traction is applied to the skeletal traction device by weights (ensuring the weights are unencumbered); the amount depends on the size of the patient and the degree of fracture displacement. The traction force is exerted along the longitudinal axis of the vertebral bodies, with the patient's neck in a neutral position. The traction is then gradually increased by adding more weights. As the amount of traction is increased, the spaces between the intervertebral discs widen and the vertebrae are given a chance to slip back into position. Reduction usually occurs after correct alignment has been restored. Once reduction is achieved, as verified by cervical spine x-rays and neurologic examination, the weights are gradually removed until the amount of weight needed to maintain the alignment is identified. Traction is sometimes supplemented with manual manipulation of the neck by a surgeon, to help achieve realignment of the vertebral bodies.

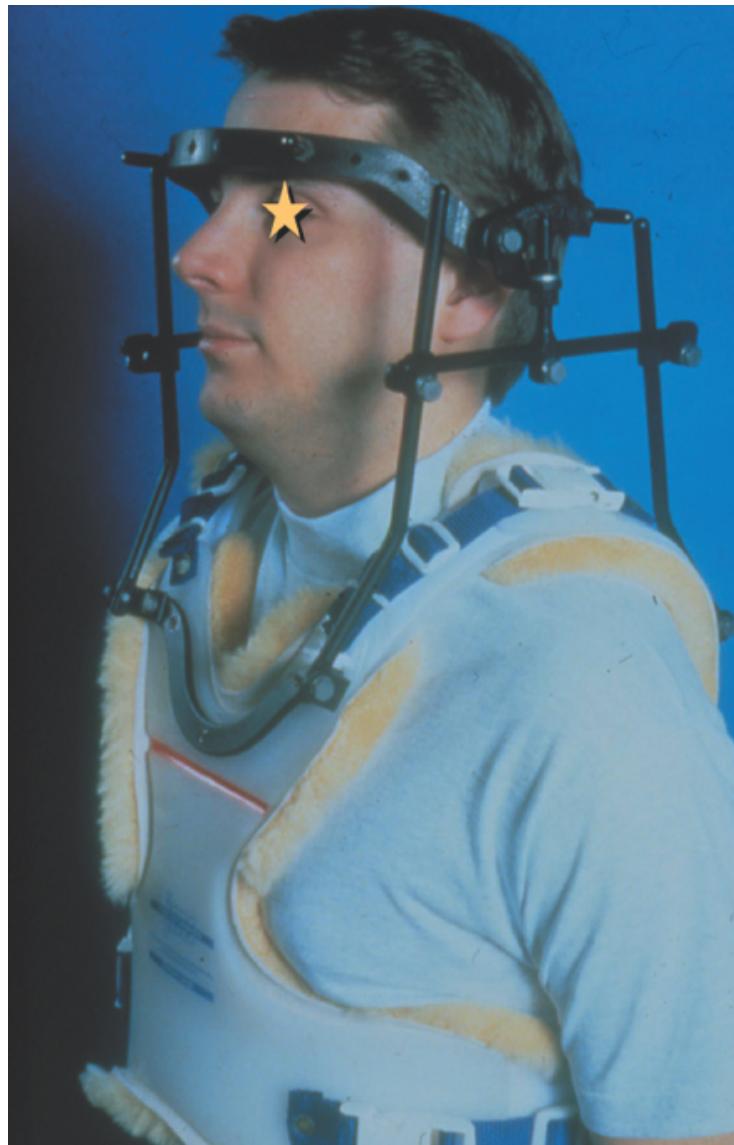
A halo device may be used initially with traction or may be applied after removal of the tongs. It consists of a titanium or stainless-steel halo ring that is fixed to the skull by four pins, which are inserted into the outer table of the skull. The ring is attached to a removable halo vest, a device that suspends the weight of the unit circumferentially around the chest. A frame connects the ring to the chest. Halo devices provide immobilization of the cervical spine while allowing early ambulation (see Fig. 63-6) for patients with adequate function.

Thoracic and lumbar injuries are usually treated with surgical intervention, followed by immobilization with a fitted brace. Traction is often not indicated either before or after surgery, due to the relative stability of the spine in these regions.



#### Quality and Safety Nursing Alert

*The patient's vital organ functions and body defenses must be supported and maintained until spinal and neurogenic shock abates and the neurologic system has recovered from the traumatic insult; this can take up to 4 months.*



**Figure 63-6 •** Halo and vest for cervical and thoracic injuries. Adapted from Schwartz, E. D., Adam, E., & Flander, S. (2007). *Spinal trauma: Imaging, diagnosis, and management*. Philadelphia, PA: Lippincott Williams & Wilkins.

## Surgical Management

Surgery is indicated in any of the following situations:

- Compression of the cord is evident.
- The injury results in a fragmented or unstable vertebral body.
- The injury involves a wound that penetrates the cord.
- Bony fragments are in the spinal canal.
- The patient's neurologic status is deteriorating.

Early surgical stabilization may improve the clinical outcome of patients compared to surgery performed later during the clinical course. The goals of surgical treatment are to preserve neurologic function by removing pressure from the spinal cord and to provide stability.

## Management of Acute Complications of Spinal Cord Injury

### Spinal and Neurogenic Shock

The spinal shock associated with SCI reflects a sudden depression of reflex activity in the spinal cord, called areflexia, that occurs below the level of injury. The muscles innervated by the part of the spinal cord segment below the level of the lesion are without sensation, paralyzed, and flaccid, and the reflexes are absent. Blood pressure may be decreased, and the patient may be bradycardic. Hypotension and shock can further damage the spinal cord; therefore, the mean arterial pressure (MAP) should be maintained at 85 mm Hg or higher during the hyperacute phase. The reflexes that initiate bladder and bowel function are affected. Bowel distention and paralytic ileus can be caused by depression of the reflexes and are treated with intestinal decompression by insertion of a nasogastric tube. A paralytic ileus most often occurs within the first 2 to 3 days after SCI and resolves within 3 to 7 days.

Neurogenic shock develops as a result of the loss of autonomic nervous system function below the level of the lesion. The vital organs are affected, causing decreases in blood pressure, heart rate, and cardiac output, as well as venous pooling in the extremities and peripheral vasodilation (Volski & Ackerman, 2020). In addition, the patient does not perspire in the paralyzed portions of the body, because sympathetic activity is blocked; therefore, close observation is required for early detection of an abrupt onset of fever. See [Chapter 11](#) for further discussion on neurogenic shock.

With injuries to the cervical and upper thoracic spinal cord, innervation to the major accessory muscles of respiration is lost and respiratory problems develop. These include decreased vital capacity, retention of secretions, increased partial pressure of arterial carbon dioxide ( $\text{PaCO}_2$ ) levels and decreased oxygen levels, respiratory failure, and pulmonary edema.

### Venous Thromboembolism

The risk for venous thromboembolism (VTE) is a potential complication of immobility and occurs in patients with SCI at the same rate as those having had other types of traumatic injuries (Wang, Strayer, Harris, et al., 2017). Patients who develop VTE are at high risk for both deep vein thrombosis (DVT) and PE due to immobility, flaccidity, and decreased vasomotor tone (Wang et al., 2017).

Manifestations of PE include pleuritic chest pain, anxiety, shortness of breath, and abnormal blood gas values (increased  $\text{PaCO}_2$  and decreased  $\text{PaO}_2$ ). A fatal PE

has been reported in up to 2% of patients with SCI within the first 3 months after injury (Hickey & Strayer, 2020).

Low-dose anticoagulation therapy usually is initiated to prevent DVT and PE, along with the use of anti-embolism stockings or sequential pneumatic compression devices (SCDs). In some cases, permanent indwelling filters may be placed prophylactically in the vena cava to prevent emboli (dislodged clots) from migrating to the lungs and causing PE. Prevention continues into the rehabilitation and chronic phases of SCI care (Abrams & Wakasa, 2019). See [Chapter 26](#) for further discussion on VTE.



#### **Quality and Safety Nursing Alert**

*The calves or thighs of a patient who is immobile should never be massaged because of the danger of dislodging an undetected thromboembolus.*

### **Other Complications**

In addition to the respiratory complications (respiratory failure, pneumonia) and autonomic dysreflexia, other complications that may occur include pressure injuries and infection (urinary, respiratory, and local infection at the skeletal traction pin sites).

## NURSING PROCESS

### The Patient with Acute Spinal Cord Injury



#### Assessment

The patient's breathing pattern and the strength of the cough are assessed, and the lungs are auscultated, because paralysis of the diaphragm, in addition to abdominal and respiratory muscles, diminishes coughing and makes clearing of bronchial and pharyngeal secretions difficult. Reduced excursion of the chest also results.

The patient is monitored closely for any changes in motor or sensory function and for symptoms of progressive neurologic damage. In the early stages of SCI, determining whether the cord has been severed may not be possible, because signs and symptoms of cord edema are indistinguishable from those of cord transection. Edema of the spinal cord may occur with any severe cord injury and may further compromise spinal cord function.

Motor and sensory functions are assessed through careful neurologic examination. These findings are recorded on a flow sheet so that changes in the baseline neurologic status can be monitored closely and accurately. The ASIA classification is commonly used to describe the level of function for patients with SCI (see Fig. 63-4). Chart 63-7 also gives examples of the effects of altered spinal cord function. At the minimum:

- Motor ability is tested by asking the patient to spread the fingers, squeeze the examiner's hand, and move the toes or turn the feet.
- Sensation is evaluated by gently pinching the skin or touching it lightly with an object such as a tongue blade, starting at shoulder level and working down both sides of the extremities. The patient should have both eyes closed so that the examination reveals true findings, not what the patient hopes to feel. The patient is asked where the sensation is felt.
- Any decrease in neurologic function is reported immediately.

The patient is also assessed for spinal shock, which is a complete loss of all reflex, motor, sensory, and autonomic activity below the level of the lesion that causes bladder paralysis and distention. The lower abdomen is palpated for signs of urinary retention and overdistention of the bladder. Further assessment is made for gastric dilation and paralytic ileus caused by an atonic bowel, a result of autonomic disruption.

Temperature is monitored because the patient may have periods of hyperthermia as a result of altered temperature control, which is due to the inability to perspire related to autonomic disruption. Body temperature becomes dependent on surroundings (poikilothermia).

#### Diagnosis

### **NURSING DIAGNOSES**

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired breathing associated with weakness or paralysis of diaphragm, abdominal, and intercostal muscles
- Impaired airway clearance associated with muscle weakness and inability to clear secretions
- Impaired mobility in bed and impaired mobility associated with motor and sensory impairments
- Risk for injury associated with motor and sensory impairment
- Risk for impaired skin integrity associated with immobility and sensory loss
- Urinary retention associated with inability to void spontaneously
- Constipation associated with presence of atonic bowel as a result of autonomic disruption
- Acute pain associated with treatment and prolonged immobility
- Autonomic dysreflexia associated with uninhibited sympathetic response of the nervous system following SCI

### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- VTE
- Orthostatic hypotension

### **Planning and Goals**

The goals for the patient may include improved breathing pattern and airway clearance, improved mobility, prevention of injury due to sensory impairment, maintenance of skin integrity, relief of urinary retention, improved bowel function, decreasing pain, early recognition of autonomic dysreflexia, and absence of complications.

### **Nursing Interventions**

#### **PROMOTING ADEQUATE BREATHING AND AIRWAY CLEARANCE**

Possible impending respiratory failure is detected by observing the patient, measuring vital capacity, monitoring oxygen saturation through pulse oximetry, and monitoring arterial blood gases. Early and vigorous attention to clearing bronchial and pharyngeal secretions can prevent retention of secretions and atelectasis. Suctioning may be indicated but should be used with caution to avoid stimulating the vagus nerve and producing bradycardia and cardiac arrest.

If the patient cannot cough effectively because of decreased inspiratory volume and inability to generate sufficient expiratory pressure, chest physiotherapy and assisted coughing may be indicated. Specific breathing exercises are supervised by the nurse to increase the strength and endurance of the inspiratory muscles, particularly the diaphragm. Assisted coughing

promotes clearing of secretions from the upper respiratory tract and is similar to the use of abdominal thrusts to clear an airway. Assisted coughing can be more effective than traditional suctioning because traditional suctioning clears the right mainstem bronchus, whereas sites for atelectasis and pneumonia are most commonly in the left lower lung lobe (Wang et al., 2017). Proper humidification and hydration are important to prevent secretions from becoming thick and difficult to remove even with coughing. The patient is assessed for signs of respiratory infection (e.g., cough, fever, dyspnea). Ascending edema of the spinal cord in the acute phase may cause respiratory difficulty that requires immediate intervention. Therefore, the patient's respiratory status must be monitored closely.

#### **IMPROVING MOBILITY**

Proper body alignment is maintained at all times. If not on a specialized rotating bed, the patient should not be turned until the primary provider has indicated that it is safe to do so. Once safe for movement, the patient is repositioned frequently and is assisted out of bed as soon as the spinal column is stabilized. Various types of splints are used to prevent footdrop, which can often occur. When used, the splints are removed and reapplied every 2 hours. Trochanter rolls, applied from the crest of the ilium to the midthigh of both legs, help prevent external rotation of the hip joints. Patients with lesions above the midthoracic level have loss of sympathetic control of peripheral vasoconstrictor activity, leading to hypotension. These patients may tolerate changes in position poorly and require monitoring of blood pressure when positions are changed.

Contractures can develop rapidly with immobility and muscle paralysis. A joint that is immobilized too long becomes fixed as a result of contractures of the tendon and joint capsule. Atrophy of the extremities results from disuse. Contractures and other complications may be prevented by range-of-motion exercises that help preserve joint motion and stimulate circulation. Passive range-of-motion exercises should be implemented as soon as possible after the injury. Toes, metatarsals, ankles, knees, and hips should be put through a full range of motion at least four, or ideally five, times daily.

For most patients who have a cervical fracture without neurologic deficit, reduction in traction followed by rigid immobilization for 6 to 8 weeks restores skeletal integrity. These patients are allowed to move gradually to an erect position. A neck brace or molded collar is applied when the patient is mobilized after traction is removed (see Fig. 63-5).

#### **PREVENTING INJURY DUE TO SENSORY AND PERCEPTUAL ALTERATIONS**

The nurse assists the patient to compensate for sensory and perceptual alterations that occur with SCI. The intact senses above the level of the injury are stimulated through touch, aromas, flavorful food and beverages, conversation, and music. Additional strategies include the following:

- Providing glasses to enable the patient to see from the supine position

- Encouraging the use of hearing aids, if indicated, to enable the patient to hear conversations and environmental sounds
- Providing emotional support to the patient and family
- Educating the patient and family about strategies to compensate for, or cope with, sensory deficits

#### **MAINTAINING SKIN INTEGRITY**

Pressure injuries are a significant complication of SCI. They may begin within hours of an acute SCI where pressure is continuous and where the peripheral circulation is inadequate as a result of spinal shock and a recumbent position. It is important to move the patient from the backboard as soon as possible and inspect the skin. In addition, patients who wear cervical collars for prolonged periods may develop breakdown from the pressure of the collar under the chin, on the shoulders, and at the occiput. Pressure injury can add substantially to the personal and economic costs of living with SCI.

The most effective approach to addressing this costly complication of SCI is prevention. The patient's position is changed at least every 2 hours. Turning not only assists in the prevention of pressure injuries but also prevents pooling of blood and edema in the dependent areas. Careful inspection of the skin is made each time the patient is turned. The skin over the pressure points is assessed for redness or breaks; the perineum is checked for soilage, and the catheter is observed for adequate drainage. The patient's general body alignment and comfort are assessed. Special attention should be given to pressure areas in contact with the transfer board.

In addition, the patient's skin should be kept clean by washing with a mild soap, rinsing well, and blotting dry. Pressure-sensitive areas should be kept well lubricated and soft with oil or emollient lotion. The patient is educated about the danger of pressure injuries and is encouraged to take control and make decisions about appropriate skin care. See [Chapter 56](#) for other aspects of care and prevention of pressure injuries.

#### **MAINTAINING URINARY ELIMINATION**

Immediately after SCI, the urinary bladder becomes atonic and cannot contract by reflex activity. Urinary retention is the immediate result. During the initial acute phase, a urinary catheter is inserted; however, prompt discontinuation is advised due to high risk for catheter-associated urinary tract infection (CAUTI). Once discontinued, the patient has no sensation of bladder distention and overstretching of the bladder and detrusor muscle may occur, delaying the return of bladder function.

Intermittent catheterization is carried out to avoid overdistention of the bladder and high risk for UTI due to retention of urine. At an early stage, family members are shown how to carry out intermittent catheterization and are encouraged to participate in this facet of care, because they will be involved in long-term follow-up and must be able to recognize complications so that treatment can be instituted.

The patient is educated to record fluid intake, voiding pattern, amounts of residual urine after voiding, characteristics of urine, and any unusual sensations that may occur. The management of a **neurogenic bladder** (bladder dysfunction that results from a disorder or dysfunction of the nervous system) is addressed during the rehabilitation phase of care. The types of neurogenic bladder differ according to which motor or sensory pathways are disrupted (Hickey & Strayer, 2020).

External catheters (condom catheters) and leg bags to collect spontaneous voidings are useful for male patients with reflex or total incontinence. The appropriate design and size must be chosen for maximal success, and the patient or caregiver must be taught how to apply the condom catheter and how to provide daily hygiene including skin inspection. Instruction on emptying the leg bag must also be provided, and modifications can be made for patients with limited hand dexterity.

#### **IMPROVING BOWEL FUNCTION**

Immediately after SCI, a paralytic ileus usually develops as a result of neurogenic paralysis of the bowel; therefore, a nasogastric tube is often required to relieve distention and to prevent vomiting and aspiration (Stoffel, Van der Aa, Wittmann, et al., 2018).

Bowel activity usually returns within the first week. With the intake of nutrition, it is important to establish a bowel program. A bowel program can help to control bowel movements by establishing a pattern of planned evacuation. The nurse administers prescribed combinations of stool softeners, stimulant laxatives, bulking laxatives, and rectal laxatives along with rectal stimulation, to counteract the effects of immobility and analgesic agents (Stoffel et al., 2018).

#### **PROVIDING COMFORT MEASURES: THE PATIENT IN TRACTION WITH TONGS OR HALO VEST**

A patient who has had pins, tongs, or calipers placed for cervical stabilization may have a headache or discomfort for several days after the pins are inserted. Patients initially may be bothered by the rather startling appearance of these devices, but usually they readily adapt to it because the device provides comfort for the unstable neck (see Fig. 63-6). The patient may complain of being caged in and of noise created by any object coming in contact with the frame of a halo device, but they can be reassured that adaptation will occur.

The areas around the four pin sites of a halo device are cleaned at least daily and observed for redness, drainage, and pain. The pins are observed for loosening, which may contribute to infection. If one of the pins becomes detached, the head is stabilized in a neutral position by one person, while another notifies the primary provider. A torque screwdriver should be readily available in case the screws on the frame need tightening.

The skin under the halo vest is inspected for excessive perspiration, redness, and skin blistering, especially on the bony prominences. The vest is opened at the sides to allow the torso to be washed. The liner of the vest should not

become wet, because dampness causes skin excoriation. Powder is not used inside the vest, because it may contribute to the development of pressure injuries. The liner should be changed periodically to promote hygiene and good skin care. If the patient is to be discharged with the vest, detailed instructions must be given to the family, with time allowed for them to demonstrate the necessary skills of halo vest care (see [Chart 63-8](#)).

#### **RECOGNIZING AUTONOMIC DYSREFLEXIA**

**Autonomic dysreflexia**, also known as autonomic hyperreflexia, is an acute life-threatening emergency that occurs as a result of exaggerated autonomic responses to stimuli that are harmless in people without SCI. It occurs only after spinal shock has resolved. This syndrome is characterized by a severe, pounding headache with paroxysmal hypertension, profuse diaphoresis above the spinal level of the lesion (most often of the forehead), nausea, nasal congestion, and bradycardia. It occurs among patients with cord lesions above T6 (the sympathetic visceral outflow level) after spinal shock has subsided. The sudden increase in blood pressure may cause retinal hemorrhage, hemorrhagic stroke, myocardial infarction, or seizures (Hickey & Strayer, 2020). A number of stimuli may trigger this reflex: distended bladder (the most common cause); distention or contraction of the visceral organs, especially the bowel (from constipation, impaction); or stimulation of the skin (tactile, pain, thermal stimuli, pressure injury). Because this is an emergency situation, the objectives are to remove the triggering stimulus and to avoid the possibility of serious complications (Eldahan & Rabchevsky, 2018).

The following measures are carried out:

- The patient is placed immediately in a sitting position to lower the blood pressure.

**Chart 63-8**



#### **HOME CARE CHECKLIST**

## The Patient with a Halo Vest

**At the completion of education, the patient and/or caregiver will be able to:**

- Name the procedure that was performed and identify any permanent changes in anatomic structure or function as well as changes in ADLs, IADLs, roles, relationships, and spirituality.
- Describe the rationale for the use of the halo vest.
- State how to contact all members of the treatment team (e.g., health care providers, home care professionals, rehabilitation team, and durable medical equipment and supply vendor).
- State changes in lifestyle (e.g., diet, ADLs, IADLs, activity) necessary for recovery and health maintenance, as applicable.
  - Demonstrate safe techniques to assist the patient with self-care, hygiene, and ambulation.
  - Demonstrate assessment of frame, traction, tongs, and pins.
  - Demonstrate pin care using correct technique.
  - Demonstrate care of skin, including assessment (e.g., reddened or irritated areas, breakdown).
  - Identify signs and symptoms of infection.
  - Explain the reasons for and the method for changing the vest liner.
  - Identify holistic measures of pain management.
- Identify signs and symptoms of complications (e.g., venous thromboembolism, respiratory impairment, urinary tract infection).
- Describe emergency measures if respiratory or other complications develop while the patient is in the halo vest or if the frame becomes dislodged.
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.
- Identify sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families.
- Identify the need for health-promotion, disease prevention, and screening activities.

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

- Rapid assessment is performed to identify and alleviate the cause.
- The bladder is emptied immediately via a urinary catheter. If an indwelling catheter is not patent, it is irrigated or replaced with another catheter.
- The rectum is examined for a fecal mass. If present, a topical anesthetic agent is inserted 10 to 15 minutes before the mass is

removed, because visceral distention or contraction can cause autonomic dysreflexia.

- The skin is examined for any areas of pressure, irritation, or broken skin.
- Any other stimulus that could be the triggering event, such as an object next to the skin or a draft of cold air, must be removed.
- If these measures do not relieve the hypertension and excruciating headache, antihypertensive medications may be prescribed and given slowly by the IV route.
- The medical record is labeled with a clearly visible indicator concerning the risk for autonomic dysreflexia.
- The patient is instructed about prevention and management measures.
- Any patient with a lesion above the T6 segment is informed that such an episode is possible and may occur even many years after the initial injury.

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Patients are at high risk for VTE after SCI. The patient must be assessed for symptoms of VTE including DVT and PE. Chest pain, shortness of breath, and changes in arterial blood gas values must be reported promptly to the primary provider. The circumferences of the thighs and calves are measured and recorded daily; further diagnostic studies are performed if a significant increase is noted. Patients remain at high risk for thrombophlebitis for several months after the initial injury. Patients with paraplegia or tetraplegia are at increased risk for the rest of their lives. Immobilization and the associated venous stasis, as well as varying degrees of autonomic disruption, contribute to the high risk and susceptibility for DVT.

Anticoagulation should be initiated within 72 hours of injury and continued for at least 3 months (Abrams & Wakasa, 2019). The use of low-molecular-weight heparin or low-dose unfractionated heparin may be followed by long-term oral anticoagulation (i.e., warfarin). Additional measures such as range-of-motion exercises, anti-embolism stockings, and adequate hydration are important preventive measures. SCDs may also be used to reduce venous pooling and promote venous return. It is also important to avoid external pressure on the lower extremities that may result from flexion of the knees while the patient is in bed.

**Orthostatic Hypotension.** For the first 2 weeks after SCI, the blood pressure tends to be unstable and can be quite low. It gradually returns to preinjury levels, but periodic episodes of severe orthostatic hypotension frequently interfere with efforts to mobilize the patient. Interruption in the reflex arcs that normally produce vasoconstriction in the upright position, coupled with vasodilation and pooling in abdominal and lower extremity vessels, can result in hypotension. Orthostatic hypotension is a particularly common problem for patients with lesions above T7. In some patients with tetraplegia, even slight elevations of the head can result in blood pressure dysregulation.

A number of techniques can be used to reduce the frequency of hypotensive episodes. Close monitoring of vital signs before and during position changes is essential. Optimization of fluid status and vasopressor medication can be used to treat the profound vasodilation. Anti-embolism stockings should be applied to improve venous return from the lower extremities. Abdominal binders may also be used to encourage venous return and provide diaphragmatic support when the patient is upright (Abrams & Wakasa, 2019). Activity should be planned in advance, and adequate time should be allowed for a slow progression of position changes from recumbent to sitting and upright. Tilt tables frequently are helpful in assisting patients to make this transition.

#### PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



**Educating Patients About Self-Care.** In most cases, patients with SCI (i.e., patients with tetraplegia or paraplegia) need long-term rehabilitation. The process begins during hospitalization as acute symptoms begin to subside or come under better control and the overall deficits and long-term effects of the injury become clear. The goals begin to shift from merely surviving the injury to learning strategies necessary to cope with the alterations that the injury imposes on activities of daily living (ADLs). The emphasis shifts from ensuring that the patient is stable and free of complications to specific assessment and planning designed to meet the patient's rehabilitation needs. Patient education may initially focus on the injury and its effects on mobility; dressing; and bowel, bladder, and sexual function. As the patient and family acknowledge the consequences of the injury and the resulting disability, the focus of education broadens to address issues necessary for carrying out the tasks of daily living and taking charge of their lives. Education must begin in the acute phase and continue throughout rehabilitation and the patient's entire life as changes occur, the patient ages, and problems arise.

Caring for the patient with SCI at home may at first seem a daunting task to the family. They will require dedicated nursing support to gradually assume full care of the patient. Although maintaining function and preventing complications will remain important, goals regarding self-care and preparation for discharge will assist in a smooth transition to rehabilitation and eventually to the community.

**Continuing and Transitional Care.** The goal of the rehabilitation process is independence. The nurse becomes a support to both the patient and the family, assisting them to assume responsibility for increasing aspects of patient care and management. Care for the patient with SCI involves members of all health care disciplines, which may include nursing, medicine, rehabilitation, respiratory therapy, physical and occupational therapy, case management, and social services. The nurse often serves as a coordinator of the management team and as a liaison with rehabilitation centers and home care agencies.

There are many challenges in providing care to the patient with SCI; meeting their psychological needs can be particularly challenging (Bibi,

Rasmussen, & McLiesh, 2018). The patient and family often require assistance in dealing with the psychological impact of the injury and its consequences; referral to a psychiatric clinical nurse specialist or other mental health care professional often is helpful. Therapeutic horseback riding may help increase balance, muscle strength, and self-esteem (Stergiou, Tzoufi, Ntzani, et al., 2017).

The nurse should reassure female patients with SCI that pregnancy is not contraindicated and fertility is relatively unaffected, but that pregnant women with acute or chronic SCI pose unique management challenges. The normal physiologic changes of pregnancy may predispose women with SCI to many potentially life-threatening complications, including autonomic dysreflexia, pyelonephritis, respiratory insufficiency, thrombophlebitis, PE, and unattended delivery. Preconception assessment and counseling are strongly recommended to ensure that the woman is in optimal health and to increase the likelihood of an uneventful pregnancy and healthy outcomes (Crane, Doody, Schiff, et al., 2019).

As more patients survive acute SCI, they face the changes associated with aging with a disability. Three common secondary health problems experienced by persons living with SCI include chronic pain, spasticity, and depression (Abrams & Wakasa, 2019). Education in the home and community focuses on health promotion and addresses the need to minimize risk factors (e.g., tobacco use, substance use disorder, obesity). Routine health-screening and preventive services are needed for the older adult with SCI for early detection of secondary health problems. Home health nurses and others who have contact with patients with SCI are in a position to educate patients about healthy lifestyles, remind them of the need for health screenings, and make referrals as appropriate. Assisting patients to identify accessible health care providers, clinical facilities, and imaging centers may increase the likelihood that they will participate in health screening.

### Evaluation

Expected patient outcomes may include:

1. Demonstrates improvement in gas exchange and clearance of secretions, as evidenced by normal breath sounds on auscultation
  - a. Breathes easily without shortness of breath
  - b. Performs hourly deep breathing exercises, coughs effectively, and clears pulmonary secretions
  - c. Is free of respiratory infection (e.g., has temperature, respiratory rate, and pulse within normal limits; breath sounds clear to auscultation; absence of purulent sputum)
2. Moves within limits of the dysfunction and demonstrates completion of exercises within functional limitations
3. Avoids injury due to sensory, motor, and perceptual alterations

- a. Uses assistive devices (e.g., glasses, hearing aids, electronic devices) as indicated
- b. Describes sensory, motor, and perceptual alterations as a consequence of injury
4. Demonstrates optimal skin integrity
  - a. Exhibits normal skin turgor; skin is free of reddened areas or breaks
  - b. Participates in skin care and monitoring procedures within functional limitations
5. Regains urinary bladder function
  - a. Exhibits no signs of UTI (e.g., has temperature within normal limits; voids clear, dilute urine)
  - b. Has adequate fluid intake
  - c. Participates in bladder training program within functional limitations
6. Regains bowel function
  - a. Reports regular pattern of bowel movement
  - b. Consumes adequate dietary fiber and oral fluids
  - c. Participates in bowel training program within functional limitations
7. Reports absence of pain and discomfort
8. Recognizes manifestations of autonomic dysreflexia if they occur (e.g., headache, diaphoresis, nasal congestion, bradycardia, or diaphoresis)
9. Is free of complications
  - a. Demonstrates no signs of thrombophlebitis, DVT, or PE
  - b. Maintains blood pressure within normal limits
  - c. Reports no lightheadedness with position changes

## Medical Management of Long-Term Complications of SCI

The patient faces a lifetime disability, requiring ongoing follow-up and care. The expertise of a number of health professionals including physicians (specifically a physiatrist), rehabilitation nurses, occupational therapists, physical therapists, psychologists, social workers, rehabilitation engineers, and vocational counselors, is necessary at different times as the need arises.

The patient with an SCI has a shorter life expectancy compared to those who have not had an SCI (Abrams & Wakasa, 2019). As patients with SCI age, they have many of the same medical problems as others. In addition, they face the threat of complications associated with their disability (Hickey & Strayer, 2020). Usually, patients are encouraged to follow-up at an outpatient spinal cord clinic when complications and other issues arise. Lifetime care includes assessment of the urinary tract at prescribed intervals because there is the likelihood of continuing alteration in detrusor and sphincter function, and the patient is prone to UTI (Abrams & Wakasa, 2019).

Long-term problems and complications of SCI include disuse syndrome, autonomic dysreflexia (discussed earlier), bladder and kidney infections, spasticity, and depression (Abrams & Wakasa, 2019). Pressure injuries with potential complications of sepsis, osteomyelitis, and fistulas occur in about 10% of patients. Spasticity may be particularly disabling. Heterotopic ossification (overgrowth of bone) in the hips, knees, shoulders, and elbows occurs in many patients after SCI. Spasticity and heterotopic ossification are painful and can produce a loss of range of motion (Abrams & Wakasa, 2019). Management includes observing for and addressing any alteration in physiologic status and psychological outlook, as well as the prevention and treatment of long-term complications. The nursing role involves emphasizing the need for vigilance in self-assessment and care.

## NURSING PROCESS

### The Patient with Tetraplegia or Paraplegia

#### Assessment

Assessment focuses on the patient's general condition, complications, and how the patient is managing at that particular point in time. A head-to-toe assessment and review of systems should be part of the database, with emphasis on the areas that are prone to problems in this population. A thorough inspection of all areas of the skin for redness or breakdown is critical. The nurse reviews the established bowel and bladder program with the patient, because the program must continue uninterrupted. Patients with tetraplegia or paraplegia have varying degrees of loss of motor power, deep and superficial sensation, vasomotor control, bladder and bowel control, and sexual function. They are faced with potential complications related to immobility, skin breakdown and pressure injuries, recurring UTIs, and contractures. Knowledge about these particular issues can further guide the assessment in any setting. Nurses in all settings, including home care, must be aware of these potential complications in the lifetime management of these patients.

An understanding of the emotional and psychological responses to tetraplegia or paraplegia is achieved by observing the responses and behaviors of the patient and family and by listening to their concerns (Bailey, Gammage, van Ingen, et al., 2017). Documenting these assessments and reviewing the plan with the entire team on a regular basis provide insight into how both the patient and the family are coping with the changes in lifestyle and body functioning. Additional information frequently can be gathered from the social worker or psychiatric/mental health worker.

It takes time for the patient and family to comprehend the magnitude of the disability. They may go through stages of grief, including shock, disbelief, denial, anger, depression, and acceptance. During the acute phase of the injury, denial can be a protective mechanism to shield the patient from the acute reality of what has happened. As the patient realizes the permanent nature of paraplegia or tetraplegia, the grieving process may be prolonged and all-encompassing because of the recognition that long-held plans and expectations are interrupted or permanently altered. A period of depression often follows as the patient experiences a loss of self-esteem in areas of self-identity, sexual functioning, and social and emotional roles. Exploration and assessment of these issues can assist in developing a meaningful plan of care.

#### Diagnosis

#### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired mobility in bed and impaired mobility associated with loss of motor function

- Risk for disuse
- Risk for impaired skin integrity associated with permanent sensory loss and immobility
- Urinary retention associated with level of injury
- Constipation associated with effects of spinal cord disruption
- Impaired sexual functioning associated with neurologic dysfunction
- Difficulty coping associated with impact of disability on daily living
- Lack of knowledge about requirements for long-term management

#### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Spasticity
- Infection and sepsis

#### **Planning and Goals**

The goals for the patient may include attainment of some form of mobility; maintenance of healthy, intact skin; achievement of bladder management without infection; achievement of bowel control; achievement of sexual expression; strengthening of coping mechanisms; knowledge of long-term management; and absence of complications.

#### **Nursing Interventions**

The patient requires extensive rehabilitation, which is less difficult if appropriate nursing management has been carried out during the acute phase of the injury or illness. Nursing care is one of the key factors determining the success of the rehabilitation program. The main objective is for the patient to live as independently as possible in the home and community.

#### **INCREASING MOBILITY**

**Exercise Programs.** The unaffected parts of the body are built up to optimal strength to promote maximum self-care. The muscles of the hands, arms, shoulders, chest, spine, abdomen, and neck must be strengthened in the patient with paraplegia, because they must bear full weight on these muscles to ambulate. The triceps and the latissimus dorsi are important muscles used in crutch walking. The muscles of the abdomen and the back also are necessary for balance and for maintaining the upright position.

To strengthen these muscles, the patient can do push-ups when in a prone position and sit-ups when in a sitting position. Extending the arms while holding weights (traction weights can be used) also develops muscle strength. Squeezing rubber balls or crumpling newspaper promotes hand strength.

With encouragement from all members of the rehabilitation team, the patient with paraplegia can develop the increased exercise tolerance needed for gait training and ambulation activities. The importance of maintaining cardiovascular fitness is stressed to the patient. Alternative exercises to increase the heart rate to target levels must be designed within the patient's abilities.

**Mobilization.** After the spine is stable enough to allow the patient to assume an upright posture, mobilization activities are initiated. A brace or vest may be used, depending on the level of the lesion. The sooner muscles are used, the less chance there is of disuse atrophy. The earlier the patient is brought to a standing position, the less opportunity there is for osteoporotic changes to take place in the long bones. Weight bearing also reduces the possibility of renal calculi and enhances many other metabolic processes.

Braces and crutches enable some patients with paraplegia to ambulate for short distances. Ambulation using crutches requires a high expenditure of energy. Motorized wheelchairs and specially equipped vans can provide greater independence and mobility for patients with high-level SCI or other lesions. Every effort should be made to encourage the patient to be as mobile and active as possible.



Long-term risks include altered body composition, a decrease in lean body mass, decreased bone mineral density, and increased body mass index (BMI). Patients are at high risk for obesity due to high fat intake combined with decreased physical activity (Silveira, Winter, Clark, et al., 2019). This puts patients at higher risk for developing comorbid conditions such as diabetes and cardiovascular diseases. Patients benefit from nutrition counseling to prevent these secondary complications. For those who are overweight or who have obesity, weight-loss programs must be designed to accommodate the dietary and physical activity barriers that are unique to this population.

#### **PREVENTING DISUSE SYNDROME**

Patients are at high risk for development of contractures as a result of disuse syndrome due to the musculoskeletal system changes (atrophy) brought about by the loss of motor and sensory functions below the level of injury. Range-of-motion exercises must be provided at least four times a day, and care is taken to stretch the Achilles tendon with exercises, to prevent footdrop. The patient is repositioned frequently and is maintained in proper body alignment whether in bed or in a wheelchair.

Contractures can complicate day-to-day care, increasing the difficulty of positioning and decreasing mobility. A number of surgical procedures have been tried, with varying degrees of success. These techniques are used if more conservative approaches fail, but the best treatment is prevention.

#### **PROMOTING SKIN INTEGRITY**

Because these patients spend a great portion of their lives in wheelchairs, pressure injuries are an ever-present threat. Contributing factors are permanent sensory loss over pressure areas; immobility, which makes relief of pressure difficult; trauma from bumps (against the wheelchair, toilet, furniture, and so forth) that cause unnoticed abrasions and wounds; loss of protective function of the skin from excoriation and maceration due to excessive perspiration and possible incontinence; and poor general health (anemia, diabetes), leading to

poor tissue perfusion. See [Chapter 56](#) for discussion on the prevention and management of pressure injuries.

The person with tetraplegia or paraplegia must take responsibility for monitoring (or directing monitoring) of their skin status. This involves relieving pressure and not remaining in any position for longer than 2 hours, in addition to ensuring that the skin receives meticulous attention and cleansing. The patient is educated that pressure injuries develop over bony prominences that are exposed to unrelieved pressure in the sitting and lying positions. The most vulnerable areas are identified. The patient with paraplegia is instructed to use mirrors, if possible, to inspect these areas morning and night, observing for redness, slight edema, or any abrasions. While in bed, the patient should turn at 2-hour intervals and then inspect the skin again for redness that does not fade on pressure. The bottom sheet should be checked for wetness and for creases. The patient with tetraplegia or paraplegia, who cannot perform these activities, is encouraged to direct others to check these areas and prevent pressure injuries from developing.

The patient is educated to relieve pressure while in the wheelchair by doing push-ups, leaning from side to side to relieve ischial pressure, and tilting forward while leaning on a table. The caregiver for the patient with tetraplegia will need to perform these activities if the patient cannot do so independently. A wheelchair cushion is prescribed to meet individual needs, which may change in time with changes in posture, weight, and skin tolerance. A referral can be made to a rehabilitation engineer, who can measure pressure levels while the patient is sitting and then tailor the cushion and other necessary aids and assistive devices to the patient's needs.

The diet for the patient with tetraplegia or paraplegia should be high in protein, vitamins, and calories to ensure minimal wasting of muscle and the maintenance of healthy skin, and high in fluids to maintain well-functioning kidneys. Excessive weight gain and obesity should be avoided because they further limit mobility.

