CHAPTER 44 Nursing Care of Clients with Intracranial Disorders

LEARNING OUTCOMES

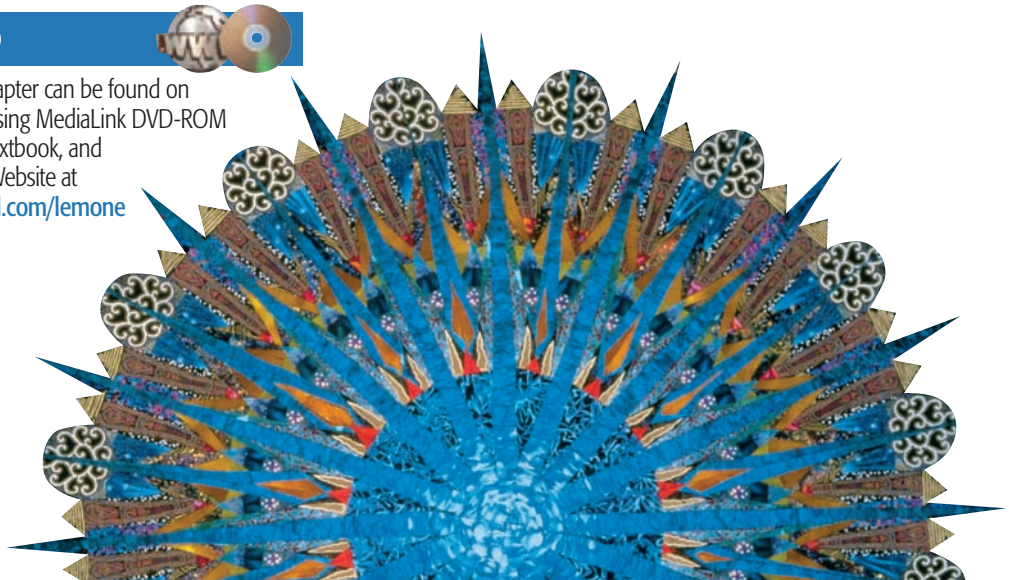
- Compare and contrast the pathophysiology, manifestations, interdisciplinary care, and nursing care of clients with alterations in level of consciousness and increased intracranial pressure.
- Explain the pathophysiology, manifestations, complications, interdisciplinary care, and nursing care of intracranial disorders, including headaches, epilepsy, traumatic brain injury, central nervous system infections, and brain tumors.
- Describe criteria for diagnosing persistent vegetative state and brain death.
- Discuss the purposes, nursing implications, and health education for the client and family for medications used to treat altered cerebral function, headaches, epilepsy, traumatic brain injury, central nervous system infections, and brain tumors.
- Discuss surgical options for the treatment of increased intracranial pressure, epilepsy, traumatic brain injury, and brain tumors.

CLINICAL COMPETENCIES

- Assess functional status of clients with intracranial disorders and monitor, document, and report abnormal manifestations.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualized nursing interventions for clients with intracranial disorders.
- Administer oral and injectable medications used to treat intracranial disorders knowledgeably and safely.
- Provide skilled care to clients having intracranial pressure monitoring, tonic-clonic seizures, and intracranial surgery.
- Integrate intradisciplinary care into care of clients with intracranial disorders.
- Provide appropriate teaching and evidence-based practice to facilitate community-based care to promote safety and prevent injury, and to provide information and support necessary for long-term care of clients with intracranial disorders.
- Revise plan of care as needed to provide effective interventions to promote, maintain, or restore functional health status to clients with intracranial disorders.

MEDIALINK

Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD-ROM accompanying this textbook, and on the Companion Website at http://www.prenhall.com/lemone



KEY TERMS

brain death, *1532*
cerebral edema, *1537*
concussion, *1559*
consciousness, *1529*
encephalitis, *1565*
epidural hematoma, *1558*
epilepsy, *1547*
hydrocephalus, *1537*
increased intracranial pressure (IICP), *1535*
locked-in syndrome, *1532*
meningitis, *1564*
persistent vegetative state, *1532*
seizures, *1547*
subdural hematoma, *1558*
traumatic brain injury (TBI), *1554*

The client with an intracranial disorder presents a unique nursing challenge. Problems the client experiences in the acute stage of the disorder are often a prelude to long-term problems requiring ongoing management. These long-term problems range from alterations in the body's basic functioning to dysfunctions in the complex processes of the human mind. Systemic problems may accompany or develop secondary to an intracranial disorder. Intracranial disorders may affect both the client's quality of life and that of the client's family. This chapter first discusses altered level of consciousness and increased intracranial pressure, followed by intracranial disorders that may manifest these and other health problems. Information specific to the client with a stroke is provided in Chapter 45 ∞.

ALTERED CEREBRAL FUNCTION

The manifestations of altered cerebral function occur as a result of illness or injury. Assessment of the patterns of those manifestations helps determine the extent of the cerebral dysfunction and improvement or deterioration of cerebral function. Except in the case of direct damage to the brainstem and reticular activating system (RAS), brain function deterioration usually follows a predictable progression, that is, a pattern in which higher levels of function are impaired initially, progressing to impairment of more primitive functions. Altered level of consciousness (LOC) and behavior changes are early manifestations of the deterioration of the function of the cerebral hemispheres. Structures in the midbrain and brainstem are affected sequentially, with characteristic changes in LOC; patterns of respiration, pupillary, and oculomotor responses; and motor function. Manifestations of progressive deterioration of cerebral function are outlined in Table 44–1.

TABLE 44–1 **Progression of Deteriorating Brain Function**

LEVEL OF CONSCIOUSNESS	PUPILLARY RESPONSE	OCULOMOTOR RESPONSES	MOTOR RESPONSES	BREATHING
Alert; oriented to time, place, and person	Brisk and equal; pupils regular	Eyes move as head turns Caloric testing (ear irrigation) produces nystagmus	Purposeful movement; responds to commands	Regular pattern with normal rate and depth
Responds to verbal stimuli; decreased concentration; agitation, confusion, lethargy; disoriented	Small and reactive	Roving eye movements; doll's eyes positive, with gaze fixed straight ahead; eye deviation away from cold caloric stimulus and toward warm stimulus	Purposeful movement in response to pain stimulus	Yawning, sighing respirations
Requires continuous stimulation to rouse			Decorticate posturing with upper extremity flexion	Cheyne-Stokes respirations with crescendo–decrescendo pattern in rate and depth followed by period of apnea
Reflexive positioning to pain stimulus	Pupils fixed (nonreactive) in midposition	Caloric testing produces nystagmus	Decerebrate posturing with adduction and rigid extension of upper and lower extremities	Central neurogenic hyperventilation with rapid, regular, and deep respirations; apneustic breathing with prolonged inspiration and pauses at full inspiration and following expiration
No response to stimuli	Pupils fixed in midposition	No spontaneous eye movement or nystagmus	Extension of upper extremities with flexion of lower extremities; flaccidity	Cluster or ataxic breathing with irregular pattern and depth of respirations; gasping respirations or apnea

THE CLIENT WITH ALTERED LEVEL OF CONSCIOUSNESS

Consciousness is a condition in which the person is aware of self and environment and is able to respond appropriately to stimuli. Full consciousness requires both normal arousal and full cognition.

- *Arousal*, or alertness, depends on the RAS, a diffuse system of neurons in the thalamus and upper brainstem.
- *Cognition* is a complex process involving all mental activities controlled by the cerebral hemispheres, including thought processes, memory, perception, problem solving, and emotion.

These two components of consciousness depend on the normal physiologic functions of and connections between the arousal mechanisms of the reticular formation and the cognitive functions of the cerebral hemispheres. Because arousal and cognition are independent components of consciousness, each can act separately on stimuli. For example, the RAS reacts to the discomfort caused by a full bladder by waking the person in the middle of the night. Once awake, however, the frontal cortex alerts the person that the bladder is full and prompts the person to go to the bathroom and empty it.

Conditions that affect either the RAS or the function of the cerebral hemispheres can interfere with the normal level of consciousness. Terms describing altered LOC are listed and defined in Table 44–2. Nurses should remember that consciousness is a dynamic state: A client may pass from full consciousness to coma within hours or experience a slow diminishment of consciousness that does not become evident for weeks or months. The nurse can help provide effective care for a client with an altered LOC by looking beyond the diagnostic labels of consciousness and accurately assessing the client's behavior and response to stimuli.

TABLE 44–2 **Terms Used to Describe Level of Consciousness**

TERM	CHARACTERISTICS OF CLIENT
Full consciousness	Alert; oriented to time, place, and person; comprehends spoken and written words
Confusion	Unable to think rapidly and clearly; easily bewildered, with poor memory and short attention span; misinterprets stimuli; judgment is impaired
Disorientation	Not aware of or not oriented to time, place, or person
Obtundation	Lethargic, somnolent; responsive to verbal or tactile stimuli but quickly drifts back to sleep
Stupor	Generally unresponsive; may be briefly aroused by vigorous, repeated, or painful stimuli; may shrink away from or grab at the source of stimuli
Semicomatose	Does not move spontaneously; unresponsive to stimuli, although vigorous or painful stimuli may result in stirring, moaning, or withdrawal from the stimuli, without actual arousal
Coma	Unarousable; will not stir or moan in response to any stimulus; may exhibit nonpurposeful response (slight movement) of area stimulated but makes no attempt to withdraw
Deep coma	Completely unarousable and unresponsive to any kind of stimulus, including pain; absence of brainstem reflexes, corneal, papillary, and pharyngeal reflexes and tendon and plantar reflexes

Pathophysiology

Level of consciousness may be altered by processes that affect the arousal functions of the brainstem, the cognitive functions of the cerebral hemispheres, or both. The major causes are (1) lesions or injuries that affect the cerebral hemispheres directly and widely or that compress or destroy the neurons of the RAS and (2) metabolic disorders.

Arousal and Cognition

The physiologic seat of consciousness, the reticular formation, is a mass of nerve cells and fibers that make up the core of the brainstem, extending from the medulla to the midbrain. The axons of reticular neurons are exceptionally long and branch outward to cells in the hypothalamus, thalamus, cerebellum, and spinal cord. A system of reticular neurons within the RAS passes steady streams of impulses through thalamic relays in order to stimulate the cerebral cortex into wakefulness. The body's sensory tracts interact with RAS neurons; this interrelationship helps control the strength of the RAS's rousing effect on the cerebrum.

Damage to the RAS impairs the person's ability to maintain wakefulness and arousal. Stroke is the most common cause of RAS destruction. Other causes include demyelinating diseases such as multiple sclerosis, tumors, abscesses, and head injury. Function of the RAS may be suppressed by compression of the brainstem, which produces edema and ischemia. Pressure and compression of the brainstem may be due to tumors, increased intracranial pressure, hematomas or hemorrhage, or aneurysm. Although it is possible to assess LOC or arousal in the client with RAS damage, the impairment in arousal may make it impossible to assess cognitive function.

The function of the brain, especially the cerebral hemispheres, depends on continuous blood flow with unimpeded supplies of oxygen and glucose. Processes that disrupt this flow of blood and nutrients may cause widespread damage to the cerebral hemispheres, impairing arousal and cognition. Bilateral hemispheric lesions (such as global ischemia), or metabolic disorders (such as hypoglycemia), are the most common causes of altered LOC related to cerebral dysfunction of the hemispheres. Localized masses, such as a hematoma or cerebral edema, that displace normal structures and cause direct or indirect pressure on the opposite hemisphere or brainstem can also affect LOC. The client who has widespread damage to the cerebral hemispheres but an intact RAS has sleep–wake cycles and may rouse in response to stimuli; the client cannot be said to be alert, however, because cognition is impaired.

Both localized neurologic processes and systemic disorders can alter LOC. Processes occurring within the brain, which

may directly destroy or compress neurologic structures, include the following:

- Increased intracranial pressure
- Stroke
- Hematoma
- Intracranial hemorrhage
- Tumors
- Infections
- Injury from excitatory amino acids
- Demyelinating disorders.

Any systemic condition that affects the delivery of blood, oxygen, and glucose to the brain or alters cell membranes may also alter LOC. If cerebral blood flow is impaired or the client becomes hypoxic or hypoglycemic, cerebral metabolism is impaired and level of consciousness declines rapidly. Severe hypoxia quickly leads to ischemia. Ischemia may be focal (for example, following a stroke) or global (as from cardiac arrest or hypovolemic shock). Widespread global ischemia causes almost immediate unconsciousness (Porth, 2005). Clients at particular risk include those with poorly controlled diabetes and those with cardiac or respiratory failure.

Other metabolic alterations that can affect LOC include fluid and electrolyte imbalances, such as hyponatremia or hyperosmolality, and acid–base alterations, such as hypercapnia (an elevated arterial carbon dioxide level). Accumulated waste products and toxins from liver or renal failure can affect neuronal and neurotransmitter function, altering LOC. Drugs that depress the central nervous system (e.g., alcohol, analgesics, anesthetics) suppress metabolic and membrane activities in the RAS and cerebral hemispheres, thereby affecting LOC. Glutamate, the main excitatory neurotransmitter in the brain, may accumulate during prolonged ischemia, resulting in acute glutamate toxicity and cell death.

Seizure activity, with abnormal electrical discharges from a local area of the brain or from the entire brain, commonly affects LOC. It appears that the spontaneous, disordered discharge of activity that occurs during a seizure exhausts energy metabolites or produces locally toxic molecules, altering LOC for a time after the seizure. Consciousness returns when the metabolic balance of the neurons is restored.

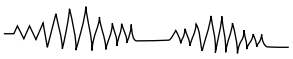
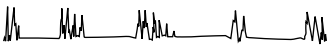
As the impairment of brain function progresses, more stimuli are required to elicit a response from the client. Initially, the client may rouse to verbal stimuli and respond appropriately to questions, remaining oriented to time, place, and person. With deterioration of neurologic function, the client becomes more difficult to rouse and may become agitated and confused when awakened. Orientation to time is lost initially, followed by orientation to place and then to person. Continuous stimulation or vigorous shaking is required to maintain wakefulness as LOC decreases. Eventually, the client does not respond, even with deep painful stimuli.

Patterns of Respirations

Progressive impairment of neural function also causes predictable changes in respiratory patterns as respiratory centers are affected. In normal respirations, a rhythmic pattern is maintained by neural centers in the pons and medulla that respond to changes in arterial levels of oxygen (Pa_{O_2}) and carbon dioxide (Pa_{CO_2}). When there is damage to the RAS or cerebral hemispheres, neural control of these centers is lost, and lower brainstem centers regulate breathing patterns by responding only to changes in Pa_{CO_2}, resulting in irregular respiratory patterns. The initial manifestations of deteriorating brain function are yawning and sighing. As outlined in Table 44–1 and illustrated in Table 44–3, progressive deterioration in brain function is accompanied by decreasing LOC and changes in breathing patterns. The type of respirations, by area of cerebral damage, are as follows:

- Diencephalon: *Cheyne-Stokes respirations* (alternating regular periods of deep, rapid breathing followed by periods of apnea)
- Midbrain: *neurogenic hyperventilation* (may exceed 40 per minute), the result of uninhibited stimulation of the respiratory centers
- Pons: *apneustic respirations*, characterized by sighing on midinspiration or prolonged inhalation and exhalation; results from excessive stimulation of the respiratory centers
- Medulla: *ataxic/apneic respirations* (totally uncoordinated and irregular), probably as a result of the loss of responsiveness to CO_2.

TABLE 44–3 Breathing Patterns Characteristic of Altered Level of Consciousness

PATTERN		DESCRIPTION
Cheyne-Stokes respirations		A regular crescendo–decrescendo pattern with increasing then decreasing rate and depth of respirations followed by a period of apnea
Central neurogenic hyperventilation		A sustained pattern of rapid, regular, deep respirations (hyperapnea)
Apneustic breathing		Prolonged inspiration with a pause at full inspiration followed by expiration and a possible pause following expiration
Cluster breathing		Clusters of several breaths with irregular periods of apnea between clusters
Ataxic respirations		Respirations that are completely irregular in pattern and depth with irregular periods of apnea

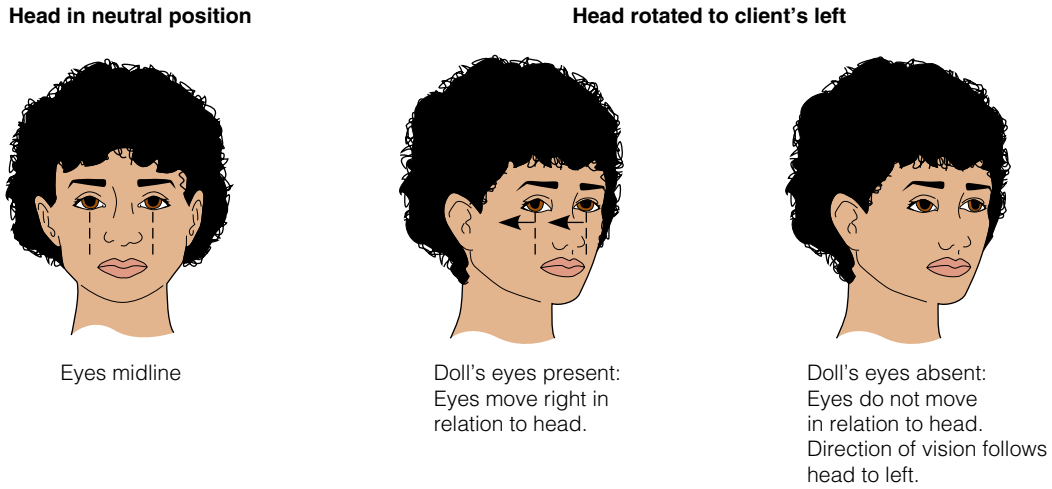


Figure 44–1 ■ Doll's eye movements characteristic of altered LOC.