#### **IMPROVING BLADDER MANAGEMENT**

The effect of the spinal cord lesion on the bladder depends on the level of injury, the degree of cord damage, and the length of time after injury. A patient with tetraplegia or paraplegia usually has either a reflex or a nonreflex bladder. Both bladder types increase the risk for UTI.

The nurse emphasizes the importance of maintaining an adequate flow of urine by encouraging a fluid intake of about 2.5 L daily. The patient should empty the bladder frequently so that there is minimal residual urine and should pay attention to personal hygiene, because infection of the bladder and kidneys almost always occurs by the ascending route. The perineum must be kept clean and dry, and attention must be given to the perianal skin after defecation. Underwear should be cotton (which is more absorbent) and should be changed at least once a day.

If an external catheter (condom catheter) is used, the sheath is removed nightly; the penis is cleansed to remove urine and is dried carefully, because warm urine on the periurethral skin promotes the growth of bacteria. Attention also is given to the collection bag. The nurse emphasizes the importance of monitoring for signs of UTI: cloudy, foul-smelling urine or hematuria (blood in the urine); fever; or chills.

The female patient who cannot achieve reflex bladder control or self-catheterization may need to wear pads or waterproof undergarments. Surgical intervention may be indicated in some patients to create a urinary diversion.

#### **ESTABLISHING BOWEL CONTROL**

The objective of a bowel training program is to establish bowel evacuation through reflex conditioning. If the SCI occurs above the sacral segments or nerve roots and there is reflex activity, the anal sphincter may be massaged (digital stimulation) to stimulate defecation. If the cord lesion involves the sacral segment or nerve roots, anal massage is not performed because the anus may be relaxed and lack tone. Massage is also contraindicated if there is spasticity of the anal sphincter. The anal sphincter is massaged by inserting a gloved finger (which has been adequately lubricated) 2.5 to 3.7 cm (1 to 1.5 inches) into the rectum and moving it in a circular motion or from side to side. It soon becomes apparent which area triggers the defecation response. This procedure should be performed at regular time intervals (usually every 48 hours), after a meal, and at a time that will be convenient for the patient at home (Schmelzer, Daniels, & Baird, 2018). The patient is educated about the symptoms of impaction (frequent loose stools; constipation) and is cautioned to watch for hemorrhoids. A diet with sufficient fluids and fiber is essential to developing a successful bowel training program, avoiding constipation, and decreasing the risk for autonomic dysreflexia.

#### **COUNSELING ON SEXUAL EXPRESSION**

Many patients with tetraplegia and paraplegia can have some form of meaningful sexual relationship, although modifications are necessary. The patient and partner benefit from counseling about the range of sexual expression possible, special techniques and positions, exploration of body sensations offering sensual feelings, and urinary and bowel hygiene as related to sexual activity. For men with erectile failure, penile prostheses enable them to have and sustain an erection, and medications for erectile dysfunction may be helpful. Sildenafil, vardenafil, and tadalafil, for example, are oral smooth muscle relaxants that cause blood to flow into the penis, resulting in an erection (see [Chapter 53](#)). Patients who are sexually active should undergo counseling in birth control methods, as some methods (e.g., oral contraceptives) may increase risks for complications such as VTE (Hickey & Strayer, 2020).

Sexual education and counseling services are included in the rehabilitation services at spinal centers. Small group meetings in which patients can share

their feelings, receive information, and discuss sexual concerns and practical aspects are helpful in producing effective attitudes and adjustments.

#### **ENHANCING COPING MECHANISMS**

The impact of the disability and loss becomes marked when the patient returns home. Each time something new enters the patient's life (e.g., a new relationship, going to work), the patient is reminded anew of their limitations. Grief reactions and depression are common.

To work through depression, the patient must have some hope for relief in the future. The nurse can encourage the patient to feel confident in their ability to achieve self-care and relative independence. The role of the nurse ranges from caretaker during the acute phase to educator, counselor, and facilitator as the patient gains mobility and independence.

The patient's disability affects not only the patient but also the entire family. In many cases, family therapy is helpful in working through issues as they arise. Adjustment to the disability leads to the development of realistic goals for the future, making the best of the abilities that are left intact, and reinvesting in other activities and relationships. Rejection of the disability causes self-destructive neglect and nonadherence to the therapeutic program, which leads to more frustration and depression. Crises for which interventions may be sought include social, psychological, marital, sexual, and psychiatric problems. The family usually requires counseling, social services, and other support systems to help them cope with the changes in their lifestyle and socioeconomic status.

A major goal of nursing management is to help the patient overcome their sense of futility and to encourage the patient in the emotional adjustment that must be made before they are willing to venture into the outside world. However, an excessively sympathetic attitude on the part of the nurse may cause the patient to develop an overdependence that defeats the purpose of the entire rehabilitation program. The patient is educated and assisted when necessary, but the nurse should avoid performing activities that the patient can do independently with a little effort. This approach to care more than repays itself in the satisfaction of seeing a patient, who is completely demoralized and helpless, become independent and find meaning in a newly emerging lifestyle.

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

**Spasticity.** Muscle spasticity can be a problematic complication of tetraplegia and paraplegia. Flexor or extensor spasms occur below the level of the spinal cord lesion and can interfere with the rehabilitation process, ADLs, and quality of life (Abrams & Wakasa, 2019). Spasticity results from an imbalance between the facilitatory and inhibitory effects on neurons that exist normally. The area of the cord distal to the site of injury or lesion becomes disconnected from the higher inhibitory centers located in the brain, so facilitatory impulses, which originate from muscles, skin, and ligaments, predominate.

Spasticity is defined as a condition of increased muscle tone in a muscle that is weak. Initial resistance to stretching is quickly followed by sudden relaxation. The stimulus that precipitates spasm can be obvious, such as movement or a position change, or subtle, such as a slight jarring of the wheelchair. Most patients with tetraplegia or paraplegia have some degree of spasticity. Because it increases muscle tone, some degree of spasticity can be beneficial in patients who are weak (Abrams & Wakasa, 2019). With SCI, the onset of spasticity usually occurs from a few weeks to 6 months after the injury. The same muscles that are flaccid during the period of spinal shock develop spasticity during recovery. The intensity of spasticity tends to peak approximately 2 years after the injury, after which the spasms tend to regress.

Management of spasticity is based on the severity of symptoms and the degree of incapacitation. Botulinum toxin injections, as well as the antispasmodic medication baclofen, available in an oral and an intrathecal form, may be indicated (Comerford & Durkin, 2020). Oral medications such as diazepam, dantrolene, and tizanidine help control spasms by decreasing sympathetic outflow from the central nervous system. Other forms of adjunctive therapy include oral and transdermal forms of clonidine (Hickey & Strayer, 2020). All of the antispasmodic medications cause drowsiness, weakness, and vertigo in some patients. Passive range-of-motion exercises and frequent turning and repositioning are helpful, because stiffness tends to increase spasticity. These activities also are essential in the prevention of contractures, pressure injuries, and bowel and bladder dysfunction.

**Infection and Sepsis.** Patients with tetraplegia and paraplegia are at increased risk for infection and sepsis from a variety of sources: urinary tract, respiratory tract, and pressure injuries. Sepsis remains a major cause of complications and death in these patients. Prevention of infection and sepsis is essential through maintenance of skin integrity, complete emptying of the bladder at regular intervals, and prevention of urinary and fecal incontinence. The risk for respiratory infection can be decreased by avoiding contact with people who have symptoms of respiratory infection, performing coughing and deep breathing exercises to prevent pooling of respiratory secretions, receiving yearly influenza vaccines, and giving up smoking. A high-protein diet is important in maintaining an adequate immune system, as is avoiding factors that may reduce immune system function, such as excessive stress, drug abuse, and excessive alcohol intake.

If infection occurs, the patient requires thorough assessment and prompt treatment. Antibiotic therapy and adequate hydration, in addition to local measures (depending on the site of infection), are initiated immediately.

UTIs are minimized or prevented by aseptic technique in catheter management, adequate hydration, a bladder training program, and prevention of overdistention of the bladder and urinary stasis.

Skin breakdown and infection are prevented by the maintenance of a turning schedule; frequent back care; regular assessment of all skin areas; regular cleaning and lubrication of the skin; passive range-of-motion exercise

to prevent contractures; pressure relief over broken skin areas, bony prominences, and heels; and wrinkle-free bed linen.

Pulmonary infections are managed and prevented by frequent coughing, turning, and deep breathing exercises and chest physiotherapy; aggressive respiratory care and suctioning of the airway if a tracheostomy is present; assisted coughing as needed; and adequate hydration.

Infections of any kind can be life-threatening. Aggressive nursing interventions are key to prevention, detection, and early management.

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



Educating Patients About Self-Care. Patients with tetraplegia or paraplegia are at risk for complications for the rest of their lives. Therefore, a major aspect of nursing care is educating the patient and family about these complications and about strategies to minimize risks. UTIs, contractures, infected pressure injuries, and sepsis may necessitate hospitalization. Other late complications that may occur include lower extremity edema, joint contractures, respiratory dysfunction, and pain. To avoid these and other complications, the patient and family members are educated about skin care, catheter care, range-of-motion exercises, breathing exercises, and other care techniques. Education is initiated as soon as possible and extends into the rehabilitation or long-term care facility and home. In all aspects of care, it is important for the nurse and patient to set mutual goals and discuss the tasks the patient is capable of doing independently and which tasks the patient needs assistance to complete. See [Chart 63-9](#) for a summary of education points for managing a therapeutic regimen at home.

**Continuing and Transitional Care.** Referral for home care is often appropriate for assessment of the home setting, patient education, and evaluation of the patient's physical and emotional status. During visits by the nurse, education about strategies to prevent or minimize potential complications is reinforced. The home environment is assessed for adequacy of care and for safety. Environmental modifications are made, and specialized equipment is obtained, ideally before the patient goes home.

The nurse also assesses the patient's and the family's adherence to recommendations and their use of coping strategies. The use of inappropriate coping strategies (e.g., drug and alcohol use) is assessed, and referrals to counseling are made for the patient and family. Appropriate and effective coping strategies are reinforced. The nurse reviews previous education and determines the need for further physical or psychological assistance. The patient's self-esteem and body image may be extremely poor at this time. Because people with high levels of social support often report feelings of well-being despite major physical disability, it is beneficial for the nurse to assess and promote further development of the support system and effective coping strategies for each patient. Caregivers and peer mentors play a critical role in

helping patients feel less dependent, experience feelings of freedom, and reintegrate into the community (Gassaway, Jones, Sweatman, et al., 2017).

**Chart 63-9**



### **HOME CARE CHECKLIST**

## Avoiding Complications of SCI

**At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of SCI and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the purpose, dose, route, schedule, side effects, and precautions for prescribed medications.
- State how to contact all members of the treatment team (e.g., health care providers, home care professionals, and durable medical equipment and supply vendor).
- State what types of environmental and safety changes or supports are needed for optimum functioning in the home.
- Demonstrate skin care:
  - Inspect bony prominences every morning and evening.
  - Identify stage I pressure injury and actions to take if present.
  - Change dressings for stage II to IV pressure injuries.
  - State dietary requirements to promote healing of pressure injuries.
  - Demonstrate pressure relief at prescribed intervals.
  - State sitting schedule and demonstrate weight lifts in wheelchair.
  - Demonstrate adherence to bed turning schedule, bed positioning, and the use of bridging techniques.
  - Apply and wear protective boots at prescribed times.
  - Demonstrate correct wheelchair sitting posture.
  - Demonstrate techniques to avoid friction and shear in bed.
  - Demonstrate proper hygiene to maintain skin integrity.
- Demonstrate bladder care:
  - State schedule for voiding, toileting, and catheterization.
  - Identify relationship of fluid intake to voiding and catheterization schedule.
  - Demonstrate clean self-intermittent catheterization and care of catheterization equipment.
  - Demonstrate indwelling catheter care.
  - Demonstrate application of external condom catheter.
  - Demonstrate application, emptying, and cleaning of urinary drainage bag.
  - Demonstrate application of incontinence pads and performance of perineal hygiene.
  - State signs and symptoms of urinary tract infection.
  - State signs and symptoms of urinary catheter blockage.
- Demonstrate bowel care:
  - State optimum dietary intake to promote evacuation.
  - Describe medication regimen for bowel care (drug names, schedule, and dosage).

- Identify schedule for optimum bowel evacuation.
- Demonstrate techniques to increase intraabdominal pressure; Valsalva maneuver; abdominal massage; leaning forward.
- Demonstrate techniques to stimulate bowel movements: ingesting warm liquids; digital stimulation; insertion of suppositories.
- Demonstrate optimum position for bowel evacuation: on toilet with knees higher than hips; left side in bed with knees flexed and head of bed elevated 30 to 45 degrees.
- Identify complications and corrective strategies for bowel retraining: constipation, impaction, diarrhea, hemorrhoids, rectal bleeding, anal tears.
- Identify community resources for peer and caregiver/family support:
  - Identify sources of support (e.g., friends, relatives, faith community).
  - Identify contact details of support services for people with disability and their caregivers/families.
- Demonstrate how to access transportation:
  - Identify locations of wheelchair accessibility for public buses or trains.
  - Identify contact details for private wheelchair van.
  - Contact Division of Motor Vehicles for handicapped parking permit.
  - Contact Division of Motor Vehicles for driving test when appropriate.
  - Identify resources for adapting private vehicle with hand controls or wheelchair lift.
- Identify vocational rehabilitation resources:
  - State the contact details for vocational rehabilitation services.
  - Identify educational opportunities that may lead to future employment.
- Identify community resources for recreation:
  - State local recreation centers that offer programs for people with disability.
  - Identify leisure activities that can be pursued in the community.
  - State how to reach primary provider with questions or if complications arise.
  - State follow-up schedule.
  - Identify the need for health promotion, disease prevention, and screening activities.

ADL, activities of daily living; IADL, instrumental activities of daily living; SCI, spinal cord injury.

The patient requires continuing, lifelong follow-up by the primary provider, physical therapist, and other rehabilitation team members, because the neurologic deficit is usually permanent and new deficits, complications, and secondary conditions can develop. These require prompt attention before they take their toll in additional physical impairment, time, morale, and financial

costs. Research suggests that education and peer mentoring may decrease complications following SCI (Abrams & Wakasa, 2019; Gassaway et al., 2017). The local counselor for the Office of Vocational Rehabilitation works with the patient with respect to job placement or additional educational or vocational training. The nurse is in a good position to remind patients and family members of the need for continuing health promotion and screening practices. Referral to accessible health care providers and imaging centers is important in health promotion and health screening. See [Chapter 7](#) for more information on chronic illness and disability.

### Evaluation

Expected patient outcomes may include:

1. Attains maximum form of mobility
2. Contractures do not develop
3. Maintains healthy, intact skin
4. Achieves bladder control, absence of UTI
5. Achieves bowel control
6. Reports sexual satisfaction
7. Shows improved adaptation to environment and others
8. Exhibits reduction in spasticity
  - a. Reports understanding of the precipitating factors
  - b. Uses measures to reduce spasticity
9. Describes long-term management required
10. Exhibits absence of complications

### CRITICAL THINKING EXERCISES

**1  ebp** You are caring for a 27-year-old woman who was involved in a motor vehicle crash. Her CT scan of the head revealed a left subdural hematoma. The Glasgow Coma Scale was 10, pupils were reactive bilaterally, and she moved all four extremities. She had no difficulties with airway control or respirations. What evidence-based nursing interventions will you implement in your care of this patient? Identify the criteria used to evaluate the strength of the evidence for these practices.

**2  pq** A 32-year-old man fell off his 10-speed bicycle, hitting the side of his head on the pavement, losing consciousness. He woke up in the emergency department confused and complained of a headache. His CT scan of the head revealed a right epidural hematoma. He was taken to the operating room for a craniotomy. What are your priority neurological assessments for this patient postoperatively? What are your priority nursing interventions to control intracranial pressure?

**3  ipc** A 22-year-old man, on a dare from his friends, attempted a backward flip from a 20-feet-high rocky cliff into shallow water that was less than 6-feet deep. He struck his back on a large protruding rock before entering the water and sustained a C-5 hyperextension injury to his neck. Upon arrival to the ED, his vital signs are stable but he is flaccid in all extremities with no sensation below nipple line. Which members of the interdisciplinary team do you anticipate will provide care for this patient? What steps will the interdisciplinary team take to address the patient's health care needs?

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference.

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## Resources

American Academy of Spinal Cord Injury Professionals, Inc.,

[www.academyscipro.org](http://www.academyscipro.org)

American Association of Neuroscience Nurses (AANN), [www.aann.org](http://www.aann.org)

Association of Rehabilitation Nurses (ARN), [www.rehabnurse.org](http://www.rehabnurse.org)

Brain Injury Association of America, [www.biausa.org](http://www.biausa.org)

Brain Trauma Foundation, [www.braintrauma.org](http://www.braintrauma.org)

Neurocritical Care Society (NCS), [www.neurocriticalcare.org](http://www.neurocriticalcare.org)

Paralyzed Veterans of America, [www.pva.org](http://www.pva.org)

United Spinal Association, [www.spinalcord.org](http://www.spinalcord.org)

World Federation of Neuroscience Nursing (WFNN), [wfnn.org](http://wfnn.org)

# **64 Management of Patients**

## **with Neurologic Infections, Autoimmune Disorders, and Neuropathies**

### **LEARNING OUTCOMES**

*On completion of this chapter, the learner will be able to:*

- 1.** Differentiate among the infectious disorders of the nervous system according to causes, manifestations, medical care, and nursing management.
- 2.** Describe the pathophysiology, clinical manifestations, and medical and nursing management of multiple sclerosis, myasthenia gravis, and Guillain–Barré syndrome.
- 3.** Use the nursing process as a framework for care of the patient with multiple sclerosis or Guillain–Barré syndrome.
- 4.** Explain disorders of the cranial nerves, their manifestations, and indicated nursing interventions.
- 5.** Apply the nursing process as a framework for care of the patient with a cranial nerve disorder.

### **NURSING CONCEPTS**

Family  
Functional Ability  
Immunity  
Infection  
Inflammation  
Mobility  
Nutrition  
Patient Education  
Sensory Perception

## GLOSSARY

- ataxia:** impaired coordination of movement during voluntary movement
- diplopia:** the awareness of two images of the same object occurring in one or both eyes (*synonym:* double vision)
- dysphagia:** difficulty swallowing
- dysphonia:** voice impairment or altered voice production
- hemiparesis:** weakness of one side of the body, or part of it, due to an injury in the motor area of the brain
- hemiplegia:** paralysis of one side of the body, or part of it, due to an injury in the motor area of the brain
- neuropathy:** general term indicating a disorder of the nervous system
- paresthesia:** numbness, tingling, or a “pins and needles” sensation
- prion:** a pathogen smaller than a virus that is resistant to standard sterilization procedures
- ptosis:** drooping of the eyelids
- spasticity:** muscular hypertonicity with increased resistance to stretch often associated with weakness, increased deep tendon reflexes, and diminished superficial reflexes

The diverse group of neurologic disorders that make up infectious and autoimmune disorders and cranial and peripheral neuropathies presents unique challenges for nursing care. The nurse who cares for patients with these disorders must have a clear understanding of the pathophysiology, diagnostic testing, medical and nursing care, and rehabilitation processes. Some of the issues nurses must help patients and families confront include adaptation to the effects of the disease, potential changes in family dynamics, and end-of-life issues.

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## INFECTIOUS NEUROLOGIC DISORDERS

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The infectious disorders of the nervous system include meningitis, brain abscesses, various types of encephalitis, Creutzfeldt–Jakob disease (CJD), and variant Creutzfeldt–Jakob disease (vCJD). The clinical manifestations, assessment and diagnostic findings, as well as the medical and nursing management, are related to the specific infectious process.

### Meningitis

Meningitis is inflammation of the meninges, which cover and protect the brain and spinal cord. The two main types of meningitis are bacterial and viral (Norris, 2019). Meningitis can be the main reason a patient is hospitalized, or it can develop during hospitalization; it is classified as septic or aseptic. Septic meningitis is caused by bacteria. The bacteria *Streptococcus pneumoniae* and *Neisseria meningitidis* are responsible for 80% to 90% of cases of bacterial meningitis in adults (Hickey & Strayer, 2020). In aseptic meningitis, the cause is viral or secondary to cancer or having a weakened immune system, such as in human immunodeficiency virus (HIV). The most common causative agents are the enteroviruses (Norris, 2019). Aseptic meningitis occurs more frequently in the summer and early fall.

First-year college students and members of the military who have not been vaccinated are at higher risk for meningococcal meningitis. Although infections occur year-round, the peak incidence is in the winter and early spring. Factors that increase the risk of bacterial meningitis include tobacco use and viral upper respiratory infection, because they increase the amount of droplet production. Otitis media and mastoiditis increase the risk of bacterial meningitis, because the bacteria can cross the epithelial membrane and enter the subarachnoid space. People with immune system deficiencies are also at greater risk for development of bacterial meningitis (Norris, 2019).

### Pathophysiology

Meningeal infections generally originate in one of two ways: through the bloodstream as a consequence of other infections or by direct spread, such as might occur after a traumatic injury to the facial bones or secondary to invasive procedures.

Once the causative organism enters the bloodstream, it crosses the blood–brain barrier and proliferates in the cerebrospinal fluid (CSF). The host immune response stimulates the release of cell wall fragments and lipopolysaccharides, facilitating inflammation of the subarachnoid and pia mater. Because the cranial vault contains little room for expansion, the

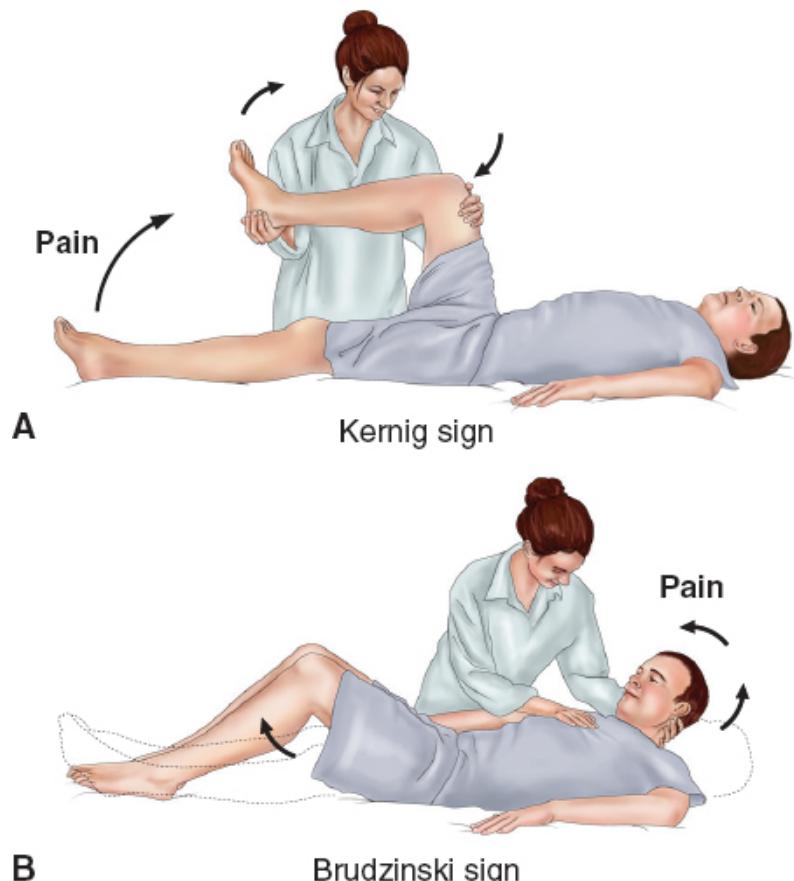
inflammation may cause increased intracranial pressure (ICP). CSF circulates through the subarachnoid space, where inflammatory cellular materials from the affected meningeal tissue enter and accumulate.

The prognosis for bacterial meningitis depends on the causative organism, the severity of the infection and illness, and the timeliness of treatment. The *N. meningitidis* bacterium results in an acute fulminant presentation approximately 10% of the time. This presentation may include adrenal damage, circulatory collapse, and widespread hemorrhages (Waterhouse–Friderichsen syndrome) (Hickey & Strayer, 2020). This syndrome is the result of endothelial damage and vascular necrosis caused by the bacteria.

## Clinical Manifestations

Headache along with fever and chills are frequent initial symptoms. Fever tends to remain high throughout the course of the illness. The headache is usually either steady or throbbing and very severe as a result of meningeal irritation. Older adults may have mental status changes and focal neurologic deficits (Mount & Boyle, 2017). Meningeal irritation results in a number of other well-recognized signs common to all types of meningitis (Norris, 2019; Weber & Kelley, 2018):

- *Neck immobility*: Nuchal rigidity (a stiff and painful neck) can be an early sign, and any attempts at flexion of the head are difficult because of spasms in the muscles of the neck. Usually, the neck is supple, and the patient can easily bend the head and neck forward.
- *Positive Kernig sign*: When the patient is lying with the thigh flexed on the abdomen, the leg cannot be completely extended (see Fig. 64-1A). When Kernig sign is bilateral, meningeal irritation is suspected.
- *Positive Brudzinski sign*: When the patient's neck is flexed (after ruling out cervical trauma or injury), flexion of the knees and hips is produced; when the lower extremity of one side is passively flexed, a similar movement is seen in the opposite extremity (see Fig. 64-1B). Brudzinski sign is a more sensitive indicator of meningeal irritation than Kernig sign.
- *Photophobia (extreme sensitivity to light)*: This finding is common due to irritation of the meninges, especially around the diaphragm sellae.
- A rash can be a striking feature of *meningococcal meningitidis* infection, occurring in about half of patients with this type of meningitis. Skin lesions develop, ranging from a petechial rash with purpuric lesions to large areas of ecchymosis.



**Figure 64-1 •** Testing for meningeal irritation. **A.** Kernig sign. Flex the patient's leg at both the hip and knee and then straighten the knee. **B.** Brudzinski sign. As the neck is flexed, watch the hips and knees for a reaction.

Disorientation and memory impairment are common early in the course of the illness. The changes depend on the severity of the infection as well as the individual response to the physiologic processes. Behavioral manifestations are also common. As the illness progresses, lethargy, unresponsiveness, and coma may develop.

Seizures can occur and are the result of areas of irritability in the brain. ICP increases secondary to diffuse brain swelling or hydrocephalus (Hickey & Strayer, 2020). The initial signs of increased ICP include decreased level of consciousness (LOC) and focal motor deficits. If ICP is not controlled, the uncus of the temporal lobe may herniate through the tentorium, causing pressure on the brainstem. Brainstem herniation is a life-threatening event that causes cranial nerve dysfunction and depresses the centers of vital functions, such as the medulla. See [Chapter 61](#) for discussion of the patient with a change in LOC or increased ICP.

An acute fulminant infection produces signs of sepsis: an abrupt onset of high fever, extensive purpuric lesions (over the face and extremities), shock,

and signs of disseminated intravascular coagulation (see [Chapter 29](#)). Death may occur within a few hours after onset of the infection.

## Assessment and Diagnostic Findings

If the clinical presentation suggests meningitis, diagnostic testing is conducted to identify the causative organism. A computed tomography (CT) scan is used to detect a shift in brain contents (which may lead to herniation) prior to a lumbar puncture in patient with altered LOC, papilledema, neurologic deficits, new onset of seizure, immunocompromised state, or history of central nervous system (CNS) disease. Bacterial culture and Gram staining of CSF and blood are key diagnostic tests (Hickey & Strayer, 2020). An overview of CSF values and alterations in bacterial, viral, and fungal meningitis is presented in [Table 64-1](#). Gram staining allows for rapid identification of the causative bacteria and initiation of appropriate antibiotic therapy.

## Prevention

The Advisory Committee on Immunization Practices of the Centers for Disease Control and Prevention (CDC) recommends that the meningococcal conjugated vaccine be given to youth at 11 to 12 years of age, with a booster dose at 16 years of age (CDC, 2020).

People in close contact with patients with meningococcal meningitis should be treated with antimicrobial chemoprophylaxis using rifampin, ciprofloxacin, or ceftriaxone. Therapy should be started within 24 hours after exposure because a delay limits the effectiveness of the prophylaxis. Vaccination should also be considered as an adjunct to antibiotic chemoprophylaxis for anyone living with a person who develops meningococcal infection. Vaccination against *Haemophilus influenzae* and *S. pneumoniae* should be encouraged for children and adults who are at-risk (CDC, 2020).

## Medical Management

Successful outcomes depend on the early administration of an antibiotic agent that crosses the blood–brain barrier into the subarachnoid space in sufficient concentration to halt the multiplication of bacteria. Penicillin G in combination with one of the cephalosporins (e.g., ceftriaxone, cefotaxime) is most often administered intravenously (IV), emergently with suspected bacterial meningitis (Hickey & Strayer, 2020).

Dexamethasone has been shown to be beneficial as adjunct therapy in the treatment of acute bacterial meningitis and in pneumococcal meningitis if it is given before or concurrently with the first dose of antibiotic and every 6 hours for the next 4 days. Research suggests that dexamethasone improves the

outcome in adults and does not increase the risk of gastrointestinal bleeding (Hickey & Strayer, 2020).

Dehydration and shock are treated with fluid volume expanders. Seizures, which may occur early in the course of the disease, are treated with anticonvulsant medications. Increased ICP is treated as necessary (see [Chapter 61](#)).



## Nursing Management

The patient with meningitis is critically ill; therefore, many of the nursing interventions are collaborative with the physician, respiratory therapist, and other members of the health care team. The patient's safety and well-being depend on sound nursing judgment. Most patients will need the following nursing interventions:

- Instituting infection control precautions until 24 hours after initiation of antibiotic therapy (oral and nasal discharge is considered infectious)
  - Assisting with pain management due to overall body aches and neck pain
  - Assisting with getting rest in a quiet, darkened room
  - Implementing interventions to treat the elevated temperature, such as antipyretic agents and cooling blankets
  - Encouraging the patient to stay hydrated either orally or peripherally
  - Ensuring close neurologic monitoring (see [Chapter 61](#))

Neurologic status and vital signs are continually assessed. Pulse oximetry and arterial blood gas values are used to quickly identify the need for respiratory support if increasing ICP compromises the brainstem. Insertion of a cuffed endotracheal tube (or tracheotomy) and mechanical ventilation may be necessary to maintain adequate tissue oxygenation.

Blood pressure (usually monitored using an arterial line) is assessed for early manifestations of shock, which precedes cardiac or respiratory failure. Rapid IV fluid replacement may be prescribed, but care is taken to prevent fluid overload. Fever also increases the workload of the heart and cerebral metabolism. ICP will increase in response to increased cerebral metabolic demands. Therefore, measures are taken to reduce body temperature as quickly as possible.

**TABLE 64-1** Cerebrospinal Fluid Values Diagnostic for Meningitis

Parameter	Normal CSF	Bacterial Meningitis	Viral Meningitis
Opening pressure (mm H <sub>2</sub> O)	100–180	Elevated >180	Variable
Leukocyte count (white blood cell/mm <sup>3</sup> )	0–5	Increased 100–5000	Increased 50–1000
Neutrophils (%)	0	≥80	<40
Protein (mg/dL)	15–50	Elevated 100–500	Normal or slightly increased
Glucose (mg/dL)	40–80; 0.6 times blood glucose level	<40; <0.4 times blood glucose level	Normal

CSF, cerebrospinal fluid.

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.

Other important components of nursing care include the following measures:

- Protecting the patient from injury secondary to seizure activity or altered LOC
- Monitoring daily body weight; serum electrolytes; and urine volume, specific gravity, and osmolality, especially if syndrome of inappropriate antidiuretic hormone (SIADH) is suspected
- Preventing complications associated with immobility, such as pressure injury and pneumonia

Any sudden, critical illness can be devastating to the family. Because the patient's condition is often critical and the prognosis guarded, the family needs to be informed about the patient's condition. Periodic family visits are essential to facilitate coping of the patient and family. An important aspect of the nurse's role is to support the family and assist them in identifying others who can be supportive to them during the crisis (Hickey & Strayer, 2020).

### Promoting Home, Community-Based, and Transitional Care

After the patient has achieved physiologic homeostasis and has demonstrated achievement of major health care goals, rehabilitation continues either in a rehabilitation facility, skilled nursing facility, or at home. Continued support and evaluation by the nurse are essential.

Because patients may have been critically ill and focused on the most obvious needs and issues, the nurse reminds the patient and family about the importance of continuing health promotion and screening practices, such as regular physical examinations and appropriate diagnostic screening tests.

## **Brain Abscess**

Brain abscesses account for less than 1% of space-occupying brain lesions in the United States (Hickey & Strayer, 2020). Brain abscesses are rare in people who are immunocompetent; they are more frequently diagnosed in people who are immunosuppressed as a result of an underlying disease or the use of immunosuppressive medications.

## **Pathophysiology**

A brain abscess is a collection of infectious material within the tissue of the brain. Bacteria are the most common causative organisms. The most common predisposing conditions for abscesses among adults who are immunocompetent are otitis media and rhinosinusitis. It is estimated that 40% of brain abscesses are otogenic in origin (Hickey & Strayer, 2020). An abscess can result from intracranial surgery, penetrating head injury, or tongue piercing. Organisms causing brain abscess may reach the brain by hematologic spread from the lungs, gums, tongue, or heart, or from a wound or intra-abdominal infection.

## **Clinical Manifestations**

The clinical manifestations of a brain abscess result from alterations in intracranial dynamics (edema, brain shift), infection, or the location of the abscess. Headache, usually worse in the morning, is the most prevalent symptom. Mental status changes may occur. Fever is present 53% of the time (Sonneville, Ruimy, Benzonana, et al., 2017). Vomiting and focal neurologic deficits occur as well. Focal deficits including weakness and decreasing vision reflect the area of brain that is involved. As the abscess expands, symptoms of increased ICP such as decreasing LOC and seizures occur (Hickey & Strayer, 2020).

## **Assessment and Diagnostic Findings**

The baseline neurologic examination may reveal a variety of signs and symptoms based on the location of the abscess (see [Chart 64-1](#)). Neuroimaging with CT scanning with contrast is used most often to identify the size and location of the abscess. Cerebritis is a small infection in the brain that can progress to an abscess if not detected or treated. Aspiration of the abscess, guided by CT or MRI, is often used to culture and identify the infectious organism. MRI is the preferred study, because it provides higher resolution of the lesion and assists with identification of additional lesions if present

(Sonneville et al., 2017). Blood cultures are obtained if the abscess is believed to arise from a distant source.

## Medical Management

Treatment is aimed at controlling increased ICP, draining the abscess, and providing antimicrobial therapy directed at the abscess and the main source of infection. Large IV doses of antibiotic agents are given to penetrate the blood–brain barrier and reach the abscess. The choice of the specific antibiotic medication is based on culture and sensitivity testing and directed at the causative organism. Antibiotics should be started as soon as possible; the initial antibiotic started typically is ceftriaxone combined with metronidazole, which will be adjusted based on the culture and sensitivity results (Brouwer & van de Beek, 2017). A stereotactic guided aspiration may be used to drain the abscess and identify the causative organism. Surgical excision is not the preferred method, except in cases where the abscess is large and multi-lobulated (Sonneville et al., 2017). Corticosteroids may be prescribed to help reduce the inflammatory cerebral edema if the patient shows evidence of an increasing neurologic deficit. Anticonvulsant medications may be prescribed to prevent or treat seizures (see [Chapter 61](#)).

Chart 64-1



### ASSESSMENT

## **Assessing for Brain Abscesses**

Be alert to the following signs and symptoms of brain abscess:

### **Frontal Lobe**

- Expressive aphasia (inability to express oneself)
- Frontal headache
- Hemiparesis (weakness on one side of the body)
- Seizures

### **Temporal Lobe**

- Changes in vision
- Facial weakness
- Localized headache
- Receptive aphasia (inability to understand language)

### **Cerebellar Abscess**

- Ataxia
- Nystagmus (rhythmic, involuntary movements of the eye)
- Occipital headache

## **Nursing Management**

Nursing care focuses on continuing to assess the neurologic status, administering medications, assessing the response to treatment, and providing supportive care.

Ongoing neurologic assessment alerts the nurse to changes in ICP, which may indicate a need for more aggressive intervention. The nurse also assesses and documents the responses to medications. Blood laboratory test results, specifically blood glucose and serum potassium levels, are closely monitored when corticosteroids are prescribed (Comerford & Durkin, 2020). Administration of insulin or electrolyte replacement may be required to return these values to within normal limits.

Patient safety is another key nursing responsibility. Injury may result from decreased LOC or falls related to motor weakness or seizures.

The patient with a brain abscess is very ill, with neurologic deficits, such as **hemiplegia** (paralysis of one side of the body, or part of it), **hemiparesis** (weakness of one side of the body, or part of it), seizures, visual deficits, and cranial nerve palsies that may persist after treatment. The nurse must assess the family's ability to express distress at the patient's condition, cope with the patient's illness and deficits, and obtain support. Treatment has improved and

70% of patients have no or minimal permanent neurologic deficits, but more research is needed on long-term function (Brouwer & van de Beek, 2017).

## Herpes Simplex Encephalitis

Encephalitis is an acute inflammatory process of the brain tissue. The herpes simplex virus (HSV) is the most common cause of acute encephalitis in the United States, accounting for 10% to 15% of cases of encephalitis (Hickey & Strayer, 2020).

## Pathophysiology

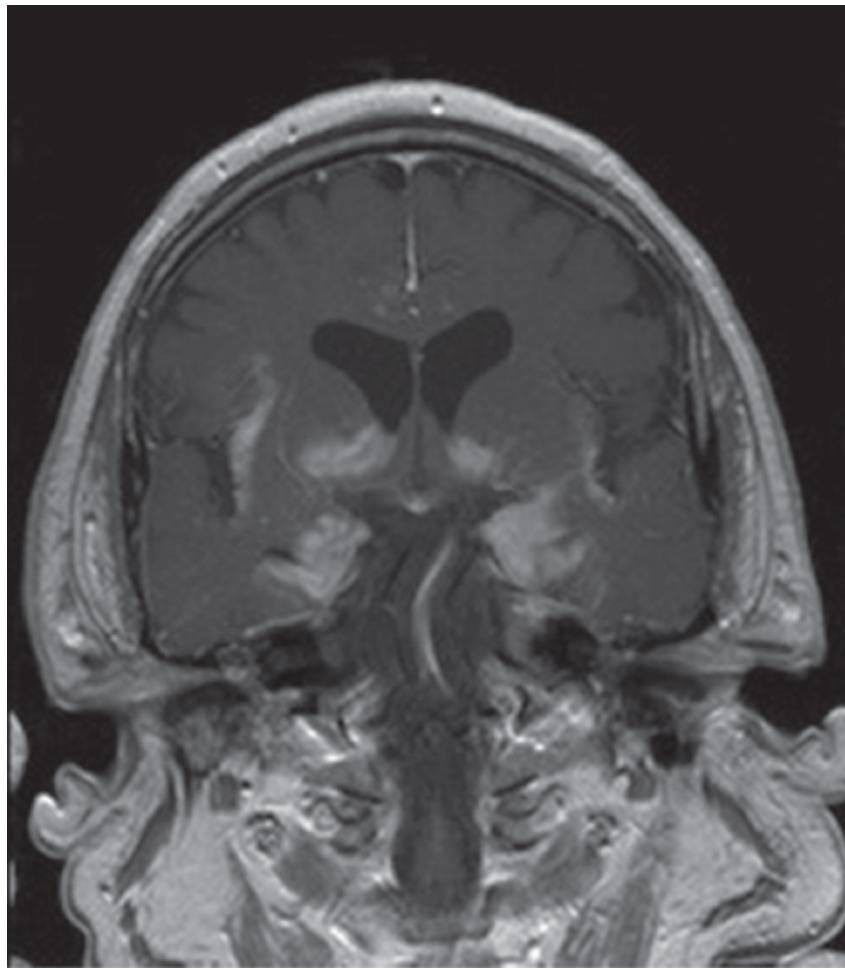
The pathology of encephalitis involves local necrotizing hemorrhage that becomes more generalized, followed by edema. There is also progressive deterioration of nerve cell bodies (Norris, 2019).

## Clinical Manifestations

The initial symptoms of herpes simplex encephalitis include fever, headache, confusion, and hallucinations. Focal neurologic symptoms reflect the areas of cerebral inflammation and necrosis and include fever, headache, behavioral changes, focal seizures, dysphasia, hemiparesis, and altered LOC (Norris, 2019).

## Assessment and Diagnostic Findings

Neuroimaging studies, such as electroencephalography (EEG), and CSF examination are used to diagnose encephalitis. MRI is used to detect inflammation that shows up as hyperintense (bright) areas (see Fig. 64-2). The EEG shows diffuse slowing or focal changes in the temporal lobe in the majority of patients. Lumbar puncture often reveals a high opening pressure, glucose within normal limits, and high protein levels in CSF samples (see Table 64-1). The polymerase chain reaction (PCR) is the standard test for early diagnosis of herpes simplex encephalitis. PCR identifies the deoxyribonucleic acid (DNA) bands of HSV-1 in the CSF with 95% sensitivity and 99% specificity rates (Hickey & Strayer, 2020).



**Figure 64-2 •** Herpes simplex virus encephalitis. Coronal view, MRI scan. Note bilateral hyperintensity (bright) areas indicative of inflammation. Reprinted with permission from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed., Fig. 28-4C). Philadelphia, PA: Wolters Kluwer.

## Medical Management

The antiviral agent acyclovir is the medication of choice in the treatment for HSV (Hickey & Strayer, 2020). Early administration of an antiviral agent (usually well tolerated) improves the prognosis associated with HSV. The mode of action is inhibition of viral DNA replication. To prevent relapse, treatment should continue for up to 3 weeks. Slow IV administration over 1 hour prevents crystallization of the medication in the urine. The usual dose of acyclovir is decreased if the patient has a history of renal insufficiency.

## Nursing Management

Assessment of neurologic function is key to monitoring the progression of disease. Comfort measures to reduce headache include dimming the lights, limiting noise and visitors, grouping nursing interventions, and administering analgesic agents. Opioid analgesic medications may mask neurologic symptoms; therefore, they are used cautiously. Seizures and altered LOC require care directed at injury prevention and safety. Nursing care addressing patient and family anxieties is ongoing throughout the illness. Monitoring of blood chemistry test results and urinary output alert the nurse to the presence of renal complications related to antiviral therapy.

## **Arthropod-Borne Virus Encephalitis**

Arthropod-borne viruses, or arboviruses, are maintained in nature through biologic transmission between susceptible vertebrate hosts by blood feeding arthropods (mosquitoes, psychodids, ceratopogonids, and ticks). Arthropod vectors transmit several types of viruses that cause encephalitis. The main vector in North America is the mosquito. Arbovirus infection (transmitted by arthropod vectors) occurs in specific geographic areas during the summer and fall. In the United States, there are five main arboviral encephalitides: eastern equine encephalitis, western equine encephalitis, St. Louis encephalitis, La Crosse encephalitis, and West Nile virus encephalitis (Hickey & Strayer, 2020).

## **Pathophysiology**

Viral replication occurs at the site of the mosquito bite. The host immune response attempts to control viral replication. If the immune response is inadequate, viremia will ensue. The virus gains access to the CNS via the olfactory tract, resulting in encephalitis. It spreads from neuron to neuron, predominantly affecting the cortical gray matter, the brainstem, and the thalamus. Meningeal exudates compound the clinical presentation by irritating the meninges and increasing ICP.

## **Clinical Manifestations**

St. Louis and West Nile virus encephalitis most commonly affect adults. An arboviral encephalitis occurs along a continuum with some cases having only flulike symptoms (i.e., headache and fever) but others progressing to specific neurologic manifestations that vary depending on the viral type (Hickey & Strayer, 2020). A unique clinical feature of arboviral encephalitis is SIADH with hyponatremia. Onset of symptoms is abrupt with fever, headache, dizziness, nausea, and malaise. If the disease spreads to the CNS, symptoms

include stiff neck, confusion, dizziness, and tremors. Coma can occur in severe cases, and mortality increases with age. Eastern equine encephalitis has the highest mortality rate at 50% (Hickey & Strayer, 2020).

## Assessment and Diagnostic Findings

Arboviral infections are seasonal. Preliminary diagnosis is based on clinical presentation and location and dates of recent travel because certain viruses are endemic to certain geographic locales. Neuroimaging and CSF evaluation are useful in the diagnosis of encephalitis. The MRI scan demonstrates inflammation of the basal ganglia in cases of St. Louis encephalitis and inflammation in the periventricular area in cases of West Nile encephalitis. EEG can identify abnormal brain waves, which can help identify some viral infections.

## Medical Management

No specific medication for arboviral encephalitis exists; therefore, treatment is supportive and symptom management is key (Hickey & Strayer, 2020). Controlling elevated ICP is a critical component of care in cases with neurologic manifestations.

## Nursing Management

Many patients, particularly those with only fever and headache, are treated on an outpatient basis. If the patient is very ill, hospitalization may be required. The nurse carefully assesses neurologic status and identifies improvement or deterioration in the patient's condition. See [Chapter 61](#) for management of the patient with increased ICP. Injury prevention is key in light of the potential for falls or seizures. Any encephalitis that progresses may result in death or lifelong residual health issues such as neurologic deficits and seizures. The family will need support and education to cope with these residual issues. The nurse may need to mobilize community support services for the patient and family, because the recovery may be long.

Public education addressing the prevention of arboviral encephalitis is a key nursing role. Clothing that provides coverage and insect repellents containing 20% to 35% diethyltoluamide (DEET) should be used on exposed clothing and skin in high-risk areas to decrease mosquito and tick bites (Hickey & Strayer, 2020). Remaining indoors at dawn and dusk when mosquito activity is highest is recommended. Screens should be in good repair in the home, and standing water should be removed. All cases of arboviral encephalitis must be reported to the local health department.

## Creutzfeldt–Jakob and Variant Creutzfeldt–Jakob Diseases

CJD and vCJD belong to a group of degenerative, infectious neurologic disorders called *transmissible spongiform encephalopathies* (TSE). CJD is rare and has no identifiable cause. vCJD, the human variation of bovine spongiform encephalopathy (BSE) (mad cow disease), results from ingesting meat infected with prions. **Prions**, which cause TSEs, are pathogens smaller than a virus that are resistant to standard methods of disinfection and sterilization (Garcia, 2019). Although CJD and vCJD have distinct clinical features, one characteristic they share is a lack of CNS inflammation. CJD may lie dormant for decades before causing neurologic degeneration. The incubation period of vCJD seems to be shorter (less than 10 years). In both diseases, the symptoms are progressive, there is no definitive treatment, and the outcome is fatal often within 1 year of symptom onset (Hickey & Strayer, 2020).

Almost all cases of vCJD have been reported in the United Kingdom, with a smaller number of cases being identified in 10 other countries worldwide. The risk of vCJD in the United States is thought to be low, because cattle are fed primarily with soy-derived feed as opposed to feed containing animal parts.

## Pathophysiology

The prion is a unique pathogen because it lacks nucleic acid, which enables the organism to withstand conventional means of disinfection and sterilization (Garcia, 2019). In both CJD and vCJD, the prion crosses the blood–brain barrier and is deposited in brain tissue and causes degeneration of brain tissue (Hickey & Strayer, 2020). Cell death occurs, and spongiform changes (spongy vacuoles) are produced in the brain. The spongiform vacuoles are surrounded by amyloid plaque.

There are three major forms of CJD (Manthorpe & Simcock, 2019). Approximately 85% of cases appear sporadically, hence this form is called sporadic CJD. The incidence is 1 case per 1 million people. Sporadic CJD occurs spontaneously with no risk factors. The second form is familial or hereditary CJD, and this type accounts for 5% to 10% of cases. The third type is acquired CJD. This form is transmitted by contaminated brain, tissue, or neurosurgical instruments and accounts for less than 1% of cases (World Health Organization [WHO]. n.d.).

The prion exists in lymphoid tissue and blood in both vCJD and CJD. Both prion diseases are believed to be bloodborne. No method is available to screen blood for infectivity. For this reason, the American Red Cross does not accept blood donation from anyone who has stayed more than 3 months in the United Kingdom (UK) between 1980 and 1996 or has received a blood transfusion in

France or the UK at any time between 1980 and the present (Miller, Grima, & Plonowski, 2020).

## Clinical Manifestations

CJD and vCJD have several clinically distinct features. Psychiatric symptoms occur early in vCJD, whereas they are a late symptom in CJD. The mean age at onset of vCJD is 27 years, whereas the mean age for CJD onset is 65 years. The presenting symptoms of vCJD include affective symptoms (i.e., behavioral changes), sensory disturbance, and limb pain. Muscle spasms and rigidity, dysarthria (difficulty speaking), incoordination, cognitive impairment, and sleep disturbances follow. Patients with sporadic CJD present with mental deterioration, **ataxia** (inability to coordinate movements), and visual disturbance. Memory loss, involuntary movement, paralysis, and mutism occur as the disease progresses. After clinical presentation, people with vCJD survive an average of 14 months; those with CJD survive for less than 1 year (Manthorpe & Simcock, 2019).

## Assessment and Diagnostic Findings

Brain biopsy, the only way to confirm diagnosis, is not recommended (Hickey & Strayer, 2020). The three diagnostic tests currently used in suspicious clinical presentations to support the diagnosis of CJD are immunologic assessment, EEG, and MRI scanning. Immunologic assessment of CSF detects a protein kinase inhibitor referred to as 14–3–3 (Hickey & Strayer, 2020). The presence of this inhibitor indicates neuronal cell death, which is not specific to CJD but does support the diagnosis. The EEG reveals a characteristic pattern over the duration of the disease. After initial slowing, the EEG shows periodic activity. Later in the course of the disease, the EEG shows burst suppressions characterized by periodic spikes alternating with slow periods. The MRI scan demonstrates symmetric or unilateral hyperintense signals arising from the basal ganglia.

## Medical Management

After the onset of specific neurologic symptoms, progression of disease occurs quickly. There is no effective treatment for CJD or vCJD. The care of the patient is supportive and palliative. Goals of the interprofessional care team include prevention of injury related to immobility and dementia, promotion of patient comfort, and provision of support and education for the family.

## Nursing Management

The nursing care of patients is primarily supportive and palliative. Psychological and emotional support of the patient and family throughout the course of the illness is needed. Care extends to providing for a dignified death and supporting the family through the processes of grief and loss. Hospice services are appropriate either at home or at an inpatient facility. See [Chapter 13](#) for an in-depth discussion of end-of-life issues.

Prevention of disease transmission is an important part of nursing care. Although patient isolation is not necessary, the use of standard precautions is important. Institutional protocols are followed for handling of brain, spinal cord, pituitary gland, and eye tissue; and for exposure and decontamination of equipment. If surgery is necessary, it is recommended that disposable instruments be used and then incinerated, because conventional methods of sterilization do not destroy the prion (Hickey & Strayer, 2020). If disposable instruments cannot be used, stringent sterilization methods such as the use of bleach for cleaning and extended sterilization time for instruments should be used (Garcia, 2019).

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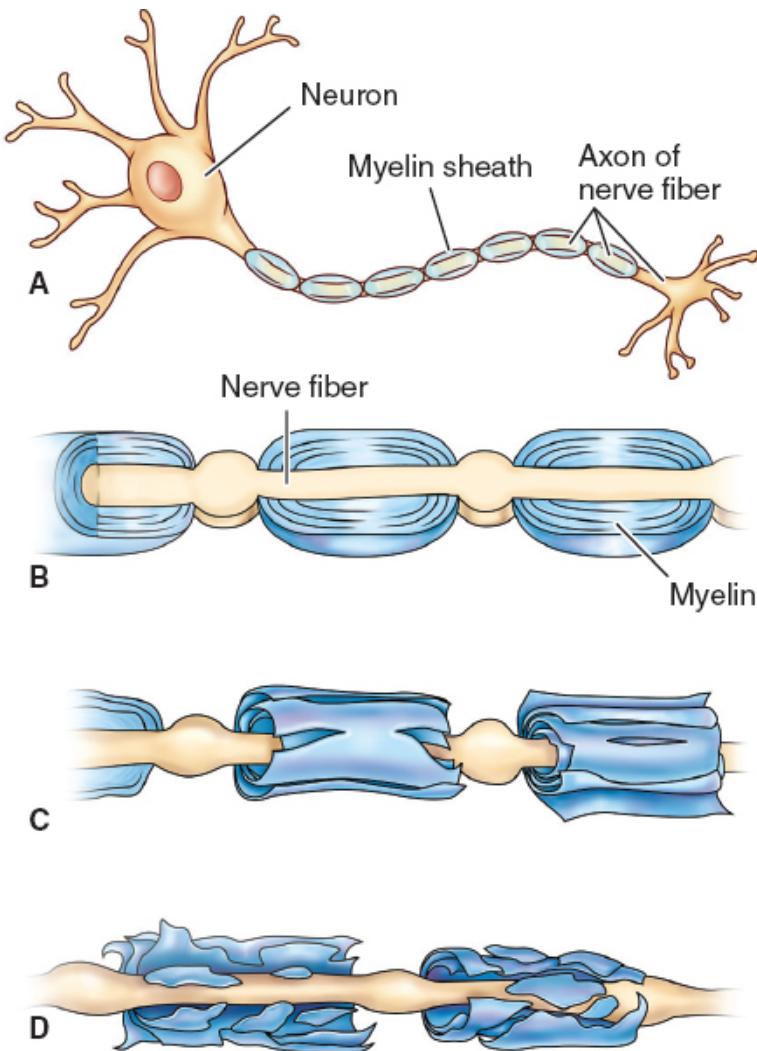
## AUTOIMMUNE PROCESSES

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Autoimmune nervous system disorders include multiple sclerosis (MS), myasthenia gravis, and Guillain–Barré syndrome (GBS).

### Multiple Sclerosis

MS is an immune-mediated, progressive demyelinating disease of the CNS. Demyelination refers to the destruction of myelin—the fatty and protein material that surrounds certain nerve fibers in the brain and spinal cord; it results in impaired transmission of nerve impulses (see [Fig. 64-3](#)). MS affects nearly 400,000 people in the United States (Hickey & Strayer, 2020; Norris, 2019). MS may occur at any age, but the age of peak onset is between 20 and 50 years; it affects women three times more than men (Hickey & Strayer, 2020).



**Figure 64-3 •** The process of demyelination. **A, B.** A normal nerve cell and axon with myelin. **C, D.** The slow disintegration of myelin, resulting in a disruption in axon function.

The cause of MS is unknown and is an area of ongoing research. Autoimmune activity results in demyelination, but the sensitized antigen has not been identified. Multiple factors play a role in the initiation of the immune process. Geographic prevalence is highest in Europe, New Zealand, southern Australia, the northern United States, and southern Canada. MS is less prevalent in Asians. There is a greater frequency in northern colder latitudes (Hickey & Strayer, 2020).

MS is considered to have many risks, including genetic factors. It has not been found to be genetically transmitted but there have been 200 genetic variations related to MS (Hickey & Strayer, 2020). A specific virus capable of initiating the autoimmune response has not been identified. It is believed that DNA on the virus mimics the amino acid sequence of myelin, resulting in an

immune system cross-reaction in the presence of a defective immune system. Environmental risks include obesity, lack of vitamin D exposure, and a high salt diet in the teenage years (Hickey & Strayer, 2020).

There are several acute and subacute forms of MS. Less severe forms include radiologically isolated syndrome (RIS) and clinically isolated syndrome (CIS). RIS consists of MS-like lesions that are identified on MRI in the absence of clinical signs and symptoms. Approximately one third of patients are diagnosed with MS within 5 years of the identification of an incidental lesion on MRI (Coyle, 2019). CIS is the presence of acute or subacute clinical findings for at least 24 hours (Coyle, 2019).

The four main clinical forms are remitting-relapsing (RRMS), secondary progressive, primary progressive (PPMS), and progressive-relapsing (Bradshaw & Houtchens, 2018).

## Pathophysiology

Sensitized T and B lymphocytes cross the blood–brain barrier; their function is to check the CNS for antigens and then leave. In MS, sensitized T cells remain in the CNS and promote the infiltration of other agents that damage the immune system. The immune system attack leads to inflammation that destroys mostly the white matter of the CNS myelin (which insulates the axon and speeds the conduction of impulses along the axon) and the oligodendroglial cells that produce myelin in the CNS (Norris, 2019).

Demyelination interrupts the flow of nerve impulses and results in a variety of manifestations, depending on the nerves affected. Plaques appear on demyelinated axons, further interrupting the transmission of impulses. Demyelinated axons are scattered irregularly throughout the CNS. The areas most frequently affected are the optic nerves, chiasm, and tracts; the cerebrum; the brainstem and cerebellum; and the spinal cord (Norris, 2019). The axons themselves begin to degenerate, resulting in permanent and irreversible damage.

## Clinical Manifestations

The course of MS assumes many different patterns. In some patients, the disease follows a benign course, and symptoms are so mild that the patient does not seek health care or treatment. The patient with RIS will have no symptoms, while the typical presentation of CIS includes unilateral optic neuritis, focal symptoms, or partial myelopathy (Coyle, 2019).

Approximately 85% of patients have RRMS (Coyle, 2019). With each relapse, recovery is usually complete; however, residual deficits may occur and accumulate over time, contributing to functional decline. Over time, most patients with RRMS move to the secondary progressive form, in which disease

progression occurs with or without relapses. Approximately 15% of patients have PPMS, in which disabling symptoms steadily increase, with rare plateaus and temporary minor improvement. PPMS may result in quadriplegia, cognitive dysfunction, visual loss, and brainstem syndromes (Coyle, 2019). The least common presentation (about 5% of cases) is progressive-relapsing, which is characterized by relapses with continuous disabling progression between exacerbations (Coyle, 2019; Norris, 2019). The signs and symptoms of MS are varied and multiple, reflecting the location of the lesion (plaque) or combination of lesions. Physical, emotional, and cognitive symptoms impact the quality of life (Debska, Milaniak, & Skorupska-Krol, 2020; Kalb, Feinstein, Rohrig, et al., 2019). Fatigue, depression, weakness, numbness, difficulty in coordination, loss of balance, spasticity, and pain are all common (Norris, 2019). Visual disturbances due to lesions in the optic nerves or their connections may include blurring of vision, **diplopia** (double vision, or the awareness of two images of the same object occurring in one or both eyes), scotoma (patchy blindness), and total blindness.

Fatigue affects most people with MS and is often the most disabling symptom (Newland, Lorenz, Smith, et al., 2019). Heat, depression, anemia, deconditioning, and medication may contribute to fatigue. Avoiding hot temperatures, effective treatment of depression and anemia, a change in medication, as well as occupational and physical therapies may help manage fatigue (Coyle, 2019).

Pain is another common symptom of MS. Lesions on the sensory pathways cause pain. Additional sensory manifestations include paresthesias, dysesthesia, and proprioception loss. Many people with MS need daily analgesic medications. In some cases, pain is managed with opioids, anticonvulsant medications, or antidepressants. Rarely, surgery may be needed to interrupt pain pathways.

Among women who are perimenopausal, those with MS are more likely to have pain related to osteoporosis. In addition to estrogen loss, immobility and corticosteroid therapy play a role in the development of osteoporosis among women with MS. Bone mineral density testing is recommended for this high-risk group. See [Chapter 36](#) for a discussion of the diagnosis of and treatment for osteoporosis.

**Spasticity** is characterized by muscle hypertonicity with increased resistance to stretch often associated with weakness, increased deep tendon reflexes, and diminished superficial reflexes. It occurs in 90% of patients with MS, most often in the lower extremities, and can include loss of the abdominal reflexes. Spasticity results from involvement of the pyramidal tracts, the main motor pathways of the spinal cord. Cognitive and psychosocial problems may reflect frontal or parietal lobe involvement. Some degree of cognitive change (e.g., memory loss, decreased concentration) occurs in about half of patients,

but severe cognitive changes with dementia (progressive organic mental disorder) are rare.

Involvement of the cerebellum or basal ganglia can produce ataxia and tremor. Loss of the control connections between the cortex and the basal ganglia may occur and cause emotional lability and euphoria. Bladder, bowel, and sexual dysfunctions are common. Additional complications include urinary tract infections (UTIs), constipation, pressure injury, contracture deformities, dependent pedal edema, pneumonia, and osteoporosis. Emotional, social, marital, economic, and vocational problems may also occur.

Exacerbations and remissions are characteristic of MS. During exacerbations, new symptoms appear and existing ones worsen; during remissions, symptoms decrease or disappear. Relapses may be associated with emotional and physical stress.



## Gerontologic Considerations

The life expectancy for patients with MS is 7 to 14 years shorter than patients without MS (Coyle, 2019). Those diagnosed with secondary progressive disease live an average of 38 years after onset. Older adult patients with MS have specific physical and psychosocial challenges. They may have chronic health problems, for which they may be taking additional medications that could interact with medications prescribed for MS. The absorption, distribution, metabolism, and excretion of medications are altered in the older adult as a result of age-related changes in kidney and liver functions. Therefore, older adult patients must be monitored closely for adverse and toxic effects of MS medications and for osteoporosis (particularly if corticosteroids have been used frequently to treat exacerbations). The cost of medications may lead to poor adherence to the prescribed regimen in older adult patients on fixed incomes.

Older adult patients with MS are particularly concerned about increasing disability, family burden, marital concern, and the possible future need for nursing home care. Immobility resulting in fewer social opportunities contributes to loneliness and depression. In addition to functional loss, the physical challenges experienced by older adults with MS include spasticity, pain, bladder dysfunction, impaired sleep, and an increased need for assistance with self-care.

## Assessment and Diagnostic Findings

The diagnosis of MS is based on clinical, imaging, and laboratory findings. An important component is the presence of plaques in the CNS disseminated in space and over time observed on MRI scans with no better explanation for the clinical presentation (Thompson, Banwell, Barkhof, et al., 2018). Additional

criteria are detailed in [Table 64-2](#). Electrophoresis of CSF identifies the presence of oligoclonal banding (several bands of immunoglobulin G bonded together, indicating an immune system abnormality) (Thompson et al., 2018). Evoked potential studies can help define the extent of the disease process and monitor changes (Coyle, 2019). Underlying bladder dysfunction is diagnosed by urodynamic studies. Neuropsychological testing may be indicated to assess cognitive impairment. A sexual history helps identify changes in sexual function.

## Medical Management

There is no cure for MS. An individual treatment program is indicated to relieve symptoms and provide continuing support, particularly for patients with cognitive changes, who may need more structure and support. The goals of treatment are to delay the progression of the disease, manage chronic symptoms, and treat acute exacerbations. Common symptoms requiring intervention include ataxia, bladder dysfunction, depression, fatigue, and spasticity. Management includes pharmacologic and nonpharmacologic strategies.

**TABLE 64-2** The 2017 McDonald Criteria for Diagnosis of Multiple Sclerosis in Patients with an Attack at Onset

	Number of Lesions with Objective Clinical Evidence	Additional Data Needed for a Diagnosis of Multiple Sclerosis
≥2 clinical attacks	≥2	None <sup>a</sup>
≥2 clinical attacks	1 (as well as clear-cut historical evidence of a previous attack involving a lesion in a distinct anatomical location <sup>b</sup> )	None <sup>a</sup>
≥2 clinical attacks	1	Dissemination in space demonstrated by an additional clinical attack implicating a different CNS site or by MRI
1 clinical attack	≥2	Dissemination in time demonstrated by an additional clinical attack or by MRI OR demonstration of CSF-specific oligoclonal bands <sup>c</sup>
1 clinical attack	1	Dissemination in space demonstrated by an additional clinical attack implicating a different CNS site or by MRI AND Dissemination in time demonstrated by an additional clinical attack or by MRI OR demonstration of CSF-specific oligoclonal bands <sup>c</sup>

If the 2017 McDonald Criteria are fulfilled and there is no better explanation for the clinical presentation, the diagnosis is multiple sclerosis. If multiple sclerosis is suspected by virtue of a clinically isolated syndrome but the 2017 McDonald Criteria are not completely met, the diagnosis is possible multiple sclerosis. If another diagnosis arises during the evaluation that better explains the clinical presentation, the diagnosis is not multiple sclerosis.

<sup>a</sup>No additional tests are required to demonstrate dissemination in space and time. However, unless MRI is not possible, brain MRI should be obtained in all patients in whom the diagnosis of multiple sclerosis is being considered. In addition, spinal cord MRI or CSF examination should be considered in patients with insufficient clinical and MRI evidence supporting multiple sclerosis, with a presentation other than a typical clinically isolated syndrome, or with atypical features. If imaging or other tests (e.g., CSF) are undertaken and are negative, caution needs to be taken before making a diagnosis of multiple sclerosis, and alternative diagnoses should be considered.

<sup>b</sup>Clinical diagnosis based on objective clinical findings for two attacks is most secure. Reasonable historical evidence for one past attack, in the absence of documented objective neurologic findings, can include historical events with symptoms and evolution characteristic for a previous inflammatory demyelinating attack; at least one attack, however, must be supported by objective findings. In the absence of residual objective evidence, caution is needed.

<sup>c</sup>The presence of CSF-specific oligoclonal bands does not demonstrate dissemination in time per se but can substitute for the requirement for demonstration of this measure.

Adapted from Thompson, A., Banwell, B., Barkhof, F., et al. (2018). Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria. *The Lancet Neurology*, 17(2), 162–173. Copyright 2018, with permission from Elsevier.

## Pharmacologic Therapy

Medications prescribed for MS include those for disease modification and those for symptom management. Disease-modifying therapies delay disease progression in many forms of MS (Rae-Grant, Day, Marrie, et al., 2018). Many types of medications are used for symptom management in MS.

### Disease-Modifying Therapies

In the past decade, the number of disease-modifying therapies has increased dramatically (Bradshaw & Houtchens, 2018). The key concept of disease-modifying therapies is to reduce the frequency of relapse, the duration of relapse, and the number and size of plaques observed on MRI in RRMS; however, these therapies are not effective in PPMS (Hickey & Strayer, 2020). There is debate about whether to use disease-modifying therapies in patients with RIS to prevent future disease progression to MS (Coyle, 2019; Rae-Grant et al., 2018).

Interferon beta-1a and interferon beta-1b are administered subcutaneously every other day. Another preparation of interferon beta-1a may be given intramuscularly once a week and pegylated interferon beta-1a can be given subcutaneously every 14 days. Side effects of all interferon-beta medications include flulike symptoms, increased liver function tests, leukopenia, headache, depression, and skin necrosis (Coyle, 2019). For optimal control of disability, disease-modifying medications should be started early in the course of the disease (Rae-Grant et al., 2018).

Glatiramer acetate also reduces the rate of relapse in RRMS and is administered subcutaneously daily. It has some adverse effects, such as injection-site reactions and flushing, but these are usually self-limiting to a few minutes. There are no monitoring parameters (Bradshaw & Houtchens, 2018).

Teriflunomide, fingolimod, and dimethyl fumarate are oral disease-modifying therapies that may be better tolerated by the patient who has difficulty with injection reactions. These medications have significantly reduced relapse rates in several types of MS (Bradshaw & Houtchens, 2018). Ocrelizumab has a 6% annual relapse reduction rate in patients with PPMS (Bradshaw & Houtchens, 2018).

IV methylprednisolone, used to treat acute exacerbations, shortens the duration of relapse but has not been found to have long-term benefit (Bradshaw & Houtchens, 2018). It exerts anti-inflammatory effects by acting on T cells and cytokines. The medication is given as 1 g IV daily for 3 to 5

days, followed by an oral taper of prednisone. Side effects include mood swings, weight gain, and electrolyte imbalances (Comerford & Durkin, 2020).

The medication mitoxantrone is given via IV infusion every 3 months. Mitoxantrone can reduce the frequency of clinical relapses in patients with secondary progressive or worsening RRMS. Patients must be very closely monitored for side effects (i.e., cardiac toxicity), and there is a maximum lifetime dose that can be given (Hickey & Strayer, 2020).

### Symptom Management

Medications are also prescribed for management of specific symptoms. Baclofen, a gamma-aminobutyric acid agonist, is the medication of choice for treating spasticity. It can be given orally or by intrathecal injection for severe spasticity (Hickey & Strayer, 2020). Benzodiazepines (e.g., diazepam), tizanidine, and dantrolene may also be used to treat spasticity and improve motor function (Hickey & Strayer, 2020). Patients with disabling spasms and contractures may require nerve blocks or surgical intervention. Fatigue that interferes with activities of daily living (ADLs) may be treated with amantadine, pemoline, or dalfampridine. Ataxia is a chronic problem most resistant to treatment. Medications used to treat ataxia include beta-adrenergic blockers (e.g., propranolol), the anticonvulsant agent gabapentin, and benzodiazepines (e.g., clonazepam).

Bladder and bowel problems are often difficult for patients, and a variety of medications (anticholinergic agents, alpha-adrenergic blockers, antispasmodic agents) may be prescribed. Nonpharmacologic strategies also assist in establishing effective bowel and bladder elimination.

UTI may be superimposed on the underlying neurologic dysfunction. Increased fluid intake and good perineal care help reduce the risk of UTI. Antibiotic agents are prescribed when appropriate. See [Chapter 49](#) for further discussion of UTI management.

## NURSING PROCESS

### The Patient with MS



#### Assessment

Nursing assessment addresses neurologic deficits and the impact of the disease on the patient and family. The patient's mobility and balance are observed to determine whether there is risk of falling. Assessment of function is carried out both when the patient is well rested and when fatigued. The patient is assessed for weakness, spasticity, visual impairment, incontinence, and disorders of swallowing and speech. Additional areas of assessment include how MS has affected the patient's quality of life, how the patient is coping, adherence to the prescribed medication regimen, and what the patient would like to improve (Debska et al., 2020; Newland et al., 2019).

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired mobility associated with weakness, muscle paresis, spasticity, increased weight
- Risk for fall associated with sensory and visual impairment, lower extremity weakness
- Fatigue associated with insufficient energy
- Difficulty coping associated with uncertainty of course of MS

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Constipation or fecal incontinence (see Chapter 41)
- Communication issues and potential for aspiration related to cranial nerve involvement (see cranial nerve discussion later in this chapter)
- Cognitive changes
- Managing therapies at home related to physical, psychological, and social limits imposed by MS
- Changes in sexuality
- Urinary incontinence (see Chapter 49)

#### Planning and Goals

The major goals for the patient may include promotion of physical mobility, avoidance of falls, decreasing fatigue, development of coping strategies, and absence of complications.

#### Nursing Interventions

An individualized program of physical, occupational, and speech-language therapy, rehabilitation, and education is combined with emotional support. An educational plan of care is developed to enable the person with MS to deal with the physiologic, social, and psychological problems that accompany chronic disease. The presence of depression, pain, fatigue, and walking difficulty all decrease physical activity. Assisting patients with management of these symptoms may help increase the level of physical activity and overall sense of well-being.

#### **PROMOTING PHYSICAL MOBILITY**

Relaxation and coordination exercises promote muscle efficiency. Progressive resistive exercises are used to strengthen weak muscles, because diminishing muscle strength is often significant in MS.

**Exercise.** Walking improves the gait, particularly the problem of loss of position sense of the legs and feet. If certain muscle groups are irreversibly affected, other muscles can be trained to compensate. Instruction in the use of assistive devices may be needed to ensure their safe and correct use.

**Minimizing Spasticity and Contractures.** Muscle spasticity is common and, in its later stages, is characterized by severe adductor spasm of the hips with flexor spasm of the hips and knees. Without relief, fibrous contractures of these joints occur. Warm packs may be beneficial, but hot baths should be avoided because of risk of burn injury secondary to sensory loss and increasing symptoms that may occur with elevation of the body temperature. Exposure to extreme cold is avoided as this may increase spasticity.

Daily exercises for muscle stretching are prescribed to minimize joint contractures. Special attention is given to the hamstrings, gastrocnemius muscles, hip adductors, biceps, and wrist and finger flexors. Muscle spasticity is common and interferes with usual function. Application of prescribed orthotics may help maintain a functional position and reduce contractures. A stretch–hold–relax routine is helpful for relaxing and treating muscle spasticity. Swimming and stationary bicycling are useful, and progressive weight bearing can relieve spasticity in the legs. The patient should not be hurried in any of these activities, because this often increases spasticity.

**Activity and Rest.** The patient is encouraged to work and exercise to a point just short of fatigue. Very strenuous physical exercise is not advisable, because it raises the body temperature and may aggravate symptoms. The patient is advised to take frequent short rest periods. Exposure to heat increases fatigue and muscle weakness, so air conditioning is recommended in at least one room.



**Nutrition.** Similar to the population at large, many patients with MS are overweight or have obesity. Contributing factors include the use of corticosteroids for exacerbations of symptoms and mobility

impairments as a result of the disease. Interventions to promote healthy eating and weight reduction need to take into account that fatigue and mobility impairments are barriers to engagement in nutritional behaviors for people with MS. Nurses also need to be certain to include family members in interventions and nutrition education, because they are often the gatekeepers for food preparation and selection. Additional strategies include avoidance of alcohol and cigarette smoking.

#### **PREVENTING FALLS**

If motor dysfunction causes problems of incoordination and clumsiness, or if ataxia is apparent, then the patient is at risk for falls. To overcome this risk, the patient is instructed to walk with feet apart to widen the base of support and to increase walking stability. If loss of position sense occurs, the patient is instructed to watch their feet while walking. Gait training may require assistive devices (walker, cane, braces, crutches, parallel bars) and instruction about their use by a physical therapist. If the gait remains inefficient, a wheelchair or motorized scooter may be the solution. The occupational therapist is a valuable resource person in suggesting and securing aids to promote independence. If incoordination is a problem and tremor of the upper extremities occurs when voluntary movement is attempted (intention tremor), weighted wrist weights or neuromodulation devices may be used. The patient is trained in transfer and ADLs.

Because sensory loss may occur in addition to motor loss, pressure injuries are a continuing threat to skin integrity. The need to use a wheelchair continuously increases the risk. See [Chapter 56](#) for a discussion of the prevention and treatment of pressure injury.

#### **MANAGING FATIGUE**

Fatigue is a common symptom reported in 60% to 90% of those with MS, but the etiology remains unclear. It is often the most disabling symptom and the most common reason patients cease employment (Hickey & Strayer, 2020; Newland et al., 2019). Many factors contribute to fatigue and the nurse helps the patient identify risks and ameliorate those that lead to fatigue. Research that identified the relationships among MS-related symptoms, sleep hygiene behaviors, and sleep quality in adults with MS who report fatigue suggests that decreasing the use of electronic devices prior to sleep can improve sleep quality and lessen fatigue (Newland et al., 2019). See the Nursing Research Profile in [Chart 64-2](#).

#### **STRENGTHENING COPING MECHANISMS**

The diagnosis of MS is distressing to the patient and family. They need to know that no two patients with MS have identical symptoms or courses of illness. Although some patients do experience significant disability, others have a near-normal lifespan with minimal disability. Some families, however, face acute frustrations and problems. MS affects people who are

often in an early stage of life and concerned about career and family responsibilities. Family conflict, disintegration, separation, and divorce are not uncommon. Often, young family members assume the responsibility of caring for a parent with MS. Nursing interventions in this area include assisting patients and families to manage or reduce stress and making appropriate referrals for counseling and support to minimize the adverse effects of dealing with chronic illness.

Chart 64-2



## NURSING RESEARCH PROFILE

## The Relationship Between MS and Sleep

Newland, P., Lorenz, R. A., Smith, J. M., et al. (2019). The relationship among multiple sclerosis-related symptoms, sleep quality, and sleep hygiene behaviors. *Journal of Neuroscience Nursing*, 51(1), 37–42.

### Purpose

Fatigue is common among those with MS and has a negative impact on many aspects of their lives. The purpose of this study was to examine the relationships among MS-related symptoms, sleep hygiene behaviors, and sleep quality in adults with MS who report fatigue.

### Design

This was a descriptive correlational study with a convenience sample of 39 community dwelling adults with MS. Data were collected about demographic characteristics. Measures used to collect data included the revised MS-Related Symptom Scale, the patient self-report version of the Expanded Disability Status Scale, a single item sleep quality scale, the Pittsburgh Sleep Quality Index (PSQI), and a sleep behavior self-rating scale.

### Findings

The mean age of participants was 45 years; 80% reported having RRMS and 20% reported PPMS. High levels of forgetfulness, anxiety, and difficulty concentrating were significantly correlated with poor sleep quality. Fatigue was higher in those who used electronic devices around bedtime and practiced poor sleep hygiene behaviors. Pain, a frequent symptom in those with MS, was not significantly related to sleep quality.

### Nursing Implications

Nurses working with patients with MS should incorporate interventions for symptoms, particularly forgetfulness, anxiety, and difficulty concentrating, as they significantly impact sleep. Education should include the recommendation to remove electronic devices from the bedroom, and to restrict the use of technology in the hours prior to sleep.

The nurse, mindful of these complex problems, initiates home care and coordinates a network of services, including social services, speech therapy, physical therapy, and homemaker services. To strengthen the patient's coping skills, as much information as possible is provided. Patients need a list of available assistive devices, services, and resources.

Coping through problem solving involves helping the patient define the problem and develop alternatives for its management. Planning carefully, maintaining flexibility, and preserving a hopeful attitude are useful for psychological and physical adaptation.

### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Complications that can occur with MS are caused by damage to the myelin in the CNS. The nurse monitors for the presence of cognitive changes, how the patient is able to manage at home, or changes in sexuality. Cognitive changes or inability to manage prescribed therapies at home may be due to psychological effects of MS. The patient is monitored for the risk of suicide as 50% of patients with MS experience major depression and the suicide rate is twice that of the general population (Kalb et al., 2019).

### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



**Educating Patients About Self-Care.** As the disease progresses, the patient and family need to learn new strategies to maintain optimal independence. Educating about self-care techniques may be initiated in the hospital or clinic setting and reinforced in the home. Self-care education may address the use of assistive devices, self-catheterization, and administration of medications that affect the course of the disease or treat complications. An education plan that addresses intramuscular or subcutaneous administration of medications (including side effects) is developed for the patient and their family or caregiver. The patient and family are educated about exercises that enable the patient to continue some form of activity or that maintain or improve function (see [Chart 64-3](#)).

**Continuing and Transitional Care.** After discharge, the nurse often provides education and reinforcement of new interventions in the patient's home. Nurses in the home setting assess for changes in the patient's physical and emotional status; provide physical care to the patient if required; coordinate outpatient services and resources; and encourage health promotion, appropriate health screenings, and adaptation. Modifications that allow independence in the home should be implemented (e.g., assistive eating devices, raised toilet seat, bathing aids, telephone modifications, long-handled comb, tongs, modified clothing).