Pupillary and Oculomotor Responses

The brainstem areas that control arousal are adjacent to areas that control the pupils. A predictable progression of pupillary and oculomotor responses occurs as level of consciousness deteriorates toward coma (see Table 44–1). If the lesion or process affecting neurologic function is localized, effects may initially be seen in the ipsilateral pupil (the pupil on the same side as the lesion). With generalized or systemic processes, pupils are affected equally. If the pupils are small and equally reactive, metabolic processes affecting LOC may be present. With compression of cranial nerve III at the midbrain, the pupils may become oval or eccentric (off center). As the level of functional impairment progresses, the pupils become fixed (unresponsive to light) and, eventually, dilated.

In deteriorating LOC and coma, spontaneous eye movement is lost and reflexive ocular movements are altered. Normally, both eyes move simultaneously in the same direction; injury to the cranial nerve nuclei in the midbrain and pons can impair normal movement. *Doll's eye movements* are reflexive movements of the eyes in the opposite direction of head rotation; they are an indicator of brainstem function (Figure 44–1 ■). As a result of the oculocephalic reflex, the eyes move upward with passive flexion of the neck and downward with passive neck extension. As brainstem function deteriorates, this reflex is lost. The eyes fail to turn together and, eventually, remain fixed in the midposition as the head is turned.

Motor Responses

The level of brain dysfunction and the side of the brain affected may be assessed by motor responses. These responses are the most accurate identifier of changes in mental status. In altered LOC, motor responses to stimuli range from an appropriate response to a command (e.g., "squeeze my hand" or "push my hands away with your feet") to flaccidity (see Table 44–1). Initially, the client may be able to move purposefully away from a noxious stimulus, for example, to brush the examiner's hand away from the face. As function declines, movements become more generalized (withdrawal, grimacing) and less purposeful. Reflexive motor responses may occur, including decorticate posturing with flexion of the upper extremities accompanied by extension of the lower extremities. With further decline, decerebrate posturing is seen, with adduction and rigid extension of the upper and lower extremities. Without intervention, the client eventually becomes flaccid, with little or no motor response to stimuli.

Coma States and Brain Death

Possible outcomes of altered LOC and coma include full recovery with no long-term residual effects, recovery with residual damage (such as learning deficits, emotional difficulties, or impaired judgment), or more severe consequences such as persistent vegetative state (cerebral death) or brain death. Resources for families are listed in Box 44–1.

BOX 44–1 Organizations Providing Information for Families of Clients in a Coma

Coma Recovery Association
807 Carman Avenue
Westbury, NY 11590
Tel: 516-997-1826

Brain Injury Association of America, Inc.
8201 Greensboro Drive, Suite 611
McLean, VA 22102
FamilyHelpline@blausa.org
Tel: 800-444-6443

Brain Trauma Foundation
523 East 72nd Street, 8th Floor
New York, NY 10021
www.braintrauma.org
Tel: 212-772-0608

Family Caregiver Alliance/National Center on Caregiving
180 Montgomery Street, Suite 1100
San Francisco, CA 94104
Tel: 800-445-8106

PERSISTENT VEGETATIVE STATE **Persistent vegetative state** (also called irreversible coma) is a permanent condition of complete unawareness of self and the environment and loss of all cognitive functions. Usually the result of severe brain trauma or global ischemia, this condition results from death of the cerebral hemispheres with continued function of the brainstem and cerebellum. While the homeostatic regulatory functions of the brain continue, the ability to respond meaningfully to the environment is lost. The diagnosis of persistent vegetative state requires that the condition has continued for at least 1 month (Porth, 2005).

The client has sleep–wake cycles and retains the ability to chew, swallow, and cough but cannot interact with the environment. When awake, the eyes may wander back and forth across the room, but they cannot track an object or person. In a minimally conscious state, the client is aware of the environment and can follow simple commands, manipulate objects, gesture or verbalize to indicate "yes/no" responses, and make meaningful movements (such as blinking or smiling) in response to a stimulus. With appropriate supportive care, the client may remain in this state for years.

LOCKED-IN SYNDROME **Locked-in syndrome** is distinctly different from persistent vegetative state in that the client is alert and fully aware of the environment and has intact cognitive abilities, but is unable to communicate through speech or movement because of blocked efferent pathways from the brain. Motor paralysis affects all voluntary muscles, although the upper cranial nerves (I through IV) may remain intact, allowing the client to communicate through eye movements and blinking. In essence, the client is "locked" inside a paralyzed body while remaining fully conscious of self and environment. Infarction or hemorrhage of the pons that disrupts outgoing nerve tracts but spares the RAS is the usual cause of locked-in syndrome. This condition may also result when the corticospinal tracts between the midbrain and pons are interrupted. Disorders of the lower motor neurons or muscles, such as acute polyneuritis, myasthenia gravis, or amyotrophic lateral sclerosis (ALS), may also paralyze motor responses, leading to locked-in syndrome.

BRAIN DEATH **Brain death** is the cessation and irreversibility of all brain functions, including the brainstem. Although the exact criteria for establishing brain death may vary somewhat from state to state, it is generally agreed that brain death has occurred when there is no evidence of cerebral or brainstem function for an extended period (usually 6 to 24 hours) in a client who has a normal body temperature and is not affected by a depressant drug or alcohol poisoning. Generally recognized criteria are:

- Unresponsive coma with absent motor and reflex movements
- No spontaneous respiration (apnea)
- Pupils fixed (unresponsive to light) and dilated
- Absent ocular responses to head turning and caloric stimulation (Caloric stimulation is performed by irrigating the ear with ice-cold water to test the oculovestibular reflex, a reflex controlled by the brainstem. Normally, the cold causes the eyes to first move toward the irrigated side, followed by a return to midline.)
- Flat electroencephalogram (EEG) and no cerebral blood circulation present on angiography (if performed)
- Persistence of these manifestations for 30 minutes to 1 hour and for 6 hours after onset of coma and apnea.

Apnea in the comatose client is determined by the apnea test. The ventilator is removed while maintaining oxygenation by tracheal cannula and allowing the P_{CO_2} to increase to 60 mmHg or higher. This level of carbon dioxide is high enough to stimulate respiration if the brainstem is functional. The EEG may be used to establish the absence of brain activity when brain death is suspected. A flat (isoelectric) EEG over a period of 6 to 12 hours in a client who is not hypothermic or under the influence of drugs that depress the central nervous system (CNS) is generally accepted as an indicator of brain death.

Prognosis

The prognosis for clients with altered levels of consciousness and coma varies according to the underlying cause and pathologic process. Age and general medical condition also play a role in determining outcome. Young adults may fully recover following deep coma from head injury, drug overdose, or other cause. Recovery of consciousness within 2 weeks is associated with a favorable outcome. In general, the prognosis is poor for clients who lack pupillary reaction or reflex eye movements 6 hours after the onset of coma.

INTERDISCIPLINARY CARE

Management of the client with an altered LOC or coma must begin immediately. The focus of management is to identify the underlying cause, preserve function, and prevent deterioration if possible. Airway and breathing must be maintained during the initial acute stage until the diagnosis and prognosis can be established. Intravenous fluids are used to support circulation and to correct fluid, electrolyte, and acid–base imbalances. Treatment protocols to reduce increased intracranial pressure or control seizure activity (discussed later in this chapter) may be initiated. Changes in LOC associated with craniocerebral trauma, such as hematomas, often require immediate surgical intervention.

Diagnosis

Although the client's history and physical examination findings often indicate the cause of alterations in LOC, several diagnostic tests may be useful in establishing the diagnosis. The tests used to evaluate for possible metabolic, toxic, or drug-induced disorders include both radiologic and laboratory tests.

CT and MRI scanning are done to detect neurologic damage due to hemorrhage, tumor, cyst, edema, myocardial infarction, or brain atrophy. These tests may also identify displacement of brain structures by large or expanding lesions. Radioisotope brain scan is performed to identify abnormal lesions in the brain and evaluate cerebral blood flow. Cerebral angiography allows radiographic visualization of the cerebral vascular system. This exam can identify lesions such as aneurysms, occluded vessels, or tumors, and may also be used to determine cessation of cerebral blood flow and brain death. Transcranial Doppler studies use an ultrasound velocity detector that records

sound waves reflected from RBCs in blood vessels to assess cerebral blood flow. Lumbar puncture with cerebrospinal fluid (CSF) analysis is performed when infection and possible meningitis are suspected as a cause of altered LOC. EEG is used to evaluate the electrical activity of the brain. (See Chapter 43 ∞ for further information and nursing implications of neurologic tests.)

Laboratory tests are used to identify and monitor altered LOC. These may include any or all of the following:

- *Blood glucose* is measured immediately when coma is of unknown origin and hypoglycemia is suspected or possible. When the blood glucose falls to less than 40 to 50 mg/dL, cerebral function declines rapidly. The client with type 1 diabetes is at particular risk for hypoglycemia-induced coma.
- *Serum electrolytes*—sodium, potassium, bicarbonate, chloride, and calcium in particular—are measured to assess for metabolic disturbances and guide intravenous therapy. Hyponatremia, in which serum sodium levels are below 115 mEq/L (normal level: 135 to 145 mEq/L), is associated with coma and convulsions, especially if it develops rapidly.
- *Serum osmolality* is evaluated. Both hyperosmolar and hypo-osmolar states may be associated with coma. Hyperosmolality (above 320 mOsm/kg H_2O) causes cellular dehydration of brain tissue as fluid is drawn into the vascular system by osmosis. Hypo-osmolality (less than 250 mOsm/kg H_2O), by contrast, leads to cerebral edema and swelling, impairing consciousness.
- *Arterial blood gases* (ABGs) are drawn to evaluate arterial oxygen and carbon dioxide levels as well as acid–base balance. Hypoxemia is a frequent cause of altered LOC; increased levels of carbon dioxide are also toxic to the brain and can induce coma, particularly when the onset of hypercapnia is acute.
- *Liver function tests,* including bilirubin, AST, ALT, LDH, serum albumin, and serum ammonia levels, are determined to evaluate hepatic function. High ammonia levels seen in hepatic failure interfere with cerebral metabolism and neurotransmitters, affecting LOC.
- *Toxicology screening* of blood and urine is done to determine if altered LOC is the result of acute drug or alcohol toxicity. Serum alcohol levels are measured and the blood is assessed for the presence of substances such as barbiturates, carbon monoxide, or lead.

Medications

Medications are used to support homeostasis and normal function for the client with altered LOC, as well as to treat specific underlying disorders. An intravenous catheter is inserted, and fluid balance is maintained using isotonic or slightly hypertonic solutions, such as normal saline or lactated Ringer's solution. The client's response to fluid administration is monitored carefully for evidence of increased cerebral edema.

If hypoglycemia is present, 50% glucose is administered intravenously to restore cerebral metabolism rapidly. Conversely, insulin is administered to the client with hyperglycemia to reduce the blood glucose level and thus the serum osmolality. With narcotic overdose, naloxone is administered. Naloxone is a narcotic antagonist that competes for narcotic receptor sites, effectively blocking the depressant effect of the narcotic. Thiamine may be administered with glucose, particularly if the client is malnourished or known to abuse alcohol, to prevent exacerbation of Wernicke's encephalopathy, a hemorrhagic encephalopathy due to thiamine deficiency that is associated with chronic alcoholism (Tierney et al., 2005).

Any underlying fluid and electrolyte imbalance is corrected by administering medications or appropriate electrolytes. For the client who is hyponatremic and has a low serum osmolality, furosemide (Lasix) or an osmotic diuretic such as mannitol may be administered to promote water excretion. Appropriate antibiotics are administered intravenously to the client with suspected or confirmed meningitis.

Surgery

Although surgery is not indicated for most clients with altered LOC, it may be necessary if the cause of coma is an intracerebral tumor, hemorrhage, or hematoma. Surgical intervention is discussed later in this chapter, in the section on brain tumors. When there is a risk of increased intracranial pressure, the client is monitored continuously. These measures are discussed in the section on increased intracranial pressure that follows.

Other Treatments

Support of the airway and respirations is vital in the client with an altered LOC. The client who is drowsy but rousable may need little more than an oral pharyngeal airway. With more severe alterations in consciousness, the client may need endotracheal intubation to maintain airway patency, particularly if the cough and gag reflexes are absent. Mechanical ventilation is indicated when hypoventilation or apnea is present. Unless a do-not-resuscitate (DNR) order is in effect, mechanical ventilation should be initiated even if it has not been established that the disorder is reversible; without ventilatory support, cerebral anoxia develops rapidly, and brain death may ensue. ABGs are monitored frequently to determine the adequacy of ventilation. Cautious hyperventilation may be used to reduce $Paco_2$ and promote cerebral vasoconstriction to reduce cerebral edema.

Nutrition

In clients with long-term alterations in consciousness, such as vegetative state or locked-in syndrome, measures to maintain nutritional status are initiated. Enteral feedings with a gastrostomy tube are preferred if the client is unable to take enough food by mouth without aspirating. In some cases, total parenteral nutrition may be used.

NURSING CARE

Nursing care of the client with an altered LOC is planned and implemented for a variety of responses of both the client and the family of the client.

Support of the Family

Family members of a client with an altered level of consciousness are often very anxious. It is difficult for the family to deal with the client's uncertain prognosis. They may experience

various conflicting emotions, such as guilt and anger. Reinforce information provided by the physician, and encourage the family to talk to the client as though he or she were able to understand. Explain that this communication may initially seem awkward, but in time it will feel appropriate. Evaluate the family's readiness to receive explanations regarding the client's treatment and care. The presence of many tubes (e.g., intravenous line, catheter, ventilator) may be overwhelming to the family. They may not perceive the seriousness of the situation if a thorough explanation is not given. Include family members in the client's care as much as they wish to be involved.

Allow significant others to stay with the client when possible. Reinforce the need for family members to care for themselves by encouraging adequate meals and rest. Offer to contact support services such as friends, neighbors, and social services that the hospital may provide. Ask family members to leave a telephone number where they can be reached, and assure them that they will be called if any significant changes occur. Encourage family members to call if they have questions or concerns.

Nursing Diagnoses and Interventions

Nursing diagnoses and interventions discussed in this section are directed toward the unconscious client and focus on problems with airway maintenance, skin integrity, contractures, and nutrition.

Ineffective Airway Clearance

Ineffective airway clearance related to loss of the cough reflex and the inability to expectorate is a major problem for the unconscious client. The cough reflex may be absent or impaired when conditions that produce coma depress the function of the medullary centers.

- Assess ability to clear secretions. Monitor breath sounds, rate and depth of respirations, dyspnea, pulse oximeter, and the presence of cyanosis. *The client's ability to clear secretions serves as the initial assessment base for developing further interventions.*
- In unconscious clients or those without an intact cough reflex, maintain an open airway by periodic suctioning, limiting the time of suctioning to 10 to 15 seconds or less. Periodic suctioning may be necessary to clear the airway of mucus, blood, or other drainage. *Suctioning for more than 15 seconds in the client with increased intracranial pressure may cause hypercapnia, which in turn vasodilates cerebral vessels, increases cerebral blood volume, and increases intracranial pressure.*

PRACTICE ALERT
If the client has a basilar skull fracture or CSF draining from the ears or nose, never suction nasally.

- Turn from side to side every 2 hours, and maintain a side-lying position with the head of the bed elevated approximately 30 degrees. Do not position the unconscious client on the back. *Turning the client from side to side facilitates respirations, prevents the tongue from obstructing the airway, and helps prevent pooling of secretions in one area of the lungs (thus decreasing the risk of pneumonia).*
- If the client has a tracheostomy, provide tracheostomy care every 4 hours and suction when secretions are present (see Chapter 37 ∞) *to maintain an open airway.*
- Monitor the results of arterial blood gas analysis and pulse oximetry. Maintain records of trends. *ABGs and pulse oximetry directly measure the oxygen content of blood and are good indicators of the lungs' ability to oxygenate the blood.*

Risk for Aspiration

The unconscious client with a depressed or absent gag and swallowing reflex is at high risk for aspiration. Drainage, mucus, or blood may obstruct the airway and interfere with oxygenation. Pooling of aspiration secretions in the lungs also increases the risk of pneumonia.