If changes in the disease or its course are noted, the nurse encourages the patient to contact the primary provider, because treatment of an acute exacerbation or new problem may be indicated. Continuing health care and follow-up are recommended.

The patient with MS is encouraged to contact the local chapter of the National MS Society for services, publications, and contact with others who have MS (see the Resources section). Local chapters also provide direct services to patients. Through group interaction, the patient has an opportunity to meet others with similar problems, share experiences, and learn self-help methods.

### **Evaluation**

Expected patient outcomes may include:

1. Improves physical mobility
  - a. Participates in gait training and rehabilitation program
  - b. Establishes a balanced program of rest and exercise
  - c. Uses assistive devices correctly and safely
2. Is free of falls
  - a. Monitors self and environment for falls risk factors
  - b. Asks for assistance when necessary
3. Reports decreased level of fatigue
  - a. Identifies strategies to decrease fatigue
  - b. Maintains appropriate sleep hygiene behaviors

Chart 64-3



### HOME CARE CHECKLIST

## The Patient with MS

**At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of MS and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the purpose, dose, route, schedule, side effects, and precautions for prescribed medications.
  - Demonstrate correct techniques of administering injectable medications, if prescribed.
- State how to contact all members of the treatment team (e.g., health care providers, home care professionals, and durable medical equipment and supply vendor).
- State changes in lifestyle (e.g., exercise, activity) necessary to maintain health.
  - Demonstrate environmental modifications and adaptive techniques for accomplishing activities of daily living.
  - Identify strategies to manage symptoms (pain, cognitive responses, dysphagia, tremors, visual disturbances).
  - State how to prevent complications (e.g., pressure injury, pneumonia, depression).
  - Identify coping strategies.
  - Identify ways to minimize fatigue.
  - Explain how to prevent injury.
  - Identify optimal nutritional intake; consider weight reduction as indicated if patient is overweight or has obesity.
  - State ways to promote sexual function.
  - Discuss ways to manage bowel and bladder function.
  - Name benefits of exercise and physical activity.
  - Identify ways to minimize immobility and spasticity.
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.
- Identify sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families (e.g., National MS Society, MS support services).
- Identify the need for health promotion, disease prevention, and screening activities.

## Resources

See [Chapter 7, Chart 7-9: Home Care Checklist: Managing Chronic Illness](#) for additional information.

ADLs, activities of daily living; IADLs, instrumental activities of daily living; MS, multiple sclerosis.

4. Demonstrates effective coping strategies
  - a. Maintains sense of control
  - b. Modifies lifestyle to fit goals and limitations
  - c. Verbalizes desire to pursue goals and developmental tasks of adulthood
  - d. Demonstrates healthy social interactions
  - e. Participates in meaningful activities
5. Understands ways to avoid complications and is free of complications
6. Explains reasons for measures to prevent complications

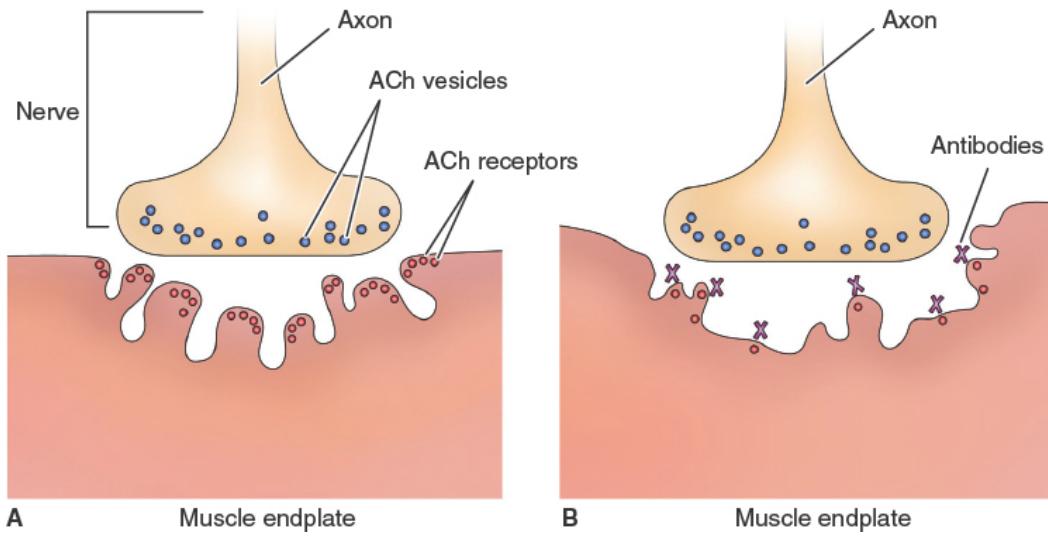
## Myasthenia Gravis

Myasthenia gravis, an autoimmune disorder affecting the myoneural junction, is characterized by varying degrees of weakness of the voluntary muscles. It is uncommon, with an incidence between 9 and 30 in 1 million people in the United States (Hickey & Strayer, 2020). It occurs more often in women during the second and third decades of life; however, after age 50, it is more common in men (Hickey & Strayer, 2020).

## Pathophysiology

Normally, a chemical impulse precipitates the release of acetylcholine from vesicles on the nerve terminal at the myoneural junction. The acetylcholine attaches to receptor sites on the motor endplate and stimulates muscle contraction. Continuous binding of acetylcholine to the receptor site is required for muscular contraction to be sustained.

In myasthenia gravis, antibodies directed at the acetylcholine receptor sites impair transmission of impulses across the myoneural junction. Therefore, fewer receptors are available for stimulation, resulting in voluntary muscle weakness that escalates with continued activity (see [Fig. 64-4](#)). These antibodies are found in 85% of people with myasthenia gravis (Hickey & Strayer, 2020). Of people with myasthenia gravis, most have either thymic hyperplasia or a thymic tumor, and the thymus gland is believed to be the site of antibody production. In patients who are acetylcholine receptor antibody negative, other antibodies appear to target a protein in the myoneural junction (Hickey & Strayer, 2020).



**Figure 64-4 • Myasthenia gravis. A.** Usual acetylcholine (ACh) receptor site. **B.** ACh receptor site in myasthenia gravis.

## Clinical Manifestations

The clinical manifestation of myasthenia gravis is highly variable. There are two clinical types: ocular and generalized. In the ocular form, only the eye muscles are involved. Diplopia and **ptosis** (drooping of the eyelids) are common (Hickey & Strayer, 2020). In the generalized form, patients experience weakness of the muscles of the face and throat (bulbar symptoms), limbs, and respiratory weakness. Weakness of the facial muscles results in a bland facial expression. Laryngeal involvement produces **dysphonia** (voice impairment) and **dysphagia** (difficulty swallowing), which increases the risk of choking and aspiration. Generalized weakness affects all extremities and may involve the intercostal muscles, resulting in decreasing vital capacity and respiratory failure. When this occurs, the patient is in a myasthenic crisis (National Institute of Neurological Disorders and Stroke [NINDS], 2020). Myasthenia gravis is purely a motor disorder with no effect on sensation or coordination.

## Assessment and Diagnostic Findings

A common test used to diagnose myasthenia gravis is the acetylcholinesterase inhibitor test. It is performed by administering edrophonium chloride IV; 30 seconds after injection, facial muscle weakness and ptosis should resolve for about 5 minutes (Hickey & Strayer, 2020). Immediate improvement in muscle strength after administration of this agent represents a positive test and usually confirms the diagnosis. Atropine should be available to control potential side

effects of this medication, which include bradycardia, asystole, bronchoconstriction, sweating, and cramping.

Another study, the ice test, is indicated in patients who have cardiac conditions or asthma. With this test, an ice pack is held over the patient's eyes for 1 minute; the ptosis should temporarily resolve in a patient with myasthenia gravis (Hickey & Strayer, 2020).

Several blood tests for acetylcholine antibodies are also used to confirm the diagnosis (Hickey & Strayer, 2020). Repetitive nerve stimulation (RNS) demonstrates a decrease in successive action potentials. A single-fiber electromyography (EMG) detects a delay or failure of neuromuscular transmission and is about 99% sensitive in confirming the diagnosis of myasthenia gravis (Hickey & Strayer, 2020). This is an uncomfortable test for the patient.

The thymus gland, a site of acetylcholine receptor antibody production, may be enlarged in myasthenia gravis and may be identified by MRI scan.

## Medical Management

Management of myasthenia gravis is directed at improving function and reducing and removing circulating antibodies. Therapeutic modalities include administration of anticholinesterase medications and immunosuppressive therapy, intravenous immune globulin (IVIG), therapeutic plasma exchange, and thymectomy. There is no cure for myasthenia gravis; treatments do not stop the production of the acetylcholine receptor antibodies.

### Pharmacologic Therapy

Pyridostigmine bromide, an anticholinesterase medication, is the first line of therapy (Hickey & Strayer, 2020). It provides symptomatic relief by inhibiting the breakdown of acetylcholine and increasing the relative concentration of available acetylcholine at the neuromuscular junction. The dosage is gradually increased to a daily maximum and is given in divided doses (usually four times a day). Adverse effects of anticholinesterase medications include diarrhea, abdominal cramps, and/or excessive saliva (Comerford & Durkin, 2020). Pyridostigmine tends to have fewer side effects than other anticholinesterase medications.

If pyridostigmine bromide does not improve muscle strength and control fatigue, the next agents used are the immunomodulating drugs. The goal of immunosuppressive therapy is to reduce production of the antibody. Corticosteroids suppress the patient's immune response, decreasing the amount of antibody production, and this correlates with clinical improvement. An initial dose of prednisone is given daily and maintained for 1 to 2 months; as symptoms improve, the medication is tapered (Hickey & Strayer, 2020). As the corticosteroid medications take effect, the dosage of anticholinesterase

medication can usually be lowered. Cytotoxic medications are used to treat myasthenia gravis if there is inadequate response to steroids. Azathioprine inhibits T lymphocytes and B-cell proliferation and reduces acetylcholine receptor antibody levels. Therapeutic effects may not be evident for 3 to 12 months. Leukopenia and hepatotoxicity are serious adverse effects, so monthly evaluation of liver enzymes and white blood cell count is necessary.

IVIG can be used to treat exacerbations; however, in select patients, it is used on a long-term adjunctive basis. IVIG treatment involves the administration of pooled human gamma-globulin and improvement occurs in a few days to a week (Hickey & Strayer, 2020). The effects of IVIG typically last only about 28 days after infusion, and complications include headache, migraine exacerbation, aseptic meningitis, and flulike symptoms (Vitiello, Emmi, Silvestri, et al., 2019).

A number of medications are contraindicated for patients with myasthenia gravis because they exacerbate the symptoms. The primary provider and the patient should weigh risks and benefits before any new medications are prescribed. Procaine should be avoided, and the patient's dentist is informed of the diagnosis of myasthenia gravis.

### **Therapeutic Plasma Exchange**

A technique called therapeutic plasma exchange, formerly referred to as plasmapheresis, is used to treat exacerbations. The patient's plasma and plasma components are removed through a centrally placed large-bore double-lumen catheter. The blood cells and antibody-containing plasma are separated, after which the cells and a plasma substitute are reinfused. Temporary reduction in the level of circulating antibodies is provided with therapeutic plasma exchange. A typical course consists of daily or alternate-day treatment, and the number of treatments is determined by the patient's response (Hickey & Strayer, 2020).



### **Surgical Management**

Thymectomy (surgical removal of the thymus gland) can produce antigen-specific immunosuppression and result in clinical improvement. Optimal outcomes of the surgery are in patients younger than 60 years who have had myasthenia gravis diagnosed within the past 3 years. It is the only treatment that can result in complete remission, which occurs in approximately 35% of patients (Hickey & Strayer, 2020). A course of preoperative IVIG or therapeutic plasma exchange decreases the time needed for postoperative mechanical ventilation. The entire thymus gland must be removed for optimal clinical outcomes.

A thymectomy should be performed at a designated center with a surgical and anesthesia staff experienced in the perioperative management of those with myasthenia gravis (Hickey & Strayer, 2020). After surgery, the patient is monitored in an intensive care unit, with special attention to respiratory function. The patient is weaned from mechanical ventilation after thorough respiratory assessment. After the thymus gland is removed, it may take up to 3 years for the patient to benefit from the procedure because of the long life of circulating T cells. Thymectomy is considered an elective surgery and best performed when the clinical course of the disease is stable for optimal outcomes (Hickey & Strayer, 2020).



## Complications

A myasthenic crisis is an exacerbation of the disease process characterized by severe generalized muscle weakness and respiratory and bulbar weakness that may result in respiratory failure. Crisis may result from disease exacerbation or a specific precipitating event. The most common precipitator is respiratory infection; others include medication change, surgery, pregnancy, and medications that exacerbate myasthenia. A cholinergic crisis caused by overmedication with cholinesterase inhibitors is rare (Hickey & Strayer, 2020).

Neuromuscular respiratory failure is the critical complication in myasthenic and cholinergic crises. Respiratory muscle and bulbar weakness combine to cause respiratory compromise. Weak respiratory muscles do not support inhalation. An inadequate cough and an impaired gag reflex, caused by bulbar weakness, result in poor airway clearance. A downward trend of two respiratory function tests, the negative inspiratory force and vital capacity, is the first clinical sign of respiratory compromise.

Endotracheal intubation and mechanical ventilation may be needed (see [Chapter 19](#)). Noninvasive positive-pressure ventilation uses an external device in the form of a vest that provides respiratory support without endotracheal intubation. Cholinesterase inhibitors are stopped when respiratory failure occurs and gradually restarted after the patient demonstrates improvement with a course of therapeutic plasma exchange or IVIG. Nutritional support may be needed if the patient is intubated for a long period or swallowing ability is affected (see [Chapter 39](#)).

## Nursing Management

Because myasthenia gravis is a chronic disease and most patients are seen on an outpatient basis, much of the nursing care focuses on patient and family education. Educational topics for outpatient self-care include medication

management, energy conservation, strategies to help with ocular manifestations, and prevention and management of complications.

Medication management is a crucial component of ongoing care. Understanding the actions of the medications and taking them on schedule is emphasized, as are the consequences of delaying medication and the signs and symptoms of myasthenic and cholinergic crises. The patient can determine the best times for daily dosing by keeping a diary to determine fluctuation of symptoms and to learn when the medication is wearing off. The medication schedule can then be manipulated to maximize strength throughout the day.



#### **Quality and Safety Nursing Alert**

*Maintenance of stable blood levels of anticholinesterase medications is imperative to stabilize muscle strength. Therefore, the anticholinesterase medications must be given on time. Any delay in administration of medications may exacerbate muscle weakness and make it impossible for the patient to take medications orally.*

Regular administration of IVIG or subcutaneous immunoglobulin may be prescribed. The patient and family are educated about managing immunoglobulin therapy.



For the procedural guidelines for management of IVIG, go to  
[thepoint.lww.com/Brunner15e](http://thepoint.lww.com/Brunner15e).

The patient is also educated about strategies to conserve energy. To do this, the nurse helps the patient identify the optimal times for rest throughout the day. If the patient lives in a two-story home, the nurse can suggest that frequently used items (e.g., hygiene products, cleaning products, snacks) be kept on each floor to minimize travel between floors. The patient is encouraged to apply for a handicapped license plate to minimize walking from parking spaces and to schedule activities to coincide with peak energy and strength levels. Using consistent routines, scheduling periods of rest, monitoring for depression, maintaining good sleep patterns, and incorporating interventions to conserve energy are all strategies to reduce fatigue (Hickey & Strayer, 2020).

To minimize the risk of aspiration, mealtimes should coincide with the peak effects of anticholinesterase medication. In addition, rest before meals is encouraged to reduce muscle fatigue. The patient is advised to sit upright during meals, with the neck slightly flexed to facilitate swallowing. Soft foods

in gravy or sauces can be swallowed more easily. Eating larger meals in the morning and smaller meals in the evening is another good strategy. Supplemental feedings may be helpful to ensure adequate nutrition.

If choking occurs frequently, the patient should be evaluated by a speech-language pathologist for formal dietary and mechanical techniques to avoid aspiration. Suction should be available at home, with the patient and family instructed in its use.

Impaired vision results from ptosis of one or both eyelids, decreased eye movement, or double vision. To prevent corneal damage when the eyelids do not close completely, the patient is instructed to tape the eyes closed for short intervals and to regularly instill artificial tears. Patients who wear eyeglasses can have “crutches” attached to help lift the eyelids. Patching of one eye or wearing prism glasses can help with double vision.

The patient is reminded of the importance of maintaining health promotion practices and following health care screening recommendations. Factors that exacerbate symptoms and potentially cause crisis should be noted and avoided: emotional stress, infections (particularly respiratory infections), vigorous physical activity, some medications, and high environmental temperature. The Myasthenia Gravis Foundation of America provides support services and educational materials for patients, families, and health care providers (see the Resources section).



### Myasthenic Crisis

Respiratory distress and varying degrees of dysphagia, dysarthria, eyelid ptosis, diplopia, and prominent muscle weakness are symptoms of myasthenic crisis. The patient is placed in an intensive care unit for constant monitoring because of associated intense and sudden fluctuations in clinical condition.

Providing ventilatory assistance takes precedence in the immediate management of the patient with myasthenic crisis. Ongoing assessment for respiratory failure is essential. The nurse assesses the respiratory rate, depth, and breath sounds and monitors pulmonary function parameters (vital capacity and negative inspiratory force) to detect pulmonary problems before respiratory dysfunction progresses. Blood is drawn for arterial blood gas analysis. Endotracheal intubation and mechanical ventilation may be needed (see [Chapter 19](#)).

If the abdominal, intercostal, and pharyngeal muscles are severely weak, the patient cannot cough, take deep breaths, or clear secretions. Chest physiotherapy, including postural drainage to mobilize secretions and suctioning to remove secretions, may have to be performed frequently. (Postural drainage should not be performed for 30 minutes after feeding.)

Assessment strategies and supportive measures include the following:

- Arterial blood gases, serum electrolytes, input and output, and daily weight are monitored.
- If the patient cannot swallow, enteral tube feedings may be prescribed (see [Chapter 39](#)).
- Sedative and tranquilizing agents are avoided, because they aggravate hypoxia and hypercapnia and can cause respiratory and cardiac depression.

## Guillain–Barré Syndrome

Guillain–Barré Syndrome (GBS), also known as acute idiopathic polyneuritis, is an autoimmune attack on the peripheral nerve myelin. The result is acute, rapid segmental demyelination of peripheral nerves and some cranial nerves, producing ascending weakness with dyskinesia (inability to execute voluntary movements), hyporeflexia, and **paresthesias** (a sensation of numbness, tingling, or a “pins and needles” sensation). An antecedent event (most often a viral infection) precipitates clinical presentation in approximately 60% to 70% of cases (CDC, 2019). *Campylobacter jejuni* (implicated in 40% of cases in the United States), cytomegalovirus, Epstein–Barr virus, *Mycoplasma pneumoniae*, *H. influenzae*, and Zika virus are the most common infectious agents that are associated with the development of GBS.

There are several subtypes of GBS (Malek & Salameh, 2019). With the most well-known type, the patient experiences weakness in the lower extremities, which progresses upward and has the potential for respiratory failure. The second type is purely motor with no altered sensation. A third type, called descending GBS, is much more difficult to diagnose; it mostly affects the head and neck muscles. The rarest type is the Miller–Fisher variant (see discussion later in this chapter) (NINDS, 2018b).

The annual incidence of GBS is 1 to 2 cases per 100,000 people, and it affects males and females equally. Death occurs in 5% to 10% of cases, resulting from respiratory failure, autonomic dysfunction, sepsis, or pulmonary embolism (PE) (Hickey & Strayer, 2020). Seventy percent of patients with GBS experience full recovery. The remaining 30% can have disability ranging from minor to major (NINDS, 2018b).

## Pathophysiology

GBS is the result of a cell-mediated and humoral immune attack on peripheral nerve myelin proteins that causes inflammatory demyelination. The best-accepted theory of cause is molecular mimicry, in which an infectious organism contains an amino acid that mimics the peripheral nerve myelin protein. The immune system cannot distinguish between the two proteins and

attacks and destroys peripheral nerve myelin. The exact location of the immune attack within the peripheral nervous system is the ganglioside GM1b. With the autoimmune attack, there is an influx of macrophages and other immune-mediated agents that attack myelin and cause inflammation and destruction, interruption of nerve conduction, and axonal loss (NINDS, 2018b).

Myelin is a complex substance that covers nerves, providing insulation and speeding the conduction of impulses from the cell body to the dendrites. The cell that produces myelin in the peripheral nervous system is the Schwann cell. In GBS, the Schwann cell can be spared, allowing for remyelination in the recovery phase of the disease. If damage has occurred to the axons, then regrowth is required and takes months or years and is often incomplete (NINDS, 2018b).

## Clinical Manifestations

GBS typically begins with muscle weakness and diminished reflexes of the lower extremities. Hyporeflexia and weakness may progress to tetraplegia. Demyelination of the nerves that innervate the diaphragm and intercostal muscles results in neuromuscular respiratory failure. Sensory symptoms include paresthesias of the hands and feet and pain related to the demyelination of sensory fibers.

The antecedent event usually occurs 1 to 3 weeks before symptoms begin. Weakness usually begins in the legs and may progress upward. Maximum weakness (the plateau) varies in length but usually includes neuromuscular respiratory failure and bulbar weakness. GBS progresses to peak severity typically within 2 weeks and no longer than 4 weeks. If progression is longer, then the patient is classified as having chronic inflammatory demyelinating polyneuropathy (Hickey & Strayer, 2020). Any residual symptoms are permanent and reflect axonal damage from demyelination.

Cranial nerve demyelination can result in a variety of clinical manifestations. Optic nerve demyelination may result in blindness. Bulbar muscle weakness related to demyelination of the glossopharyngeal and vagus nerves results in the inability to swallow or clear secretions. Vagus nerve demyelination results in autonomic dysfunction, manifested by instability of the cardiovascular system. The presentation is variable and may include tachycardia, bradycardia, hypertension, or orthostatic hypotension. The symptoms of autonomic dysfunction occur and resolve rapidly. GBS does not affect cognitive function or LOC.

Although the classic clinical features include areflexia and ascending weakness, variations in the clinical presentation occurs. There may be a sensory presentation, with progressive sensory symptoms; an atypical axonal

destruction; or the Miller–Fisher variant, which includes paralysis of the ocular muscles, ataxia, and areflexia (Malek & Salameh, 2019; NINDS, 2018b).

## Assessment and Diagnostic Findings

The patient presents with symmetric weakness, diminished reflexes, and upward progression of motor weakness. A history of a viral illness in the previous few weeks suggests the diagnosis. Changes in vital capacity and negative inspiratory force are assessed to identify impending neuromuscular respiratory failure. Serum laboratory tests are not useful in the diagnosis. However, elevated protein levels are detected in CSF evaluation, without an increase in other cells. Electrophysiology studies demonstrate a progressive loss of nerve conduction velocity (Malek & Salameh, 2019).



## Medical Management

Because of the possibility of rapid progression and neuromuscular respiratory failure, GBS is a medical emergency that may require management in an intensive care unit. After baseline values are identified, assessment of changes in muscle strength and respiratory function alert the clinician to the physical and respiratory needs of the patient. Respiratory therapy or mechanical ventilation may be necessary to support pulmonary function and adequate oxygenation. Some clinicians recommend elective intubation before the onset of extreme respiratory muscle fatigue. Emergent intubation may result in autonomic dysfunction, and mechanical ventilation may be required for an extended period. The patient is weaned from mechanical ventilation after the respiratory muscles can again support spontaneous respiration and maintain adequate tissue oxygenation.

Other interventions are aimed at preventing the complications of immobility. These may include the use of anticoagulant agents and sequential compression boots to prevent venous thromboembolism (VTE), including deep vein thrombosis (DVT) and PE.

Therapeutic plasma exchange and IVIG are used to directly affect the peripheral nerve myelin antibody level. Both therapies decrease circulating antibody levels and reduce the amount of time the patient is immobilized and dependent on mechanical ventilation. The cardiovascular risks posed by autonomic dysfunction require continuous electrocardiographic (ECG) monitoring. Tachycardia and hypertension are treated with short-acting medications such as alpha-adrenergic blocking agents. The use of short-acting agents is important, because autonomic dysfunction is very labile. Hypotension is managed by increasing the amount of IV fluid administered.

## NURSING PROCESS

### The Patient with GBS

#### Assessment

Ongoing assessment for disease progression is critical. The patient is monitored for life-threatening complications (respiratory failure, cardiac arrhythmias, VTE [including DVT or PE]) so that appropriate interventions can be initiated. Because of the threat to the patient in this sudden, potentially life-threatening disease, the nurse must assess the patient's and family's ability to cope and their use of coping strategies.

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired breathing associated with rapidly progressive weakness and impending respiratory failure
- Impaired mobility associated with paralysis
- Impaired nutritional intake associated with inability to swallow
- Impaired verbal communication associated with cranial nerve dysfunction
- Anxiety associated with loss of control and paralysis
- Fatigue associated with physical deconditioning and stressors

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Respiratory failure
- Autonomic dysfunction

#### Planning and Goals

The major goals for the patient may include improved respiratory function, increased mobility, improved nutritional status, effective communication, decreased anxiety, decreased fatigue, and absence of complications.

#### Nursing Interventions

##### MAINTAINING RESPIRATORY FUNCTION

Respiratory function can be maximized with incentive spirometry and chest physiotherapy. Monitoring for changes in vital capacity and negative inspiratory force is key to early intervention for neuromuscular respiratory failure. Mechanical ventilation is required if the vital capacity falls, making spontaneous breathing impossible and tissue oxygenation inadequate.

The potential need for mechanical ventilation should be discussed with the patient and family on admission to provide time for psychological

preparation and decision making. Intubation and mechanical ventilation result in less anxiety if they are initiated on a nonemergency basis to a patient who has been well informed. The patient may require mechanical ventilation for a long period. See [Chapter 19](#) for the nursing management of the patient requiring mechanical ventilation.

Bulbar weakness that impairs the ability to swallow and clear secretions is another factor in the development of respiratory failure in the patient with GBS. Suctioning may be needed to maintain a clear airway.

The nurse assesses the blood pressure and heart rate frequently to identify autonomic dysfunction so that interventions can be initiated quickly if needed. Medications are given or a temporary pacemaker placed for clinically significant bradycardia.

#### **ENHANCING PHYSICAL MOBILITY**

Nursing interventions to enhance physical mobility and prevent the complications of immobility are key to the function and survival of patients. The paralyzed extremities are supported in functional positions, and passive range-of-motion exercises are performed at least twice daily. DVT and PE are threats to the patient who is paralyzed. Nursing interventions are aimed at preventing VTE. Range-of-motion exercises, position changes, anticoagulation, the use of anti-embolism stockings and sequential compression boots, and adequate hydration decrease the risk of VTE.

Padding may be placed over bony prominences, such as the elbows and heels, to reduce the risk of pressure injury. The need for frequent position changes cannot be overemphasized. The nurse evaluates laboratory test results that may indicate malnutrition or dehydration, both of which increase the risk of pressure injury and decrease mobility. The nurse collaborates with the primary provider and dietitian to develop a plan to meet the patient's nutritional and hydration needs.

#### **PROVIDING ADEQUATE NUTRITION**

Paralytic ileus may result from insufficient parasympathetic activity. In this event, the nurse administers IV fluids and parenteral nutrition as a supplement and monitors for the return of bowel sounds. If the patient cannot swallow because of bulbar paralysis (immobility of muscles), a gastrostomy tube may be placed to administer nutrients. The nurse carefully assesses the return of the gag reflex and bowel sounds before resuming oral nutrition.

#### **IMPROVING COMMUNICATION**

Because of paralysis, the patient cannot talk, laugh, or cry and therefore has no method for communicating needs or expressing emotion. Although the patient may be unable to speak, cognition is completely intact. Establishing some form of communication with picture cards or an eye blink system

provides a means of communication. Collaboration with the speech therapist may be helpful in developing a communication mechanism that is most effective for a specific patient.

#### **DECREASING ANXIETY**

The patient and family are faced with a sudden, potentially life-threatening disease; therefore, their levels of anxiety may be high. The impact of disease on the family depends on the patient's role within the family. Referral to a support group may provide information and support to the patient and family.

The family may feel helpless in caring for the patient. Mechanical ventilation and monitoring devices may frighten and intimidate them. Family members often want to participate in physical care; with education and support by the nurse, they should be allowed and encouraged to do so.

In addition the patient may experience isolation, loneliness, lack of control, and fear. Nursing interventions that increase the patient's sense of control include providing information about the condition, emphasizing a positive appraisal of coping resources, and providing education about relaxation exercises and distraction techniques. The positive attitude and atmosphere of the multidisciplinary team are important to promote a sense of well-being.

Diversional activities are encouraged to decrease loneliness and isolation. Encouraging visitors (when possible), engaging visitors or volunteers to read to the patient, listening to musical recordings or audiobooks, assisting with audio visual communication with friends and family through a phone or electronic device, and watching television or movies are ways to alleviate the patient's sense of isolation.

#### **REDUCING FATIGUE**

The disease process increases disability and dependence on others for simple activities of living. As the patient begins to recover and regains the ability to perform activities on their own, they will find increased rest is needed to maintain this independence. The patient may need only a little help at the beginning of the day, but as the day progresses may require more assistance. Too much exertion will result in fatigue. The patient will need assistance learning how to pace their daily activities to incorporate periods of rest, both physically and mentally. The nurse can help identify activities that are physically demanding, assess the amount of sleep the patient is getting and needs, assist the patient and family to ensure time for self-care, and provide education about healthy eating to maintain strength.

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Thorough assessment of respiratory function at regular and frequent intervals is essential, because respiratory insufficiency and subsequent failure due to weakness or paralysis of the intercostal muscles and

diaphragm may develop quickly. Respiratory failure is the major cause of mortality, although rare. In addition to the respiratory rate and the quality of respirations, vital capacity is monitored frequently and at regular intervals so that respiratory insufficiency can be anticipated. Decreasing vital capacity with associated muscle weakness indicates impending respiratory failure. Signs and symptoms include breathlessness while speaking, shallow and irregular breathing, the use of accessory muscles, tachycardia, weak cough, and changes in respiratory pattern.

Other complications include cardiac arrhythmias (which necessitate ECG monitoring), transient hypertension, orthostatic hypotension, DVT, PE, urinary retention, and other threats to any patient who is immobilized and paralyzed. These complications require monitoring and attention to prevent them and prompt treatment if indicated.

#### PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



**Educating Patients About Self-Care.** Patients with GBS and their families are usually frightened by the sudden onset of life-threatening symptoms and their severity. Therefore, educating the patient and family about the disorder and its generally favorable prognosis is important (see [Chart 64-4](#)).

During the acute phase of the illness, the patient and family are educated about strategies that can be implemented to minimize the effects of immobility and other complications. As function begins to return, family members and other home care providers are educated about care of the patient and their role in the rehabilitation process. Preparation for discharge is an interdisciplinary effort requiring family or caregiver education by all team members, including the nurse, physician, occupational and physical therapists, speech therapist, and respiratory therapist.

**Chart 64-4**



#### HOME CARE CHECKLIST

## The Patient with GBS

**At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of GBS and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the purpose, dose, route, schedule, side effects, and precautions for prescribed medications.
- State how to contact all members of the treatment team (e.g., health care providers, home care professionals, and durable medical equipment and supply vendor).
- State what types of environmental and safety changes or supports are needed for optimum functioning in the home.
  - State changes in lifestyle (e.g., nutrition, skin care, exercise, activity) necessary during recovery period and to maintain health as indicated.
  - Demonstrate environmental modifications and adaptive techniques for accomplishing ADLs (e.g., bathing, hygiene, grooming, dressing) and safely managing self-care.
  - Manage respiratory needs—tracheostomy care, suctioning.
  - Verbalize dietary adjustments and optimum nutrition during recovery.
  - Demonstrate proper body mechanics regarding lifting and transfers.
  - Practice gait training and strength endurance.
  - Perform range-of-motion exercises.
  - Discuss bowel and bladder management.
  - Operate and explain function of medical equipment and mobility aids—walkers, wheelchairs, bedside commodes, tub transfer benches, adaptive devices.
  - Describe coping mechanisms and diversional activities appropriately.
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.
- Identify sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families (e.g., GBS Foundational International).
- Identify the need for health promotion, disease prevention, and screening activities.

ADLs, activities of daily living; GBS, Guillain–Barré syndrome; IADLs, instrumental activities of daily living.

**Continuing and Transitional Care.** Most patients with GBS experience complete recovery. Patients who have experienced total or prolonged paralysis require intensive rehabilitation; the extent depends on the patient's needs. Approaches include a comprehensive inpatient program if deficits are significant, an outpatient program if the patient can travel by car, or a home program of physical and occupational therapy. The recovery phase may be long and requires patience as well as involvement on the part of the patient and family.

During acute care, the focus is on immediate issues and deficits. The nurse needs to remind or educate patients and family members of the need for continuing health promotion and screening practices after this initial phase of care.

### Evaluation

Expected patient outcomes may include the following:

1. Maintains effective respirations and airway clearance
  - a. Has clear breath sounds on auscultation
  - b. Demonstrates gradual improvement in respiratory function
  - c. Breathes spontaneously
  - d. Has vital capacity within normal range
  - e. Exhibits arterial blood gases and pulse oximetry within normal limits
2. Shows increasing mobility
  - a. Regains use of extremities
  - b. Participates in rehabilitation program
  - c. Demonstrates no contractures and minimal muscle atrophy
3. Receives adequate nutrition and hydration
  - a. Consumes diet adequate to meet nutritional needs
  - b. Swallows without aspiration
4. Demonstrates recovery of speech
  - a. Communicates needs through alternative strategies
  - b. Practices exercises recommended by the speech therapist
5. Shows less anxiety
6. Experiences fewer episodes of fatigue
  - a. Verbalizes a plan to reduce fatigue and increase energy
  - b. Takes rest periods during the day
  - c. Identifies activities with higher importance during periods of high energy
7. Has absence of complications

- a. Maintains intact skin integrity
  - b. Does not develop VTE
  - c. Voids without difficulty
- 

## CRANIAL NERVE DISORDERS

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Because the brainstem and cranial nerves involve vital motor, sensory, and autonomic functions of the body, these nerves may be affected by conditions arising primarily within these structures or in secondary extension from adjacent disease processes. The cranial nerves are examined separately and in sequence (see [Chapter 60](#), [Table 60-2](#)). Some cranial nerve deficits can be detected by observing the patient's face, eye movements, speech, and swallowing. EMG is used to investigate motor and sensory dysfunction. An MRI scan is used to obtain images of the cranial nerves and brainstem. An overview of disorders that may affect each of the cranial nerves, including clinical manifestations and nursing interventions, is presented in [Table 64-3](#). The following discussion centers on the most common disorders of the cranial nerves: trigeminal neuralgia, a condition affecting the fifth cranial nerve, and Bell palsy, caused by involvement of the seventh cranial nerve.

**TABLE 64-3** Disorders of Cranial Nerves

Disorder	Clinical Manifestations	Nursing Interventions
<b>Olfactory Nerve—I</b>		
Head trauma Intracranial tumor Intracranial surgery	Unilateral or bilateral anosmia (temporary or persistent) Diminished taste for food	Assess sense of smell Assess for cerebrospinal fluid rhinorrhea if patient has sustained head trauma
<b>Optic Nerve—II</b>		
Optic neuritis Increased intracranial pressure Pituitary tumor	Lesions of optic tract producing homonymous hemianopsia	Assess visual acuity Restructure environment to prevent injuries Educate patient to accommodate for visual loss
<b>Oculomotor Nerve—III</b>		Assess extraocular movement and for nonreactive pupil
<b>Trochlear Nerve—IV</b>		Assess extraocular movement and for nonreactive pupil
<b>Abducens Nerve—VI</b>		
Vascular Brainstem ischemia Hemorrhage and infarction Neoplasm Trauma Infection	Dilation of pupil with loss of light reflex on one side Impairment of ocular movement Diplopia Gaze palsies Ptosis of eyelid	Assess extraocular movement and for nonreactive pupil
<b>Trigeminal Nerve—V</b>		
Trigeminal neuralgia Head trauma Cerebellopontine lesion Sinus tract tumor and metastatic disease Compression of trigeminal root by tumor or blood vessel	Pain in face Diminished or loss of corneal reflex Chewing dysfunction	Assess for pain and triggering mechanisms for pain Assess for difficulty in chewing Discuss trigger zones and pain precipitants with patient Protect cornea from abrasion Ensure good oral hygiene Educate patient about medication regimen
<b>Facial Nerve—VII</b>		
Bell palsy Facial nerve tumor Intracranial lesion Herpes zoster	Facial dysfunction; weakness and paralysis Hemifacial spasm Diminished or absent taste Pain	Recognize facial paralysis as emergency; refer for treatment as soon as possible Discuss protective care for eyes Select easily chewed foods; patient should eat and drink from unaffected side of mouth Emphasize importance of oral hygiene

		Provide emotional support for changed appearance of face
<b>Vestibulocochlear Nerve—VIII</b>		
Tumors and acoustic neuroma Vascular compression of nerve Ménière syndrome	Tinnitus Vertigo Hearing difficulties	Assess pattern of vertigo Provide for safety measures to prevent falls Ensure that patient can maintain balance before ambulating Caution patient to change positions slowly Assist with ambulation Encourage the use of assistive devices
<b>Glossopharyngeal Nerve—IX</b>		
Glossopharyngeal neuralgia from neurovascular compression of cranial nerves IX and X Trauma Inflammatory conditions Tumor Vertebral artery aneurysms	Pain at base of tongue Difficulty in swallowing Loss of gag reflex Palatal, pharyngeal, and laryngeal paralysis	Assess for paroxysmal pain in throat, decreased or absent swallowing, and gag and cough reflexes Monitor for dysphagia, aspiration, and nasal dysarthric speech. Position patient upright for eating or tube feeding
<b>Vagus Nerve—X</b>		
Spastic palsy of larynx; bulbar paralysis; high vagal paralysis Guillain–Barré syndrome Vagal body tumors Nerve paralysis from malignancy, surgical trauma such as carotid endarterectomy	Voice changes (temporary or permanent hoarseness) Vocal paralysis Dysphagia	Assess for airway obstruction/provide airway management Prevent aspiration Support patient having voice reconstruction procedures
<b>Spinal Accessory Nerve—XI</b>		
Spinal cord disorder Amyotrophic lateral sclerosis Trauma Guillain–Barré syndrome	Drooping of affected shoulder with limited shoulder movement Weakness or paralysis of head rotation, flexion, extension; shoulder elevation	Support patient undergoing diagnostic tests
<b>Hypoglossal Nerve—XII</b>		
Medullary lesions	Abnormal movements of	Observe swallowing ability

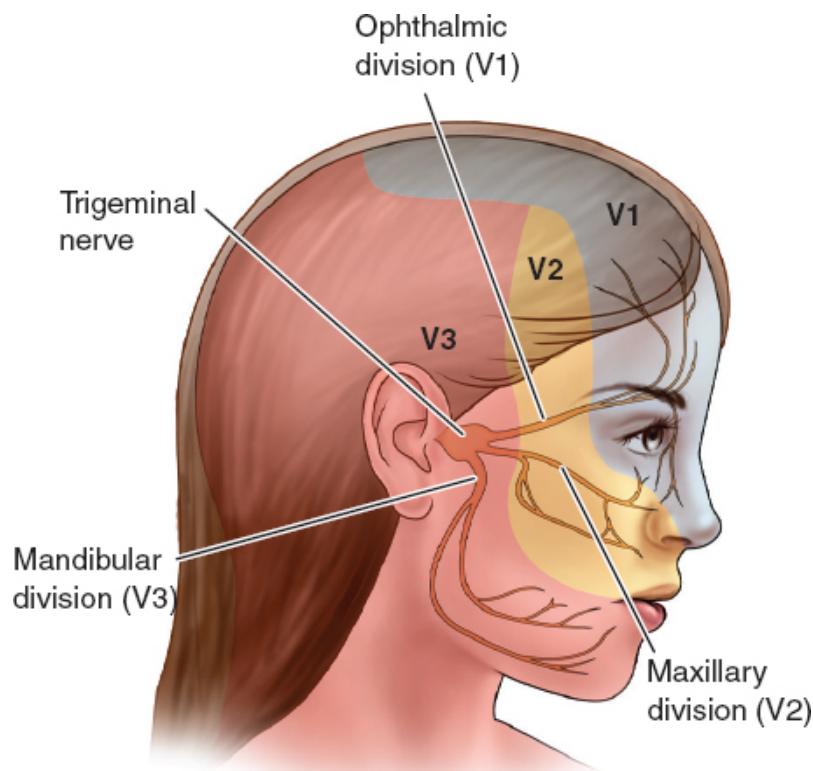
Amyotrophic lateral sclerosis	tongue	Observe speech pattern
Polio and motor system disease, which may destroy hypoglossal nuclei	Weakness or paralysis of tongue muscles	Be aware of swallowing or vocal difficulties
Multiple sclerosis	Difficulty in talking, chewing, and swallowing	Prepare for alternative feeding methods (tube feeding) to maintain nutrition
Trauma		

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological & neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.

## Trigeminal Neuralgia

Trigeminal neuralgia, formerly known as *tic douloureux*, is a condition of the fifth cranial nerve that is characterized by paroxysms of sudden pain in the area innervated by any of the three branches of the nerve (Hickey & Strayer, 2020; see Fig. 64-5). The pain ends as abruptly as it starts and is described as a unilateral shooting and stabbing or burning sensation. The unilateral nature of the pain is an important feature. Associated involuntary contraction of the facial muscles can cause sudden closing of the eye or twitching of the mouth, hence the former name *tic douloureux* (painful twitch). Although the cause is not certain, it is thought to be demyelination of axons in the trigeminal ganglion, root, and nerve by pressing vessels or a demyelinating disease such as MS (Hickey & Strayer, 2020).

Trigeminal neuralgia occurs most often as people age, most commonly between the fifth and sixth decade of life. It is more common in women and in people with MS compared to the general population (Hickey & Strayer, 2020). Patients who develop trigeminal neuralgia before age 50 years should be evaluated for the coexistence of MS, because trigeminal neuralgia occurs more often in patients with MS (Hickey & Strayer, 2020). Pain-free intervals may be measured in terms of minutes, hours, days, or longer. With advancing years, the painful episodes tend to become more frequent and agonizing. The patient lives in constant fear of attacks.



**Figure 64-5 •** Distribution of trigeminal nerve branches—the fifth cranial nerve.

Paroxysms can occur with any stimulation of the terminals of the affected nerve branches, such as washing the face, shaving, brushing the teeth, eating, and drinking. A draft of cold air or direct pressure against the nerve trunk may also cause pain. Certain areas are called *trigger points* because the slightest touch immediately starts a paroxysm or episode. To avoid stimulating these areas, patients with trigeminal neuralgia try not to touch or wash their faces, shave, chew, or do anything else that might cause an attack. These behaviors are a clue to the diagnosis.

## Medical Management

### Pharmacologic Therapy

Anticonvulsant agents, such as carbamazepine, relieve pain in most patients with trigeminal neuralgia by reducing the transmission of impulses at certain nerve terminals. Carbamazepine is taken with meals. Serum levels must be monitored to avoid toxicity in patients who require high doses to control the pain. Side effects include nausea, dizziness, drowsiness, and aplastic anemia (Hickey & Strayer, 2020). The patient is monitored for bone marrow depression during long-term therapy. Gabapentin and baclofen are also used

for pain control. If pain control is still not achieved, phenytoin may be used as adjunctive therapy.

## Surgical Management

If pharmacologic management fails to relieve pain, a number of surgical options are available. Although these procedures may relieve facial pain for a few years, recurrence is possible (Hickey & Strayer, 2020). The choice of procedure depends on the patient's preference and health status. The procedures are designed to either decompress the nerve and save nerve function, or to damage the nerve and destroy nerve function to keep it from malfunctioning (Hickey & Strayer, 2020).

### Microvascular Decompression of the Trigeminal Nerve

An intracranial approach is used to relieve the contact between the cerebral vessel and the trigeminal nerve root entry. With the aid of an operating microscope, the artery loop is lifted from the nerve to relieve the pressure, and a small prosthetic device is inserted to prevent recurrence of impingement on the nerve. The postoperative management is the same as for other intracranial surgeries (see [Chapter 61](#)).

### Radiofrequency Thermal Coagulation

Percutaneous radiofrequency produces a thermal lesion on the trigeminal nerve. Although immediate pain relief is experienced, dysesthesia of the face and loss of the corneal reflex may occur. MRI is used for identification of the trigeminal nerve followed by gamma knife radiosurgery. Gamma knife radiosurgery is a noninvasive method of delivering focused radiation to the trigeminal nerve (Obermann, 2019).

### Percutaneous Balloon Microcompression

Percutaneous balloon microcompression disrupts large myelinated fibers in all three branches of the trigeminal nerve. After its placement, the balloon is filled with a contrast material for fluoroscopic identification. The balloon compresses the nerve root for 1 minute and provides microvascular decompression (Hickey & Strayer, 2020).

## Nursing Management

### Preventing Pain

Preoperative management of a patient with trigeminal neuralgia occurs mostly on an outpatient basis and includes recognizing factors that may aggravate excruciating facial pain, such as food that is too hot or too cold or jarring of the patient's bed or chair. Even washing the face, combing the hair, or brushing

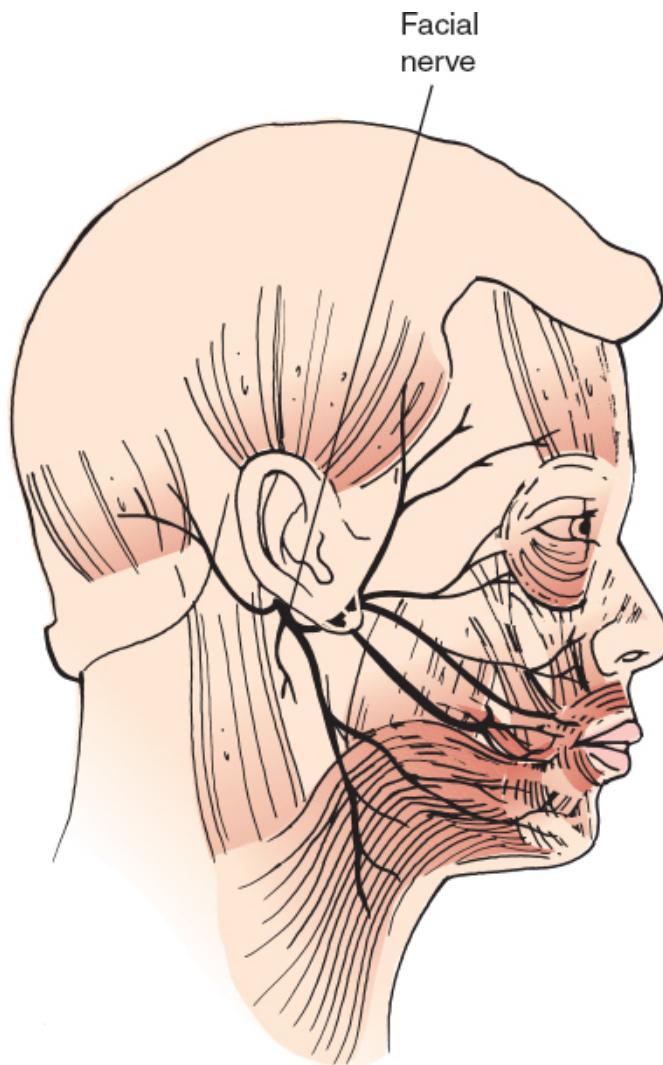
the teeth may produce acute pain. The nurse can assist the patient in preventing or reducing this pain by providing education about preventive strategies. Providing cotton pads and room temperature water for washing the face, instructing the patient to rinse with mouthwash after eating if toothbrushing causes pain, and performing personal hygiene during pain-free intervals are all effective strategies. The patient is instructed to take food and fluids at room temperature, to chew on the unaffected side, and to ingest soft foods. The nurse recognizes that anxiety, depression, and insomnia often accompany chronic painful conditions and uses appropriate interventions and referrals. See [Chapter 9](#) for management of patients with chronic pain.

### Providing Postoperative Care

Postoperative neurologic assessments are conducted to evaluate the patient for facial motor and sensory deficits in each of the three branches of the trigeminal nerve. If the surgery results in sensory deficits to the affected side of the face, the patient is instructed not to rub the eye because the pain of a resulting injury will not be detected. The eye is assessed for irritation or redness. Artificial tears may be prescribed to prevent dryness in the affected eye. The patient is cautioned not to chew on the affected side until numbness has diminished. The patient is observed carefully for any difficulty in eating or swallowing foods of different consistencies.

### Bell's Palsy

Bell's palsy (idiopathic facial paralysis) is caused by unilateral inflammation of the seventh cranial nerve, which results in weakness or paralysis of the facial muscles on the affected side (see [Fig. 64-6](#)). Although the cause is unknown, theories include reactivation of a dormant viral infection (herpes simplex, herpes zoster) or autoimmune syndromes (NINDS, 2018a). Those most commonly affected with Bell's palsy are between the ages of 15 and 45 years (Hickey & Strayer, 2020).



**Figure 64-6 •** Distribution of the facial nerve—the seventh cranial nerve.

Bell's palsy may be a type of pressure paralysis. The inflamed, edematous nerve becomes compressed to the point of damage, or its blood supply is occluded, producing ischemic necrosis of the nerve. The face is distorted from paralysis of the facial muscles which ranges in severity. Other symptoms may include drooping of the mouth, drooling, excessive lacrimation (tearing), and the patient may experience painful sensations in the face, behind the ear, and in the eye (NINDS, 2018a). The patient may also experience speech difficulties and may be unable to eat on the affected side because of weakness or paralysis of the facial muscles. The majority of patients recover completely and Bell's palsy rarely recurs (Somasundara, Sullivan, & Cheesbrough, 2017).

## Medical Management

The objectives of treatment are to maintain the muscle tone of the face and to prevent or minimize denervation. The patient should be reassured that no stroke has occurred and that spontaneous recovery occurs within 3 to 5 weeks in most patients.

Corticosteroid therapy (prednisone) may be prescribed to reduce inflammation and edema; this reduces vascular compression and permits restoration of blood circulation to the nerve. Early administration of corticosteroid therapy, started within 72 hours of symptom onset, is highly effective in diminishing the severity of the disease, relieving the pain, and preventing or minimizing denervation (NINDS, 2018a).

Facial pain is controlled with analgesic agents. Electrical stimulation may be applied to the face to prevent muscle atrophy. Although most patients recover with conservative treatment, surgical decompression of the facial nerve is controversial as there is minimal evidence that this is helpful (NINDS, 2018a; Somasundara et al., 2017).

## Nursing Management

While the paralysis is present, nursing care involves protection of the eye from injury. Frequently, the eyelid does not close completely and the blink reflex is diminished, so the eye is vulnerable to injury from dust and foreign particles. Corneal irritation and ulceration may occur. Distortion of the lower lid alters the proper drainage of tears. To prevent injury, the eye should be covered with a protective shield at night. The eye patch may abrade the cornea, however, because there is some difficulty in keeping the partially paralyzed eyelids closed. Moisturizing eye drops during the day and eye ointment at bedtime may help prevent injury (Somasundara et al., 2017). The patient can be educated to close the paralyzed eyelid manually before going to sleep. Wraparound sunglasses or goggles may be worn during the day to decrease evaporation from the eye.

After the sensitivity of the nerve to touch decreases and the patient can tolerate touching the face, the nurse can suggest massaging the face several times daily, using a gentle upward motion, to maintain muscle tone. Facial exercises, such as wrinkling the forehead, blowing out the cheeks, and whistling, may be performed with the aid of a mirror to prevent muscle atrophy. Exposure of the face to cold and drafts is avoided.

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## DISORDERS OF THE PERIPHERAL NERVOUS SYSTEM

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### Peripheral Neuropathies

A peripheral **neuropathy** (disorder of the nervous system) is a disorder affecting the peripheral motor and sensory nerves. Peripheral nerves connect the spinal cord and brain to all other organs. They transmit motor impulses from the brain and relay sensory impulses to the brain. Peripheral neuropathies are characterized by bilateral and symmetric disturbance of function, usually beginning in the feet and hands. The most common cause of peripheral neuropathy is diabetes with poor glycemic control (Hickey & Strayer, 2020). Many drugs, such as antineoplastic agents, also cause peripheral neuropathies (Hickey & Strayer, 2020). The major symptoms of peripheral nerve disorders are loss of sensation, muscle atrophy, weakness, diminished reflexes, pain, and paresthesia of the extremities.

Peripheral nerve disorders are diagnosed by history, physical examination, and electrodiagnostic studies such as EEG. The diagnosis of peripheral neuropathy in the older adult population is challenging because many symptoms, such as decreased reflexes, can be associated with the normal aging process (Eliopoulos, 2018).

No specific treatment exists for peripheral neuropathy. Elimination or control of the cause may slow progression. Patients with peripheral neuropathy are at risk for falls, thermal injuries, and skin breakdown. The plan of care includes inspection of the lower extremities for skin breakdown. Assistive devices such as a walker or cane may decrease the risk of falls. Bathwater temperature is checked to avoid thermal injury. Footwear should be accurately sized. Driving may be limited or eliminated, thereby disrupting the patient's sense of independence.

## Mononeuropathy

Mononeuropathy is limited to a single peripheral nerve and its branches. It arises when the trunk of the nerve is compressed or entrapped (as in carpal tunnel syndrome), traumatized (as when bruised by a blow), overstretched (as in joint dislocation), punctured by a needle used to inject a drug or damaged by the drugs thus injected, or inflamed because an adjacent infectious process extends to the nerve trunk. Mononeuropathy is frequently seen in patients with diabetes.

Pain is seldom a major symptom of mononeuropathy when the condition is due to trauma, but in patients with complicating inflammatory conditions such as arthritis, pain is prominent. Pain is increased with all body movements that tend to stretch, strain, or cause pressure on the injured nerve and sudden jarring of the body (e.g., from coughing or sneezing). The skin in the areas supplied by nerves that are injured or diseased may become reddened and glossy, the subcutaneous tissue may become edematous, and the nails and hair

in this area are altered. Chemical injuries to a nerve trunk, such as those caused by drugs injected into or near it, are often permanent.

The objective of treatment of mononeuropathy is to remove the cause, if possible (e.g., freeing the compressed nerve). Local corticosteroid injections may reduce inflammation and the pressure on the nerve. Aspirin or codeine may be used to relieve pain. Chronic pain can be treated with neuropathic pain medications such as gabapentin (Comerford & Durkin, 2020).

Nursing care involves protection of the affected limb or area from injury, as well as appropriate patient education about mononeuropathy and its treatment. The nurse assesses the impact of pain and weakness on the patient's quality of life and suggests interventions to cope with concerns the patient expresses (Girach, Julian, Varrassi, et al., 2019). Referrals to physical therapy for exercises to prevent muscle wasting and to occupational therapy to evaluate for splints that may help with appropriate positioning may be warranted.

### CRITICAL THINKING EXERCISES

**1 ipc** You are participating in morning rounds in the ICU where you work. The team is discussing a patient who has been assigned to you for the day, a 20-year-old woman admitted last night with meningitis. What members of the interprofessional care team are essential to include in the care of this patient? How will you, as this patient's nurse, facilitate an interprofessional discussion to help facilitate her recovery?

**2 pq** You are caring for a 60-year-old man with a recent diagnosis of CJD. His wife of 30 years visits every day. What are the priorities for care of this patient? What will be your priorities when educating this patient and his wife?

**3 ebp** In the outpatient clinic where you work, a woman with a new diagnosis of MS received treatment and is back to her baseline function. She tells you that she has been "cured." What evidence-based education would you provide the patient about potential triggers that may cause a return of symptoms?

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## Resources

Creutzfeldt–Jakob Disease Foundation, [www.cjdfoundation.org](http://www.cjdfoundation.org)  
Guillain–Barré Syndrome Foundation International, [www.gbs-cidp.org](http://www.gbs-cidp.org)  
Myasthenia Gravis Foundation of America (MGFA), [www.myasthenia.org](http://www.myasthenia.org)  
National Multiple Sclerosis Society, [www.nationalmssociety.org](http://www.nationalmssociety.org)  
The Foundation for Neuropathy, [www.foundationforpn.org](http://www.foundationforpn.org)

# 65 Management of Patients with Oncologic or Degenerative Neurologic Disorders

## LEARNING OUTCOMES

*On completion of this chapter, the learner will be able to:*

1. Describe brain and spinal cord tumors: their classification, pathophysiology, clinical manifestations, diagnosis, and medical and nursing management.
2. Use the nursing process as a framework for care of the patient with nervous system metastases or primary brain tumor.
3. Explain the pathophysiologic processes responsible for various neurodegenerative disorders.
4. Apply the nursing process as a framework for care of the patient with Parkinson's disease or the patient who has had a cervical discectomy.

## NURSING CONCEPTS

Family  
Fluids and Electrolytes  
Infection  
Intracranial Regulation  
Mobility  
Nutrition  
Patient Education  
Sensory Perception  
Stress and Coping

## GLOSSARY

**bradykinesia:** abnormally slow voluntary movements and speech

**chorea:** rapid, jerky, involuntary, purposeless movements of the extremities or facial muscles, including facial grimacing

**dementia:** broad term for a syndrome characterized by a general decline in higher brain functioning, such as reasoning, with a pattern of eventual decline in ability to perform even basic activities of daily living, such as toileting and eating

**dyskinesia:** impaired ability to execute voluntary movements

**dysphonia:** voice impairment or altered voice production

**neurodegenerative:** deterioration of cells or function of the nervous system

**papilledema:** edema of the optic nerve usually due to increased intracranial pressure (ICP)

**paresthesia:** numbness, tingling, or a “pins and needles” sensation

**sciatica:** pain and tenderness that radiates along the sciatic nerve that runs through the thigh and leg

**spondylosis:** degenerative changes occurring in a disc and adjacent vertebral bodies; can occur in the cervical or lumbar vertebrae

The occurrence of oncologic or degenerative disease processes in the neurologic system produces a unique set of nursing challenges. Thus, nurses who care for patients with these disorders must have a clear understanding of the pathophysiology, diagnostic testing, medical and nursing care, and rehabilitation processes. Nurses provide care for patients with oncologic or degenerative disease processes in many inpatient and outpatient settings. Oncologic disorders include brain and spinal cord tumors. Degenerative neurologic disorders include Parkinson’s disease (PD), Huntington disease,

amyotrophic lateral sclerosis (ALS), muscular dystrophies, and degenerative disc disease. Post-polio syndrome (PPS) is thought to be degenerative in nature and is included in this chapter.

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## ONCOLOGIC DISORDERS OF THE BRAIN AND SPINAL CORD

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There are many types of brain and spinal cord tumors, each with its own biology, prognosis, and treatment options. Because of the unique anatomy and physiology, tumors of the central nervous system (CNS) are challenging to diagnose and treat.

### Brain Tumors

A brain tumor occupies space within the skull, growing as a spherical mass or diffusely infiltrating tissue. The effects of brain tumors are caused by inflammation, compression, and infiltration of tissue. A variety of physiologic changes result, causing any or all of the following pathophysiologic events (Hickey & Strayer, 2020):

- Increased intracranial pressure (ICP) and cerebral edema
  - Focal neurologic signs such as headache
  - Seizure activity
  - Hydrocephalus
  - Altered pituitary function

Neoplastic lesions in the brain ultimately cause death by increasing ICP and impairing vital functions, such as respiration.

There are over 100 types of brain tumors with an estimated 78,000 new cases each year. These include 25,000 malignant and 53,000 nonmalignant brain tumors (American Association of Neuroscience Nurses [AANN], 2016). Brain tumors are classified as primary or secondary. Primary brain tumors originate from cells within the brain. In adults, most primary brain tumors originate from glial cells (cells that make up the structure and support system of the brain and spinal cord) and are supratentorial (located above the covering of the cerebellum). Primary tumors progress locally, rarely metastasize outside the CNS, and have a 5-year survival rate of 33.4% (Garcia, Slone, Dolecek, et al., 2019).

Developed countries have a higher incidence of primary brain tumors, with rates of 5.1 per 100,000 compared to 3.0 per 100,000 in less developed countries. This is most likely due to more frequent diagnosis with improved imaging modalities. Although many risk factors have been investigated,

exposure to ionizing radiation is the only known modifiable risk factor (AANN, 2016). Many genetic factors and genetic syndromes (such as neurofibromatosis) are associated with brain tumor risk in families (AANN, 2016).

Secondary, or metastatic, brain tumors develop from structures outside the brain and are twice as common as primary brain tumors (AANN, 2016). Metastatic lesions to the brain can occur from the lung, breast, lower gastrointestinal tract, pancreas, kidney, and skin (melanomas) neoplasms. Single or multiple metastases may occur, and brain metastases may be found at any time during the disease course, even at initial diagnosis of the primary disease. Patient survival rates from primary brain cancers are improving, however, the incidence of brain metastases is increasing (AANN, 2016).

The highest incidence of brain tumors in adults occurs in the fifth through seventh decades of life (Young, Chmura, Wainwright, et al., 2017). There is a slight male predominance in the incidence of malignant brain tumors.

## Types of Primary Brain Tumors

Brain tumors may be classified into several groups: those arising from the coverings of the brain (e.g., dural meningioma), those developing in or on the cranial nerves (e.g., acoustic neuroma), those originating within brain tissue (e.g., glioma), and metastatic lesions originating elsewhere in the body. Tumors of the pituitary and pineal glands and of cerebral blood vessels are also types of brain tumors. Relevant clinical considerations include the location and the histologic character of the tumor. About 70% of the time tumors are benign but even benign tumors, such as colloid cysts, can occur in vital areas and can grow large enough to have serious effects (Hickey & Strayer, 2020). See [Chart 65-1](#) for the classification of brain tumors.

### Chart 65-1

## Classification of Brain Tumors in Adults

### I. Intracerebral Tumors

- A. Gliomas—invade any portion of the brain; most common type of brain tumor
  - 1. Astrocytomas (grades I and II)
  - 2. Glioblastoma (astrocytoma grades III and IV)
  - 3. Oligodendrogloma (low and high grades)
  - 4. Ependymoma (grades I to IV)
  - 5. Medulloblastoma

### II. Tumors Arising From Supporting Structures

- A. Meningiomas
- B. Neuromas (acoustic neuroma, schwannoma)
- C. Pituitary adenomas

### III. Developmental Tumors

- A. Angiomas
- B. Dermoid, epidermoid, teratoma, craniopharyngioma

### IV. Metastatic Lesions

Adapted from Hickey, J. V., & Strayer, A. L. (2020). *The clinical practice of neurological and neurosurgical nursing* (8th ed.). Philadelphia, PA: Wolters Kluwer.

## Gliomas

In adults, gliomas (principally astrocytoma) account for approximately 25% of symptomatic primary brain tumors. Glial tumors, the most common type of intracerebral brain neoplasm, are divided into many categories (McFadie-Figueroa & Lee, 2018). Astrocytomas, arising from astrocytic cells, are the most common type of glioma and are graded from I to IV, indicating the degree of malignancy (McFadie-Figueroa & Lee, 2018). The grade is based on cellular density, cell mitosis, and degree of differentiation from the original cell type. Grades III and IV tumors are known as glioblastomas and have little resemblance to the original cell type. Astrocytomas infiltrate into the surrounding neural connective tissue and therefore cannot be totally removed without causing considerable damage to vital structures.

Oligodendroglial tumors, arising from oligodendroglial cells, represent about 1.4% of gliomas (Hickey & Strayer, 2020). Most oligodendroglomas occur in adults aged 50 to 60, are found in men more often than in women, and are categorized as low or high grade (anaplastic) (Young et al., 2017). The histologic distinction between astrocytomas and oligodendroglomas is difficult to make but is important, because oligodendroglomas are more sensitive than astrocytomas to chemotherapy. Tumors originating from

ependymal cells, another type of glial cell, are known as ependymomas and are more common in children than adults. Glial tumors may be treated with a combination of surgery, radiation therapy, and chemotherapy, depending on specific cell and patient characteristics as well as the location of the tumor (Young et al., 2017).

## Meningiomas

Meningiomas, which represent 37% of all primary brain tumors, are common benign encapsulated tumors of arachnoid cells on the meninges (McFaline-Figueroa & Lee, 2018). They are slow growing, occur most often in middle-aged adults, and are more common in women. Meningiomas often occur in areas proximal to the venous sinuses. Manifestations depend on the area involved and are often the result of compression rather than invasion of brain tissue. Preferred treatment for symptomatic lesions is surgery with complete removal or partial dissection, although radiation therapy may be useful for some patients. Metastasis is rare with meningiomas but benign meningiomas may be challenging to remove surgically without causing neurologic deficits if the tumor is located at the base of the skull or surrounds the optic nerve, or in the rare case if the tumor is invasive. Multiple meningiomas may occur with neurofibromatosis type 2 (Euskirchen & Peyre, 2018).

## Acoustic Neuromas

Acoustic neuromas account for 16% of brain tumors, with men and women equally affected, and occur most commonly in the fifth decade of life (Hong & Moliterno, 2019).

An acoustic neuroma is a tumor of the eighth cranial nerve—the cranial nerve most responsible for hearing and balance. It usually arises just within the internal auditory meatus, where it frequently expands before filling the cerebellopontine recess. An acoustic neuroma may grow slowly and attain considerable size before it is diagnosed. The patient usually experiences loss of hearing, tinnitus, and episodes of vertigo and staggering gait. As the tumor becomes larger, painful sensations of the face may occur on the same side as a result of the tumor's compression of the fifth cranial nerve (Hong & Moliterno, 2019). Many acoustic neuromas are benign and can be managed conservatively. Many that continue to grow can be surgically removed and have a good prognosis (see [Chapter 59](#)). Some acoustic neuromas may be suitable for stereotactic radiotherapy rather than open craniotomy. Stereotactic radiotherapy is discussed later in this chapter.

## Pituitary Adenomas

Pituitary tumors account for about 16% of all primary brain tumors (AANN, 2016). They can occur at any age but are more common in older adults.

Women are affected more often than men, particularly during the childbearing years. Pituitary tumors are rarely malignant but cause symptoms as a result of pressure on adjacent structures or hormonal changes (Jang, Oh, Lee, et al., 2020).

### Pressure Effects of Pituitary Adenomas

Pressure from a pituitary adenoma may be exerted on the optic nerves, optic chiasm, or optic tracts or on the hypothalamus or the third ventricle if the tumor invades the cavernous sinuses or expands into the sphenoid bone. These pressure effects produce headache, visual dysfunction, hypothalamic disorders (disorders of sleep, appetite, temperature, and emotions), increased ICP, and enlargement and erosion of the sella turcica (Donovan & Welch, 2018; Jang et al., 2020; Molitch, 2017).

### Hormonal Effects of Pituitary Adenomas

Functioning pituitary tumors can produce one or more hormones normally produced by the anterior pituitary. Hormonal hypersecretion is caused only by pituitary adenomas (Molitch, 2017). Many adenomas (50%) secrete an excess amount of hormone including prolactin (prolactinomas), growth hormone (GH) producing acromegaly in adults, adrenocorticotrophic hormone (ACTH) resulting in Cushing's disease, or thyroid-stimulating hormone (TSH) (Molitch, 2017). Adenomas that secrete TSH or follicle-stimulating hormone and luteinizing hormone occur infrequently, whereas adenomas that produce both GH and prolactin are relatively common.

The female patient whose pituitary gland is secreting excessive quantities of prolactin presents with amenorrhea or galactorrhea (excessive or spontaneous flow of milk). Male patients with prolactinomas may present with impotence and hypogonadism. Acromegaly, caused by excess GH, produces enlargement of the hands and feet, distortion of the facial features, and pressure on peripheral nerves (entrapment syndromes). The clinical features of Cushing's disease, a condition associated with prolonged overproduction of cortisol, occur with excessive production of ACTH. Manifestations include a form of obesity with redistribution of fat to the facial, supraclavicular, and abdominal areas; hypertension; purple striae and ecchymoses; osteoporosis; elevated blood glucose levels; and emotional disorders. See [Chapter 45](#) for a discussion of endocrine disorders resulting from these tumors.



## Gerontologic Considerations

The incidence of all brain tumors increases with age (Young et al., 2017). Intracranial tumors can produce personality changes, confusion, speech dysfunction, or disturbances of gait. In older adult patients, early signs and symptoms of intracranial tumors can be easily overlooked or incorrectly

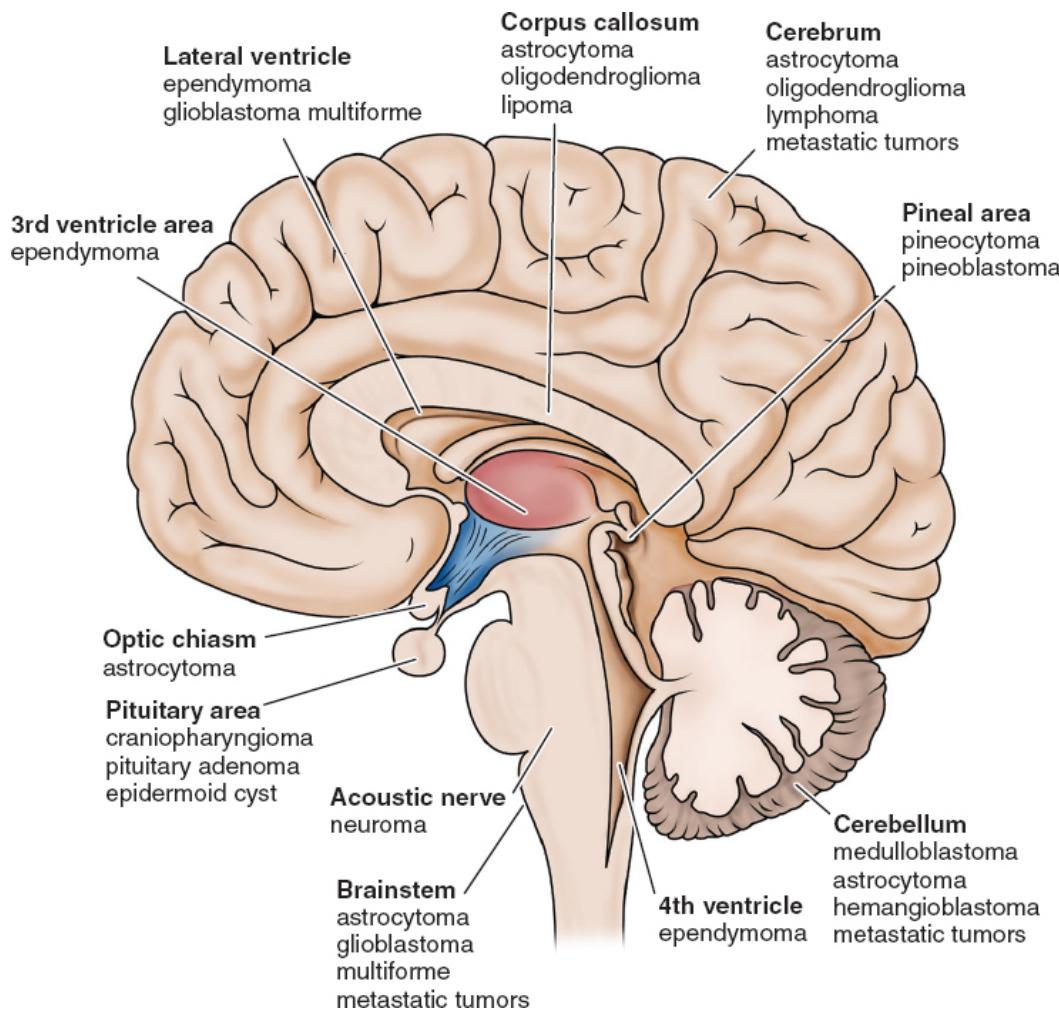
attributed to cognitive and neurologic changes associated with normal aging (Eliopoulos, 2018). Neurologic signs and symptoms in the older adult must be carefully evaluated, because brain metastases occur in patients with a history of prior cancer. Regardless of the age of the patient or the decision to proceed or not with treatment, the nurse provides supportive care. Researchers have reported that the degree of frailty influences clinical outcomes of older adult patients undergoing surgery for brain tumor resection (Harland, Wang, Gunaydin, et al., 2020). Patients identified as moderately frail or frail are at increased risk for a longer length of hospital stay and tend to require discharge to a long-term care facility rather than home (Harland et al., 2020).

## Clinical Manifestations

Brain tumors can produce both focal or generalized neurologic signs and symptoms. Generalized symptoms reflect increased ICP, and the most common focal or specific signs and symptoms result from tumors that interfere with functions in specific brain regions. [Figure 65-1](#) indicates common brain tumor sites.

### Increased Intracranial Pressure

As discussed in [Chapter 61](#), the skull is a rigid compartment containing essential noncompressible contents: brain matter, intravascular blood, and cerebrospinal fluid (CSF). The Monro-Kellie hypothesis or doctrine explains the dynamic equilibrium of the cranial contents. According to this hypothesis, if any one of these skull components increases in volume, ICP increases unless one of the other components decreases in volume. Consequently, any change in volume occupied by the brain (as occurs with disorders such as brain tumor or cerebral edema) produces signs and symptoms of increased ICP (Witherspoon & Ashby, 2017).



**Figure 65-1 • Common brain tumor sites.**

The enlarging tumor and its associating edema disrupts the equilibrium between the brain, blood, and CSF. As the tumor grows, compensatory adjustments may occur through compression of intracranial veins, reduction of CSF volume (by increased absorption or decreased production), a modest decrease in cerebral blood flow, or reduction of intra- and extracellular brain tissue mass. When these compensatory mechanisms fail, the patient develops signs and symptoms of increased ICP, most often including headache, nausea with or without vomiting, and **papilledema** (swelling of the optic nerve) (Hickey & Strayer, 2020). Personality changes and a variety of focal deficits, including motor, sensory, and cranial nerve dysfunction, are common.

## Headache

One third of patients with brain tumors report headache as an early symptom (Hickey & Strayer, 2020). Headache is most commonly reported in the early morning and is made worse by coughing, straining, or sudden movement

(Norris, 2019). It is thought to be caused by the tumor invading, compressing, or distorting the pain-sensitive structures or by edema that accompanies the tumor. The headaches may be generalized or localized to the site of the tumor. As the edema increases, headache is generally bifrontal or bioccipital regardless of the tumor location (Hickey & Strayer, 2020).

### Vomiting

Vomiting, seldom related to food intake, is usually the result of irritation of the vagal centers in the medulla (Hickey & Strayer, 2020). Forceful vomiting is described as projectile vomiting. Headache may be relieved by vomiting.

### Visual Disturbances

The tumor itself or the surrounding edema can compress the third cranial nerve, causing optic disc swelling or papilledema. This limits the visual acuity along the visual pathway, mildly or profoundly, as diplopia (double vision), hemianopsia (visual field deficits), or varying levels of blindness (Jang et al., 2020).

### Seizures

Seizures are common in patients with brain tumors either initially or throughout their disease process (McFadie-Figueroa & Lee, 2018). Seizures may be focal or generalized. Tumors of the frontal, parietal, and temporal lobes carry the greatest risk of seizures; seizures are unusual with brainstem or cerebellar tumors. See [Chapter 61](#) for discussion about seizures and related management.

If not the initial presentation, seizures can be a result of metabolic factors (electrolyte imbalances, liver failure or kidney disease, radiation or chemotherapy side effects), structural causes (parenchymal metastases, leptomeningeal disease, dural metastases) or a new hemorrhage, thrombosis, or development of meningitis (Hickey & Strayer, 2020). If the patient presents with a seizure, then anticonvulsant drugs are prescribed. Medications with the best evidence for controlling seizure activity include levetiracetam, carbamazepine, phenytoin, and zonisamide (Comerford & Durkin, 2020).

### Localized Symptoms

When specific regions of the brain are affected, local signs and symptoms occur, such as sensory or motor abnormalities, visual alterations, alterations in cognition, or language disturbances (e.g., aphasia). Identifying the signs and symptoms is important, because it can help identify tumor location. Some tumors are not easily localized because they lie in so-called silent areas of the brain (i.e., areas in which functions are not well defined). Many tumors can be

localized by correlating the signs and symptoms to specific areas in the brain, as follows (Hickey & Strayer, 2020):

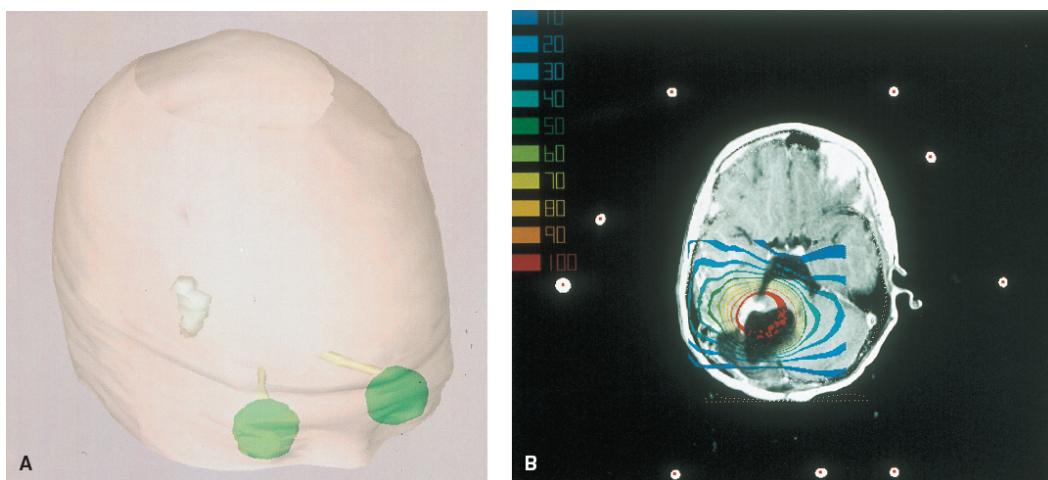
- A tumor in the motor cortex of the frontal lobe produces hemiparesis and partial seizures on the opposite side of the body or generalized seizures. A frontal lobe tumor may also produce changes in emotional state and behavior, as well as an apathetic mental attitude. The patient often becomes impulsive, inappropriate in speech, gestures, and behavior.
- A parietal lobe tumor may cause decreased sensation on the opposite side of the body or generalized seizures.
- A temporal lobe tumor may cause seizures as well as psychological disorders.
- An occipital lobe tumor produces visual manifestations: contralateral homonymous hemianopsia (visual loss in half of the visual field on the opposite side of the tumor) and visual hallucinations.
- A cerebellar tumor causes dizziness; an ataxic or staggering gait with a tendency to fall toward the side of the lesion; marked muscle incoordination; and nystagmus (involuntary rhythmic eye movements), usually in the horizontal direction.
- A cerebellopontine angle tumor usually originates in the sheath of the acoustic nerve and gives rise to a characteristic sequence of symptoms. Tinnitus and vertigo appear first, soon followed by progressive nerve deafness (eighth cranial nerve dysfunction). Numbness and tingling of the face and tongue occur (due to involvement of the fifth cranial nerve). Later, weakness or paralysis of the face develops (seventh cranial nerve involvement). Finally, because the enlarging tumor presses on the cerebellum, abnormalities in motor function may be present.
- Brainstem tumors may be associated with cranial nerve deficits along with complex motor and sensory function impairments (see [Chapter 64, Table 64-3](#)).