- Assess swallowing and gag reflexes every shift as appropriate to the client's level of consciousness. *Deepening levels of unconsciousness may cause a loss in swallow and gag reflexes.*
- Monitor for and report manifestations of aspiration: crackles and wheezes, dullness to percussion over an area of the lungs, dyspnea, tachypnea, and cyanosis. *Early recognition facilitates prompt intervention.*
- Provide interventions to prevent aspiration:
  - Maintain NPO status.
  - Place in the side-lying position.
  - Provide oral hygiene and suctioning as needed.

  The side-lying position allows secretions to drain from the mouth rather than into the pharynx. Oral hygiene and suctioning remove secretions that might otherwise be aspirated.

PRACTICE ALERT
Never give unconscious clients oral food and fluids because of the risk of aspiration.

Risk for Impaired Skin Integrity

The unconscious client is at risk for impaired skin integrity as a result of immobility and the inability to provide self-care. On average, healthy people change positions during sleep every 11 minutes; the unconscious client often cannot maintain the movement needed to prevent pressure on the skin, especially over bony prominences. As a result, the skin and subcutaneous tissues may become ischemic and prone to develop pressure ulcers. Perspiration and incontinence of urine and stool may exacerbate the problem. Nursing interventions are directed to maintaining the integrity not only of the skin, but also of the lips and mucous membranes.

- Assess skin every shift, especially over bony prominences, the back of the scalp, and around genitals and buttocks. *The large surface area of the skin bears weight and is in constant contact with the surface of the bed. The skin, subcutaneous tissue, and muscles, especially those tissues over bony prominences, undergo constant pressure. This impairs normal capillary blood flow, which interferes with the exchange of nutrients and waste products. Tissue ischemia and necrosis may result and lead to the development of pressure ulcers.*
- Provide proper positioning. Reposition bed-ridden clients at least every 2 hours if this is consistent with the overall treatment goals. Keep the head of the bed elevated no higher than

30 degrees unless prescribed differently. Provide special pads and mattresses that distribute weight more evenly (e.g., silicone-filled pads, egg-crate cushions, turning frames, flotation pads). Consider requesting/using a special therapeutic bed that automatically turns the client at regular intervals. Lift the client instead of dragging the client across the sheet. *When the head of the bed is elevated above 30 degrees, the client's torso tends to slide down toward the foot of the bed. Friction and perspiration cause the skin and superficial fascia to remain fixed against the bed linens while the deep fascia and skeleton slide downward. When a person is pulled rather than lifted, the skin remains fixed to the sheet while the fascia and muscles are pulled upward. These shearing forces promote tissue breakdown.*

- Provide interventions to prevent breakdown of the skin and mucous membranes:
  - Keep bed linens clean, dry, and wrinkle free.
  - Provide daily bath with mild soap.
  - Cleanse the skin after urine and fecal soiling with a mild cleansing agent.
  - Provide oral care and lubricate the lips every 2 to 4 hours.
  - Maintain accurate intake and output records.
  - Keep the cornea moist by instilling methyl cellulose solution (0.5% to 1%) and apply protective eye shields or close the eyelids with adhesive strips if the corneal reflex is absent.

  Keeping linens clean, dry, and wrinkle free decreases the risk of injury from the shearing force of bed rest and protects against environmental factors that cause drying. Adequate hydration of the stratum corneum appears to protect the skin against mechanical insult. Preventing dehydration maintains circulation and decreases the concentration of urine, thereby minimizing skin irritation in people who are incontinent. Proper eye care prevents corneal abrasion and irritation.

Impaired Physical Mobility

Clients who are unconscious are unable to maintain normal musculoskeletal movement and are at high risk for contractures related to decreased movement. Because the flexor and adductor muscles are stronger than the extensors and abductors, flexor and adductor contractures develop quickly without preventive measures. Passive ROM exercises must be performed routinely to maintain muscle tone and function, to prevent additional disability, and to help restore impaired motor function.

- Maintain extremities in functional positions by providing proper support devices. Remove support devices every 4 hours for skin care and passive ROM exercises. Provide pillows for the axillary region; rolled washcloths may be placed in elevated hands; use splints to prevent plantar flexion (foot drop). *Pillows in the axillary region help prevent adduction of the shoulder. Rolled washcloths help decrease edema and flexion contracture of the fingers. Splints are useful in preventing plantar flexion.*
- Collaborate with a physical therapist to develop and implement passive range-of-motion (ROM) exercises (unless contraindicated, as for the client with increased intracranial pressure) at least four times a day, keeping the following principles in mind:
  - Place one hand above the joint being exercised. The other hand gently moves the joint through its normal range of motion.
  - Move the body part to the point of resistance, and stop.

  Placing one hand above the joint provides support against gravity and prevents unwanted movement. ROM exercises help prevent contractures by stretching muscles and tendons and maintaining joint mobility.

Risk for Imbalanced Nutrition: Less than Body Requirements

The unconscious client is at risk for an alteration in nutrition related to a reduced or complete inability to eat. This is especially true for the client who is unconscious as the result of an infection or trauma, both of which increase metabolic requirements.

- Monitor nutritional status through daily weights (on bed scales) and laboratory data. For accuracy, weigh the client at the same time each day, using the same scales. Ensure that the client wears the same clothing. *Changes in laboratory data with decreased nutrition include a decrease in the levels of serum prealbumin and serum transferrin.*
- Assess the need for alternative methods of nutritional support (tube feeding or total parenteral nutrition) through collaboration with dietitian. *Clients unable to take oral food require parenteral nutrition or liquid feedings through a nasogastric, gastrostomy, or jejunostomy tube. Needs for protein, calories, zinc, and vitamin C increase during wound healing.*

THE CLIENT WITH INCREASED INTRACRANIAL PRESSURE

Increased intracranial pressure (IICP) (also labeled *intracranial hypertension*) is sustained elevated pressure (10 mmHg or higher) within the cranial cavity (Wilensky & Bloom, 2005). Transient increases in ICP occur with normal activities such as coughing, sneezing, straining, or bending forward. These transient increases are not harmful; however, sustained IICP can result in significant tissue ischemia and damage to delicate neural tissue. Cerebral edema is the most frequent cause of sustained increases in ICP. Other causes include head trauma, tumors, abscesses, stroke, inflammation, and hemorrhage.

Pathophysiology

In the adult, the rigid cranial cavity created by the skull is normally filled to capacity with three essentially noncompressible elements: the brain (80%), cerebrospinal fluid (8%), and blood (12%). A state of dynamic equilibrium exists; if the volume of any of the three components increases, the volume of the others must decrease to maintain normal pressures within the cranial cavity. This is known as the *Monro-Kellie hypothesis.* The normal intracranial pressure is 5 to 10 mmHg (measured intracranially with a pressure transducer while the client is lying with the head elevated 30 degrees) or 60 to 180 cm H_2O (measured with a water manometer while the client is lying in a lateral recumbent position).

Cerebral blood flow and perfusion are important concepts for understanding the development and effects of increased intracranial pressure. Whereas blood and CSF contribute an

equal percentage to normal intracranial volume, vascular factors account for twice the amount of increase in ICP that CSF does. The brain requires a constant supply of oxygen and glucose to meet its metabolic demands; 15% to 20% of the resting cardiac output goes to the brain to meet its metabolic needs. Interruption of the cerebral blood flow leads to ischemia and disruption of the cerebral metabolism.

Pressure and chemical autoregulation are compensatory mechanisms in which cerebral arterioles change diameter to maintain cerebral blood flow when ICP increases. In pressure autoregulation, stretch receptors within small blood vessels of the brain cause smooth muscle of the arterioles to contract. Increased arterial pressure stimulates these receptors, leading to vasoconstriction; when arterial pressure is low, stimulation of these receptors decreases, causing relaxation and vasodilation. Chemical, or metabolic, autoregulation works in much the same way as pressure autoregulation. In this case, the stimulus is a buildup of metabolic by-products of cell metabolism, including lactic acid, pyruvic acid, carbonic acid, and carbon dioxide. Carbon dioxide and increased hydrogen ion concentration are potent cerebral vasodilators that may act locally or systemically to increase cerebral blood flow. Conversely, a fall in $Paco_2$ causes cerebral vasoconstriction. Arterial oxygen tension (Pao_2) also affects cerebral blood flow, although it is a less powerful mechanism than that exerted by carbon dioxide and hydrogen ions.

IICP may result from an increase in intracranial contents from a space-occupying lesion, hydrocephalus, cerebral edema (swelling), excess cerebrospinal fluid, or intracranial hemorrhage. Displacement of some CSF to the spinal subarachnoid space and increased CSF absorption are early compensatory mechanisms. The low-pressure venous system is also compressed, and cerebral arteries constrict to reduce blood flow. Brain tissue's ability to accommodate change is relatively restricted. The relationship between the volume of the intracranial components and intracranial pressure is known as *compliance*. When the capacity to compensate for increased intracranial pressure is exceeded, increased intracranial pressure (hypertension) develops. Intracranial hypertension is a sustained state of IICP and is potentially life threatening.

Autoregulatory mechanisms have a limited ability to maintain cerebral blood flow. When autoregulation fails, cerebrovascular tone is reduced and cerebral blood flow becomes dependent on changes in blood pressure. Autoregulation may be lost either locally or globally because of several factors, including increasing intracranial pressure, local or diffuse cerebral tissue ischemia or inflammation, prolonged hypotension, and hypercapnia or hypoxia.

Manifestations

With loss of autoregulation, intracranial pressure continues to rise and cerebral perfusion falls. Cerebral tissue becomes ischemic, and manifestations of cellular hypoxia appear. The manifestations of IICP are listed in the box on this page.

Level of Consciousness

Because the neurons of the cerebral cortex are most sensitive to oxygen deficit, changes in cortical function are the earliest manifestations of increasing ICP (Porth, 2005). Behavior and personality changes occur; the client may become irritable and agitated. Memory and judgment are impaired, and speech pattern changes may be noted. The client's LOC decreases. As cerebral hypertension and hypoxia progress, the LOC continues to decrease in a predictable pattern to coma and unresponsiveness.

MANIFESTATIONS of Increased Intracranial Pressure

- Decreased level of consciousness. *Early*: Confusion; restlessness, lethargy; disorientation, first to time, then to place and person. *Late:* Comatose with no response to painful stimuli.
- Pupillary dysfunction. Sluggish response to light progressing to fixed pupils; with a localized process, pupillary dysfunction is first noted on the ipsilateral side.
- Oculomotor dysfunction. Inability to move eye(s) upward; ptosis (drooping) of the eyelid.
- Visual abnormalities. Decreased visual acuity, blurred vision, diplopia.
- Papilledema. May be late sign.
- Motor impairment. *Early*: Hemiparesis or hemiplegia of the contralateral side. *Late*: Abnormal responses such as decorticate or decerebrate positioning; flaccidity.
- Headache. Uncommon but may occur with processes that slowly increase ICP; worse on rising in the morning and with position changes.
- Projectile vomiting without nausea.
- Cushing's response. Increased systolic blood pressure, widening pulse pressure, bradycardia.
- Respirations. Altered respiratory pattern related to level of brain dysfunction.
- Temperature. May be significantly elevated as compensatory mechanisms fail.

Motor Responses

Pressure on the pyramidal tract often causes weakness (hemiparesis) on the contralateral side early in IICP. As ICP continues to increase, hemiplegia and abnormal motor responses, such as decorticate or decerebrate posturing, develop (see Chapter 43 ∞ for an illustration of these postures).

Vision and Pupils

Altered vision is an early manifestation of IICP; it is caused by pressure on the visual pathways and cranial nerves. Blurred vision, decreased visual acuity, and diplopia are common. Pupillary and oculomotor responses are affected as well. Because the cause of IICP is often localized at first, pupillary changes, including gradual dilation and sluggish response to light, may initially be limited to the ipsilateral side.

Vital Signs

Ischemia of the vasomotor center in the brainstem triggers the CNS ischemic response, a late sign of IICP. Neuronal ischemia in the vasomotor center causes a marked increase in the mean arterial pressure (MAP), with a significant increase in systolic blood pressure and increased pulse pressure. The increased

MAP causes reflexive slowing of the cardiac rate. This trio of manifestations (increased MAP, increased pulse pressure, and bradycardia) is known as *Cushing's response* (or triad), and represents the brainstem's final effort to maintain cerebral perfusion (Porth, 2005). The respiratory pattern also changes, often in the predictable progression outlined earlier in Table 44–1. Although the temperature is usually normal in early stages, as ICP continues to increase, hypothalamic function is impaired and the temperature may rise dramatically.

Other Manifestations

Additional manifestations of IICP include headache, particularly on rising, that worsens with position changes. Headache is more common with slowly developing IICP and occurs because of pressure on pain-sensitive structures, such as the middle meningeal arteries, the venous sinuses, and the dura at the base of the skull. Papilledema (edema and swelling of the optic disk) may be noted on funduscopic examination. Vomiting, often projectile and occurring without warning, may develop.

Cerebral Edema

Cerebral edema is an increase in the volume of brain tissue due to abnormal accumulation of fluid. Cerebral edema is often associated with increased intracranial pressure; it may occur as a local process in the area of a tumor or injury, or it may affect the entire brain. Two types of cerebral edema have been identified and are described as follows (Porth, 2005):

- *Vasogenic edema*, an increase in the capillary permeability of cerebral vessels, occurs with impairment of the blood–brain barrier, allowing diffusion of water and protein into the interstitial spaces of the brain. A variety of pathologies, such as ischemia, hemorrhage, brain tumors and injuries, and infections (such as meningitis), may cause the increase in capillary permeability. The site of the brain injury, the level of increase in capillary permeability, and the client's systemic blood pressure influence the rate and extent of the edema's spread. Vasogenic edema is manifested by focal (localized) neurologic deficits, altered levels of consciousness, and severe intracranial hypertension.
- *Cytotoxic edema*, actual swelling of the brain cells from an increase in intracellular fluid, involves changes in the functional or structural integrity of cell membranes due to pathologies such as water intoxication (such as from the syndrome of inappropriate secretion of antidiuretic hormone [SIADH]) or severe ischemia, intracranial hypoxia, acidosis, and brain trauma. With abnormally low cerebral perfusion, oxygen and nutrients are depleted, intracranial cells switch to anaerobic metabolism, and the sodium-potassium pump in the cell walls is impaired. Sodium diffuses into the cells, pulling fluid with it. The cells swell, and intracranial pressure rises. Accumulated metabolic waste products, such as lactic acid, contribute to a rapid deterioration of cell function. Cytotoxic edema is a slowly progressive process that results in altered consciousness. The edema may be so severe that it causes cerebral infarction with brain tissue necrosis.

Cerebral edema tends to be proportional to the extent of the pathology precipitating it. Brain function is not disrupted by cerebral edema unless the edema causes an increase in ICP. When it does, a vicious cycle can ensue: Cerebral edema increases ICP, which in turn decreases cerebral blood flow. Brain tissue becomes hypoxic and ischemic, increasing toxic metabolic by-products, hydrogen ion concentration, and carbon dioxide levels in the tissue. Autoregulatory mechanisms cause vasodilation and increase cerebral blood flow, further increasing cerebral edema and intracranial pressure. Without effective intervention, the client's condition can deteriorate rapidly; intracranial pressure increases to the point where brain structures herniate.

Hydrocephalus

Hydrocephalus refers to a progressive dilatation of the ventricular system, which becomes dilated as the production of CSF exceeds its absorption (Hickey, 2003). Hydrocephalus may increase ICP when it develops acutely. It is generally classified as either noncommunicating or communicating hydrocephalus. Noncommunicating hydrocephalus occurs when CSF drainage from the ventricular system is obstructed. It may develop when a mass or tumor, inflammation or hemorrhage, or congenital malformation obstructs the ventricular system. Communicating hydrocephalus is a condition in which CSF is not effectively reabsorbed through the arachnoid villi. It may occur secondarily to subarachnoid hemorrhage or scarring from infection. In *normal pressure hydrocephalus*, seen most often in adults age 60 or older, ventricular enlargement causes cerebral tissue compression but the CSF pressure on lumbar puncture is normal. This condition may follow cerebral trauma or surgery, or the cause may not be known.

Manifestations of hydrocephalus depend on the rate of its development. They may be mild and insidious in onset, presenting as progressive cognitive dysfunctions, gait disruptions, and urinary incontinence. If the process causing hydrocephalus is an acute one, the manifestations are those of IICP.

Brain Herniation

If IICP is not treated, cerebral tissue is displaced toward a more compliant area. This can result in brain herniation, the displacement of brain tissue from its normal compartment under dural folds of the falx cerebri or through the tentorial notch or incisura of the tentorium cerebelli (Porth, 2005). Herniation of the cerebellum through the tentorium exerts pressure on the brainstem, with subsequent herniation through the foramen magnum. This is a lethal complication of IICP because it puts pressure on the vital centers of the medulla.

Brain herniation syndromes are generally categorized as supratentorial or infratentorial, depending on their location above or below the tentorium cerebelli (Figure 44–2 ■). Supratentorial herniation syndromes include cingulate herniation, central or transtentorial herniation, uncal or lateral transtentorial herniation, and infratentorial.