## Assessment and Diagnostic Findings

The history of the illness, the manner, and the time frame in which the symptoms evolved are key components in the diagnosis of brain tumors. A neurologic examination indicates the involved areas of the CNS. To assist in the precise localization of the lesion, a battery of tests is performed. Computed tomography (CT) scans, enhanced by a contrast agent, can give specific information concerning the number, size, and density of the lesions, and the extent of secondary cerebral edema. CT can provide information about the ventricular system. A magnetic resonance imaging (MRI) scan is the most

helpful diagnostic tool for detecting brain tumors, particularly smaller lesions, and tumors in the brainstem and pituitary regions, where bone is thick. MRI is also useful in monitoring response to treatment.

Computer-assisted stereotactic (three-dimensional) biopsy is used to diagnose deep-seated brain tumors and to provide a basis for treatment and prognosis. Stereotactic approaches involve the use of a three-dimensional frame that allows very precise localization of the tumor; a stereotactic frame and multiple imaging studies (x-rays, CT scans, or MRIs) are used to localize the tumor and verify its position (see Fig. 65-2). Brain-mapping technology helps determine the proximity of diseased areas of the brain to structures essential for normal brain function.



**Figure 65-2 •** **A.** Using stereotactic or “brain-mapping” guided approach, a three-dimensional computer image fuses the computed tomography image and magnetic resonance image to pinpoint the exact location of the brain tumor. This low-grade astrocytoma is localized adjacent to the brainstem, is inoperable, and is treated with radiation. Note the optic chasm and optic nerves. **B.** Computerized image of the prescribed radiation dose.

Positron emission tomography (PET) is used to supplement MRI scanning in many centers. On PET scans, low-grade tumors are associated with hypometabolism, and high-grade tumors show hypermetabolism. This information can be useful in making treatment decisions (Achrol, Rennert, Anders, et al., 2019). An electroencephalogram can detect abnormal brain waves in regions occupied by or adjacent to tumor; it is used to evaluate temporal lobe seizures and to assist in ruling out other disorders. Cytologic studies of the CSF may be performed to detect malignant cells as CNS tumors can shed cells into the CSF resulting in metastasis.

## Medical Management

A variety of medical management approaches, including surgery, chemotherapy, and external-beam radiation therapy, are used alone or in combination (AANN, 2016; Hickey & Strayer, 2020). A relatively new treatment option for glioblastomas is tumor-treating fields. This device provides alternating electric field therapy that disrupts the mitotic process and is worn on the head (McFaline-Figueroa & Lee, 2018). The main side effect is skin irritation.

Secretory tumors may be treated with medications that suppress hormones. Nonfunctioning tumors may have no effect on pituitary function or may suppress hormone production and release. Hormone replacement may be necessary for these patients to restore normal endocrine function. The management of brain tumors is complex and requires an interdisciplinary approach to care to optimize patient outcomes (Achrol et al., 2019).

## Surgical Management

The objective of surgical management is to remove as much tumor as possible without increasing the neurologic deficit (paralysis, blindness), or to relieve symptoms by partial removal (decompression). Surgery also provides the opportunity to biopsy tissue to establish a definitive diagnosis. A variety of surgical approaches may be used; the specific approach depends on the type of tumor, its location, and its accessibility. Conventional surgical approaches require a craniotomy (incision into the skull). See [Chapter 61](#) for a discussion of care of the patient who has undergone a craniotomy. This approach is used in patients with meningiomas, acoustic neuromas, cystic astrocytomas of the cerebellum, colloid cysts of the third ventricle, congenital tumors such as dermoid cyst, and some of the granulomas. With improved imaging techniques and the availability of the operating microscope and microsurgical instrumentation, even large tumors can be removed through a relatively small craniotomy. For patients with malignant glioma, complete removal of the tumor and cure are not possible, but the rationale for resection includes relief of ICP, removal of any necrotic tissue, and reduction in the bulk of the tumor, which theoretically leaves behind fewer cells to become resistant to radiation or chemotherapy. Most pituitary adenomas are treated by transsphenoidal microsurgical removal (see [Chapter 61](#)), and the remainder of tumors that cannot be removed completely are treated by radiation (Hickey & Strayer, 2020).

## Radiation Therapy

Radiation therapy—the cornerstone of treatment for many brain tumors—decreases the incidence of recurrence of incompletely resected tumors (AANN, 2016). Gamma radiation is delivered via an external beam to the

tumor in multiple fractions. Brachytherapy (the surgical implantation of radiation sources to deliver high doses at a short distance) is an option for some types of tumors depending on their location. It is usually used as an adjunct to conventional radiation therapy or as a rescue measure for recurrent disease. Radioisotopes such as iodine 131 ( $^{131}\text{I}$ ) are used to minimize effects on surrounding brain tissue.

Stereotactic procedures may be performed using a linear accelerator or gamma knife to perform radiosurgery (Hickey & Strayer, 2020). These procedures allow treatment of deep, inaccessible tumors, often in a single session. Precise localization of the tumor is accomplished by the stereotactic approach and by minute measurements and precise positioning of the patient. Multiple narrow beams then deliver a very high dose of radiation. An advantage of this method is that no surgical incision is needed. Disadvantages include the lag time between treatment and the desired result as well as the potential for developing radiation necrosis (AANN, 2016).

## Chemotherapy

Chemotherapy may be used in conjunction with radiation therapy, or as the sole therapy, with the goal of increasing survival time. The greatest challenge in chemotherapy of brain tumors is that the blood–brain barrier prevents drugs from getting to the tumor in effective doses without causing systemic toxicity (AANN, 2016).

Malignant glioma is usually treated with 6 weeks of oral temozolomide during radiation therapy, followed by 6 to 12 months of oral temozolomide. Low-grade gliomas may be treated with 6 months of oral temozolomide alone. Temozolomide is an oral chemotherapy that crosses the blood–brain barrier (McFaline-Figueroa & Lee, 2018). Several other chemotherapy agents are used alone or in combination depending on the type of tumor.

Autologous bone marrow transplantation is used in some patients who will receive chemotherapy or radiation therapy, because it can “rescue” the patient from the bone marrow toxicity associated with high doses of chemotherapy and radiation. A fraction of the patient’s bone marrow is aspirated, usually from the iliac crest, and stored. The patient receives large doses of chemotherapy or radiation therapy to destroy large numbers of malignant cells. The marrow is then reinfused intravenously after treatment is completed. See [Chapter 12](#) for discussion of bone marrow transplant.

## Pharmacologic Therapy

Corticosteroids are useful in relieving headache and alterations in level of consciousness. Corticosteroids such as dexamethasone are thought to reduce inflammation and edema around tumors (AANN, 2016). Other medications used include osmotic diuretics (e.g., mannitol and hypertonic saline) to

decrease the fluid content of the brain, which leads to a decrease in ICP. Anticonvulsant medications are used to treat and control seizures (Comerford & Durkin, 2020).

## Nursing Management

The characteristics of headache, when present, should be assessed. Upright positioning and pain medications may be useful in managing pain; nurses should evaluate the effectiveness of pain management interventions (Ijzerman-Korevaar, Snijers, Saskia, et al., 2018). Even if seizure history is absent, the patient and family should be educated about the possibility of seizure and the need to adhere to prophylactic anticonvulsant medications, if prescribed. The patient with a brain tumor may be at increased risk for aspiration as a result of cranial nerve dysfunction. Medications to alleviate nausea and to prevent vomiting should be considered (Ijzerman-Korevaar et al., 2018). Preoperatively, the gag reflex and ability to swallow are evaluated. In patients with diminished gag response, care includes educating the patient to direct food and fluids toward the unaffected side, having the patient sit upright to eat, offering a semisoft diet, and having suction readily available. The effects of increased ICP caused by the tumor mass are reviewed in [Chapter 61](#). The nurse performs neurologic checks; monitors vital signs; maintains a neurologic observation record (see [Chapter 61, Fig. 61-6](#)); spaces nursing interventions to prevent rapid increase in ICP; and reorients the patient when necessary to person, time, and place. The use of corticosteroids to control headache and neurologic symptoms requires astute nursing assessment and intervention because many adverse effects can occur, including hyperglycemia, electrolyte abnormalities, and muscle weakness (see [Chapter 45, Table 45-3](#)). Patients with changes in cognition caused by their lesion require frequent reorientation and the use of orienting devices (e.g., personal possessions, photographs, lists, a clock), supervision of and assistance with self-care, and ongoing monitoring and intervention for prevention of injury. Patients with seizures are carefully monitored and protected from injury. Motor function is checked at intervals because specific motor deficits may occur, depending on the tumor's location. When muscle weakness is present, an interprofessional approach, including the nurse and physical and occupational therapists, can be used to preserve muscle strength, promote range of motion, and facilitate independence in self-care. Sensory disturbances are assessed and any area of numbness should be protected from injury. Speech is evaluated, and patients with speech deficits can be educated to use alternative forms of communication. Eye movement and pupillary size and reaction may be affected by cranial nerve involvement. Fatigue is common during therapy; efforts should be made to conserve energy and promote rest.

The psychosocial effects on family caregivers of a family member who has brain metastases may be significant (Ketcher, Otto, & Reblin, 2020). Caregivers should be included in the plan of care.

The nursing process for patients undergoing neurosurgery is discussed in [Chapter 61](#). The patient's functional abilities should be reassessed postoperatively, because changes can occur.

## Cerebral Metastases

A significant number of patients with cancer experience neurologic deficits caused by metastasis to the nervous system, which can include the brain, CSF, and meninges. Metastatic lesions to the brain are more common than primary brain tumors and have an associated 2-year survival rate of less than 10% (Achrol et al., 2019). The high number of metastatic brain tumors is clinically important, as more patients with all forms of cancer live longer because of improved therapies. Neurologic signs and symptoms include headache, gait disturbances, visual impairment, personality changes, altered mentation (memory loss and confusion), focal weakness, paralysis, aphasia, and seizures (McFaline-Figueroa & Lee, 2018). These signs and symptoms can be devastating to both patient and family. Metastases to the CSF and meninges, known as leptomeningeal metastases, can produce symptoms of headache and isolated cranial nerve deficits.

## Medical Management

The treatment of metastatic nervous system cancer is palliative and involves eliminating or reducing serious symptoms. Even when palliation is the goal, distressing signs and symptoms can be relieved, thereby improving the quality of life for both patient and family (McFaline-Figueroa & Lee, 2018). Patients with intracerebral metastases who are not treated have a poor prognosis with a limited survival time. The therapeutic options include whole brain radiation therapy (the foundation of treatment) for multiple metastases or stereotactic radiosurgery for up to three sites of metastases. Surgery may be considered for a single symptomatic metastasis. Systemic chemotherapy directed at the primary cancer may be ineffective in crossing the blood-brain barrier, but chemotherapy that crosses this barrier may be added. Intrathecal chemotherapy, with direct injection of chemotherapy agents into the CSF of the brain or spinal canal, may be useful in persons with metastases (Song, Li, Yin, et al., 2018). Some combination of these treatments is usually the optimal method.

Pain can be a significant problem and is managed by means of a stepped progression in the doses and type of analgesic agents needed for relief. If the

patient has severe pain, morphine can be infused into the epidural or subarachnoid space through a spinal needle and a catheter placed as near as possible to the spinal segment where the pain is projected. Small doses of morphine are given at prescribed intervals (see [Chapter 9](#)).

## NURSING PROCESS

### The Patient with Nervous System Metastases or Primary Brain Tumor

#### Assessment

The nursing assessment includes a baseline neurologic examination and focuses on how the patient is functioning, moving, and walking; adapting to weakness or paralysis and to loss of vision and speech; and dealing with seizures. Assessment addresses symptoms that cause distress to the patient and affect the quality of life, including pain, respiratory problems, bowel and bladder disorders, and sleep disturbances, as well as impairment of skin integrity, fluid balance, and temperature regulation (Hickey & Strayer, 2020). Nutritional status is assessed, because cachexia and cancer-related anorexia-cachexia syndrome are common (see [Chapter 12](#)).

The nurse takes a dietary history to assess food intake, intolerance, and preferences. Calculation of body mass index can confirm the loss of subcutaneous fat and lean body mass (see [Chapter 4](#)). Biochemical measurements are reviewed to assess the degree of malnutrition, impaired cellular immunity, and electrolyte balance (normal laboratory values are found in Appendix A on [thePoint](#)). A dietitian assists in determining the caloric needs of the patient.

The nurse works with other members of the health care team to assess the impact of the illness on the family in terms of home care, altered relationships, financial problems, time pressures, and family problems. This information is important in helping family members cope with the diagnosis and the changes associated with it.

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired self-feeding, impaired ability to perform hygiene, impaired ability to dress, and impaired self-toileting associated with loss or impairment of motor and sensory functions and decreased cognitive abilities
- Impaired nutritional status associated with cachexia due to treatment and tumor effects, decreased nutritional intake, and malabsorption
- Impaired nutritional status associated with increased nutritional intake and impaired metabolism
- Anxiety associated with uncertainty, change in appearance, or altered lifestyle

- Interrupted family process associated with situational crisis imposed by the care of the person with a terminal illness

#### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Seizures (see [Chapter 61](#))
- Headaches (see [Chapter 61](#))

#### **Planning and Goals**

The goals for the patient include compensating for self-care deficits, improving nutrition, reducing anxiety, enhancing family coping skills, and absence of complications.

#### **Nursing Interventions**

##### **COMPENSATING FOR SELF-CARE DEFICITS**

The patient may have difficulty participating in goal setting depending on the tumor location and if cognitive function is affected. The nurse encourages the family to assist the patient to be as independent as possible for as long as possible (Hickey & Strayer, 2020). Increasing assistance with self-care activities is to be expected. Because the patient with nervous system metastasis and the family live with uncertainty, they are encouraged to plan for each day and to make the most of each day. The tasks and challenges are to assist the patient to find useful coping mechanisms, adaptations, and compensations for solving problems that arise. Use of an interprofessional health care team is helpful. An individualized exercise program helps maintain strength, endurance, and range of motion. Eventually, referral for home or hospice care may be necessary (see [Chapter 13](#)).

##### **IMPROVING NUTRITION**

Patients with nausea, vomiting, diarrhea, breathlessness, and pain are rarely interested in eating. These symptoms are managed or controlled through assessment, planning, and care. The nurse educates the family about how to position the patient for comfort and safety during meals. A dietitian may help with alternative food choices that are easily tolerated. Meals are planned for times when the patient is rested and in less distress from pain or the effects of treatment.

The patient needs to be clean, comfortable, and free of pain for meals in an environment that is as attractive as possible. Oral hygiene before meals helps to improve appetite. Offensive sights, sounds, and odors are eliminated. Creative strategies may be required to make food more palatable, provide enough fluids, and increase opportunities for socialization during meals. The family may be asked to keep a log of daily weights and to record the quantity of food eaten to determine the daily calorie count. Dietary supplements, if acceptable to the patient, can be

provided to meet increased caloric needs. If the patient is not interested in most usual foods, those foods preferred by the patient should be offered. When the patient shows marked deterioration as a result of tumor growth and effects, some other form of nutritional support (e.g., tube feeding, parenteral nutrition) may be indicated if consistent with the patient's end-of-life preferences (AANN, 2016) (see [Chapter 39](#) for discussion of tube feeding and [Chapter 41](#) for discussion of parenteral nutrition). Nursing interventions include assessing the patency of the central and intravenous lines or feeding tube, monitoring the insertion site for infection, checking the infusion rate, monitoring intake and output, and changing the intravenous tubing and dressing. Family members are instructed in these techniques if they will be providing care at home. Parenteral nutrition can be provided at home if indicated. The patient's quality of life may guide the selection, initiation, maintenance, and discontinuation of nutritional support. The nurse and family should not place too much emphasis on eating or on discussions about food, because the patient may not desire aggressive nutritional intervention. The subsequent course of action must be congruent with the wishes and choices of the patient and family.

Increased appetite may occur in patients receiving steroids and may result in weight gain as well as hyperglycemia. Patients and family members should be instructed to monitor weight and blood glucose (if appropriate), as well as maintain a healthy diet with caloric intake appropriate to patient needs.

#### **RELIEVING ANXIETY**

Patients may be restless, with changing moods that may include intense depression, euphoria, paranoia, and severe anxiety. The patient's response to a life-threatening illness reflects their pattern of reaction to other crisis situations. Serious illness imposes additional strains that often bring other unresolved problems to light. The patient's own coping strategies can help deal with anxious and depressed feelings. Health care providers need to be sensitive to the patient and caregivers' concerns and fears (Ketcher et al., 2020).

Patients need the opportunity to exercise some control over their situation. A sense of mastery can be gained as they learn to understand the disease and its treatment and how to deal with their feelings. Researchers have reported that resilience positively influences problem-based coping strategies (Liang, Liu, Lu, et al., 2020). The presence of family, friends, a spiritual advisor, and health care professionals may be supportive. Brain tumor support services may provide a feeling of meaning and strength (see the Resources section).

Spending time with patients allows them time to talk and to communicate their fears and concerns. Open communication and acknowledgment of fears are often therapeutic. Touch is also a form of

communication. These patients need reassurance that continuing care will be provided and that they will not be abandoned. If a patient's emotional reactions are very intense or prolonged, additional help from a spiritual advisor, social worker, or mental health professional may be indicated.

#### **ENHANCING FAMILY PROCESSES**

The family needs to be reassured that their loved one is receiving optimal care and that attention will be paid to the patient's changing symptoms and concerns. If the patient can no longer carry out self-care, the family and additional support systems (social worker, home health aide, home health nurse, hospice nurse) may be needed. Assessment and education of family caregivers about neurologic and cognitive symptoms is necessary, acute, and challenging (Boele, Terhorst, Prince, et al., 2019). Investigating resources that are available may decrease the burden on the caregivers. These efforts improve the psychosocial well-being of the patient and family caregivers (Boele et al., 2019; Ketcher et al., 2020).

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



**Educating Patients About Self-Care.** The patient and family often have major responsibility for care at home. Caregivers may struggle with a new "normal" while their support systems fluctuate as their relationship with the patient they are caring for is strengthened, maintained, or strained (Ketcher et al., 2020). In addition to the psychological aspect, education includes pain management strategies, prevention of complications related to treatment strategies, and methods to ensure adequate fluid and food intake (see [Chart 65-2](#)). Educational needs of the patient and family regarding care priorities are likely to change as the disease progresses; the nurse should assess the changing needs of the patient and the family and inform them about resources and services early and frequently to assist them (see the Resources section).

**Continuing and Transitional Care.** Home health services are valuable resources that should be made available to the patient and the family early in the course of an illness. Anticipating needs before they occur can assist in smooth initiation of services. Home health focuses on the areas of symptom and pain control, assistance in self-care, control of treatment complications, and administration of specific forms of treatment (e.g., parenteral nutrition). The nurse assesses pain management, respiratory status, complications of the disorder and its treatment, and the patient's cognitive and emotional status. In addition, the nurse assesses the family's ability to perform necessary care and notifies the primary provider about changing needs or complications, if indicated.

Steps to initiate hospice care, including discussion of hospice care as an option, should not be postponed until death is imminent for the patient with a metastasis or high-grade tumor. Exploration of hospice care as an option

should be initiated at a time when hospice services can provide support and care to the patient and family consistent with their end-of-life decisions and can assist in allowing death with dignity. See [Chapter 13](#) for in-depth discussion of end-of-life care.

### Evaluation

Expected patient outcomes may include:

1. Engages in self-care activities to extent possible
  - a. Uses assistive devices or accepts assistance as needed
  - b. Schedules periodic rest periods to permit maximal participation in self-care
2. Maintains as optimal a nutritional status as possible
  - a. Eats and accepts food within limits of condition and preferences

**Chart 65-2**



### HOME CARE CHECKLIST

## The Patient with Nervous System Metastases or Primary Brain Tumor

**At the completion of education, the patient and/or caregiver will be able to:**

- State effects of the tumor according to its type and location in the brain or spinal cord.
- State the impact of the tumor and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
  - Describe goals and side effects of treatment and suggested management approaches.
- State the name, dose, side effects, frequency, and schedule for all medications.
  - When indicated, use nonpharmacologic pain management techniques in addition to prescribed pharmacologic methods.
- State how to contact all members of the treatment team (e.g., health care providers, home care professionals, and durable medical equipment and supply vendor).
- State changes in lifestyle (e.g., nutritional needs, ADL assistance) necessary to maintain health.
- Identify coping strategies, such as:
  - Taking control, setting daily goals, and staying positive
  - Participating in rehabilitation to improve self-care
  - Engaging in relaxation techniques
  - Utilizing family support
  - Contacting support services (e.g., American Brain Tumor Association)
  - Participating in faith community/religious practices
- Identify community resources, including palliative care, home health service, or hospice, as appropriate.
- List complications of medications/therapeutic regimen necessitating a call to the nurse or primary provider.
- List complications of medications/therapeutic regimen necessitating a visit to the emergency department.
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.

### Resources

See [Chapter 12, Chart 12-10: Home Care Checklist: The Patient Receiving Care for an Oncologic Disorder](#).

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

- b. Accepts alternative methods of providing nutrition if indicated
3. Reports being less anxious
  - a. Is less restless and is sleeping better
  - b. Verbalizes concerns and fears
  - c. Participates in activities of personal importance
4. Family members seek help as needed
  - a. Demonstrate ability to bathe, feed, and care for the patient and participate in pain management and prevention of complications
  - b. Express feelings and concerns to appropriate health professionals
  - c. Discuss and seek hospice care as needed
5. Understands ways to avoid complications and is free of complications
  - a. Explains reasons for measures to prevent complications
  - b. Seizures and headaches are controlled to the extent possible

## Spinal Cord Tumors

Tumors within the spinal canal are classified according to their anatomic relation to the spinal cord (Hickey & Strayer, 2020). They include intramedullary lesions (within the spinal cord), extramedullary-intradural lesions (within or under the spinal dura), and extramedullary-extradural lesions (outside the dural membrane). Primary tumors are usually intramedullary, consisting of astrocytoma or ependymoma; meningiomas can occur as extramedullary-intradural lesions (Epstein, 2018). Secondary tumors are far more common and are usually extramedullary-extradural lesions. Tumors that occur within the spinal canal or exert pressure on it cause symptoms ranging from localized or shooting pains, weakness, and loss of reflexes below the tumor level to progressive loss of motor function and paralysis. Usually, sharp pain occurs in the area innervated by the spinal roots that arise from the cord in the region of the tumor. In addition, increasing sensory deficits develop below the level of the lesion. Loss of bowel and bladder function is common.

## Assessment and Diagnostic Findings

Neurologic examination and diagnostic studies are used to make the diagnosis. Neurologic examination focuses on assessing pain and identifying loss of reflexes, sensation, or motor function. Helpful diagnostic studies include CT scans, MRI scans, and biopsy. The MRI scan is the most commonly used and the most sensitive diagnostic tool (Hickey & Strayer, 2020) and it is

particularly helpful in detecting epidural spinal cord compression and metastases (Kaplow & Iyere, 2016).

## Medical Management

Treatment of specific intraspinal tumors depends on the type and location of the tumor, the presenting symptoms, and the patient's physical status. Surgical intervention is the primary treatment for most spinal cord tumors followed by chemotherapy and radiation therapy.

Extramedullary-extradural spinal cord compression occurs in 5% to 7% of patients who die of cancer and is considered a neurologic emergency (Kaplow & Iyere, 2016). For the patient with spinal cord compression resulting from metastatic cancer (most commonly from breast, prostate, or lung), high-dose dexamethasone combined with radiation therapy is effective in relieving pain (see [Chapter 12](#) for a discussion of care of the patient with spinal cord compression). Palliative care may be an option for the medical management of some patients. Chemotherapy specific to the tumor type may be considered (AANN, 2016).

## Surgical Management

Tumor removal is desirable but not always possible. The goal is to remove as much tumor as possible while sparing uninvolved portions of the spinal cord. Sudden decrease or loss of motor, sensory, and bowel and bladder function indicates the need for emergent surgery to reestablish function and protect the cord from further damage (Hickey & Strayer, 2020). Microsurgical techniques have improved the prognosis for patients with intramedullary tumors. Extramedullary-intradural tumors may be completely resected. Prognosis is related to the degree of neurologic impairment at the time of surgery, the speed with which symptoms occurred, and the origin of the tumor. Patients with extensive neurologic deficits before surgery are less likely to regain full functional recovery after successful tumor removal.

## Nursing Management

### Providing Preoperative Care

The objectives of preoperative care include recognition of neurologic changes through ongoing assessments, pain control, and management of altered activities of daily living (ADLs) resulting from sensory and motor deficits and bowel and bladder dysfunction. The nurse assesses for weakness, muscle wasting, spasticity, sensory changes, bowel and bladder dysfunction, and potential respiratory problems, especially if a cervical tumor is present. The patient is also evaluated for coagulation deficiencies. A history of

anticoagulation medication use is obtained and reported, because the use of these may impede hemostasis postoperatively. The patient is educated about breathing exercises, and they are demonstrated preoperatively. Postoperative pain management strategies are discussed with the patient before surgery.

### **Assessing the Patient After Surgery**

The patient is monitored for deterioration in status with frequent and targeted assessments of vital signs and neurologic examinations (Hickey & Strayer, 2020). A sudden onset of neurologic deficit is an ominous sign and may be due to spinal cord ischemia or infarction. Frequent neurologic checks are carried out, with emphasis on movement, strength, and sensation of the upper and lower extremities. Assessment of sensory function involves pinching the skin of the arms, legs, and trunk to determine if there is loss of feeling and, if so, at what level. Vital signs are monitored at regular intervals to detect and treat potential complications early (Hemmer, 2018).

### **Managing Pain**

The prescribed pain medication should be given in adequate amounts and at appropriate intervals to relieve pain and prevent its recurrence. Pain is the hallmark of spinal metastasis. Patients with sensory root involvement may suffer excruciating pain, which requires effective pain management.

The bed is usually kept flat initially. The nurse turns the patient as a unit, keeping shoulders and hips aligned and the back straight (also referred to as logrolling) (see discussion later in chapter). The side-lying position is usually the most comfortable, because this position imposes the least pressure on the surgical site. Placement of a pillow between the knees of the patient in a side-lying position helps to prevent extreme knee flexion.

### **Monitoring and Managing Potential Complications**

If the tumor was in the cervical area, respiratory compromise due to postoperative edema may occur (Kaplow & Iyere, 2016). The nurse monitors the patient for asymmetric chest movement, abdominal breathing, and abnormal breath sounds. For a high cervical lesion, the endotracheal tube remains in place until adequate respiratory function is ensured. The patient is encouraged to perform deep-breathing and coughing exercises.

The area over the bladder is palpated or a bladder scan performed to assess for urinary retention (see [Chapter 47](#), Fig. 47-8). The nurse also monitors for incontinence, because urinary dysfunction usually implies significant decompensation of spinal cord function. An intake and output record is maintained. In addition, the abdomen is auscultated for bowel sounds.

Staining of the dressing may indicate leakage of CSF from the surgical site, which may lead to serious infection or to an inflammatory reaction in the

surrounding tissues that can cause severe pain in the postoperative period. Bulging at the incision may indicate contained CSF leak. The site should be monitored for increasing bulging, known as pseudomeningocele, which may require surgical repair (Alattar, Hirshman, McCutcheon, et al., 2018; Hickey & Strayer, 2020).

## Promoting Home, Community-Based, and Transitional Care



### Educating Patients About Self-Care

In preparation for discharge, the patient is assessed for the ability to function independently in the home and for the availability of resources such as family members to assist in caregiving. Patients with residual sensory involvement are cautioned about the dangers of extremes in temperature. They should be educated about the dangers of heating devices (e.g., hot water bottles, heating pads, space heaters) as their sensory integration may be impaired, causing them to lose the ability to detect dangerous stimulations and to react appropriately. The patient is educated to check skin integrity daily. Patients with impaired motor function related to motor weakness or paralysis may require training in ADLs and safe use of assistive devices, such as a cane, walker, or wheelchair. The patient and family members are educated about pain management strategies, bowel and bladder management, and assessment for signs and symptoms that should be reported promptly (see [Chart 65-2](#)).

### Continuing and Transitional Care

Referral for inpatient or outpatient rehabilitation may be warranted to improve self-care abilities. A consultation for home, community-based, or transitional care may be indicated and provides the nurse with the opportunity to assess the patient's physical and psychological status and the patient's and family's ability to adhere to recommended management strategies. During the home visit, the nurse determines whether changes in neurologic function have occurred. The patient's respiratory status and nutritional status are assessed. The adequacy of pain management is assessed, and modifications are made to ensure adequate pain relief. The need for hospice services or placement in an extended care facility is discussed with the patient and family if warranted, and the patient is asked about preferences for end-of-life care. In addition, social workers may be consulted to assist the patient and family members in identifying support services that can provide help in coping with the disease process (Hickey & Strayer, 2020).

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## DEGENERATIVE DISORDERS

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Disorders of the central and peripheral nervous system that are **neurodegenerative** (leading to deterioration of normal cells or function of the nervous system) are characterized by the slow onset of signs and symptoms. Patients are managed at home for as long as possible and are admitted to the acute care setting for exacerbations, treatments, and surgical interventions as needed.

## Parkinson's Disease

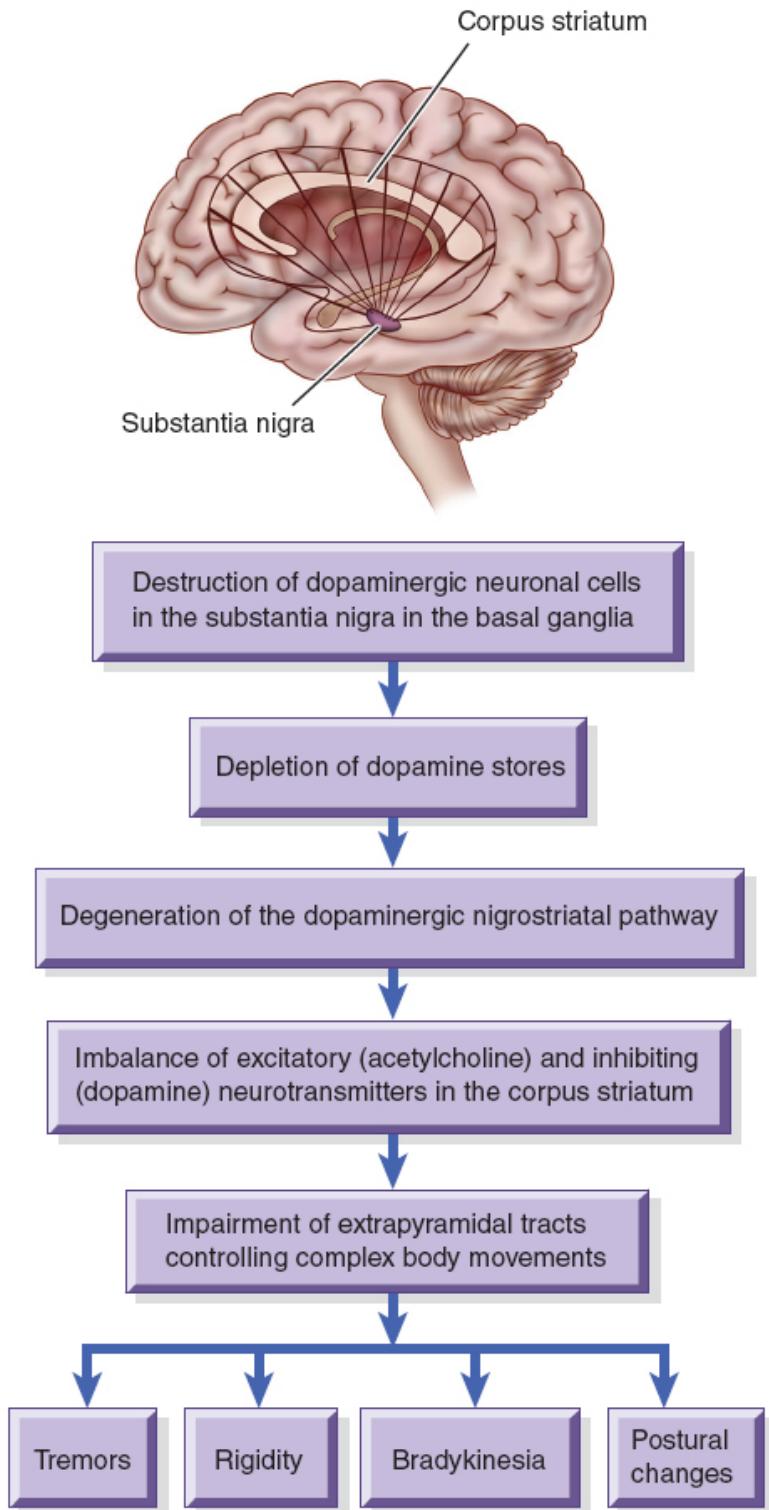
PD is a slowly progressing neurologic movement disorder that eventually leads to disability. It affects about 1 million patients who are hospitalized in the United States each year (Moore, Smith, & Cho, 2017). The disease affects men more often than women. Symptoms usually first appear in the fifth decade of life; however, cases have been diagnosed as early as 30 years of age. The degenerative or idiopathic form of PD is the most common; there is also a secondary form with a known or suspected cause. Although the cause of most cases is unknown, research suggests a multifactorial combination of age, environment, and heredity (AANN, 2019).

## Pathophysiology

PD is associated with decreased levels of dopamine resulting from degeneration of dopamine storage cells in the substantia nigra in the basal ganglia region of the brain (see Fig. 65-3). Fibers or neuronal pathways project from the substantia nigra to the corpus striatum, where neurotransmitters are vital to the control of complex body movements. Through the neurotransmitters acetylcholine (excitatory) and dopamine (inhibitory), striatal neurons relay messages to the higher motor centers that control and refine motor movements. The loss of dopamine stores in this area of the brain results in more excitatory neurotransmitters than inhibitory neurotransmitters, leading to an imbalance that affects voluntary movement (Hickey & Strayer, 2020).

Clinical symptoms do not appear until 60% of the pigmented neurons are lost and the striatal dopamine level is decreased by 80%. Cellular degeneration impairs the extrapyramidal tracts that control semiautomatic functions and coordinated movements; motor cells of the motor cortex and the pyramidal tracts are not affected. Researchers are working on uncovering the exact mechanism of neurodegeneration. Current theories suggest a combined and complicated interweaving of both environmental and genetic factors that affect numerous fundamental cellular processes. Fifteen percent of early PD cases are associated with multiple genetic mutations (Poewe, Seppi, Tanner, et al., 2017). Ongoing research includes recognition of biomarkers and development of individualized treatment options (Poewe et al., 2017).

## Physiology/Pathophysiology



**Figure 65-3 •** Pathophysiology of Parkinson's disease. The nuclei in the substantia nigra project fibers to the corpus striatum. The

nerve fibers carry dopamine to the corpus striatum. The loss of dopamine nerve cells from the brain's substantia nigra is thought to be responsible for the symptoms of Parkinson's disease.

## Clinical Manifestations

PD has a gradual onset, and symptoms progress slowly over a chronic, prolonged course. The cardinal signs are tremor, rigidity, bradykinesia/akinesia, and postural instability (Hickey & Strayer, 2020). Two major subtypes of PD are tremor dominant (most other symptoms are absent) and nontremor dominant (akinetic-rigid and postural instability).

### Tremor

Although symptoms are variable, a slow, unilateral resting tremor is present in the majority of patients at the time of diagnosis. Resting tremor characteristically disappears with purposeful movement and during sleep but is evident when the extremities are motionless or at rest. The tremor may manifest as a rhythmic, slow turning motion (pronation-supination) of the forearm and the hand and a motion of the thumb against the fingers as if rolling a pill between the fingers.

### Rigidity

Resistance to passive limb movement characterizes muscle rigidity. Passive movement of an extremity may cause the limb to move in jerky increments, referred to as lead-pipe or cogwheel movements. Involuntary stiffness of the passive extremity increases when another extremity is engaged in voluntary active movement. Stiffness of the arms, legs, face, and posture are common. Early in the disease, the patient may complain of shoulder pain due to rigidity (Hickey & Strayer, 2020).

### Bradykinesia

A common feature of PD is **bradykinesia**, which refers to the overall slowing of active movement (Bronner & Korczyn, 2017). Patients may also take longer to complete activities and have difficulty initiating movement, such as rising from a sitting position or turning in bed.

### Postural Instability

The patient commonly develops postural and gait problems. Due to a loss of postural reflexes, the patient stands with the head bent forward and walks with a propulsive gait. The posture is caused by the forward flexion of the neck, hips, knees, and elbows. The patient may walk faster and faster, trying to move the feet forward under the body's center of gravity (shuffling gait). Difficulty

in pivoting causes loss of balance, either forward (propulsion) or backward (retropulsion). Gait impairment and postural instability place the patient at increased risk for falls (Hickey & Strayer, 2020).

### Other Manifestations

The effect of PD on the basal ganglia often produces autonomic symptoms that include excessive and uncontrolled sweating, drooling, paroxysmal flushing, orthostatic hypotension, gastric and urinary retention, constipation, and sexual dysfunction (Bronner & Korczyn, 2017). Dysphagia is a substantial problem, with more than 50% of patients reporting choking as well as vision and olfactory changes (AANN, 2019). Neurogenic orthostatic hypotension occurs in 30% to 50% of patients with PD (Sin & Khemani, 2020).

Psychiatric changes include depression, anxiety, dementia, delirium, hallucinations, and psychosis. Depression and anxiety are common; whether these are reactions to the disorder or related to a biochemical abnormality is uncertain (AANN, 2019). Stress, medications, and depression contribute to the cognitive changes of diminished executive functions, attention difficulties, decreased thinking, and word-finding challenges. More than 80% of patients with a 20-year disease duration of PD experience **dementia**, a broad term for a syndrome characterized by a general decline in higher brain functioning, such as reasoning, with a pattern of eventual decline in ability to perform even basic ADLs, such as toileting and eating (Gale, Acar, & Daffner, 2018). In addition, auditory and visual hallucinations have been reported in people with PD and may be associated with depression, dementia, lack of sleep, or adverse effects of medications.