- *Cingulate herniation* (Figure 44–2A) occurs when the cingulate gyrus is displaced under the falx cerebri. Local blood supply and cerebral tissue are compressed, resulting in ischemia and further increases in intracranial pressure.

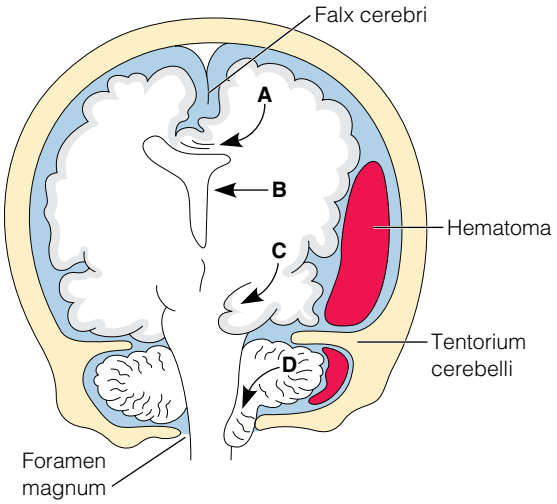


Figure 44–2 ■ Forms of brain herniation due to intracranial hypertension. *A*, Cingulate herniation occurs when the cingulate gyrus is compressed under the falx cerebri. *B*, Central herniation occurs when a centrally located lesion compresses central and midbrain structures. *C*, Lateral herniation occurs when a lesion at the side of the brain compresses the uncus or hippocampal gyrus. *D*, Infratentorial herniation occurs when the cerebellar tonsils are forced downward, compressing the medulla and top of the spinal cord.

- *Central or transtentorial herniation* is the downward displacement of brain structures, including the cerebral hemispheres, basal ganglia, diencephalon, and midbrain through the tentorial incisura (Figure 44–2B). The client's neurologic signs may deteriorate rapidly, with decreased LOC progressing to coma, Cheyne-Stokes respirations progressing to central neurogenic hyperventilation, and pupils progressing from small and reactive to midsize and fixed. The client may demonstrate abnormal motor responses with unilateral decorticate posturing.
- *Uncal or lateral transtentorial herniation* occurs when a lateral mass displaces cerebral tissue centrally, forcing the medial aspect of the temporal lobe under the edge of the tentorial incisura (Figure 44–2C). The oculomotor nerve (cranial nerve III) often becomes trapped between the uncus and the tentorium, causing ipsilateral pupillary dilation. Other manifestations include alterations in LOC, motor deficits (which may occur on the same side as the herniation because of compression of the cerebral peduncle on the opposite side), decreased sensation, respiratory changes, abnormal positioning, and eventual respiratory arrest.
- *Infratentorial herniation* results from increased pressure within the infratentorial compartment. Herniation may occur either upward, with structures displaced through the tentorial incisura, or downward, with displacement through the foramen magnum (Figure 44–2D). Downward displacement compresses the medulla, including its centers for controlling vital functions. Manifestations associated with medullary compression include coma, altered respiratory patterns, fixed pupils, and decorticate or decerebrate posturing. Respiratory or cardiac arrest may occur.

INTERDISCIPLINARY CARE

Management of the client with IICP is directed toward identifying and treating the underlying cause of the disorder, and controlling ICP to prevent herniation syndrome. IICP is a medical emergency, and there is little time to complete lengthy diagnostic tests. The diagnosis must be made on the basis of observation and neurologic assessment; even subtle changes may be clinically significant.

Diagnosis

Diagnostic tests focus on identifying the presence of IICP and its underlying cause. A CT scan or MRI is generally the initial test used to identify the possible causes of IICP (such as space-occupying lesions or hydrocephalus) and to evaluate therapeutic options. In general, a lumbar puncture is not performed when IICP is suspected because the sudden release of the pressure in the skull may cause cerebral herniation.

In addition to the diagnostic tests listed in the previous section for altered LOC, the following specific tests are usually ordered and their results closely monitored:

- Serum osmolality is an indicator of hydration status in the client with IICP. The test measures the number of dissolved particles (electrolytes, urea, glucose) in the serum. The normal range for the adult is 280 to 300 mOsm/kg H_2O. In addition to the restriction of fluids in the client with IICP, serum osmolality is maintained at a slightly elevated level (325 mOsm/kg H_2O) to draw excess intracellular fluid into the vascular system.
- ABGs are monitored frequently to assess pH and levels of oxygen and carbon dioxide. Hydrogen ions and carbon dioxide are both potent vasodilators; hypoxemia also causes vasodilation, although to a lesser degree.

Medications

Medications play an important role in the management of IICP. Diuretics, particularly osmotic diuretics, are commonly used to reduce ICP and are the mainstays of pharmacologic treatment. Nursing implications for these medications are described in the Medication Administration box on the next page.

Osmotic diuretics work by increasing the osmolarity of the blood, thereby drawing water out of edematous brain tissue and into the vascular system for elimination via the kidneys. The effects of these drugs vary with the type of injury. Regardless of the agent used, the optimal dose is the lowest that reduces ICP. Mannitol is the most commonly employed osmotic diuretic. Glucose, urea, and glycerol are other osmotic diuretics that may be used. Urine output by Foley catheter is monitored. Electrolyte levels are carefully assessed and potassium is replaced as indicated.

Loop diuretics, such as furosemide (Lasix) (the drug of choice) and ethacrynic acid (Edecrin), may be prescribed for some clients with IICP. These diuretics act on the renal tubule and are extremely effective in promoting diuresis. Additionally, loop diuretics may be used to manage the rebound effect that may occur with mannitol administration.

Sedation and paralysis are used as chemical restraints to control restlessness and agitation, because these movements increase blood pressure, ICP, and cerebral metabolism. Paralysis

MEDICATION ADMINISTRATION Increased Intracranial Pressure

OSMOTIC DIURETICS

Mannitol (Osmitrol)
Urea
Glucose

Osmotic diuretics (hyperosmotic agents) draw fluid out of brain cells by increasing the osmolality of the blood. The effects of these drugs vary with the type of injury. Mannitol therapy is often initiated if the client's ICP has exceeded 15 to 20 mmHg for at least 10 minutes. Both intravenous bolus and continuous infusion techniques are used. Repeated use of mannitol can lead to continual elevations in serum osmolality, with attendant risk of seizures and serious fluid and electrolyte imbalance. Urea is seldom administered intravenously because a severe local reaction may result if leakage occurs at the injection site. Mannitol and urea are used cautiously if renal disease is present.

Note: Because the client with increased intracranial pressure often has an altered level of consciousness, client and family teaching are not discussed in this box.

Nursing Responsibilities

- Monitor vital signs, urinary output, central venous pressure (CVP), and pulmonary artery pressures (PAP) before and every hour throughout administration.
- Assess client for manifestations of dehydration.
- Assess client for muscle weakness, numbness, tingling, paresthesia, confusion, and excessive thirst.
- Assess client for pulmonary edema while administering the medication.
- Monitor neurologic status and intracranial pressure readings.
- Monitor renal function and serum electrolytes throughout therapy.
- Do not administer the medication if crystals are present in solution. Administer with an in-line filter. Observe infusion site frequently for infiltration.
- Do not administer mannitol solution with blood.
- Do not discontinue medication abruptly. Rebound migraine headaches may occur.

LOOP DIURETICS

Furosemide (Lasix)
Ethacrynic acid (Edecrin)

Loop diuretics such as furosemide and ethacrynic acid inhibit sodium and chloride reabsorption at the ascending loop of Henle. They cause a reduction in the rate of CSF production, thus reducing the ICP.

Nursing Responsibilities

- Monitor vital signs and electrolyte values closely.
- Assess fluid status throughout therapy.
- Monitor blood pressure and pulse before and during administration.
- Monitor renal laboratory studies closely.
- Use infusion pump to ensure accurate dosage.

INTRAVENOUS FLUIDS

Keeping the client moderately dehydrated to maintain serum osmolality can be effective in reducing cerebral edema. When giving intravenous fluids, closely monitor the osmolality of the solutions; if clients with increased ICP are given hypo-osmolar solutions, increased cerebral edema can occur. Preferred solutions include 0.45% to 0.9% sodium chloride solutions.

Nursing Responsibilities

- Monitor fluid status closely.
- Monitor neurologic status closely.
- Avoid administering hypo-osmolar solutions, such as 5% dextrose in water.
- Half-strength normal saline (0.45% sodium chloride) is considered a suitable fluid for a client who has increased intracranial pressure.
- Take care not to restrict fluids excessively in clients receiving dehydrating agents (such as osmotic or loop diuretics).

OTHER PHARMACOLOGIC INTERVENTIONS FOR ICP

- Antipyretics, such as acetaminophen, are used to reduce hyperthermia, thereby decreasing the high cerebral metabolism that contributes to ICP.
- Antiulcer drugs, such as histamine H_2 antagonists (for example, ranitidine [Zantac]) or sucralfate (Carafate), are used in clients with ICP to decrease the development of stress ulcers.
- Antihypertensive agents, such as beta-adrenergic blocking agents, may be used if the mean arterial pressure is high.
- Vasopressors may be used if the mean arterial pressure is low.
- Anticonvulsants may be given to prevent or treat seizures.

with neuromuscular blockage is most often accomplished with pancuronium. Clients must be closely monitored during treatment for residual muscle weakness and signs of respiratory distress. A peripheral nerve stimulator may be used for this purpose.

Antipyretics, such as acetaminophen, are used alone or in combination with a hypothermia blanket to treat hyperthermia. Hyperthermia increases the cerebral metabolic rate and exacerbates an existing increase in ICP. Anticonvulsants are often required to manage seizure activity associated with brain injury and IICP. Gastrointestinal prophylaxis with intravenous histamine H_2 antagonists or proton pump inhibitors are often used, because clients with IICP are at increased risk for the development of stress gastritis and ulcers (Tierney et al., 2005).

Intravenous fluids are usually necessary to maintain the client's fluid and electrolyte balance as well as vascular volume. If the client's blood pressure is unstable, vasoactive medications may be administered to maintain the MAP in a range that supports cerebral perfusion while minimizing increases in ICP. When enteral feeding is not possible, total parenteral nutrition may be administered.

Surgery

Clients with IICP may undergo various intracranial surgical techniques to treat the underlying cause (see the discussion in the later section on brain tumors). In addition, infarcted or necrotic tissue may be resected to reduce brain mass. A drainage catheter or shunt may be inserted laterally via a burr hole into a ventricle to drain excess cerebrospinal fluid and reduce hydrocephalus. The removal of even a small amount of CSF may dramatically reduce IICP and restore cerebral perfusion pressure.

ICP Monitoring

Critical to preserving brain function and preventing secondary brain damage from IICP are careful assessments and monitoring with ICP monitors, measuring cerebral blood flow and cerebral perfusion pressure, and measuring oxygen levels of brain tissue. Intracranial pressure monitors facilitate continual assessment of ICP and the effects of medical therapy and nursing interventions on ICP. In addition, cerebral perfusion pressure (the difference between MAP and ICP) can be readily calculated, allowing more precise manipulation of therapeutic measures to maintain cerebral perfusion and thereby prevent ischemia. The criteria for ICP monitoring depends on the client, but in general, clients who are comatose and have a Glasgow Coma Score (described in Chapter 43 ∞) of 8 or less should be monitored.

Basic monitoring systems include an intraventricular catheter, subarachnoid bolt or screw, and epidural probe (Figure 44–3 ■). Intraventricular fluid-filled catheters are placed in the anterior horn of the lateral ventricle (most often in the right side). Ventricular catheters can both drain CSF and measure ICP. The ICP value is measured deep in the brain and is considered the most reflective of the whole brain pressure. Subarachnoid devices are placed in the subarachnoid space. A fiber-optic transducer-tipped catheter can be placed in the epidural, subdural, or parenchymal space, with ICP values considered very accurate. Once the intracranial sensor is implanted, it is connected to a transducer that converts the impulses to a signal that the recording device can translate into an oscilloscope tracing, digital value, or graphic recording. Factors that increase the risk for infection during ICP monitoring are listed in Table 44–4.

Transcranial blood flow is monitored with transcranial Doppler studies (TCD) to measure the velocity of blood flow in the cerebral vessels. Cerebral perfusion pressure (CPP) is the pressure it takes for the heart to provide the brain with blood, calculated by subtracting the ICP from mean arterial pressure (normal CPP is 70 to 95 mmHg). Brain oxygenation monitoring may be conducted by using a jugular bulb oxygen saturation (SjO_2) monitor connected to a small fiber-optic catheter inserted into the jugular vein. (Normal SjO_2 is 50% to 75%.) Another device used to monitor brain tissue oxygenation is the LICOX system, which includes information about oxygen status and temperature status within the brain tissue itself (Brettler, 2004). In addition, cerebral microdialysis catheters can provide information about the nature of the cerebral interstitial fluid.

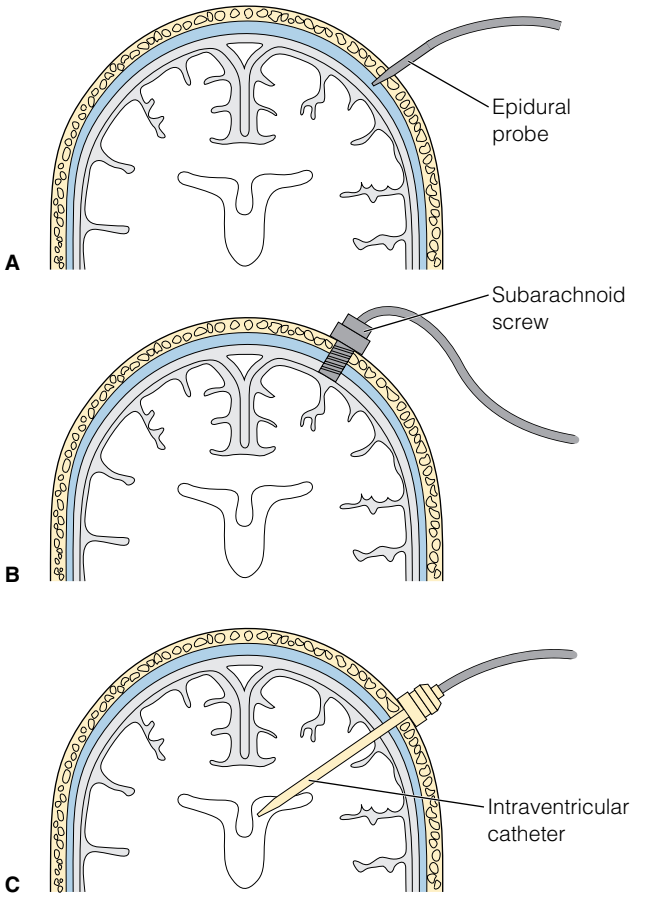


Figure 44–3 ■ Types of intracranial pressure monitoring. *A*, Epidural probe. *B*, Subarachnoid screw. *C*, Intraventricular catheter.

Mechanical Ventilation

Clients with ICP often require intubation and are placed on a ventilator for respiratory management. Mechanical ventilation may be used to maintain partial pressure of oxygen and carbon dioxide, thus preventing hypoxemia and hypercapnia, both of which can increase intracranial pressure. It is important to maintain adequate oxygenation with a partial pressure of arterial oxygen at about 100 mmHg and a partial pressure of arterial carbon dioxide of about 35 mmHg. The client with IICP and signs of impending herniation may be judiciously hyperventilated to cause cerebral vasoconstriction; however, this also increases cerebral ischemia.

TABLE 44–4 **Risk Factors for Infection with Intracranial Pressure Monitoring**

FACTOR	RATIONALE
Intraventricular catheter	Is more invasive than other monitoring devices
Open head trauma or neurosurgery	Disrupts protective skin and skeletal barriers
Intracranial hemorrhage	Necessitates frequent flushing of catheter to maintain patency
Older adult	Tends to have impaired immune defenses
Monitoring for more than 3 to 5 days; or open system or frequent irrigation	Offers increased opportunity for pathogens to enter and grow

NURSING CARE

The nursing care of clients with IICP involves identifying those at risk and managing factors known to increase intracranial pressure. A major focus is protecting the client from sudden increases in ICP or a decrease in cerebral blood flow.

Nursing Diagnoses and Interventions

Nursing interventions include performing neurologic assessments, maintaining the patency of the airway, ensuring adequate ventilation, positioning and moving, instituting seizure precautions, and monitoring fluids and electrolytes. Additionally, both client and family need emotional support during this period. The client with IICP has varied responses to actual or potential changes in physiologic processes.

Ineffective Tissue Perfusion: Cerebral

A number of disorders may lead to IICP, including cerebral edema, hydrocephalus, space-occupying lesions and hemorrhage, herniation syndromes, and changes in carbon dioxide concentrations. Increasing intracranial pressure alters cerebral perfusion and oxygenation of brain cells. The client with IICP requires intensive care, and often needs ventilator assistance.

- Assess for and report manifestations of IICP every 15 minutes to 1 hour and as necessary. Assessment areas include LOC, behavior, motor/sensory functions, pupillary size and reaction to light, and vital signs, including temperature. Look for trends, because vital signs alone do not correlate well with early deterioration. *Assessment of neurologic status establishes the client's clinical condition and provides a baseline to measure changes. Sudden changes in neurologic signs often indicate deterioration. An elevated temperature with increased oxygen consumption further increases intracranial pressure. Pupillary responses mirror the status of the midbrain and pons. Pressure on the brainstem may compromise the function of cranial nerves IX and X and protective mechanisms, such as the gag and cough reflexes.*

PRACTICE ALERT

Often, the earliest manifestations of a change in intracranial pressure are alterations in the level of consciousness and respirations.

- For the client on a ventilator: Maintain patency of the airway; preoxygenate with 100% oxygen before suctioning; limit suctioning to 10 seconds; suction gently. *Preoxygenation helps maintain oxygen levels during suctioning. Suctioning stimulates the cough reflex and Valsalva maneuver. Correct suctioning minimizes the risk of hypoxemia.*
- Monitor ABGs. *ABGs provide a reliable indicator of oxygen and carbon dioxide levels. If oxygen concentration is low, oxygen may be given or increased.*
- Elevate head of the bed to 30 degrees or keep flat, as prescribed; maintain the alignment of the head and neck to avoid hyperextension or exaggerated neck flexion; avoid prone position. *Keeping the head of the bed elevated facilitates venous drainage from the cerebrum. Obstruction of jugular veins can impede venous drainage from the brain.*
- Monitor bladder distention and bowel constipation. Administer stool softeners and use the Credé technique (applying pressure to the suprapubic region with the fingers of one or both hands) to empty the bladder. If the Credé technique is not effective, evaluate the pros and cons of urinary catheterization if the bladder remains distended. *Constipation and bladder distention increase intrathoracic or intra-abdominal pressure and place the client at risk for impaired venous drainage from the brain.*
- If alert, assist in moving up in bed. Do not ask to push with heels or arms or push against a footboard. Avoid a footboard and restraints. *Moving up in bed requires pushing. Helping the client move prevents initiation of the Valsalva maneuver, which increases intracranial pressure.*
- Plan nursing care so that activities are not clustered together; avoid turning the client, getting the client on the bedpan, or suctioning within the same time period. Schedule nursing care to provide rest periods between procedures. *Multiple procedures, including certain nursing care activities, can increase ICP. Constant stimulation tends to increase ICP. Individualized nursing care ensures optimal spacing of activities and rest.*
- Provide a quiet environment, limiting noxious stimuli. Avoid jarring the bed. Try to limit situations that cause emotional upset; maintain a calm, reassuring manner; caution family members to refrain from unpleasant conversations or conversations that may be emotionally stimulating to the client. *Noxious stimuli and emotional upsets cause an elevation in ICP.*
- Maintain fluid limitations, if prescribed. *Restricting fluids helps decrease cerebral edema by reducing total body water.*

Risk for Infection

Although any client with an open head wound is at risk for infection, the interventions discussed here are for the client with an intracranial monitoring device. Most clinical units have written protocols for managing these systems. The following nursing actions serve only as a general guide.

- Keep dressings over the catheter dry, and change dressings on a prescribed basis (usually every 24 to 48 hours). *Wet dressings are conducive to bacterial growth.*
- Monitor the insertion site for leaking CSF, drainage, or infection. Monitor for manifestations of infection, including changes in vital signs, chills, increased WBC counts, and positive cultures of drainage. *Close monitoring helps detect the earliest signs of infection and helps prevent major complications. Fever is usually considered the key assessment. However, fever in a client with a neurologic disorder may be due to damage to the hypothalamus. Headache, generalized muscle aches, shivering, and chills may also be seen in the client with infection.*
- Use strict aseptic technique when in contact with the device. Check drainage system for loose connections. *The use of aseptic technique and monitoring drainage systems for loose connections help prevent nosocomial infections. Most nosocomial infections are transmitted by healthcare workers who fail to wash their hands properly, to change gloves between clients, or to follow aseptic technique protocols. Invasive procedures provide an excellent opportunity for microbes to enter the body.*

Client and Family Education

Teach the client at risk for or having IICP (and able to follow instructions) to avoid coughing, blowing the nose, straining to have a bowel movement, pushing against the bed rails, or performing isometric (muscle contracting) exercises. Advise the client to maintain head and neck alignment when turning in bed and to take rest periods.

Encourage the family to talk to the client, but maintain a quiet environment with a minimum of stimuli. Inform family members that upsetting the client may increase intracranial pressure and that they should avoid discussions that may distress the client. For clients unable to make decisions about treatment and to sign informed consent, the family must carry out these functions.

THE CLIENT WITH A HEADACHE

Headache, one of the most frequent manifestations of a health problem people experience, is pain within the cranial vault. Headaches may occur as a result of benign or pathologic conditions, intracranial or extracranial conditions, diseases of other body systems, stress, musculoskeletal tension, or a combination of these factors.

Most headaches are mild, transient, and relieved by a mild analgesic. However, some headaches are chronic, intense, and recurrent. Manifestations of headache vary according to the cause, type, and precipitating symptoms.

Pathophysiology

The bones and brain tissue itself lack pain-sensitive nerve fibers, but selected structures within the cranial vault are sensitive to pain. Headache is experienced when there is traction, pressure, displacement, inflammation, or dilation of nociceptors (nerve endings that are receptors of noxious stimuli) in areas sensitive to pain (Hickey, 2003). Pain-sensitive structures include supporting structures, such as the skin, muscles, and periosteum; the nasal cavities and sinuses; portions of the meninges, cranial nerves II, III, IV, V, VI, IX, and X; and cerebral vessels, including extracranial arteries and the venous sinuses. Most facial and scalp structures are sensitive to pain. The most common types of headaches are migraine, cluster, and tension headaches (Table 44–5).

Migraine Headache

Migraine headache is a recurring vascular headache lasting from 4 to 72 hours, often initiated by a triggering event and usually accompanied by a neurologic dysfunction. It affects about 15% of the population, with three times as many women as men having migraines (eMedicine Health, 2006). It is more common between the ages of 25 and 55 years. More than 80% of people with migraines (called *migraineurs*) have family members with them too. Migraine headaches may occur daily or as infrequently as once a year.

FAST FACTS

Migraine Headache

- Eight out of 10 migraineurs have severe or extremely severe attacks.
- One out of 4 migraineurs seek emergency room treatment.
- Fifty-one percent of migraineurs say that the headache reduces their work and/or school productivity by at least 50%.
- The direct and indirect annual costs of migraines is approximately $13 billion (National Headache Foundation, 2005).

There are two types of migraine headaches: common migraine (without an aura) and classic migraine (with an aura; most often experienced as a visual disturbance prior to the

TABLE 44–5 **Comparison of Migraine, Cluster, and Tension Headaches**

TYPE	RISK FACTORS	FREQUENCY AND DURATION	DESCRIPTION	PRODROMAL AND ASSOCIATED MANIFESTATIONS
Migraine	Female Family history of migraine headache.	Episodic: ■ Tends to occur with stress and crisis. ■ Often correlates with menstrual cycle. ■ Can last hours to days.	Slow onset; pain becomes more severe, involving one side of head more than other.	Prodromal manifestations: visual defects, confusion, paresthesias. Associated manifestations: nausea, vomiting, chills, fatigue, irritability, sweating.
Cluster	Male Use of alcohol or nitrates. May begin in early childhood.	Episodes are clustered together in rapid succession for a few days or weeks with remissions that last for months. Can last a few minutes to a few hours.	May begin in infraorbital region and spread to head and neck; throbbing, deep pain, often unilateral.	Prodromal manifestations: uncommon. Associated manifestations: flushing, tearing of eyes, nasal congestion, sweating and swelling of temporal vessels.
Tension	Related to tension and anxiety. No family history. Often begins in adolescence.	Episodic: ■ Varies with amount of stress. ■ Duration also varies; can be constant.	Tight, pressing, viselike; may involve neck and shoulders.	Prodromal manifestations: uncommon. Associated manifestations: sustained contraction of neck muscles.

pain). Common migraines occur in 80% of the people who are affected by this disorder. Headaches classified as migraines may differ in intensity, duration, and frequency. The exact causes of migraine are not fully understood, but they are believed to be the result of abnormalities in cerebrovascular blood flow, a reduction in brain and electrical activity, or increased release of sensory substances such as serotonin, norepinephrine, substance P, nitric oxide, or glutamate.

A variety of factors are believed to trigger the onset of a migraine headache. Rapid changes in blood glucose levels, stress, emotional excitement, fatigue, hormonal changes due to menstruation, stimuli such as bright lights, and food high in tyramine or other vasoactive substances (e.g., aged cheese, nuts, chocolate, and alcoholic beverages) have been associated with migraine attacks. Hypertension and fever may make the disorder worse.

COMMON MIGRAINE This type is the most common and is associated with hereditary factors. The aura stage is absent; clients are aware only that a headache is eminent. The headache develops gradually, lasting hours to days, and may occur in women during periods of premenstrual tension and fluid retention. Chills, nausea and vomiting, fatigue, and nasal congestion are often present.

CLASSIC MIGRAINE The classic migraine headache has several stages, as follows:

- The aura stage is characterized by sensory manifestations, usually visual disturbances such as bright spots or flashing lights zigzagging across the visual fields. This stage lasts from 5 to 60 minutes. Less common sensory symptoms include numbness or tingling of the face or hand, weakness of an arm or leg, mild aphasia, confusion, drowsiness, and lack of coordination. Additionally, some clients experience a premonition the day prior to an attack. They may feel nervous or have other mood changes. The aura period corresponds with the initial physiologic change of vasoconstriction.
- The headache stage is characterized by vasodilation, a decline in serotonin levels, and the onset of throbbing headache. It appears that the pain is related to increased vessel permeability and polypeptide exudation by perivascular nerve endings rather than the vasodilation itself. Cerebral arteries are dilated and distended, with walls that are edematous and rigid. Beginning unilaterally, the headache eventually may involve both sides as it increases in intensity during the next several hours. Nausea and vomiting often occur. The client may be acutely ill and is often extremely irritable. The sensory organs often become hypersensitive, and the client withdraws from sound and light. The scalp is tender. The headache may last from several hours to a day or two.
- During the post-headache phase, the headache area is sensitive to touch, and a deep aching is present. The client is exhausted. Vessel size and serotonin levels return to normal.

Cluster Headache

A *cluster headache* is an extremely severe, unilateral, burning pain located behind or around the eyes. The cluster headache is predominantly experienced by men between the ages of 20 and 40 years. The headaches occur in groups or "clusters" of one to eight each day for several weeks or months, followed by remission lasting months to years (Hickey, 2003). The physiologic mechanism underlying cluster headaches is not well understood, but involves a vascular disorder, a disturbance of serotonergic mechanisms, a sympathetic defect, or dysregulation of the hypothalamus.

Although the headache may occur at any time, it typically begins 2 to 3 hours after falling asleep, awakens the person, and then lasts from 15 to 180 minutes. Prodromal signs are absent. Intense unilateral pain around or behind one eye is accompanied by rhinorrhea, lacrimation, flushing, sweating, facial edema, and possible miosis or ptosis on the affected side. The same side of the head is involved in each cluster of attacks.

The headaches often occur in the spring and fall and then disappear for an extended period. Attacks may be triggered by drinking alcohol, eating specific foods, medications such as nitroglycerin, or there may be no known precipitating event.

Tension Headache

Tension headache is characterized by bilateral pain, with a sensation of a band of tightness or pressure around the head. Sharply localized painful spots (trigger points) may be present. The onset is gradual, and the intensity, frequency, and duration of the attack vary greatly. This type of headache is caused by sustained contraction of the muscles of the head and neck. It is often precipitated by stressful situations and anxiety. Secondary causes include prolonged computer use and disorders of the eyes, ears, sinuses, or cervical vertebrae. Abnormal posture associated with occupations that require bending over a desk (e.g., office workers, students) often precipitates tension-type headache. Additionally, slouching while reading or watching television can lead to muscle contraction. Most headaches are tension-type headaches.

INTERDISCIPLINARY CARE

Identifying the underlying cause(s) of the headache is the initial focus of interdisciplinary care. If the underlying cause is treatable, the headache will often decrease or disappear. An accurate diagnosis of the type of headache is key to the treatment.

Therapeutic management for migraine headache includes a combination of client teaching, medications, and measures to control contributing factors. Dietary changes such as eliminating caffeine, cured meats, monosodium glutamate (MSG), and foods containing tyramine (red wine, aged cheese, and others) may be necessary. Stress management or biofeedback is also part of the overall strategy. Treatment protocols for cluster headache include eliminating aggravating factors (e.g., consumption of alcohol) and using medications and oxygen inhalation. The management of tension headaches is directed toward reducing the client's level of stress and relieving pain with ice and aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs).

Diagnosis

Diagnosis and treatment are based on history, identifying triggering or precipitating events, and the type of headache. A thorough history and physical examination are integral parts of the assessment. Neurodiagnostic testing may be done to rule out a structural disease process. Testing may include a brain scan,

MRI, x-ray studies of the skull and cervical spine, EEG, or lumbar puncture for CSF if inflammation is suspected. Serum metabolic screens and hypersensitivity testing also may be performed if systemic problems are suspected.

Medications

Pharmacologic management depends on the type of headache. The goals of treatment are to reduce the frequency and severity of headaches and to limit or relieve a headache that is beginning or in progress.

The management of migraine headache includes administering medications to prevent pain (prophylactic therapy) as well as drugs to stop (or abort) a headache in progress. The client with frequent migraine headaches is a candidate for prophylactic therapy. Drugs used to reduce the frequency and severity of migraine follow:

- Methysergide maleate (Sansert) is a serotonin antagonist that competitively blocks serotonin receptors in the CNS and is also a potent vasoconstrictor.
- Propranolol hydrochloride (Inderal) is a beta-blocker that prevents dilation of vessels in the pia mater and inhibits serotonin uptake.
- Topiramate (Topamax) and valproic acid (Depakote) are CNS agents and anticonvulsants. They have been approved by the FDA for use to prevent migraines (National Headache Foundation, 2005).

When the manifestations of migraine are recognized early, several medications may be used to abort or limit the severity and duration of the headache. Ergotamine tartrate (Cafergot) is a complex drug that reduces extracranial blood flow, decreases the amplitude of cranial artery pulsation, and decreases basal artery hyperperfusion. Administered at the onset of an attack, ergotamine controls up to 70% of acute attacks. Sumatriptan (Imitrex) is available in oral, nasal spray, or subcutaneous injection forms. It binds with serotonin$_1$ receptors and is rapidly effective. Zolmitriptan (Zomig), a selective serotonin$_1$ receptor agonist, is administered orally and is effective in the treatment of acute headache. Once a migraine is in progress, a narcotic analgesic such as codeine or meperidine (Demerol) may be required. Antiemetics may be prescribed to control nausea and vomiting.

Many of the same medications used for migraine also prevent or treat cluster headache. Because the onset of cluster headaches is abrupt, abortive therapy is not possible. Medications such as ergotamine tartrate may be given in suppository form at bedtime to prevent headache during the episodic attacks. Clients may find that inhaling 100% oxygen at 7 L/min for 15 minutes at the onset of an attack relieves their headache (Tierney et al., 2005).

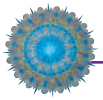
Nonnarcotic analgesics such as aspirin or acetaminophen may relieve tension headaches. Additionally, tranquilizers such as diazepam may reduce muscle tension.

Nursing implications for drugs commonly prescribed for headaches are described in the Medication Administration on the next page.

Alternative and Complementary Therapies

The following alternative and complementary therapies are used to relieve the pain of headaches.

- Vitamin D, elemental calcium, riboflavin (vitamin B), and magnesium
- Acupuncture
- Relaxation, guided imagery, massage
- Melatonin, 5-HTP, CoQ10
- Magnetic field therapy
- Herbal therapy
- Osteopathic manipulation



NURSING CARE

In addition to the nursing care discussed in this section, a Nursing Care Plan for a client with migraine headaches is found on page 1547.

Health Promotion

Teach clients with tension headaches relaxation techniques, such as massage and biofeedback. Counseling for chronic anxiety may also be helpful. Triggers for migraine or cluster headache should be identified and, if possible, eliminated. For example, avoiding physical and emotional stress, having regular and consistent sleep patterns, eating meals regularly, and avoiding specific foods or alcohol can be incorporated into daily life and are helpful. Specific suggestions are outlined in Box 44–2.

Assessment

Collect the following data through the health history and physical examination.

- *Health history:* History of intracerebral trauma, tumor, or infection; detailed history and description of headache characteristics; family history; triggering factors; usual diet; effects of recurring headaches on lifestyle, activities of daily living (ADLs), and role performance.
- *Physical assessment:* Skin (diaphoresis, pallor, flushing), eyes (sensitivity to light, tearing), muscle strength and movement.