Hypokinesia (abnormally diminished movement) is also common and may appear after the tremor. The freezing phenomenon refers to a transient inability to perform active movement and is thought to be an extreme form of bradykinesia. The patient tends to shuffle and exhibits a decreased arm swing as well. As dexterity declines, micrographia (small handwriting) develops. The face becomes increasingly masklike and expressionless, and the frequency of blinking decreases. **Dysphonia** (voice impairment or altered voice production) may occur as a result of weakness and incoordination of the muscles responsible for speech. In many cases, the patient develops dysphagia, begins to drool, and is at risk for choking and aspiration (AANN, 2019).

Complications associated with PD are common and are typically related to disorders of movement. As the disease progresses, patients are at risk for respiratory and urinary tract infection, skin breakdown, and injury from falls. The adverse effects of medications used to treat the symptoms are associated with numerous complications such as **dyskinesia** (impaired ability to execute voluntary movements) or orthostatic hypotension.

## **Assessment and Diagnostic Findings**

Although laboratory tests and imaging studies are not helpful to the provider in diagnosing PD, ongoing research with PET and single-photon emission CT scanning has been helpful in understanding the disease and advancing treatment. Currently, the disease is diagnosed clinically from the patient's history and the presence of two of the four cardinal manifestations: tremor, rigidity, bradykinesia, and postural changes.

Early diagnosis can be challenging because patients rarely are able to pinpoint when the symptoms started. Often, a family member notices a change such as stooped posture; a stiff arm; a slight limp; tremor; or slow, small handwriting. The medical history, presenting symptoms, neurologic examination, and response to pharmacologic management are carefully evaluated when making the diagnosis. Diagnosis is often confirmed by a positive response to a levodopa trial (Hickey & Strayer, 2020).

The Revised Movement Disorder Society Unified Parkinson Disease Rating Scale (MDS-UPDRS) is a helpful assessment tool as it measures the disease progression including motor and nonmotor symptoms, and includes treatment complications (AANN, 2019).

## **Medical Management**

Treatment is directed toward controlling symptoms and maintaining functional independence, because no medical or surgical approaches in current use prevent disease progression (AANN, 2019). Care is individualized for each patient based on presenting symptoms and social, occupational, and emotional needs. Pharmacologic management is the mainstay of treatment, although advances in research have led to more surgical options. Patients are usually cared for at home and are admitted to the hospital only for complications or to initiate new treatments.

### **Pharmacologic Therapy**

Antiparkinsonian medications act by increasing striatal dopaminergic activity; reducing the excessive influence of excitatory cholinergic neurons on the extrapyramidal tract, thereby restoring a balance between dopaminergic and cholinergic activities; or acting on neurotransmitter pathways other than the dopaminergic pathway.

Levodopa is the most effective agent and the mainstay of treatment. Levodopa is converted to dopamine in the basal ganglia, producing symptom relief. Carbidopa is often added to levodopa to avoid metabolism of levodopa before it can reach the brain. The beneficial effects of levodopa therapy are most pronounced in the first year or two of treatment. Benefits begin to wane and adverse effects become more severe over time (Hickey & Strayer, 2020).

Within 5 to 10 years, most patients develop a response to the medication characterized by dyskinesia including facial grimacing, rhythmic jerking movements of the hands, head bobbing, chewing and smacking movements, and involuntary movements of the trunk and extremities. The patient may experience an on-off syndrome in which sudden periods of near immobility (“off effect”) are followed by a sudden return of effectiveness of the medication (“on effect”). Changing the drug dosing regimen or switching to other drugs may be helpful in minimizing the on-off syndrome. Other potential adverse effects include nausea, vomiting, appetite loss, decreased BP, dystonia, dyskinesia, and confusion (Comerford & Durkin, 2020). To minimize adverse effects of levodopa over time, current practice includes delaying use of levodopa-containing drugs as long as possible, with the use of other drugs for symptom control in the interim. [Table 65-1](#) provides a summary of select medications used in PD.

## Surgical Management

The limitations of levodopa therapy, improvements in surgical techniques, and new approaches in transplantation have renewed interest in the surgical treatment of PD. In patients with disabling tremor, rigidity, or severe levodopa-induced dyskinesia, surgery may be considered. Although surgery provides symptom relief in select patients, it has not been shown to alter the course of the disease or to produce permanent improvement.

### Stereotactic Procedures

Thalamotomy and pallidotomy are ablative procedures that were formerly used to relieve symptoms of PD such as tremors. However, these procedures permanently destroy brain tissue and are rarely used today. Deep brain stimulation (DBS) has largely replaced ablative procedures in the surgical treatment of PD. DBS involves surgical implantation of an electrode into the brain in either the globus pallidus or subthalamic nucleus. Stimulation of these areas may increase dopamine release or block anticholinergic release, thereby improving tremor and rigidity. Levodopa medication dose may be able to be reduced, thus improving dyskinesias.

Patients eligible for DBS are those who have responded to levodopa but are impaired by dyskinesias, have had the disease for at least 5 years, and are disabled by tremor. Patients with dementia and atypical PD are usually not considered for surgical procedures. PD rating scales and specific neurologic tests are used to identify patients who are eligible. Surgical treatment typically occurs 10 to 13 years after diagnosis (AANN, 2019).

**TABLE 65-1** Select Medications Used to Treat Parkinson's Disease

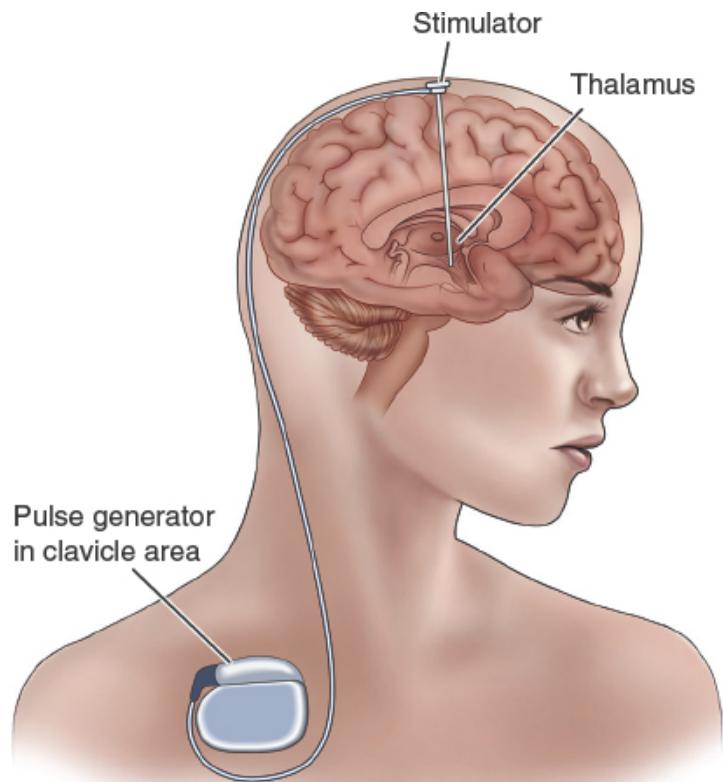
Medications	Indications and Therapeutic Effects	Common Side Effects
<b>Anticholinergic Agents</b>		
trihexyphenidyl hydrochloride	Control of tremor in patients with early-onset disease	Blurred vision, flushing, rash, constipation, urinary retention, and acute confusional states
benztropine mesylate	Counteract the action of acetylcholine	Contraindicated in patients with narrow-angle glaucoma
<b>Antiviral Agent</b>		
amantadine hydrochloride	Reduce rigidity, tremor, bradykinesia, and postural changes in early Parkinson's disease (PD)	Psychiatric disturbances (mood changes, confusion, depression, hallucinations), lower extremity edema, nausea, epigastric distress, urinary retention, headache, and visual impairment
<b>Dopamine Agonists</b>		
bromocriptine mesylate pergolide	Early PD as well as secondary drug therapy after carbidopa or levodopa loses effectiveness	Nausea, vomiting, diarrhea, lightheadedness, hypotension, impotence, and psychiatric effects
<b>Nonergot Derivatives</b>		
ropinirole hydrochloride pramipexole	Early stages of PD	May cause drowsiness or dizziness
<b>Monoamine Oxidase-Inhibitors</b>		
selegiline rasagiline	Inhibit dopamine breakdown	Agitation, dizziness, nausea, headache, rhinitis, back pain, stomatitis, orthostatic hypotension, insomnia
<b>Catechol-O-Methyltransferase Inhibitors</b>		
entacapone	Increase the duration of action of carbidopa or levodopa	Abdominal pain, back pain, constipation, nausea, diarrhea, blood in urine
tolcapone	Reduce motor fluctuations in patients with advanced PD	

Adapted from Moore, D. J., Smith, B. M., & Cho, M. H. (2017). Managing medications for hospital patients with Parkinson disease. *American Nurse Today*, 12(1), 9–12.

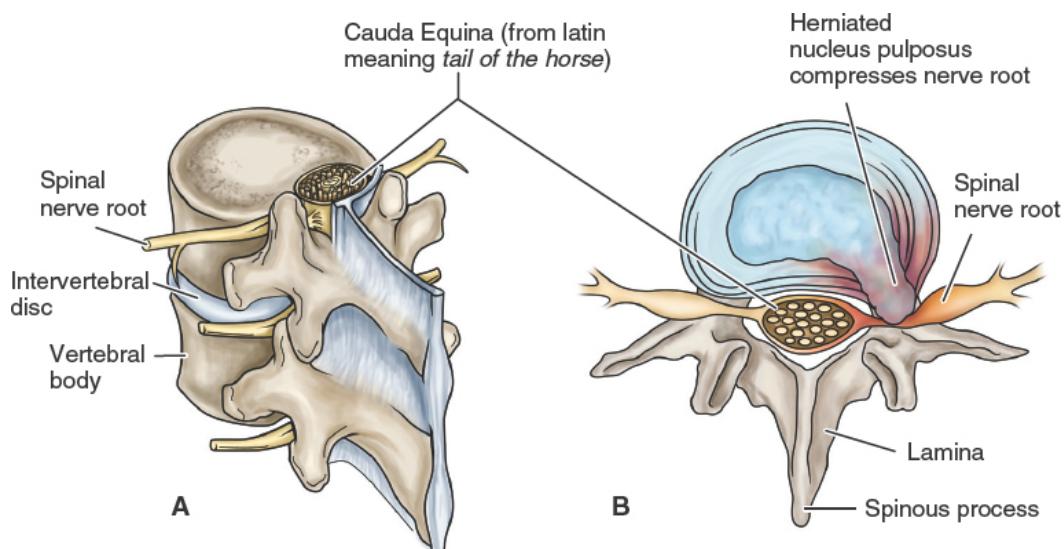
A CT or MRI scan is used to localize the appropriate surgical site in the brain. Then, the patient's head is positioned in a stereotactic frame (see Fig. 65-4). After the surgeon makes an incision in the skin and a burr hole, an electrode is passed through to the target area to the subthalamic nuclei or globus pallidus. The desired response of the patient to the electrical stimulation (i.e., a decrease in rigidity) is used to confirm electrode placement. Electrode placement is completed on one side of the brain at a time; bilateral electrodes are usually placed (AANN, 2019). Electrodes are then connected to a pulse generator that is implanted in a subcutaneous subclavicular or abdominal pouch (see Fig. 65-5). The battery-powered pulse generator sends high-frequency electrical impulses through a wire placed under the skin to a lead anchored to the skull (see Fig. 65-6). These devices are not without complications that can result from both the surgical procedure needed for implantation (e.g., weakness, paresthesias, confusion, hemorrhage) and the device itself (e.g., infection, lead leakage) (Hickey & Strayer, 2020).



**Figure 65-4 •** A stereotactic frame is applied to a patient's head in preparation for deep brain stimulation. The frame provides external points of reference.



**Figure 65-5 •** Deep brain stimulation is provided by a pulse generator surgically implanted in a pouch beneath the clavicle. The generator sends high-frequency electrical impulses to the thalamus, thereby blocking the nerve pathways associated with tremors in Parkinson's disease.



**Figure 65-6 • A.** Normal lumbar spine vertebrae, intervertebral discs, and spinal nerve root. **B.** Ruptured vertebral disc.

## Neural Transplantation

Ongoing research is exploring transplantation of porcine neuronal cells, human fetal cells, and stem cells to replace degenerated striatal cells (Kirkeby, Parmar, & Barker, 2017). Legal, ethical, and political concerns surrounding the use of fetal brain cells and stem cells have limited the exploration of these procedures.

## NURSING PROCESS

### The Patient with Parkinson's Disease

#### Assessment

The nurse gathers information focusing on how the disease has affected the patient's ADLs and functional abilities. The patient is observed for degree of disability and functional changes that occur throughout the day, such as responses to medication. Almost every patient with a movement disorder has some functional alteration and may have some type of behavioral dysfunction. The following questions may be useful to assess alterations:

- Do you have leg or arm stiffness?
- Have you experienced any irregular jerking of your arms or legs?
- Have you ever been "frozen" or rooted to the spot and unable to move?
- Does your mouth water excessively? Have you (or others) noticed yourself grimacing or making faces or chewing movements?
- What specific activities do you have difficulty doing?
- Have you had any recent falls?

During this assessment, the nurse observes the patient for quality of speech, loss of facial expression, swallowing deficits (drooling, poor head control, coughing), tremors, slowness of movement, weakness, forward posture, rigidity, evidence of mental slowness, and confusion. PD symptoms, as well as side effects of medications, put these patients at high risk for falls; therefore, a fall risk assessment should be conducted (Hickey & Strayer, 2020).

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired mobility associated with muscle rigidity and postural impairment
- Impaired self-feeding, impaired ability to dress, impaired ability to perform hygiene, impaired self-toileting associated with tremor and muscle rigidity
- Constipation associated with medication and reduced activity
- Impaired nutritional intake associated with tremor, slowness in eating, difficulty in chewing and swallowing
- Impaired verbal communication associated with decreased speech volume, slowness of speech, inability to move facial muscles
- Difficulty coping associated with depression and dysfunction due to disease progression

### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Sleep disturbances
- Psychiatric disturbances

### **Planning and Goals**

The goals for the patient may include improving functional mobility, maintaining independence in ADLs, achieving adequate bowel elimination, attaining and maintaining acceptable nutritional status, achieving effective communication, and developing positive coping mechanisms.

### **Nursing Interventions**

#### **IMPROVING MOBILITY**

A progressive program of daily exercise will increase muscle strength, improve coordination and dexterity, reduce muscular rigidity, and prevent contractures that occur when muscles are not used (Hickey & Strayer, 2020). Walking, riding a stationary bicycle, swimming, and gardening are all exercises that help maintain joint mobility. Stretching (stretch–hold–relax) and range-of-motion exercises promote joint flexibility. Postural exercises are important to counter the tendency of the head and neck to be drawn forward and down. A physical therapist may be helpful in developing an individualized exercise program and can provide instruction to the patient and caregiver on exercising safely. Faithful adherence to an exercise and walking program helps delay the progress of the disease. Warm baths and massage, in addition to passive and active exercises, help relax muscles and relieve painful muscle spasms that accompany rigidity.

Balance may be adversely affected because of the rigidity of the arms (arm swinging is necessary in normal walking). Special walking techniques must be learned to offset the shuffling gait and the tendency to lean forward. The patient is educated to concentrate on walking erect, to watch the horizon, and to use a wide-based gait (i.e., walking with the feet separated). A conscious effort must be made to swing the arms, raise the feet while walking, and use a heel–toe placement of the feet with long strides. The patient is advised to practice walking to marching music or to the sound of a ticking metronome, because this provides sensory reinforcement. Performing breathing exercises while walking provides additional movement of the ribcage and aerates larger parts of the lungs. Frequent rest periods aid in preventing frustration and fatigue.

#### **ENHANCING SELF-CARE ACTIVITIES**

Encouraging, educating, and supporting the patient during ADLs promote self-care (Hickey & Strayer, 2020). Environmental modifications are necessary to compensate for functional disability. Patients may have severe mobility problems that make normal activities impossible. Adaptive or assistive devices may be useful. A hospital bed at home with bedside rails,

an over-bed frame with a trapeze, or a rope tied to the foot of the bed can provide assistance in pulling up without help. An occupational therapist can evaluate the patient's needs in the home, make recommendations regarding adaptive devices, and educate the patient and caregiver how to improvise.

#### **IMPROVING BOWEL ELIMINATION**

The patient may have severe problems with constipation. Among the factors causing constipation are weakness of the muscles used in defecation, lack of exercise, inadequate fluid intake, and decreased autonomic nervous system activity. The medications used for the treatment of the disease also inhibit normal intestinal secretions. A regular bowel routine may be established by encouraging the patient to follow a regular time pattern, consciously increase fluid intake, and eat foods with moderate fiber content. Laxatives should be avoided. Psyllium, for example, decreases constipation but carries the risk of bowel obstruction (Comerford & Durkin, 2020). A raised toilet seat is useful because the patient has difficulty in moving from a standing to a sitting position. Additional time may be needed for the patient to navigate to the toilet due to gait disturbances.

#### **IMPROVING NUTRITION**

Patients may have difficulty maintaining their weight. Eating becomes a very slow process, requiring concentration due to a dry mouth from medications and difficulty chewing and swallowing. These patients are at risk for aspiration because of impaired swallowing and the accumulation of saliva. They may be unaware that they are aspirating; subsequently, pneumonia may develop (Aslam, Simpson, Baugh, et al., 2019).

Monitoring weight on a weekly basis indicates whether caloric intake is adequate. Supplemental feedings increase caloric intake. As the disease progresses, a nasogastric or percutaneous endoscopic gastrostomy (PEG) tube may be necessary to maintain adequate nutrition. A consultation with dietitian may be indicated.

#### **ENHANCING SWALLOWING**

Swallowing difficulties and choking are common in PD, leading to aspiration and pneumonia. To offset the aspiration risk, the patient should sit in an upright position during mealtime (Aslam et al., 2019). A semisolid diet with thick liquids is easier to swallow than solids; thin liquids should be avoided. The patient is instructed to think through the swallowing sequence, place the food on the tongue, close the lips and teeth, lift the tongue up and then back, and swallow. The patient is encouraged to chew first on one side of the mouth and then on the other. To control the buildup of saliva, the patient is reminded to hold the head upright and make a conscious effort to swallow. Massaging the facial and neck muscles before meals may be beneficial.

### **ENCOURAGING THE USE OF ASSISTIVE DEVICES**

An electric warming tray keeps food hot and allows the patient to rest during the prolonged time that it may take to eat. Special utensils also assist at mealtime. A plate that is stabilized, a nonspill cup, and eating utensils with built-up handles are useful self-help devices. The occupational therapist can assist in identifying appropriate adaptive devices.

### **IMPROVING COMMUNICATION**

Speech disorders are present in most patients with PD. The low-pitched, monotonous, soft speech of patients requires that they make a conscious effort to speak slowly, with deliberate attention to what they are saying. The patient is reminded to face the listener, exaggerate the pronunciation of words, speak in short sentences, and take a few deep breaths before speaking. A speech therapist may be helpful in designing speech improvement exercises and assisting the family and health care personnel to develop and use a method of communication that meets the patient's needs. A small electronic amplifier is helpful if the patient has difficulty being heard.

### **SUPPORTING COPING ABILITIES**

Support can be given by encouraging the patient and pointing out that activities will be maintained through active participation. Patients with PD can become socially and emotionally withdrawn. It is best if patients are active participants in their therapeutic program, including social and recreational events.

Patients often feel embarrassed, apathetic, inadequate, bored, and lonely. In part, these feelings may result from physical slowness and the great effort that even small tasks require. The patient is assisted and encouraged to set achievable goals (e.g., improvement of mobility). Every effort should be made to encourage patients to carry out the tasks involved in meeting their own daily needs and to remain independent. Doing things for the patient merely to save time undermines the basic goal of improving coping abilities and promoting a positive self-concept.

### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

A planned program of activity throughout the day prevents too much daytime sleeping as well as disinterest and apathy. When sleep disturbances occur, several interventions can improve sleep, including limiting caffeine intake before bedtime and assessing for nocturia.

The patient with PD is monitored regularly for signs and symptoms of depression. An interprofessional approach with a combination of physiotherapy, psychotherapy, medication therapy, and support group participation is used to treat depression when diagnosed (Hickey & Strayer, 2020).

The patient in late stages of PD is particularly at risk for psychiatric disturbances such as hallucinations, psychosis, and paranoid delusions (Hickey & Strayer, 2020). Interventions to manage psychiatric disturbances include low doses of antipsychotic drugs, minimizing medications with psychiatric side effects (see [Table 65-1](#)), and avoidance of dopamine depleting medications (Hickey & Strayer, 2020). Patients and their caregivers benefit from an interprofessional approach to care when psychiatric disturbances are present.

#### **PROMOTING HOME, COMMUNITY-BASED, OR TRANSITIONAL CARE**



**Educating Patients About Self-Care.** Patient and family education is important in the management of PD. The needs depend on the severity of symptoms and the stage of the disease.

Care must be taken not to overwhelm the patient and family with too much information early in the disease process. Ongoing assessment, intervention, and evaluation of the patient and family's adaptations and education requirements are necessary with every encounter. The strategies to promote health include a clear explanation of the disease and the goal of assisting the patient to remain functionally independent as long as possible (see [Chart 65-3](#)). The patient and family must be educated about the effects and side effects of medications and about the importance of reporting side effects to the primary provider. Nurses in the home help patients and their families enhance self-management and quality of life.

**Continuing and Transitional Care.** Family members and partners often serve as caregivers, with home, community-based, or transitional services available to assist in meeting health care needs as the disease progresses. The caregiver may be under considerable stress from living with and caring for a person with a significant disability. Providing information about treatment and care helps anticipate future needs. The caregiver is included in the plan and may be advised to learn stress reduction techniques, to include others in the caregiving process, to obtain periodic relief from responsibilities, and to have a yearly health assessment. Allowing family members and partners to express feelings of frustration, anger, and guilt is often helpful to them.

The patient should be evaluated in the home for adaptation and safety needs and adherence to the plan of care. In the advanced stages, patients usually enter long-term care facilities. Periodically, admission to an acute care facility may be necessary for changes in medical management or treatment of complications. Nurses provide support, education, and monitoring of patients over the course of the illness.

**Chart 65-3**  **HEALTH PROMOTION**

## **Strategies for the Patient with PD**

To promote optimal health, the nurse works closely with the patient and family to ensure that they understand:

- How PD and its treatment impact physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality
- The importance of adhering to the prescribed medication regimen; including knowing the purpose, dose, route, schedule, side effects, and precautions for all medications
- How and when to contact all members of the treatment team (e.g., health care providers, home care professionals, and durable medical equipment and supply vendors)
- Specific types of environmental and safety changes or support that will allow optimum functioning in the home
- The risk of falls/injury and how to implement fall prevention and adaptive measures in the home

In addition, the nurse advises the patient and family about lifestyle changes that are necessary to maintain health and promote self-care and independence. These include:

- Ensuring nutritional needs, including adhering to dietary restrictions, managing dysphagia, and preventing aspiration
- Promoting speech and communication skills: speech exercises, communication techniques, breathing exercises
- Managing constipation: fluid intake, bowel routine
- Managing urinary problems: functional incontinence, retention (indwelling urinary catheter care, suprapubic catheter care)
- Avoiding the effects of immobility and promoting the advantages of preventive care: skin breakdown (frequent turning, pressure release, skin care), pneumonia (deep breathing, movement), contractures (range-of-motion exercises)
- Promoting the benefits of a daily exercise program
- Ensuring safe walking and balancing
- Using appropriate coping mechanisms and diversional activities

ADLs, activities of daily living; IADLs, instrumental activities of daily living; PD, Parkinson's disease.

The nurse involved in home and continuing care educates the patient and family members about the importance of addressing health promotion needs such as screening for hypertension and stroke risk assessments in this predominantly older adult population. Patients are referred to appropriate health care providers. Information is available from both the Parkinson's Foundation and the American Parkinson Disease Association (see the Resources section).

### Evaluation

Expected patient outcomes may include:

1. Strives toward improved mobility
  - a. Participates in exercise program daily
  - b. Walks with wide base of support; exaggerates arm swinging when walking
  - c. Takes medications as prescribed
2. Progresses toward self-care
  - a. Allows time for self-care activities
  - b. Uses self-help devices
3. Maintains bowel function
  - a. Consumes adequate fluid
  - b. Increases dietary intake of fiber
  - c. Reports regular pattern of bowel function
4. Attains improved nutritional status
  - a. Swallows without aspiration
  - b. Takes time while eating
5. Achieves a method of communication
  - a. Communicates needs
  - b. Practices speech exercises
6. Copes with effects of PD
  - a. Sets realistic goals
  - b. Demonstrates persistence in meaningful activities
  - c. Verbalizes feelings to appropriate person

## Huntington Disease

Huntington disease is a chronic, progressive, hereditary disease of the nervous system that results in progressive involuntary choreiform movement and dementia. The disease affects approximately 1 in 10,000 men or women of all races at midlife. Every person has the gene that causes Huntington disease;

however, only those who inherit the expansion of the gene will develop the disease and pass it onto their children. Because it is transmitted by an autosomal dominant gene, each child of a parent with Huntington disease has a 50% risk of inheriting the disorder (Smedley & Coulson, 2019).

A genetic mutation in Huntington disease, the presence of a repeat in the Huntington gene (*HTT*), has been identified (Smedley & Coulson, 2019). Genetic testing can identify people who will develop this disease but cannot predict timing of disease onset. Although the gene was mapped in 1983, patients may choose not to be tested because of concerns about employment and health care discrimination. People of childbearing age with a family history of Huntington disease often seek information about their risk of disease transmission. Genetic counseling is crucial, and patients and their families may require long-term psychological counseling and emotional, financial, and legal support (see [Chapter 6](#)).

## Pathophysiology

The basic pathology involves premature death of cells in the striatum (caudate and putamen) of the basal ganglia, the region deep within the brain that is involved in the control of movement. Cells also are lost in the cortex, the region of the brain associated with thinking, memory, perception, judgment, and behavior, and in the cerebellum, the area that coordinates voluntary muscle activity. Why the protein destroys only certain brain cells is unknown, but several theories have been proposed to explain the phenomenon. One possible theory is that glutamine, a building block for protein, abnormally collects in the cell nucleus, causing cell death (McColgan & Tabrizi, 2017).

## Clinical Manifestations

This condition is characterized by a triad of symptoms that includes motor dysfunction (the most prominent is **chorea**, or rapid, jerky, involuntary, purposeless movements); cognitive impairment (problems with attention and emotion recognition); and behavioral features, such as apathy and blunted affect (McColgan & Tabrizi, 2017). As the disease progresses, constant writhing, twisting, and uncontrollable movements may involve the entire body. These motions are devoid of purpose or rhythm, although patients may try to turn them into purposeful movement. All of the body musculature is involved. Facial movements produce tics and grimaces. Speech becomes slurred, hesitant, often explosive, and eventually unintelligible. Chewing and swallowing are difficult, and there is a constant danger of choking and aspiration. Choreiform movements persist during sleep but are diminished (Mestre & Shannon, 2017).

As with speech, the gait becomes disorganized to the point that ambulation eventually is impossible. Although independent ambulation should be encouraged for as long as possible, a wheelchair usually becomes necessary. Eventually, the patient is confined to bed when the chorea interferes with walking, sitting, and all other activities. Bladder and bowel control are lost.

Cognitive impairment, such as problems with attention or recognizing emotions, occurs early on; in later stages, marked dementia is present. Initially, the patient is aware that the disease is responsible for the myriad dysfunctions that are occurring.

The behavioral changes may be more devastating to the patient and family than the abnormal movements. Personality changes may result in nervous, irritable, or impatient behaviors. In the early stages, patients are particularly subject to uncontrollable fits of anger, profound and often suicidal depression, apathy, anxiety, psychosis, or euphoria. Judgment and memory are impaired, and dementia eventually ensues (Mestre & Shannon, 2017). Hallucinations, delusions, and paranoid thinking may precede the appearance of disjointed movements. Emotional and cognitive symptoms often become less acute as the disease progresses (Mestre & Shannon, 2017).

Onset usually occurs between 35 and 45 years of age, although about 10% of patients are children. The disease progresses slowly. Despite a ravenous appetite, patients usually become emaciated and exhausted. Patients succumb in 10 to 20 years to heart failure, pneumonia, or infection, or as a result of a fall or choking (Mestre & Shannon, 2017).

## Assessment and Diagnostic Findings

The diagnosis is made based on the clinical presentation of characteristic symptoms, a positive family history, and the known presence of the genetic marker cytosine-adenine-guanine (CAG) repeating on the Huntington gene (HTT)(Smedley & Coulson, 2019). CT or MRI scans show symmetrical striatal atrophy before motor symptoms appear (Urrutia, 2019).

## Medical Management

No treatment halts or reverses the underlying process; therefore, the focus is on optimizing quality of life with available medication and supportive treatment. Patients experience the most benefit when they have integrated expertise of a Huntington disease multidisciplinary team (Urrutia, 2019). The only medication that is approved by the U.S. Food and Drug Administration to treat the symptom of chorea is tetrabenazine (Comerford & Durkin, 2020).

Benzodiazepines and neuroleptic drugs have also been reported to control chorea. Motor signs must be assessed and evaluated on an ongoing basis so that optimal therapeutic drug levels can be reached. Akathisia (motor

restlessness) in the patient who is overmedicated is dangerous because it may be mistaken for the restless fidgeting of the illness and consequently may be overlooked. In certain types of the disease, hypokinetic motor impairment resembles PD. In patients who present with rigidity, some temporary benefit may be obtained from antiparkinson medications, such as levodopa.

Selective serotonin reuptake inhibitors and tricyclic antidepressants have been recommended for control of psychiatric symptoms. The threat of suicide is present particularly early in the course of the disease. Psychotic symptoms usually respond to antipsychotic medications. Psychotherapy aimed at allaying anxiety and reducing stress may be beneficial. Nurses must look beyond the disease to focus on the patient's needs and capabilities (see [Chart 65-4](#)).

## Promoting Home, Community-Based, and Transitional Care



### Educating Patients About Self-Care

The needs of the patient and family for education depend on the nature and severity of the physical, cognitive, and psychological changes experienced by the patient. The patient and family members are educated about the medications prescribed and about signs indicating a need for change in medication or dosage. The educational plan addresses strategies to manage symptoms such as chorea, swallowing problems, limitations in ambulation, memory loss, irritability, depression, and loss of bowel and bladder function. Consultation with a speech therapist may be indicated to assist in identifying alternative communication strategies if speech is affected. A PEG tube may be considered for nutritional support later in the disease (Eliopoulos, 2018).

### Continuing and Transitional Care

A program combining medical, nursing, psychological, social, occupational, speech, and physical rehabilitation services and palliative care is needed to help the patient and family cope with this severely disabling illness. Huntington disease exacts enormous emotional, physical, social, and financial tolls on every member of the family. The family needs supportive care as they adjust to the impact of the illness. Regular follow-up visits help allay the fear of abandonment.

#### Chart 65-4

## Care of the Patient with Huntington Disease

**Nursing Diagnosis:** Risk for injury from falls and possible skin breakdown (pressure injury abrasions), resulting from constant movement

### Nursing Interventions

Pad the sides and head of the bed; ensure that the patient can see over the sides of bed.

Use padded heel and elbow protectors.

Keep the skin meticulously clean.

Apply emollient cleansing agent and skin lotion as needed.

Use soft sheets and bedding.

Have patient wear football padding or other forms of padding.

Encourage ambulation with assistance to maintain muscle tone.

Secure the patient (only if necessary) in bed or chair with padded protective devices, making sure that they are loosened frequently.

**Nursing Diagnosis:** Impaired nutritional intake due to inadequate ingestion and dehydration resulting from swallowing or chewing disorders and danger of choking or aspirating food

### Nursing Interventions

Administer phenothiazines (chlorpromazine) as prescribed before meals (calms some patients).

Talk to the patient before mealtime to promote relaxation; use mealtime for social interaction. Provide undivided attention and help the patient enjoy the mealtime experience.

Use a warming tray to keep food warm.

Learn the position that is best for *this* patient. Keep patient as close to upright as possible while feeding. Stabilize patient's head gently with one hand while feeding.

Show the food, explain what the foods are, and temperature (e.g., whether hot or cold).

Encircle the patient with one arm and get as close as possible to provide stability and support while feeding. Use pillows and wedges for additional support.

Do not interpret stiffness, turning away, or sudden turning of the head as rejection; these are uncontrollable choreiform movements.

For feeding, use a long-handled spoon (iced tea spoon). Place spoon on middle of tongue and exert slight pressure.

Place bite-sized food between patient's teeth. Serve stews, casseroles, and thick liquids.

Disregard messiness, and treat the person with dignity.

Wait for the patient to chew and swallow before introducing another spoonful. Make sure that bite-sized food is small.

Give between-meal feedings. Constant movement expends more calories.

Patients often have voracious appetites, particularly for sweets.

Use blenderized meals if patient cannot chew; do not repeatedly give the same strained baby foods. Gradually introduce increased textures and consistencies to the diet.

For swallowing difficulties:

Apply gentle deep pressure around the patient's mouth.

Rub fingers in circles on the patient's cheeks and then down each side of the patient's throat.

Develop skill in the abdominal thrust (to be used in the event of choking).

**Nursing Diagnosis:** Impaired verbal communication from excessive grimacing and unintelligible speech

### Nursing Interventions

Read to the patient.

Employ biofeedback and relaxation therapy to reduce stress.

Consult with speech therapist to help maintain and prolong communication abilities.

Try to devise a communication system, perhaps using cards with words or pictures of familiar objects, before verbal communication becomes too difficult. Patients can indicate correct card by hitting it with hand, grunting, or blinking the eyes.

Learn how this particular patient expresses needs and wants—particularly nonverbal messages (widening of eyes, responses).

Patients can understand even if unable to speak. Do not isolate patients by ceasing to communicate with them.

**Nursing Diagnosis:** Acute confusion and impaired socialisation

### Nursing Interventions

Reorient the patient after awakening.

Have clock, calendar, and wall posters in view to assist in orientation.

Use every opportunity for one-to-one contact.

Use music for relaxation.

Have the patient wear a medical identification bracelet.

Keep the patient in the social mainstream.

Recruit and train volunteers for social interaction. Role-model appropriate and creative interactions.

Do not abandon a patient because the disease is terminal. Patients are *living* until the end.

Home care assistance, day care centers, respite care, and eventually skilled long-term care can assist the patient and family in coping with the constant strain of the illness. Although the relentless progression of the disease cannot be halted, families can benefit from supportive care. Planning for end-of-life care should occur early in the disease (see [Chapter 13](#)).

Voluntary organizations can be major aids to families and have been largely responsible for bringing the illness to national attention. The Huntington Disease Society of America helps patients and families by providing information, referrals, family and public education, and support for research (see the Resources section).

## Amyotrophic Lateral Sclerosis

ALS is a disease of unknown cause in which there is a loss of motor neurons (nerve cells controlling muscles) in the anterior horns of the spinal cord and the motor nuclei of the lower brainstem. It is often referred to as Lou Gehrig disease, after the famous baseball player who suffered from the disease. As motor neuron cells die, the muscle fibers that they supply undergo atrophic changes. Neuronal degeneration may occur in both the upper and lower motor neuron systems (see [Chapter 60](#)). The leading theory held by researchers is that overexcitation of nerve cells by the neurotransmitter glutamate results in cell injury and neuronal degeneration. Risk factors are noted in [Chart 65-5](#).

ALS most commonly occurs between 40 and 60 years of age and affects all social, racial, and ethnic backgrounds, with men being affected at slightly higher rates than women. The majority of cases of ALS arise sporadically, but 5% to 10% of cases are familial ALS resulting from an autosomal dominant trait carried by one parent. Familial ALS occurs 10 years earlier than the ALS average, and those afflicted tend to have a shorter life span (Hardiman, Al-Chalabi, Chio, et al., 2017).

## Clinical Manifestations

Clinical manifestations depend on the location of the affected motor neurons, because specific neurons activate specific muscle fibers. The chief symptoms are fatigue, progressive muscle weakness, cramps, fasciculations (twitching), and lack of coordination. Loss of motor neurons in the anterior horns of the spinal cord results in progressive weakness and atrophy of the muscles of the arms, trunk, or legs. Spasticity usually is present, and the deep tendon stretch reflexes become brisk and overactive. Usually, the function of the anal and bladder sphincters remains intact, because the spinal nerves that control muscles of the rectum and urinary bladder are not affected.

### Chart 65-5 RISK FACTORS

### **Amyotrophic Lateral Sclerosis**

- Age
- Autoimmune disease
- Environmental exposures to toxins
- Family history
- Smoking
- Viral infections

Adapted from Vacca, V. M. (2020). Amyotrophic lateral sclerosis: Nursing care and considerations. *Nursing*, 50(6), 32–39.

In about 25% of patients, weakness starts in the muscles supplied by the cranial nerves, and difficulty in talking, swallowing, and ultimately breathing occurs (Conde, Martin, & Winck, 2019; Vacca, 2020). When the patient ingests liquids, soft palate and upper esophageal weakness cause the liquid to be regurgitated through the nose. Weakness of the posterior tongue and palate impairs the ability to laugh, cough, or even blow the nose. If bulbar muscles are impaired, speaking and swallowing are progressively difficult, and aspiration becomes a risk. The voice assumes a nasal sound, and articulation becomes so disrupted that speech is unintelligible. Some emotional lability may be present. It was traditionally believed that ALS spared cognitive function, but it is now recognized that some patients experience cognitive impairment.

The prognosis generally is based on the area of CNS involvement and the speed with which the disease progresses. Eventually, respiratory function is compromised (Conde et al., 2019). Death usually occurs as a result of infection, respiratory insufficiency, or aspiration.

## **Assessment and Diagnostic Findings**

ALS is diagnosed on the basis of the signs and symptoms, because no clinical or laboratory tests are specific to this disease. Electromyography and muscle biopsy studies of the affected muscles indicate reduction in the number of functioning motor units. An MRI scan may show high signal intensity in the corticospinal tracts; this differentiates ALS from a multifocal motor neuropathy. Neuropsychological testing can assist in assessment and diagnosis (Hickey & Strayer, 2020).

## **Management**

No cure exists for ALS (Vacca, 2020). The main focus of medical and nursing management is on interventions to maintain or improve function, well-being, and quality of life. Because ALS is a progressive disease, the therapeutic needs

are different than those of patients with acute processes. Insurance carriers tend to limit the number of therapy sessions, but with early integration into ALS clinics, alliances are developed for future contact with therapists familiar with the disease process (Hogden, Foley, Henderson, et al., 2017).

Two drugs are used to treat ALS, riluzole and edaravone (Vacca, 2020). The precise action of these drugs is not clear, but both are considered disease modifying treatments for ALS (Vacca, 2020).

Symptomatic treatment and rehabilitative measures are used to support the patient and improve the quality of life. Baclofen, dantrolene sodium, or diazepam may be useful for patients troubled by spasticity, which causes pain and interferes with self-care. Modafinil may be used for fatigue, and additional medications may be added to manage the pain, depression, drooling, and constipation that often accompany the disease. Research suggests that greater functional impairment is associated with greater depressive symptoms; consequently, managing depression helps to maintain a better quality of life (Soofi, Bello-Haas, Kho, et al., 2018). Many clinical trials and an ALS registry contribute to the ongoing study of this devastating disease (Vacca, 2020).