Nursing Diagnoses and Interventions

The primary response of the client requiring nursing interventions is acute pain. Develop nursing interventions to help the client identify strategies for controlling the pain and discomfort of the headache.

BOX 44–2 Suggestions to Decrease Incidence of Migraine Headaches

- Wake up at the same time each morning.
- Exercise at least three times a week.
- No smoking or caffeine after 3 P.M.
- No artificial sweeteners.
- No MSG.
- Reduce or eliminate red wine, cheese, alcohol, chocolate, and caffeine.
- Try a gluten-free diet.

MEDICATION ADMINISTRATION Headaches

BETA-BLOCKERS

Propanolol hydrochloride (Inderal)
Nadolol (Corgard)
Atenolol (Tenormin)
Timolol maleate (Blocadren)

Beta-blockers are effective in the prophylactic treatment of headache. They act by combining with beta-adrenergic receptors to block the response to sympathetic nerve impulses, circulating catecholamines, or adrenergic drugs.

Nursing Responsibilities

- Before beginning therapy, determine pulse and blood pressure in both arms with client lying, sitting, and standing.
- Assess baseline and monitor serum glucose level, CBC, electrolytes, and liver and renal function studies.
- Note any history of diabetes or impaired renal function.
- Note the rate and quality of respirations; drugs in this category may cause dyspnea and bronchospasm.
- Administer the drug with meals to prevent gastrointestinal disturbances.
- Be alert that beta-blockers cause bradycardia and the heart rate may not rise in response to stress, such as exercise or fever. Notify the primary healthcare provider if pulse falls below 50 or if blood pressure changes significantly.
- Teach the client or family member how to take a pulse and blood pressure reading.

Health Education for the Client and Family

- Take the medication with meals to provide a coating for the gastrointestinal tract and prevent gastrointestinal disturbances.
- Return for blood work as prescribed.
- Take the last dose of the day at bedtime.
- Rise from a sitting or lying position to a standing position slowly to avoid dizziness and falls.
- Take pulse and blood pressure each day and maintain a record of readings.
- Avoid excessive intake of alcohol, coffee, tea, or cola. Consult with the healthcare provider before taking any over-the-counter medications.
- Report any cough, nasal stuffiness, or feelings of depression to the healthcare provider.

TRICYCLIC ANTIDEPRESSANTS

Imipramine hydrochloride (Tofranil)
Amitriptyline hydrochloride (Elavil)

The tricyclic antidepressants have been successful in the prophylaxis of cluster and migraine headaches. Although the exact mechanism is not known, they do prevent the reuptake of norepinephrine or serotonin, or both. They are chemically related to the phenothiazines, and as such they exhibit many of the same pharmacologic effects (e.g., anticholinergic, antiserotonin, sedative, antihistaminic, and hypotensive effects).

NURSING RESPONSIBILITIES

- Assess baseline CBC and liver function studies, heart sounds, and neurologic status before initiating prescribed therapy.

Health Education for the Client and Family

- Make position changes slowly.
- Chew sugarless gum to relieve dry mouth.
- Do not abruptly quit taking the medication.

ERGOT ALKALOID DERIVATIVES

Methysergide maleate (Sansert)

Methysergide is an ergot alkaloid derivative structurally related to LSD. It acts by stimulating smooth muscle, leading to vasoconstriction. It is thought that methysergide prevents headaches by blocking the effects of serotonin, a powerful vasodilator believed to play a role in vascular headaches. It also inhibits the release of histamine from mast cells and prevents the release of serotonin from platelets.

Nursing Responsibilities

- Note any history of renal or hepatic disease.
- Assess baseline eosinophil and neutrophil counts before beginning therapy.
- Administer the drug with meals or milk to minimize gastrointestinal irritation due to increased hydrochloric acid production.
- Assess for renal, CNS, and cardiovascular complications.
- Drug dosage should be gradually reduced over 2 to 3 weeks to prevent rebound headaches. A drug-free interval of 3 to 4 weeks is required with each 6-month course of therapy to prevent complications.
- Monitor for signs of ergotism, such as coldness or numbness of the fingers and toes, nausea, vomiting, headache, muscle pain, and weakness. Vasoconstriction may further impair peripheral circulation and increase blood pressure.

Health Education for the Client and Family

- Take the medication with meals or milk to minimize gastrointestinal upset.
- Report to the primary care provider nervousness, weakness, rashes, hair loss, or swelling of the extremities.
- Weigh daily and report any unusual weight gain to the primary care provider.
- Return to the primary care provider for a checkup at least every 6 months or as instructed. Do not take the drug on a regular basis for longer than 6 months, but do not abruptly stop taking it.
- Return for follow-up blood work as ordered.

SEROTONIN SELECTIVE AGONIST

Sumatriptan succinate (Imitrex)
Zolmitriptan (Zomig)
Rizatriptan Benzoate (Maxalt)

Binds to vascular receptors to vasoconstrict cranial blood vessels and relieve migraine headache.

Nursing Responsibilities

- Assess for history of peripheral vascular disease, renal or hepatic problems, and pregnancy.
- Evaluate relief of migraine headache, and assess for side effects of photophobia, sound sensitivity, and nausea and vomiting.

Health Education for the Client and Family

- Do not use more than two injections in a 24-hour period, and allow at least 1 hour between injections.
- Use the autoinjector to administer the medication, and follow instructions for proper method of giving the injection and disposing of the syringe.
- Report wheezing, heart palpitations, skin rash, swelling of the eyelids or face, or chest pain to the healthcare provider immediately.

CALCIUM CHANNEL BLOCKERS

Verapamil (Isoptin)
Nifedipine (Procardia)

(continued)

MEDICATION ADMINISTRATION Headaches (continued)

The calcium channel blockers may have value in controlling cerebral vasospasms by two mechanisms: inhibiting the influx of calcium into the cerebral artery and interfering with the destruction of erythrocytes and aggregation of platelets.

Nursing Responsibilities

- These drugs cause peripheral vasodilation. Therefore, monitor blood pressure and pulse during the initial administration of the drug. Any excessive hypotensive response and tachycardia may precipitate angina. Request written parameters for safe drug administration.
- Monitor intake and output and daily weights. Assess for manifestations of congestive heart failure: weight gain, peripheral edema, dyspnea, rales, and jugular vein distention.
- Teach client and family members how to take pulse and blood pressure readings.

Health Education for the Client and Family

- Take the medication with meals to reduce gastrointestinal irritation.
- Take pulse and blood pressure before taking medications each day at the same time, and follow instructions regarding when to withhold medication and when to contact the provider. Keep a record of pulse and blood pressure readings.
- Report any side effects, such as dizziness, vertigo, unusual flushing, facial, warmth, or headaches, to the primary care provider.
- Report immediately any swelling of the hands or feet, pronounced dizziness, or chest pain accompanied by sweating, shortness of breath, or severe headaches.

NONSTEROIDAL ANTI-INFLAMMATORY DRUG (NSAID): SALICYLATE

Acetylsalicylic acid (Ecotrin, Bufferin)

Acetylsalicylic acid, or aspirin, is a nonnarcotic analgesic, antipyretic, anti-inflammatory agent used to relieve headache pain.

Nursing Responsibilities

- Determine the type and pattern of pain. If aspirin was used in the past for pain control, note its effectiveness.
- Note any history of peptic ulcers or other conditions that may suggest potential problems with the use of salicylates.
- Assess clients receiving anticoagulant therapy for bruises, bleeding of the mucous membranes, or blood in the urine or stool.

Health Education for the Client and Family

- Take aspirin after meals or before meals with an antacid and a full glass of water to minimize gastric irritation.
- Report ringing in the ears, unusual bleeding of gums, bruising, or black tarry stools to the primary healthcare provider.
- Monitor blood glucose levels carefully (if you have diabetes), and report hypoglycemia if it occurs.

ERGOTAMINE

Caffeine-ergotamine tartrate combination (Cafergot)
Ergotamine tartrate (Gynergen)

Ergot alkaloids vasoconstrict the cerebral blood vessels, decreasing the amplitude of the pulsations of the cranial arteries. The major use of ergot alkaloids is the treatment of migraine headaches. Cafergot has the same actions as Gynergen, in addition, the caffeine it contains provides a vasoconstrictive action, enhancing the effects of ergotamine.

Nursing Responsibilities

- Because the drug accumulates in the body and is eliminated slowly, ergotamine poisoning may occur. Sepsis, renal and vascular disease, heavy smoking, malnutrition, pregnancy, contraceptive hormones, and fever can increase the risk of ergotamine poisoning.
- These drugs are contraindicated in clients with diabetes mellitus, sepsis, hepatic or renal disease, peripheral and coronary artery disease, hypertension, and pregnancy.

Health Education for the Client and Family

- Take the drug immediately at onset of headache.
- Report the following to your healthcare provider: pain in the leg muscles, weakness, and coldness or numbness of fingers or toes.
- A dose of Cafergot taken late in the day may prevent sleep because of the effects of caffeine.

Acute Pain

Headaches originate from both intracranial and extracranial sources and range in severity from benign, transient discomfort to severe, incapacitating pain. Interventions focus on teaching the client self-care measures to control or relieve the pain, and reducing any associated problems, such as nausea and vomiting or anxiety.

- Teach to maintain a diary of headaches, including duration, onset, location, relation to menstruation or food intake, and related manifestations such as factors that relieve or intensify the pain. *A thorough assessment of the headache is essential for both the client and the healthcare provider to identify the circumstances and patterns of headache occurrence.*
- Ask the client to rate the pain or discomfort on a scale of 0 to 10 (with 10 being the worst pain). *Using a scale to rate the pain provides an objective measure of the client's subjective experience of the pain or discomfort. The scale can also be used to evaluate the effectiveness of pain relief measures.*
- Teach to minimize light, noise, and activity and rest in a quiet, nonstimulating environment when experiencing a headache. *Manipulating the environment helps reduce noxious stimuli that may increase pain.*
- Teach to use noninvasive and nonpharmacologic pain relief measures such as deep breathing or relaxation to facilitate self-management of pain (see Chapter 9 ∞). *Alternative strategies to control pain can help reduce tension and may help to increase the client's sense of control over the pain.*
- Suggest application of cold compresses or dry heat to the head and neck. *The application of cold causes vasoconstriction, which helps reduce pain in vascular headaches. Application of heat can reduce muscle tension and improve circulation.*
- Teach to follow good nutrition guidelines, get regular exercise and sleep, and minimize stress. *Headaches are more likely to occur when ill, tired, or under stress.*

NURSING CARE PLAN A Client with a Migraine Headache

Betty Friedman is a 25-year-old grade-school teacher. Her friends and the other teachers regard Ms. Friedman as an enthusiastic person who sets high standards for herself and strives for perfection. During the spring semester, Ms. Friedman begins to miss work and sometimes appears very nervous. One day, another teacher notices Ms. Friedman running down the hall and into the restroom; the teacher finds Ms. Friedman vomiting. As she washes up, Ms. Friedman tells the other teacher that she has been having headaches since she began menstruating, but that they have never been as intense and frequent as during this past year. Ms. Friedman agrees to see the nurse practitioner, Jane Schickadanz, at the school clinic for evaluation.

ASSESSMENT

During her health history, Ms. Friedman relates that each month before her menstrual cycle she becomes nervous and sees flashing lights. She also has difficulty expressing herself and thinking clearly. The next day she develops a "sick headache." She states that the headache can last 1 to 2 days and that afterwards she cannot brush her hair because her scalp hurts. Ms. Friedman attributes these symptoms to PMS and adds that she thinks she is allergic to cheese and nuts because she gets sick after eating them. After assessment, and in consultation with the physician, Ms. Schickadanz diagnoses Ms. Friedman's problem as a migraine with aura headache. Sumatriptan succinate (Imitrex) is prescribed.

DIAGNOSES

- *Acute Pain* related to vasodilation of cerebral vessels and a decreased serotonin level
- *Deficient Knowledge* of pain management
- *Altered Role Performance* related to pain

EXPECTED OUTCOMES

- Experience reduced frequency and duration of pain.
- Identify available resources for helping with self-management of pain.

PLANNING AND IMPLEMENTATION

- Ask to keep a diary of her headaches for the next month, noting times of their occurrence, location and duration of pain, and factors that trigger the onset, such as her menstrual period or certain foods.
- Teach to take medication at the first awareness of an impending attack.
- Suggest an appointment with a counselor to learn methods of relaxation and stress relief.
- Request dietary referral for elimination of foods that might precipitate headaches.

EVALUATION

Four weeks after beginning medication therapy with Imitrex and relaxation techniques, Ms. Friedman has noted a decrease in the intensity of the headaches. She reports that the medication has stopped the headaches, which, she has noted, tend to occur more frequently immediately before her menstrual period. She is walking for 30 minutes each day and has made changes in her usual diet. Ms. Friedman states, "I feel good about going to work with my kids at school and knowing I can control my pain."

CRITICAL THINKING IN THE NURSING PROCESS

1. List the questions you would include in a health history that would identify stressors consistent with migraine headaches.
2. Develop a teaching plan for Ms. Friedman that includes methods of reducing fluid retention before her menstrual period, as well as a suggested diet based on the food guide pyramid.
3. Design a plan of care for Ms. Friedman for the nursing diagnosis *Disturbed Sleep Pattern.*

See Evaluating Your Response in Appendix C.

Community-Based Care

In addition to implementing comfort measures, client education has a high priority. Develop a teaching plan to help the client learn how to limit attacks (e.g., by avoiding precipitating factors) and reduce the effects of the headache. Provide specific information about prescribed medications. Referrals for methods of stress reduction may be necessary for clients with long-term or migraine headaches.

THE CLIENT WITH EPILEPSY

Epilepsy (also called *seizure disorder*) is a chronic disorder of abnormal recurring, excessive, and self-terminating electrical discharge from neurons. Epilepsy is characterized by recurring seizures accompanied by some type of change in behavior. A **seizure** (sometimes called a convulsion) is a single event of abnormal electrical discharge in the brain resulting in an abrupt and temporary altered state of cerebral function (Hickey, 2003). This abnormal neuronal activity, which may involve all or part of the brain, disturbs skeletal motor function, sensation, autonomic function of the viscera, behavior, and/or consciousness. Epilepsy is categorized as a paroxysmal disorder because its manifestations are discontinuous; that is, minutes, days, weeks, or even years may elapse between seizures.

Incidence and Prevalence

Epilepsy is one of the most common neurologic conditions, affecting up to 2.5 million people in the United States. There is a strong genetic component. Although people of any age may be affected, the prevalence and incidence of epilepsy increases dramatically in older adults (see the Meeting Individualized Needs box on page 1548).

FAST FACTS

Epilepsy

- Epilepsy affects people of all ages, races, and ethnic backgrounds.
- Every year, more than 181,000 people develop epilepsy for the first time (Epilepsy Foundation, 2005a).
- Epilepsy is not a mental disorder.

MEETING INDIVIDUALIZED NEEDS **Epilepsy in Older Adults**

For years, epilepsy was believed to be a disease that only affected children. Research has now found that the incidence of epilepsy in adults age 75 and over is higher than in the first 10 years of life, with 7% of all older adults having epilepsy (Spitz, 2005). These data have important implications for nursing assessments and care.

- The most common cause of epilepsy in older adults is arteriosclerosis of the cerebrovascular system (with up to 80% of the older population having arteriosclerosis).
- The manifestations of epilepsy in older adults are different than in younger adults and children. Although 60% of younger people have generalized tonic-clonic seizures, only 30% of older adults have generalized tonic-clonic seizures. The most common type of seizure in older adults is a complex partial seizure.
- Older adults tend to have longer postseizure manifestations than do younger adults.
- Epilepsy that begins in older adults is often easier to control with antiepileptic drugs (AEDs) than that in younger people. However, some AEDs decrease the effect of statins used to treat arteriosclerosis (the most common cause of epilepsy in the elderly).

The incidence of epilepsy is increasing. Researchers have suggested that the increase may be due to technologic advances in obstetric and pediatric care that allow extremely high-risk neonates to survive and to other technologic advances that have improved survival rates after craniocerebral trauma.

Isolated seizure episodes may occur in otherwise healthy people for a variety of reasons, including an acute febrile state, infection, metabolic or endocrine disorder (such as hypoglycemia), or exposure to toxins. Epilepsy may be idiopathic (that is, it may have no identifiable cause), or it may be secondary to birth injury, infection, vascular abnormalities, trauma, or tumors.

Pathophysiology

Normally, when the mind is actively working, electrical activity in the brain is unsynchronized; when the mind is at rest, electrical activity is mildly synchronized. It is believed that most seizures arise from a few unstable, hypersensitive, and hyperreactive neurons in the brain. During a seizure, these neurons produce a rhythmic and repetitive hypersynchronous discharge. Although the exact initiating factor for seizure activity has not been identified, several theories have been proposed (Porth, 2005):

- Alterations in the permeability of, or ion distribution across, cell membranes
- Alterations in the excitability of neurons resulting from neuroglia (neuroglia are CNS connective or supporting cells; they include astrocytes, oligodendroglia, and microglia)
- Scarring or decreased inhibition of activity in the cerebral cortex or thalamic region
- Imbalances of excitatory and inhibitory neurotransmitters such as acetylcholine (ACh) or gamma aminobutyric acid (GABA).

All people have a seizure threshold; when this threshold is exceeded, a seizure may result. In some people, the seizure threshold may be abnormally low, increasing their risk for seizure activity; in other people pathologic processes may alter the seizure threshold (Porth, 2005). The neurons that initiate seizure activity are called the *epileptogenic focus*. Abnormal neuronal activity may remain localized, causing a partial or focal seizure, or it may spread to involve the entire brain, causing generalized seizure activity.

Seizures may also be provoked or unprovoked. Unprovoked (primary or idiopathic) seizures have no identifiable cause, with multiple episodes diagnosed as a seizure disorder. Provoked (secondary) seizure etiologies include febrile seizures in children, toxemia of pregnancy, rapid withdrawal from alcohol or barbiturates, systemic metabolic conditions (such as hypoglycemia, hypoxia, uremia, and electrolyte imbalances), and pathologies of the brain (such as meningitis, cerebral bleeding, or cerebral edema).

Metabolic needs of the brain increase dramatically during seizure activity. The demand for adenosine triphosphate (ATP), the energy source of the brain, increases by approximately 250%. Consequently, the demand for glucose and oxygen (which are needed to produce ATP) increases, and oxygen consumption increases by about 60%. To supply this increased oxygen need and remove carbon dioxide and other metabolic by-products, cerebral blood flow increases to about 2.5 times that of the normal rate. As long as oxygenation, blood glucose levels, and cardiac function remain normal, cerebral blood flow can respond to this increased metabolic demand of the brain. If cerebral blood flow cannot meet these needs, however, cellular exhaustion and cellular destruction may result.

Manifestations

Although seizures may be categorized in several different ways, the classification developed by the International League Against Epilepsy is the most useful clinically (Tierney et al., 2005). In this classification, seizures are divided into those that affect only part of the brain (partial seizures) and those that are generalized (affect all of the brain). An individual may have more than one type, in what are called mixed seizures.

Partial Seizures

Partial seizures involve the activation of only a restricted part of one cerebral hemisphere. A partial seizure accompanied by no alteration in consciousness is called a *simple partial seizure*; one in which consciousness is impaired is called a *complex partial seizure.*

SIMPLE PARTIAL SEIZURES The manifestations of simple partial seizures depend on the involved area of the brain. Manifestations may include alterations in motor function, sensory signs, or autonomic or psychic symptoms. Typically, the motor

portion of the cortex is affected, causing recurrent muscle contractions of the face or a contralateral part of the body, such as a finger or hand. This motor activity may stay confined to one area or spread sequentially to adjacent parts, a phenomenon known as a *Jacksonian march* or *Jacksonian seizure*. Manifestations of a simple partial seizure involving the sensory portion of the brain may include abnormal sensations or hallucinations. Disruptions in the function of the autonomic nervous system, with resulting tachycardia, flushing, hypotension, and hypertension, or psychic manifestations, such as a sense of déjà vu (a feeling that "this has happened before") or inappropriate fear or anger, may also be experienced during a simple partial seizure.

COMPLEX PARTIAL SEIZURES During a complex partial seizure, consciousness is impaired and the client may engage in repetitive, nonpurposeful activity, such as lip smacking, aimless walking, or picking at clothing. These behaviors are known as *automatisms*. During the seizure, the client loses conscious contact with the environment; amnesia is common after the seizure, and several hours may elapse before the client regains full consciousness. Complex partial seizures usually originate in the temporal lobe and may be preceded by an aura, such as an unusual smell, a sense of déjà vu, or a sudden intense emotion.

Generalized Seizures

Generalized seizures involve both hemispheres of the brain as well as deeper brain structures, such as the thalamus, basal ganglia, and upper brainstem. Consciousness is always impaired with generalized seizures. Absence and tonic-clonic seizures are the common forms of generalized seizure activity; they occur more frequently (especially in children) than partial seizures.

ABSENCE SEIZURES *Absence (petit mal) seizures* are characterized by a sudden brief cessation of all motor activity accompanied by a blank stare and unresponsiveness. Absence seizures are more common in children than in adults. The seizure typically lasts only 5 to 10 seconds, although some may last for 30 seconds or more. Movements such as eyelid fluttering or automatisms such as lip smacking may occur during an absence seizure. Seizure activity may vary from occasional episodes to several hundred per day.

TONIC-CLONIC SEIZURES *Tonic-clonic seizures* (grand mal) are the most common type of seizure activity in adults. This type of seizure activity follows a typical pattern. A warning *aura* may precede generalized seizure activity. The aura may be a vague sense of uneasiness or an abnormal gustatory, visual, auditory, or visceral sensation (such as a metallic taste in the mouth, a smell of burning rubber, or seeing a bright light). Often, however, the seizure occurs without warning.

The seizure begins with a sudden loss of consciousness and sharp tonic muscle contractions (the tonic phase of the seizure). With the muscle contraction, air is forced out of the lungs, and the client may cry out. Postural control is lost, and the client falls to the floor in the opisthotonic posture (Figure 44–4A ■). Muscles are rigid, with the arms and legs extended and the jaw clenched. Urinary incontinence is common; bowel incontinence may also occur. Breathing ceases and cyanosis develops during the tonic phase of a seizure. The pupils are fixed and dilated. The tonic phase lasts an average of 15 seconds, although it may persist for up to a minute.

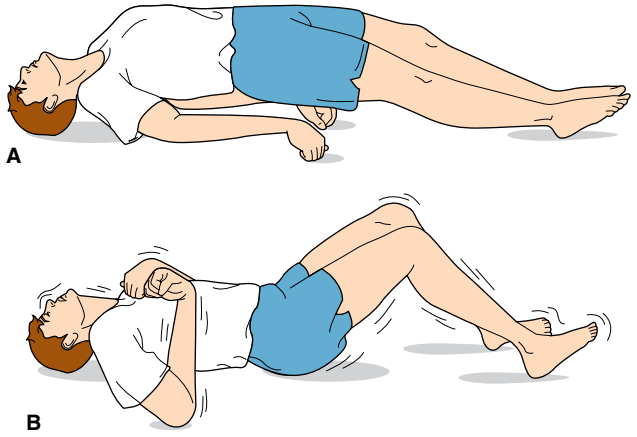


Figure 44–4 ■ Tonic-clonic seizures in grand mal seizures. *A*, Tonic phase. *B*, Clonic phase.

The clonic phase, which follows the tonic phase, is characterized by alternating contraction and relaxation of the muscles in all the extremities along with hyperventilation (Figure 44–4B). The eyes roll back, and the client froths at the mouth. The clonic phase varies in duration and subsides gradually. The entire tonic-clonic portion of the seizure generally lasts no more than 60 to 90 seconds.

Following the clonic phase of seizure activity, the client remains unconscious and unresponsive to stimuli. This period is known as the postictal period or phase. The client is relaxed and breathes quietly. The client regains consciousness gradually and may be confused and disoriented on waking. Headache, muscle aches, and fatigue often follow the seizure, and the client may sleep for several hours. Amnesia of the seizure is usual; the client also may not recall events just prior to the seizure activity.

Because of the lack of warning with tonic-clonic seizures, the client may experience injury. Head injury, fractures, burns, or motor vehicle crashes may occur secondarily to seizure activity.

Status Epilepticus

Status epilepticus can develop during seizure activity. In this case, the seizure activity becomes continuous, with only very short periods of calm between intense and persistent seizures. The repetitive seizures may be of any type, although they are usually generalized tonic-clonic (Porth, 2005). Repeated seizures have a cumulative effect, producing muscular contractions that can interfere with respirations. The client is in great danger of developing hypoxia, acidosis, hypoglycemia, hyperthermia, and exhaustion if the convulsive activity is not halted. Status epilepticus is considered a life-threatening medical emergency that requires immediate treatment.

INTERDISCIPLINARY CARE

Initial treatment focuses on controlling the seizure; the long-term goal is to determine the cause and prevent future seizures.

Interdisciplinary care includes diagnostic testing, medications, and, in some cases, surgery.

Diagnosis

Diagnostic testing is performed to confirm the seizure diagnosis and to determine any treatable causes and precipitating factors. (See Chapter 43 ∞ for a description of neurologic tests and related nursing care.) Radiologic examinations include an MRI or CT scan to determine abnormalities in the brain and a skull x-ray to identify any bony abnormalities. An electroencephalogram (EEG) helps localize any brain lesions and confirm the diagnosis. A lumbar puncture may be performed to assess spinal fluid for CNS infections (increased WBCs) or tumors (increased protein levels). Blood studies are used to assess blood count, electrolytes, blood urea, and blood glucose.

Medications

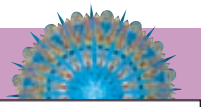
Antiepileptic drugs (AEDs) (also called anticonvulsant drugs) can reduce or control most seizure activity. More than 20 drugs are used in the treatment of epilepsy. These medications do not cure the disorder; they only manage its manifestations. AEDs generally act in one of two ways: by raising the seizure threshold or by limiting the spread of abnormal activity within the brain.

The goals of medications for epilepsy are to protect the client from harm and to reduce or prevent seizure activity without impairing cognitive function or producing undesirable side effects. Ideally, the lowest possible dose of a single medication that will control the client's seizures is prescribed; often, however, several medications must be tried before the most effective is identified, and a combination of drugs may be needed to manage the client's seizures. Therapy is individualized, based on the type of seizure activity and the client's response to the medication. Four drugs are recommended by the American Academy of Neurology as monotherapy (taking one drug at a time) for newly diagnosed adults with either partial or mixed seizures. These are gabapentin (Neurontin), lamotrigine (Lamictal), oxcarbazepine (Trileptal), and topiramate (Topamax). Examples of, and nursing implications for AEDs are described in the Medication Administration below; drug interactions are listed in Box 44–3.

If the client has been seizure free for at least 3 years, withdrawal of medications may be considered, with the dose of one drug at a time reduced over weeks or months. There is no way to predict which clients can remain seizure free without medication, but if seizures reoccur, the same medications usually provide good control.

Status epilepticus requires immediate intervention to preserve life. Establishing and maintaining the airway is a priority. A solution of 50% dextrose is administered intravenously to prevent hypoglycemia. Diazepam (Valium) or lorazepam (Ativan) is given intravenously, and the dose repeated in 10 minutes

MEDICATION ADMINISTRATION **Seizures**



ANTIEPILEPTIC DRUGS (AEDS)

Examples of AEDs are:

Phenytoin (Dilantin)
Phenobarbital
Primidone (Mysoline)
Carbamazepine (Tegretol)
Valproic acid (Depakene)
Ethosuximide (Zarontin)
Clonazepam (Klonopin)
Gabapentin (Neurontin)
Lamotrigine (Lamictal)
Tiagabine HCL (Gabitril)

AEDs are used to control chronic seizures and involuntary muscle spasms or movements characteristic of certain neurologic diseases. These drugs act in the motor cortex of the brain to reduce the spread of electrical discharges from the rapidly firing epileptic foci in this area. These agents control seizures without impairing the normal functions of the CNS. Drugs effective against one type of seizure may not be effective against another; anticonvulsant therapy must be individualized.

Nursing Responsibilities

- Monitor blood pressure, pulse, and respirations.
- Note evidence of CNS side effects, such as blurred vision, dimmed vision, slurred speech, nystagmus, or confusion. Gingival hyperplasia may be noted in clients taking phenytoin.
- Recognize that if clients are to be on prolonged therapy, they may need a diet rich in vitamin D.
- Monitor the serum calcium level as ordered; phenytoin can contribute to demineralization of bone.
- When administering anticonvulsants intravenously, monitor closely for respiratory depression and cardiovascular collapse.
- Administer gabapentin 2 hours after antacids.
- Administer tiagabine HCL with food.

Health Education for the Client and Family

- Take the exact dosage prescribed. Do not increase, decrease, or discontinue the dosage without obtaining the primary care provider's approval; doing so may lead to convulsions.
- Avoid hazardous tasks until the drug has been regulated. AEDs may at first decrease mental alertness and cause drowsiness, headache, dizziness, and incoordination of muscles. These effects are usually dose related and may disappear with a change of dosage or continued therapy.
- If you are taking phenytoin (Dilantin), maintain good oral hygiene: Use a soft toothbrush, massage the gums, and floss daily.
- It is very important to obtain liver function studies regularly as ordered by the primary care provider. This will help detect early signs of hepatitis and other liver problems. Report for all scheduled laboratory studies, including complete blood count, kidney and liver function studies, and drug levels.
- Carry identification indicating the type of seizures for which you are being treated.
- Do not take gabapentin 1 hour before or less than 2 hours after an antacid.
- If you are taking lamotrigine and develop a rash, tell your healthcare provider.
- Take tiagabine HCL (Gabitril) with food.

BOX 44–3 Drug Interactions with AEDs

- *Valproic acid (Depakene) and phenobarbital.* Blood levels of phenobarbital may rise significantly when valproic acid is added to the client's medication regimen.
- *Phenobarbital and digoxin*. This combination may increase the metabolism of digoxin, resulting in decreased digoxin levels.
- *Phenobarbital and sodium warfarin (Coumadin).* Phenobarbital may decrease the absorption of sodium warfarin from the gastrointestinal tract and decrease the drug's anticoagulant response.
- *Disulfiram (Antabuse) and phenobarbital.* This combination may inhibit the metabolism of the anticonvulsant drug and increase the incidence of side effects associated with the anticonvulsant drug.
- *Carbamazepine and oral contraceptives.* Carbamazepine decreases the effectiveness of oral contraceptives.
- *Other drugs.* Other drugs reported to interact with anticonvulsant drugs include aspirin, certain antibiotics, isoniazid, acetazolamide (Diamox), antacids, folic acid, and narcotics.

if necessary to stop seizure activity. Phenytoin (Dilantin) is administered intravenously for longer term control of seizures. Phenobarbital may also be administered to clients in status epilepticus.

Surgery

Resective surgery, with removal of the epileptogenic focus, is an option that is still in its early stages. Candidates for this type of surgery include those who are unresponsive to medical management, who have a unilateral focus, and who have impaired quality of life from seizures. Resections of the temporal lobe are most commonly performed and are most effective for partial complex seizures. An estimated 5% of clients with epilepsy may be candidates for surgery. The goal of surgery is to reduce the client's uncontrollable seizures.

To be selected as a candidate for surgery, the client must be highly motivated and psychologically prepared. A psychologic screening is required because the preoperative preparation is extensive and time consuming and because the surgery is long and requires that the client remain awake during surgery so that he or she can cooperate and respond to commands. The EEG is monitored during surgery to identify the epileptogenic focus and evaluate the effect of surgical intervention.

General postoperative care for the client with intracranial surgery follows the nursing management guidelines outlined later in the chapter. Specific preoperative and postoperative care for a client with a seizure disorder is described in the box below.

Vagal Nerve Stimulation Therapy

Vagal nerve stimulation (VNS) therapy is approved as a treatment for clients with partial-onset seizures who do not respond to AEDs. The therapy does not stop the seizures, but rather reduces the number and improves the client's quality of life. It is almost always necessary to continue taking AEDs. The therapy is designed to prevent seizures by sending regular small pulses of electrical energy to the brain via the vagus nerve. A flat, round battery (about the size of a silver dollar) is implanted in the chest wall, and electrodes are threaded under the skin and wound around the vagus nerve in the neck. The battery is programmed to deliver a few seconds of electrical energy every few seconds. If the person feels that a seizure is about to happen, a discharge can be activated by passing a small magnet over the battery. In some people this stops the seizures. Side effects are hoarseness and throat discomfort.

NURSING CARE

See the next page for a Nursing Care Plan for a client with seizures.

Health Promotion

Health promotion activities for the client with seizures focus on teaching to reduce the incidence of seizure activity and to promote safety. Stress the following:

- Know the importance of follow-up care, of keeping medical appointments, and of continuing to take AEDs as prescribed even when no seizures are experienced.
- Review any state and local laws that apply to people with seizure disorders. Driving a motor vehicle is usually prohibited for 6 months to 2 years after a seizure episode. Usually, a driver's license can be reinstated or obtained after a seizure-free period and a letter from the nurse practitioner or physician.

NURSING CARE OF THE CLIENT WITH Seizures Who Is Having Surgery

PREOPERATIVE CARE

- For most clients, AEDs are withheld the morning or evening of the day before surgery. *AEDs may interfere with intraoperative EEG monitoring*.
- For clients with frequent and/or severe seizures, however, a partial dose of medication may be administered. *This prevents seizures or status epilepticus during surgery*.
- A low dose of analgesics is administered before surgery. *The client must remain awake throughout the lengthy procedure to respond to commands during EEG recording*.

POSTOPERATIVE CARE

- AEDs are administered parenterally until the client can tolerate oral fluids; medications are then continued orally. *It is common for the client to have seizures in the early postoperative period*.
- Steroids are administered for the first 3 days after surgery and are tapered and then discontinued during the following week. *Steroids are given to decrease cerebral edema*.