Most patients with ALS are managed at home and in the community, with hospitalization for acute problems. The most common reasons for hospitalization are dehydration and malnutrition, pneumonia, and respiratory failure; recognizing these problems at an early stage in the illness allows for the development of preventive strategies. End-of-life issues include pain, dyspnea, and delirium (Hickey & Strayer, 2020).

Mechanical ventilation (using negative-pressure ventilators) is an option if alveolar hypoventilation develops. Noninvasive positive-pressure ventilation is also an option. The use of noninvasive positive-pressure ventilation is particularly helpful at night and postpones the decision about whether to undergo a tracheotomy for long-term mechanical ventilation (Conde et al., 2019). A patient experiencing aspiration and swallowing difficulties may require enteral feeding. A PEG tube is inserted before the forced vital capacity drops below 50% of the predicted value. The tube can be safely placed in patients who are using noninvasive positive-pressure ventilation for ventilatory support (Hickey & Strayer, 2020).

Decisions about life support measures are made by the patient and family and should be based on a thorough understanding of the disease, the prognosis, and the implications of initiating such therapy. Patients are encouraged to complete an advance directive to preserve their autonomy in decision making. See [Chapter 13](#) for additional discussion of end-of-life care.

The ALS Association has broad programs of research funding, patient and clinical services, patient information and support, and medical and public information (see the Resources section). The *ALS Association Newsletter* is a source of practical information.

## Muscular Dystrophies

The muscular dystrophies are a group of incurable muscle disorders characterized by progressive weakening and wasting of the skeletal or voluntary muscles with 30 different types to date. Most of these diseases are inherited. Duchenne muscular dystrophy, the most common and severe type, occurs in 1 of every 3500 male births (Norris, 2019). The pathologic features include degeneration and loss of muscle fibers, variation in muscle fiber size, phagocytosis and regeneration, and replacement of muscle tissue by connective tissue. The common characteristics of these diseases include varying degrees of muscle wasting and weakness and abnormal elevation in serum levels of muscle enzymes. Differences among these diseases center on the genetic pattern of inheritance, the muscles involved, the age at onset, and the rate of disease progression. The symptoms can be diverse and may include muscle stiffness or weakness, decreased respiratory reserve, or cardiomyopathy. Prognosis depends on the type of muscular dystrophy. Regardless of age of onset, this disease varies in progression (Birnkrant, Bushby, Bann, et al., 2018b). The unique needs of these patients, who in the past did not live to adulthood, must be addressed, as they are living longer because of better supportive care and the emergence of disease modifying therapies (Birnkrant et al., 2018b; Trout, Case, Clemens, et al., 2018).

## Medical Management

Treatment of the muscular dystrophies focuses on supportive care and prevention of complications in the absence of a cure (Birnkrant et al., 2018b). The goal of supportive management is to keep the patient active and functioning as normally as possible and to minimize functional deterioration. An individualized therapeutic exercise program is prescribed to prevent muscle tightness, contractures, and disuse atrophy. Night splints and stretching exercises are used to delay contractures of the joints, especially the ankles, knees, and hips. Braces may compensate for muscle weakness.

Spinal deformity is a severe problem. Weakness of trunk muscles and spinal collapse occur almost routinely in patients with severe neuromuscular disease. To help prevent spinal deformity, the patient is fitted with an orthotic jacket to improve sitting stability and reduce trunk deformity. This measure also supports cardiovascular status. In time, spinal fusion is performed to maintain spinal stability. Other procedures may be carried out to correct deformities.

Compromised pulmonary function may result either from progression of the disease or from deformity of the thorax secondary to severe scoliosis (Norris, 2019; Trout et al., 2018). Upper respiratory infections and fractures from falls must be vigorously treated in a way that minimizes immobilization because

joint contractures become worse when the patient's activities are more restricted than usual.

Other difficulties may manifest in relation to the underlying disease. Weakness of the facial muscles makes it difficult to attend to dental hygiene and to speak clearly, and impairs the ability to swallow safely (Birnkrant, Bushby, Bann, et al., 2018a). Gastrointestinal tract problems may include gastric dilation, rectal prolapse, and fecal impaction. Finally, cardiomyopathy appears to be a common complication in all forms of muscular dystrophy (Birnkrant et al., 2018a).

Genetic counseling is advised for parents and siblings of the patient because of the genetic nature of this disease. The Muscular Dystrophy Association (MDA) works to combat neuromuscular disease through research, programs of patient services and clinical care, and professional and public education (see the Resources section).

## Nursing Management

The goals of the patient and the nurse are to maintain function at optimal levels and to enhance the quality of life. Therefore, the patient's physical requirements, which are considerable, are addressed without losing sight of emotional and developmental needs. The patient and family are actively involved in decision making, including end-of-life decisions (see [Chapter 13](#)).

During hospitalization for treatment of complications, the knowledge and expertise of the patient and family members responsible for caregiving in the home are assessed. Because the patient and family caregivers often have developed caregiving strategies that work effectively for them, these strategies need to be acknowledged and accepted, and provisions must be made to ensure that they are maintained during hospitalization (Birnkrant et al., 2018b).

Families of adolescents and young adults with muscular dystrophy need assistance to shift the focus of care from pediatric to adult care and to understand the usual disease course (Lindsay, Cagliostro, & McAdam, 2019; Trout et al., 2018). Nursing goals include assisting the adolescent to make the transition to adult values and expectations while providing age-appropriate ongoing care. The nurse may need to help build the confidence of an older adolescent or adult patient by encouraging them to pursue job training to become economically independent. Other nursing interventions might include guidance in accessing adult health care and finding appropriate programs in sex education.

## Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

Management goals are addressed in special rehabilitation programs or in the patient's home and community. Therefore, the patient and family require information and education about the disorder, its anticipated course, and care and management strategies that will optimize the patient's growth and development and physical and psychological status. Members of a variety of health-related disciplines are involved in patient and family education; recommendations are communicated to all members of the health care team so that they may work toward common goals.

### Continuing and Transitional Care

Both the neuromuscular disease and the associated deformities may progress in adolescence and adulthood. Self-help and assistive devices can aid in maintaining maximum independence. These devices, recommended by physical and occupational therapists, often become necessary as more muscle groups are affected.

The family is instructed to monitor the patient for respiratory problems, because respiratory infection and cardiac failure are the most common causes of death. As respiratory difficulties develop, patients and their families need information regarding respiratory support. Options currently exist that can provide ventilatory support (e.g., negative-pressure devices, positive-pressure ventilators) while allowing mobility (Birnkrant et al., 2018a). Patients can remain relatively independent in a wheelchair, for example, while being maintained on a ventilator at home for many years.

The patient is encouraged to continue with range-of-motion exercises to prevent contractures, which are particularly disabling. Practical adaptations must be made, however, to cope with the effects of chronic neuromuscular disability. The patient at various stages of the disease may require a manual or an electric wheelchair, gait aids, upper and lower extremity and spinal orthotics, seating systems, bathroom equipment, lifts, ramps, and additional assistive devices, all of which require a team approach. The nurse assesses how the patient and family are managing, makes referrals, and coordinates the activities of the physical therapist, occupational therapist, and social services.

Patients who express concerns about increasing disability and dependence on others, as well as significant deterioration in health-related quality of life, benefit from an interprofessional approach to care (Trout et al., 2018). The patient is faced with a progressive loss of function, leading eventually to death. Feelings of helplessness and powerlessness are common. Each functional loss is accompanied by grief and mourning. The patient and family are assessed for depression, anger, or denial. The patient and family are assisted and encouraged to address decisions about end-of-life options before their need arises (see [Chapter 13](#)).

A psychiatric nurse clinician or other mental health professional may assist the patient to cope and adapt to the disease. By understanding and addressing

clinical concerns that are important for patients and their families, while at the same time assessing caregiver knowledge and burden during every encounter, the nurse provides a hopeful, supportive, and nurturing environment.

## Degenerative Disc Disease

Low back pain is the second most common neurologic disorder in the United States, with one quarter of all adults reporting recent (within 3 months) back pain (Hickey & Strayer, 2020). It is frequently associated with depression, anxiety, smoking, alcohol abuse, obesity, and stress; and it is the most common reason for missed work and decreased productivity while at work (Ramanathan, Hibbert, Wiles, et al., 2018). Low back pain results in significant economic costs to patients, their families, and society. Acute pain lasts less than 3 months, whereas chronic pain has a duration of 3 months or longer. Approximately 90% of patients with low back pain recover spontaneously within 4 to 6 weeks (Hickey & Strayer, 2020). See [Chapter 36, Chart 36-2](#) for further discussion of strategies to prevent acute low back pain.

## Pathophysiology

The intervertebral disc is a cartilaginous plate that forms a cushion between the vertebral bodies (see [Fig. 65-6A](#)). This tough, fibrous material is incorporated in a capsule. A ball-like cushion in the center of the disc is called the *nucleus pulposus*. In herniation of the intervertebral disc (ruptured disc), the nucleus of the disc protrudes into the annulus (the fibrous ring around the disc), with subsequent nerve compression (Norris, 2019). Protrusion or rupture of the nucleus pulposus usually is preceded by degenerative changes that occur with aging. Loss of protein polysaccharides in the disc decreases the water content of the nucleus pulposus. The development of radiating cracks in the annulus weakens resistance to nucleus herniation. After trauma (falls and repeated minor stresses such as lifting incorrectly), the cartilage may be injured.

For most patients, the immediate symptoms of trauma are short-lived, and those resulting from injury to the disc do not appear for months or years. Disc degeneration pushes the capsule back into the spinal canal. It could also rupture and allow the nucleus pulposus to be pushed back against the dural sac or against a spinal nerve as it emerges from the spinal column (see [Fig. 65-6B](#)). This disease of the spinal root produces pain and extreme sensitivity to touch due to radiculopathy (pressure in the area of distribution of the involved nerve endings). Continued pressure may produce degenerative changes in the involved nerve, such as changes in sensation and deep tendon reflexes.



## Gerontologic Considerations

Low back pain is commonly reported in older adults (Eliopoulos, 2018). Degenerative disc disease is also prevalent among older adults, but age alone should not exclude a patient from undergoing lumbar fusion if indicated (Badhiwala, Karmur, Hachem, et al., 2019). Researchers studied a group of 2238 patients ( $n = 1119$ , age  $<70$ ;  $n = 1119$ , age  $\geq 70$ ) who underwent spinal fusion. The groups were balanced for factors including sex, race, diabetes, hypertension, congestive heart failure, smoking, chronic steroid use, type of fusion, and number of levels (see later discussion). Rates of all complications were similar between younger and older age groups, except urinary tract infection, which was more frequent among the  $\geq 70$  age group (OR 2.32,  $p = .009$ ). Those in the older age group were more likely to be discharged to a rehabilitation or skilled care facility, rather than directly home (Badhiwala et al., 2019).

## Clinical Manifestations

A herniated disc with accompanying pain may occur in any portion of the spine: cervical, thoracic (rare), or lumbar. The clinical manifestations depend on the location, the rate of development (acute or chronic), and the effect on the surrounding structures. Functional limitations are the main manifestation reported by patients. Researchers also report that pain intensity in patients with low back pain has a direct effect on ADLs and sleep quality (Kose, Tastan, Temiz, et al., 2019).

## Assessment and Diagnostic Findings

A thorough health history and physical examination are important to rule out potentially serious conditions that may manifest as low back pain, including fracture, tumor, infection, or cauda equina syndrome (Hickey & Strayer, 2020).

The MRI scan has become the diagnostic tool of choice for localizing even small disc protrusions, particularly for lumbar spine disease. If the clinical symptoms are not consistent with the pathology seen on MRI, CT scanning and myelography are performed. A neurologic examination is carried out to determine whether reflex, sensory, or motor impairment from root compression is present and to provide a baseline for future assessment. Electromyography may be used to localize the specific spinal nerve roots involved. See [Chapter 36, Chart 36-1](#) for a summary of additional diagnostic studies that may be used to evaluate low back pain.

## Medical Management

Herniations of the cervical and the lumbar discs occur most commonly and are usually managed conservatively with medication and physical therapy or exercise (Hickey & Strayer, 2020). Surgery is sometimes necessary.

## Surgical Management

Surgical excision of a herniated disc is performed if there is evidence of a progressing neurologic deficit (muscle weakness and atrophy, loss of sensory and motor function, loss of sphincter control) and radicular pain (pain that follows the dermatomal distribution [see [Chapter 60, Fig. 60-9](#)] of the compressed nerve) that are unresponsive to conservative management. The goal of surgical treatment is to reduce the pressure on the nerve root to relieve pain and reverse neurologic deficits. Microsurgical techniques make it possible to remove only the amount of tissue that is necessary, which preserves the integrity of normal tissue better and imposes less trauma on the body. During these procedures, spinal cord function can be monitored electrophysiologically.

Some of the surgical techniques available include (Hickey & Strayer, 2020):

- *Microdiscectomy*: Removal of herniated or extruded fragments of intervertebral disc material
- *Laminectomy*: Removal of the bone between the spinal process and facet pedicle junction to expose the neural elements in the spinal canal; this allows the surgeon to inspect the spinal canal, identify and remove pathologic tissue, and relieve compression of the cord and roots
- *Hemilaminectomy*: Removal of part of the lamina and part of the posterior arch of the vertebra
- *Partial laminectomy or laminotomy*: Creation of a hole in the lamina of a vertebra
- *Discectomy with fusion*: Fusion of the vertebral spinous process with a bone graft (from iliac crest or bone bank), with the object of spinal fusion being to bridge over the defective disc to stabilize the spine and reduce the rate of recurrence
- *Foraminotomy*: Enlargement of the intervertebral foramen to increase the space for exit of a spinal nerve, resulting in reduced pain, compression, and edema

## Herniation of a Cervical Intervertebral Disc

The cervical spine is subjected to stresses that result from disc degeneration (due to aging, improper body mechanics) and **spondylosis** (degenerative changes occurring in a disc and adjacent vertebral bodies). Cervical disc

degeneration may lead to lesions that can cause damage to the spinal cord and its roots (Hickey & Strayer, 2020).

## Clinical Manifestations

A cervical disc herniation usually occurs at the C5–C6 or C6–C7 interspaces and compresses a unilateral nerve root (Hickey & Strayer, 2020). Pain and stiffness may occur in the neck, the top of the shoulders, and the region of the scapulae. Patients sometimes interpret these signs as symptoms of heart trouble or bursitis. Pain may also occur in the arm and hand, accompanied by **paresthesia** (numbness, tingling, or a “pins and needles” sensation) of the upper extremity. Cervical MRI usually confirms the diagnosis. Occasionally, the disc herniates centrally onto the spinal cord, causing Lhermitte syndrome, an electriclike shock sensation in the extremities or spine with neck flexion or straining, and myelopathy (bilateral arm and leg weakness). Myelopathy indicates compression of the spinal cord. The patient may exhibit a decrease in fine motor skills, difficulty with ambulation, difficulty with bowel and bladder control, and respiratory impairment if compression has occurred high in the cervical spine (Hemmer, 2018).

## Medical Management

The goals of treatment are to rest and immobilize the cervical spine to give the soft tissues time to heal and to reduce inflammation in the supporting tissues and the affected nerve roots in the cervical spine (Hickey & Strayer, 2020). It also reduces inflammation and edema in soft tissues around the disc, relieving pressure on the nerve roots. Proper positioning on a firm mattress may bring dramatic relief from pain.

The cervical spine may be rested and immobilized by a cervical collar, cervical traction, or a brace. A collar allows maximal opening of the intervertebral foramina and holds the head in a neutral or slightly flexed position. The patient may have to wear the collar 24 hours a day during the acute phase. The skin under the collar is inspected for irritation. After the patient is free of pain, cervical isometric exercises are started to strengthen the neck muscles.

## Pharmacologic Therapy

Analgesic agents (nonsteroidal anti-inflammatory drugs [NSAIDs], acetaminophen/oxycodone, or acetaminophen/hydrocodone) are prescribed during the acute phase to relieve pain, and sedative agents may be given to control the anxiety that is often associated with cervical disc disease. Muscle relaxants (cyclobenzaprine, methocarbamol, metaxalone) are prescribed for less than 1 week to interrupt muscle spasm and to promote comfort (Hickey &

Strayer, 2020). NSAIDs (aspirin, ibuprofen, naproxen) or corticosteroids are prescribed to treat the inflammation and swelling that usually occurs in the affected nerve roots and supporting tissues. Occasionally, a corticosteroid is injected into the epidural space for relief of radicular pain. NSAIDs are given with food to prevent gastrointestinal irritation (Comerford & Durkin, 2020). Hot, moist compresses (for 10 to 20 minutes) applied to the back of the neck several times daily increase blood flow to the muscles and help relax the patient and reduce muscle spasm.

## Surgical Management

Surgical excision of the herniated disc may be necessary if there is a significant neurologic deficit, progression of the deficit, evidence of cord compression, or pain that either worsens or fails to improve. A cervical discectomy, with or without fusion, may be performed to alleviate symptoms. An anterior surgical approach may be used through a transverse incision to remove disc material that has herniated into the spinal canal and foramina, or a posterior approach may be used at the appropriate level of the cervical spine. Potential complications with the anterior approach include carotid or vertebral artery injury, recurrent laryngeal nerve dysfunction, esophageal perforation, and airway obstruction (Hickey & Strayer, 2020). Complications of the posterior approach include damage to the nerve root or the spinal cord due to retraction or contusion of either of these structures, resulting in weakness of muscles supplied by the nerve root or cord.

Microsurgery, such as endoscopic microdiscectomy, may be performed in select patients through a small incision, using magnification techniques. This usually results in less tissue trauma and pain, and patients consequently have a shorter length of hospital stay compared with those who have conventional surgery (Hickey & Strayer, 2020).

## NURSING PROCESS

### The Patient Undergoing a Cervical Discectomy

#### Assessment

The patient is asked about past injuries to the neck, such as whiplash injury, because unresolved trauma can cause persistent discomfort, pain and tenderness, and symptoms of arthritis in the injured joint of the cervical spine. Assessment includes determining the onset, location, and radiation of pain and assessing for paresthesias, limited movement, and diminished function of the neck, shoulders, and upper extremities. It is important to determine whether the symptoms are bilateral; with large herniations, bilateral symptoms may be caused by cord compression. The area around the cervical spine is palpated to assess muscle tone and tenderness. Range of motion in the neck and shoulders is evaluated.

The patient is asked about any health issues that may influence the postoperative course and quality of life. It is also important to assess mood and stress levels. The nurse determines the patient's need for information about the surgical procedure and reinforces what the primary provider has explained. Strategies for pain management are discussed with the patient (Hickey & Strayer, 2020).

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Acute pain associated with the surgical procedure
- Impaired mobility associated with the postoperative surgical regimen
- Lack of knowledge about the postoperative course and home care management

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Hematoma at the surgical site, resulting in cord compression and neurologic deficit
- Recurrent or persistent pain after surgery

#### Planning and Goals

The goals for the patient may include relief of pain, improved mobility, increased knowledge and self-care ability, and prevention of complications.

#### Nursing Interventions

##### RELIEVING PAIN

Incisional pain is expected. Radicular pain improves over time as the nerve recovers. If the patient has had a bone fusion with bone removed from the iliac crest, considerable pain may be experienced at the donor site. Interventions consist of monitoring the donor site for hematoma formation, administering the prescribed postoperative analgesic agent, positioning for comfort, and reassuring the patient that the pain can be relieved. If the patient experiences a sudden increase in pain, extrusion of the graft may have occurred, requiring reoperation. A sudden increase in pain should be promptly reported to the surgeon (Lall, 2018).

The patient may experience a sore throat, hoarseness, and dysphagia due to temporary edema. These symptoms are relieved by throat lozenges, voice rest, and humidification. A pureed diet may be given if the patient has dysphagia.

#### **IMPROVING MOBILITY**

Postoperatively, a cervical collar (neck orthosis) may be worn, which contributes to limited neck motion and altered mobility. The patient is instructed to turn the body instead of the neck when looking from side to side. The neck should be kept in a neutral (midline) position. The patient is assisted during position changes to make sure that the head, shoulders, and thorax are kept aligned. When assisting the patient to a sitting position, the nurse supports the patient's neck and shoulders. To increase stability, the patient should wear shoes when ambulating. The patient is encouraged not to lift more than 10 pounds.

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

The patient is evaluated for bleeding and hematoma formation by assessing for swelling, excessive pressure in the neck, or severe pain in the incision area. The dressing is inspected for serosanguineous drainage, which suggests a dural leak. If this occurs, meningitis is a threat. A complaint of headache requires careful evaluation. Neurologic checks are made for swallowing deficits and upper and lower extremity weakness, because cord compression may produce rapid or delayed onset of paralysis (Hickey & Strayer, 2020). The patient who has had an anterior cervical discectomy is also assessed for a sudden return of radicular (spinal nerve root) pain, which may indicate instability of the spine (Hemmer, 2018).

Throughout the postoperative course, the patient is monitored frequently to detect any signs of respiratory difficulty, because retractors used during surgery may injure the laryngeal nerve, resulting in hoarseness and the inability to cough effectively and clear pulmonary secretions. In addition, the blood pressure and pulse are monitored to evaluate cardiovascular status and optimal circulation to the surgical site.

Bleeding at the surgical site and subsequent hematoma formation may occur. Severe localized pain not relieved by analgesic agents should be

reported. A change in neurologic status (motor or sensory function) should be reported promptly, because it suggests hematoma formation that may necessitate surgery to prevent irreversible motor and sensory deficits (Hickey & Strayer, 2020).

#### PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



**Educating Patients About Self-Care.** The patient's length of hospital stay is likely to be short; therefore, the patient and family should understand the care that is important for a smooth recovery. A cervical collar, if prescribed, is usually worn for about 6 weeks. The patient is educated in the use and care of the cervical collar. The patient will need to alternate tasks that involve minimal body movement (e.g., reading) with tasks that require greater body movement.

The patient is educated about strategies to manage the incision and to manage pain, and about signs and symptoms that may indicate complications that should be reported to the primary provider. The nurse assesses the patient's understanding of these management strategies, limitations, and recommendations. In addition, the nurse assists the patient in identifying strategies to cope with ADLs (e.g., self-care, child care) and minimize risks to the surgical site (see [Chart 65-6](#)). A discharge educational plan is developed collaboratively by members of the health care team to decrease the risk of recurrent disc herniation. Topics include those previously discussed as well as proper body mechanics, maintenance of optimal weight, proper exercise techniques, and modifications in activity.

**Continuing and Transitional Care.** The patient is instructed to see the primary provider at prescribed intervals so that the provider can document the disappearance of old symptoms and assess the range of motion of the neck. Recurrent or persistent pain may occur despite removal of the offending disc or disc fragments. Patients who undergo discectomy usually have consented to surgery after prolonged pain; they have often undergone repeated courses of ineffective conservative management and previous surgeries to relieve the pain. Therefore, the recurrence or persistence of symptoms postoperatively, including pain and sensory deficits, is often discouraging for the patient and family. The patient who experiences recurrence of symptoms requires emotional support and understanding. In addition, the patient is assisted in modifying activities and in considering options for subsequent treatment. The nurse educates the patient and family members about the need to participate in health-promotion and health-screening practices.

#### Evaluation

Expected patient outcomes may include:

1. Reports decreasing frequency and severity of pain

2. Demonstrates improved mobility
  - a. Demonstrates progressive participation in self-care activities
  - b. Identifies prescribed activity limitations and restrictions
  - c. Demonstrates proper body mechanics
3. Is knowledgeable about postoperative course, medications, and home care management
  - a. Lists the signs and symptoms to be reported postoperatively
  - b. Identifies dose, action, and potential side effects of medications
  - c. Identifies appropriate home care management activities and any restrictions
4. Has absence of complications
  - a. Reports no increase in incision pain or sensory symptoms
  - b. Demonstrates normal findings on neurologic assessment

## Herniation of a Lumbar Disc

Most lumbar disc herniations occur at the L5–S1 region (Hickey & Strayer, 2020). A herniated lumbar disc produces low back pain accompanied by varying degrees of sensory and motor impairment.

Chart 65-6



### HOME CARE CHECKLIST

## The Patient with Cervical Discectomy and Cervical Collar

**At the completion of education, the patient and/or caregiver will be able to:**

- Name the procedure that was performed and identify any permanent changes in anatomic structure or function as well as changes in ADLs, IADLs, roles, relationships, and spirituality.
- Identify interventions and strategies (e.g., durable medical equipment, adaptive equipment) used in adapting to any permanent changes in structure or function to promote safety and optimum functioning.
  - Use adequate mattress and chair support.
- State the name, dose, side effects, frequency, and schedule for all medications.
  - Describe nonpharmacologic interventions for pain relief used in conjunction to prescribed pain relief preparations.
- State how to obtain medical supplies and carry out dressing changes, wound care, and other prescribed regimens.
- State how to contact all members of the treatment team (e.g., health care providers, home care professionals, and durable medical equipment and supply vendor).
- Describe ongoing postoperative therapeutic regimen, including diet and activities to perform (e.g., exercises) and to limit or avoid (e.g., lifting, stair climbing, driving a car) during rehabilitation.
- Describe care for the surgical incision site.
  - Keep staples or sutures clean and dry and cover with dry dressing.
- Demonstrate proper body mechanics and prescribed exercise techniques.
  - Describe how to modify activity for optimum functioning.
  - Avoid sitting or standing for more than 30 minutes.
  - Avoid twisting, flexing, extending, or rotating the neck.
  - Avoid sleeping in a prone position or the use of pillows to minimize neck flexion in bed; keep head in a neutral position.
  - Wear low-heeled shoes.
  - Place a wrinkle-free silk scarf under the collar to increase comfort.
  - **For men:** Shave without twisting or moving the neck. This may be done with help while lying flat or sitting. Remove only the front part of the collar for shaving.
- Practice stress reduction and relaxation techniques.
- Care of the cervical collar:
  - Wear the collar at all times until directed otherwise by the primary provider.
  - Wash the neck under the collar twice a day with mild soap.
  - Keep the neck still while the collar is open.

- With the assistance of a helper, wash the neck in steps.
- Lie flat and supine.
- Open the Velcro tabs on each side of the collar and remove its front portion.
- Gently wash and dry the neck.
- Replace the front part of the collar and refasten the tabs.
- Turn to one side with a thin pillow under the head.
- Open one tab.
- Gently wash and dry the back of the neck. Refasten the tab.
- Turn to the other side and wash and dry this side. Refasten the tab.
- Notify primary provider if any signs or symptoms of infection occur, such as fever, redness or irritation, drainage, increased pain.
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.
- Identify sources of support for patient and caregivers (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families.
- Identify the need for health promotion, disease prevention, and screening activities.

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

## Clinical Manifestations

The patient complains of low back pain with muscle spasm and **sciatica** (pain and tenderness that radiates along the sciatic nerve that runs through the thigh and leg). Pain is aggravated by actions that increase intraspinal fluid pressure, such as bending, lifting, or straining (as in sneezing or coughing), and usually is relieved by bed rest. There is often some type of postural deformity, because pain causes an alteration of the normal spinal mechanics. If the patient lies on the back and attempts to raise a leg in a straight position, pain radiates into the leg; this maneuver, called the *straight-leg raising test*, stretches the sciatic nerve. Additional signs include muscle weakness, alterations in tendon reflexes, and sensory loss.

## Assessment and Diagnostic Findings

The diagnosis of lumbar disc disease is based on the history and physical findings, specifically the location, quality, severity of pain, and the use of imaging techniques such as MRI and CT scans as well as myelography.

## Medical Management

The objectives of treatment are to relieve pain, slow disease progression, and increase the patient's functional ability. Bed rest is discouraged because it may weaken muscles, but activities that exacerbate pain should be avoided.

Because muscle spasm is prominent during the acute phase, muscle relaxants are used. NSAIDs and systemic corticosteroids may be given to counter the inflammation and swelling that usually occurs in the supporting tissues and the affected nerve roots. Moist heat and massage help relax muscles. Strategies for increasing the patient's functional ability include weight reduction, physical therapy, and biofeedback. Exercises, prescribed by physical therapy, can help strengthen back muscles and decrease pain (Hickey & Strayer, 2020). See [Chapter 9](#) for descriptions of nursing interventions for the patient with pain.

## Surgical Management

In the lumbar region, surgical treatment includes lumbar disc excision through a posterolateral laminotomy and the techniques of microdiscectomy and percutaneous discectomy. In microdiscectomy, an operating microscope is used to visualize the offending disc and compressed nerve roots; it permits a small incision (2.5 cm [1 inch]) and minimal blood loss and takes about 30 minutes of operating time. In general, the length of hospital stay is short, and the patient makes a rapid recovery. Several minimally invasive techniques in spinal surgery have led to improved patient outcomes and lower hospital costs (Hickey & Strayer, 2020).

A patient undergoing a disc procedure at one level of the vertebral column may have a degenerative process at other levels. A herniation relapse may occur at the same level or elsewhere, so the patient may become a candidate for another disc procedure. Arachnoiditis (inflammation of the arachnoid membrane) may occur after surgery (and after myelography); it involves an insidious onset of diffuse, frequently burning pain in the lower back, radiating into the buttocks. Disc excision can leave adhesions and scarring around the spinal nerves and dura, which then produce inflammatory changes that create chronic neuritis and neurofibrosis. Disc surgery may relieve pressure on the spinal nerves, but it does not reverse the effects of neural injury and scarring and the pain that results. Failed disc syndrome (recurrence of sciatica after lumbar discectomy) remains a cause of disability (Hickey & Strayer, 2020).

## Nursing Management

### Providing Preoperative Care

Most patients fear surgery on any part of the spine and therefore need explanations about the surgery and reassurance that it will not weaken the back. When data are being collected for the health history, any reports of pain,

paresthesia, or muscle spasm are recorded to provide a baseline for comparison after surgery. Health issues that may influence the postoperative course and quality of life (e.g., fatigue, mood, stress, patient expectations, smoking) are important to assess. Preoperative assessment also includes an evaluation of movement of the extremities as well as bladder and bowel function (Hickey & Strayer, 2020). To facilitate the postoperative turning procedure, the patient is instructed to turn as a unit (called logrolling) as part of the preoperative preparation. Before surgery, the patient is also encouraged to take deep breaths, cough, and perform muscle setting exercises to maintain muscle tone.

Research suggests that the use of motivational interviewing helps build self-confidence with self-care management of symptoms in the postoperative period (Scheffel, Amidei, & Fitzgerald, 2019). See the Nursing Research Profile in [Chart 65-7](#).

### Assessing the Patient After Surgery

After lumbar disc excision, vital signs are checked frequently and the wound is inspected for hemorrhage, because vascular injury is a complication of disc surgery. Because postoperative neurologic deficits may occur from nerve root injury, the sensation and motor strength of the lower extremities are evaluated at specified intervals, along with the color and temperature of the legs and sensation of the toes. It is important to assess for urinary retention, which is another sign of neurologic deterioration (Hickey & Strayer, 2020). In discectomy with fusion, the patient has an additional surgical incision if bone fragments were taken from the iliac crest or fibula to serve as wedges in the spine. The recovery period is longer for patients who undergo discectomy with spinal fusion, because bony union must take place.

### Positioning the Patient

To position the patient, a pillow is placed under the head, and the knee rest is elevated slightly to relax the back muscles. When the patient is lying on one side, however, extreme knee flexion must be avoided. The patient is encouraged to move from side to side to relieve pressure and is reassured that no injury will result from moving. When the patient is ready to turn, the bed is placed in a flat position and a pillow is placed between the patient's legs. The patient turns as a unit (logrolls) without twisting the back.

Chart 65-7



NURSING RESEARCH PROFILE

## **Improving Confidence with Self-Care Management**

Scheffel, K., Amidei, C., & Fitzgerald, K. A. (2019). Motivational interviewing: Improving confidence with self-care management in postoperative thoracolumbar spine patients. *Journal of Neuroscience Nursing*, 51(3), 113–117.

### **Purpose**

Patients undergoing spine surgery often lack confidence in self-care management of symptoms such as pain, lack of sleep, depression, and immobility. The purpose of this study was to examine whether a targeted motivational interview would improve confidence with self-care management of symptoms following spine surgery.

### **Design**

This pilot study used a quasi-experimental, one group, pretest–posttest design with 15 participants who were undergoing spine surgery. The two main instruments used to gather data included the 10-item Oswestry Disability Index (ODI) and the Health Confidence Index (HCI).

### **Findings**

Paired sample *t*-tests of the pre- and postintervention scores on both the ODI and HCI showed statistically significant differences. The HCI showed a statistically significant increase in scores with mean preintervention scores of 6.73 ( $SD = 2.12$ ) and mean postintervention scores of 8.73 ( $SD = 1.43$ ) indicating a significant increase in confidence in self-care of symptom-related disability.

### **Nursing Implications**

Motivational interviewing is an effective strategy for implementing health promoting behaviors. This study adds evidence that motivational interviewing is a strategy nurses can use to improve patients' confidence in self-care management of symptoms following spine surgery.

To get out of bed, the patient lies on one side while pushing up to a sitting position. At the same time, the nurse or family member eases the patient's legs over the side of the bed. Coming to a sitting or standing posture is accomplished in one long, smooth motion. Most patients walk to the bathroom on the same day as the surgery. Sitting is discouraged except for defecation.

## **Promoting Home, Community-Based, and Transitional Care**



Educating Patients About Self-Care

The patient is instructed to increase activity gradually, as tolerated, because it takes up to 6 weeks for the ligaments to heal. Excessive activity may result in spasm of the paraspinal muscles (Hickey & Strayer, 2020).

Activities that produce flexion strain on the spine (e.g., driving a car) should be avoided until healing has taken place. Heat may be applied to the back to relax muscle spasms. Scheduled rest periods are important, and the patient is advised to avoid heavy work for 2 to 3 months after surgery. Exercises are prescribed to strengthen the abdominal and erector spinal muscles. A back brace or corset may be necessary if back pain persists.

### **Continuing and Transitional Care**

Referral for inpatient or outpatient rehabilitation may be warranted to improve self-care abilities after medical or surgical treatment for herniation of a lumbar disc. A home care referral may be indicated and provides the nurse with the opportunity to assess the patient's physical and psychological status, as well as their ability to adhere to recommended management strategies. During the home visit, the nurse determines whether changes in neurologic function have occurred. The adequacy of pain management is assessed, and modifications are made to ensure adequate pain relief (Hickey & Strayer, 2020).

## **Post-Polio Syndrome**

Polio has mostly been eradicated globally due to concerted vaccination efforts. However, people who survived the polio epidemic of the 1940s and 1950s, many of whom are now older adults, are developing new symptoms of weakness, fatigue, and musculoskeletal pain identified as PPS. This phenomenon, which occurs at least 15 years after the polio exposure, affects 15 to 20 million people worldwide (Shing, Chipika, Finegan, et al., 2019). Men and women appear to be equally at risk for this condition.

## **Pathophysiology**

The exact cause of PPS is not known, but researchers suspect that with aging or muscle overuse, the neurons not destroyed by the poliovirus continue generating axon sprouts (Shing et al., 2019). These new terminal axon sprouts reinnervate the affected muscles after the initial insult but may become more vulnerable as the body ages.

## **Assessment and Diagnostic Findings**

No specific diagnostic test exists for PPS. Clinical diagnosis is made on the basis of the history and physical examination and exclusion of other medical

conditions that could be causing the new symptoms. Patients report a history of paralytic poliomyelitis followed by partial or complete recovery of function, with a plateau of function and then the recurrence of symptoms. Signs and symptoms may occur decades after the original onset of poliomyelitis (Shing et al., 2019).

## Management

No specific medical or surgical treatment is available for this syndrome, and therefore nurses play a pivotal role in the team approach to assisting patients and families in dealing with the symptoms of progressive loss of muscle strength and significant fatigue (Shing et al., 2019; Pastuszak, Stepien, Tomeczykiewicz, et al., 2017). Pain and weakness may be improved with infusion of intravenous immunoglobulin (Shing et al., 2019).



For the procedural guidelines for managing immunoglobulin therapy, go to [thepoint.lww.com/Brunner15e](http://thepoint.lww.com/Brunner15e).

Nursing interventions are aimed at maintaining the patient's strength as well as physical, psychological, and social well-being. Other health care professionals who may assist in patient care include physical, occupational, speech, and respiratory therapists; social workers; and chaplains.

The patient needs to plan and coordinate activities to conserve energy and reduce fatigue. Rest periods should be planned and assistive devices used to reduce weakness and fatigue. Important activities should be planned for the morning, because fatigue often increases in the afternoon and evening.

Pain in muscles and joints may be a problem. Nonpharmacologic techniques such as the application of heat and cold are appropriate, because older adults may not tolerate or may react to medications, particularly when they are taking multiple medications (Eliopoulos, 2018).

Maintaining a balance between adequate nutritional intake and avoiding excess calories that can lead to obesity is a challenge in this group of patients who are sedentary. Pulmonary hygiene and adequate fluid intake can help with airway management. Several interventions can improve sleep, including limiting caffeine intake before bedtime and assessing for nocturia. The patient may need to be evaluated for obstructive sleep apnea. Supportive ventilation may be appropriate, with continuous positive airway pressure if sleep apnea is a problem (see Chapter 18).

Bone density testing in patients with PPS has revealed low bone mass and osteoporosis. Therefore, the importance of identifying risks, preventing falls,

and treating osteoporosis must be discussed with patients and families. Families also need to be made aware of the possibility of changes in individual and family relationships due to the many symptoms of PPS. The nurse also needs to remind patients and family members of the need for health-promotion activities and health screening (Shing et al., 2019).

### CRITICAL THINKING EXERCISES

**1  ebp** A 64-year-old man has been admitted to the unit where you work after having a seizure and has been newly diagnosed with a brain tumor. Identify the evidence-based practices for the management of brain tumors. Describe the evidence base for the practices that you have identified and the criteria used to evaluate the strength of that evidence. Identify the health-promotion activities you would recommend to this patient and the rationale for your recommendations.

**2  pq** You are caring for a 55-year-old woman newly diagnosed with Parkinson's disease. Assess and prioritize the patient's physiologic and psychosocial needs. What nursing interventions and actions would you suggest to assist in managing the treatment of and coping with Parkinson's disease? How would this be different if the patient lives alone?

**3  ipc** You are participating in morning rounds on the surgery unit where you work. The team is discussing one of the patients who has been assigned to you for the day, a 75-year-old man admitted yesterday following a lumbar disc excision. What members of the interprofessional care team should be included in the care of this patient? How will you, as this patient's nurse, facilitate an interprofessional discussion to assist with his discharge?

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## Resources

- ALS Association, [www.alsa.org](http://www.alsa.org)  
American Association of Neuroscience Nurses, [www.aann.org](http://www.aann.org)  
American Brain Tumor Association, [www.abta.org](http://www.abta.org)  
American Parkinson Disease Association, [www.apdaparkinson.org](http://www.apdaparkinson.org)  
Family Caregiver Alliance, [www.caregiver.org](http://www.caregiver.org)  
Huntington's Disease Society of American, [www.hdsa.org](http://www.hdsa.org)  
Muscular Dystrophy Association, [www.mda.org](http://www.mda.org)  
National Brain Tumor Society, [www.braintumor.org](http://www.braintumor.org)  
Parkinson's Foundation, [www.parkinson.org](http://www.parkinson.org)  
Spinal Cord Tumor Association, INC, [www.spinalcordtumor.org](http://www.spinalcordtumor.org)  
The Michael J. Fox Foundation, [www.michaeljfox.org](http://www.michaeljfox.org)