NURSING CARE PLAN A Client with a Seizure Disorder

Janet Carlson is a 19-year-old college student who lives with her parents and one younger sister. Although Janet had seizures while she was in grade school, they have been controlled with medication. However, she had a tonic-clonic seizure yesterday and immediately made an appointment with her family physician. She is currently taking phenytoin (Dilantin) 300 mg/day as a maintenance medication to prevent seizures.

ASSESSMENT

Evita Farias, RN, completes a health history for Ms. Carlson. During the history, she tells Ms. Farias that she has been under stress because of difficulties in completing her course requirements this semester. She has not been sleeping as many hours per night, and sometimes she forgets to take her medication. Janet's serum phenytoin level is 8 mg/mL. Therapeutic level is 10 to 20 mg/mL.

DIAGNOSES

- *Risk for Injury* related to recurrence of generalized tonic-clonic seizure activity and low serum phenytoin levels
- *Deficient Knowledge* of activities that may trigger seizure occurrence, the effect of stress on seizures, and medication information

EXPECTED OUTCOMES

- Verbalize precipitating and triggering factors related to the onset of seizures.
- Verbalize the relationship between emotional and physical stress and seizures.
- Verbalize the importance of taking AEDs.

PLANNING AND IMPLEMENTATION

- Teach client and her family the following:
  - Current information about seizures
  - Care during and after a seizure
  - Medication protocols
  - Factors and activities that can trigger seizures
  - The importance of follow-up care.
- Refer client and her family to a local epilepsy support group.
- Recommend that she purchase and wear a Medic-Alert bracelet.

EVALUATION

Ms. Carlson is instructed to continue taking Dilantin 300 mg/day. She states the importance of nutrition, rest, and measures to reduce stress. She also discusses the importance of maintaining the proper blood levels of her medication, stating that too little or too much of the medication could cause problems. Ms. Carlson recognizes that the seizures had recurred during a busy time in school during which she had forgotten to take her medication. She is now wearing a Medic-Alert bracelet. Ms. Farias provides the Carlsons with the telephone number of the Epilepsy Foundation of America.

CRITICAL THINKING IN THE NURSING PROCESS

1. If you were Ms. Carlson's nurse, would your teaching differ if she were living alone? If so, how?
2. Ms. Carlson tells you that although she knows she should not drive a car, she often drives her friend to work. How would you approach this problem?
3. Ms. Carlson states that "it's embarrassing to wear a Medic-Alert bracelet." How would you respond, and what recommendation(s) would you make?

See Evaluating Your Response in Appendix C.

- Know drug interactions with other prescribed drugs, over-the-counter (OTC) drugs, street drugs, and alcohol.
- Teach family members first aid for a seizure:
  - Cushion the head.
  - Loosen anything tight around the neck.
  - Turn on the side.
  - Nothing in the mouth.
  - Don't hold down.
- Teach family members when to call for medical assistance:
  - If the seizure lasts for more than 5 minutes
  - If there is slow recovery, a second seizure, or difficulty breathing after the seizure
  - If there are signs of injury (such as bleeding from the mouth).

Assessment

Collect the following data through the health history and physical examination:

- *Health history:* Past seizures; age when the first seizure occurred, most recent seizure, factors precipitating a seizure, any warning signs (aura), prophylactic anticonvulsant therapy, and specific concerns the client may have about the seizures.
- *Physical assessment:* Important data used in determining an accurate diagnosis describes manifestations obtained from nursing assessments before, during, and after a seizure. (Table 44–6 lists nursing assessments with rationale.)

Nursing Diagnoses and Interventions

Nursing care of clients with a seizure disorder focuses on providing care during and immediately after the seizure and on client/family teaching. The client with seizures has a wide variety of responses to actual or potential changes in health status; interventions discussed in this section focus on facilitating physical and psychologic comfort and safety.

Risk for Ineffective Airway Clearance

During a seizure, the tongue may fall back and obstruct the airway, the gag reflex may be depressed, and secretions may pool at the back of the throat. These may put the client at risk for an obstructed airway. Most seizures occur in the home or community; therefore, teach these interventions to the client's family:

- Provide interventions to maintain a patent airway:
  - Loosen clothing around the neck.
  - Turn on the side.
  - Do not force anything into the mouth.
  - If prescribed and available, administer oxygen by mask.

TABLE 44–6 Nursing Assessments Before, During, and After a Seizure

ASSESSMENT	RATIONALE
What was the client's level of consciousness? If consciousness was lost, at what point?	Indicates area of brain involved and type of seizure.
What was the client doing just before the attack?	May suggest precipitating factors.
In what part of the body did the seizure start?	May indicate the site of seizure activity in the brain tissue; for example, if jerking movements were first observed in right hand, the seizure focus may be in left motor cortex.
Was there an epileptic cry?	Usually indicates the tonic stage of a generalized tonic-clonic seizure.
Were any automatisms such as eyelid fluttering, chewing, lip smacking, or swallowing observed?	Often seen in complex, partial, and absence seizures.
How long did movements last? Did the location or character change (tonic to clonic)? Did movements involve both sides of the body or just one?	Indicates areas in which focal activity originated.
Did the head and/or eyes turn to one side and, if so, which side?	Helps localize the focus of the seizure. During the seizure, the head and eyes typically will turn away from the side of the epileptogenic focus.
Were there changes in pupillary reactions?	Indicates involvement of the autonomic nervous system.
If the client fell, was the head hit?	Skull x-ray studies may be needed to rule out subdural hematoma or fracture.
Was there foaming or frothing from the mouth?	Usually indicates a tonic-clonic seizure.

Although it was at one time believed that it was necessary to place a padded tongue blade in the client's mouth during a seizure, this is no longer recommended; an improperly placed tongue blade can obstruct the airway. Turning the client on the side allows secretions to drain from the mouth.

- Teach family members or significant others how to care for the client during a seizure to prevent airway obstruction. *Family members are often the only people present to provide this emergency intervention.*

Anxiety

The client with a seizure disorder is understandably anxious about the future, with questions about ability to go to school, work, have a family, and drive a car. Feelings of embarrassment about having a seizure in public and rejection by others are common and also increase the client's anxiety.

- Provide support by explaining that concerns are normal. *It is important to be sensitive to the effect of seizures on the client's self-concept and body image; alterations in these areas not only increase anxiety but also cause withdrawal from socialization with others. Demonstrating acceptance of the client's concerns allows further discussion.*
- Help identify safe leisure activities. *Worrying about being hurt if a seizure occurs may cause withdrawal from social activities that are pleasurable.*
- Provide information about sources and support groups. *Sharing information with other people with similar health problems allows for a more realistic viewpoint; accurate information can clear up misconceptions that cause anxiety.*
- Provide accurate information about hiring practices and legal limitations on driving or operating heavy or dangerous machinery. *Accurate information decreases anxiety about the unknown. The Americans with Disabilities Act prohibits discrimination; however, there are legal limitations on driving until the person is proved free of seizures.*

Using NANDA, NIC, and NOC

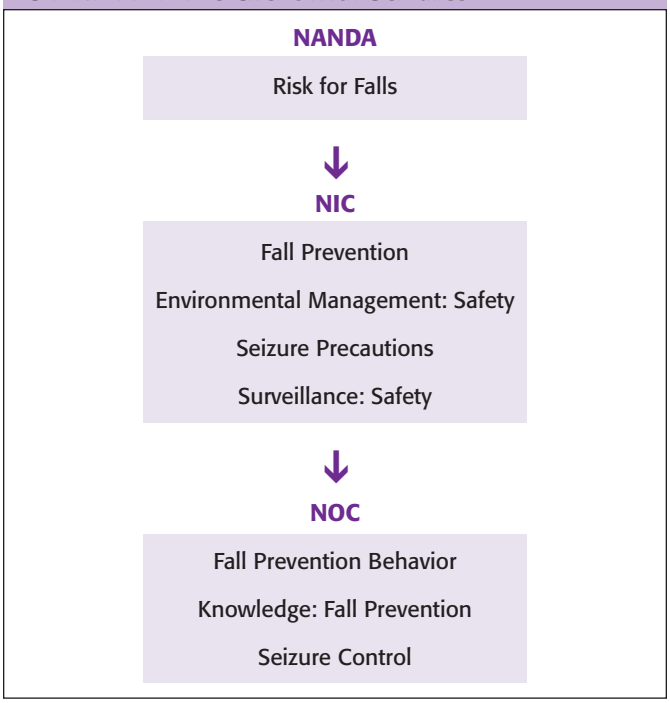
Chart 44–1 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with a seizure disorder.

Community-Based Care

Teaching follows a systematic assessment of the needs of both the client and family. Include family members so that they can learn seizure management, including the care and observations necessary before and during a seizure. Stress the importance of safety and keeping the airway patent.

NANDA, NIC, AND NOC LINKAGES

CHART 44–1 The Client with Seizures



Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

Help both the client and family adjust to a diagnosis of epilepsy. Address the following topics:

- The importance of wearing a Medic-Alert band or carrying a medical alert card at all times
- Avoiding alcoholic beverages and limiting coffee intake
- Taking showers versus tub baths, because of safety issues during a generalized seizure
- Factors that may trigger a seizure, such as abrupt withdrawal from medication, constipation, fatigue, excessive stress, fever, menstruation, sights and sounds such as television, flashing video, and computer screens
- Helpful resources include:
  - American Epilepsy Society
  - Epilepsy Foundation.

TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) (also called *craniocerebral trauma*) refers to any injury of the scalp, skull (cranium or facial bones), or brain. TBI is a leading cause of death and disability in the United States. The National Head Injury Foundation defines TBI as a traumatic insult to the brain capable of causing physical, intellectual, emotional, social, and vocational changes. A TBI may be classified as a penetrating (open) head injury (e.g., resulting from a knife, bullet, or baseball bat) or a closed head injury (a blunt injury to the brain that does not result in an open skull fracture). TBI may cause problems with cognition, movement, sensation, and emotions. Even mild brain injuries, if repeated over an extended period of time, can result in cumulative neurologic and cognitive deficits.

Each year, 1.5 million Americans sustain a TBI. Of those who survive, one of every six people is unable to return to school or work when discharged from the hospital (Centers for Disease Control and Prevention [CDC], 2004b).

FAST FACTS

TBI

- Of the 1.5 million people who sustain a TBI each year, 235,000 are hospitalized and survive.
- Each year, 50,000 people die of TBI.
- Each year, 80,000 to 90,000 people experience the onset of long-term or lifelong disability associated with a TBI (CDC, 2006).

The leading causes of TBI are falls, followed by motor vehicle crashes and assaults. Elevated blood alcohol levels, not wearing motorcycle helmets, and not wearing seat belts contribute significantly to the risk of crashes and subsequent injury. Firearm use is the leading cause of death related to TBI, causing 10% of all TBI but resulting in 44% of TBI-related deaths. Nine out of 10 people with firearm-related TBI die; of those, nearly 66% are classified as suicidal in intent (CDC, 2004). Other causes of head injury include sports injuries and occupational injuries. Adults ages 15 to 44 are at the greatest risk, with the male-to-female ratio of 3-to-1 (Hickey, 2003). Other risk factors include being over the age of 75 and living in a high-crime area.

Specific damage following craniocerebral injuries is related to the mechanism of the injury (how it occurs), the nature of the injury (type), and the location of the injury (where it occurs).

Head injuries may be classified as blunt or penetrating, and can occur through several mechanisms:

- Acceleration injury is sustained when the head is struck by a moving object, such as a swinging bat.
- Deceleration injury occurs when the head hits a stationary object, such as a concrete wall.
- Acceleration-deceleration injury (also called a *coup-contrecoup* phenomenon) occurs when the head hits an object and the brain "rebounds" within the skull (Figure 44–5 ■). The brain is injured at the point of impact (the coup) and on the opposite side of the impact (the contrecoup). Two or more areas of the brain can be injured as a result of this phenomenon.
- Deformation injuries are those in which the force deforms and disrupts the integrity of the impacted body part (e.g., skull fracture).

Types of craniocerebral trauma include injuries to the skull (including fractures), injuries to the brain (including concussion and contusion), and intracranial hemorrhage (including hematomas). Brain injury can result either from the direct effects of the trauma on brain tissue or from secondary responses to trauma, such as cerebral edema, hematoma (blood clot), swelling, or increased intracranial pressure.

THE CLIENT WITH A SKULL FRACTURE

A *skull fracture* is a break in the continuity of the skull. It may occur with or without damage to the brain; however, intracranial trauma often results from skull fractures. The considerable force of impact significantly increases the risk of underlying hematoma formation. Disruption of the skull can also cause cranial nerve injury, allow bacteria to enter the cranial vault, and/or allow CSF to leak out.

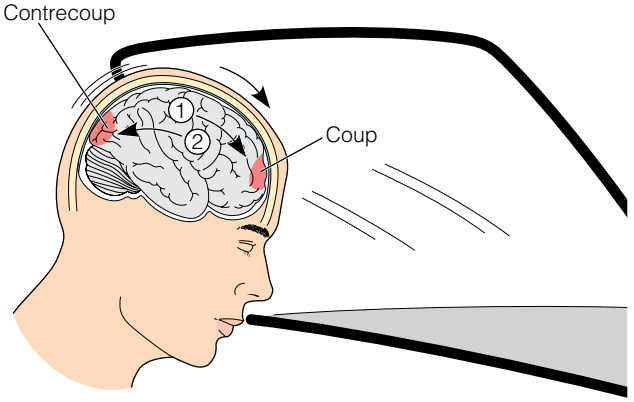


Figure 44–5 ■ Coup-contrecoup head injury. Following the initial injury (coup), the brain rebounds within the skull and sustains additional injury (contrecoup) in the opposite part of the brain.

MEDIALINK Coup-contrecoup Animation

Pathophysiology

Skull fractures are classified as open or closed. In an open fracture, the dura is torn, and in a closed fracture, the dura is not torn. Skull fractures are further classified into one of four categories: linear, comminuted, depressed, or basilar (Table 44–7).

Linear Fractures

Linear fractures are the most common, accounting for 80% of all skull fractures. They typically extend from the point of impact toward the base of the skull. Although the risk of infection or CSF leakage is minimal with this type of fracture because the dura usually remains intact, subdural or epidural hematomas (a collection of blood) frequently underlie the fracture. A hematoma (discussed later in this chapter) places pressure on underlying brain tissue, increasing both intracranial pressure and the risk of brain damage.

Comminuted and Depressed Fractures

Comminuted and depressed skull fractures increase the risk of direct damage to brain tissue from bruising (contusion) and bone fragments. However, the risk of secondary brain injury may be reduced in these fractures, because in breaking the bone, the traumatic impact energy is distributed and dissipated. If the skin overlying the fracture is lacerated or the dura is torn, the risk of infection is increased.

Basilar Fractures

Basilar skull fractures involve the base of the skull and usually are extensions of adjacent fractures, although they may occur independently. Although most basilar skull fractures are uncomplicated, they may involve the sinuses of the frontal bone or the petrous portion of the temporal bone (middle ear). If the dura is disrupted, CSF may leak through the tear. CSF leakage may include *rhinorrhea* (CSF leakage through the nose) or *otorrhea* (CSF leakage from the ear). Blood may be visible behind the tympanic membrane (hemotympanum), or ecchymosis may be noted over the mastoid process (Battle's sign). Bilateral periorbital ecchymosis ("raccoon eyes") is another possible manifestation. If CSF leakage is present, the risk of infection is high. Other complications of basilar skull fractures include injury to the internal carotid artery and compression of cranial nerve I, II, III, IV, V, VII, or VIII.

TABLE 44–7 **Types of Skull Fractures**

TYPE	DESCRIPTION
Linear (simple)	Simple, clean break in skull. Occurs with low-velocity injuries.
Comminuted	Bone is crushed into small, fragmented pieces. Usually seen with high-impact injuries.
Depressed	Inward depression of bone fragments. Usually due to a powerful blow to the skull. The dura may or may not be intact. Bone fragments may penetrate into the brain tissue.
Basilar	Occurs at the base of the skull. May be linear, comminuted, or depressed.

INTERDISCIPLINARY CARE

Treatment of a client with a skull fracture depends on the type and location of the fracture. Skull fracture may be only one of several head injuries.

A simple linear fracture generally requires bed rest and observation for underlying injury to brain tissue or hematoma formation. No specific treatment is required. Depressed skull fractures require surgical intervention, usually within 24 hours of the injury, to debride the wound completely and remove bone fragments, which may become embedded in brain tissue or cerebral blood vessels. If depressed deeply, the bone may be elevated. If cerebral edema is not present, a cranioplasty with insertion of acrylic bone may be performed. Basal skull fractures do not require surgery unless CSF leakage persists. Regular neurologic assessments and observation for manifestations of meningitis are required for the hospitalized client. Antibiotics may be administered prophylactically.

NURSING CARE

The client with a craniocerebral trauma may have a variety of responses and healthcare needs, depending on the location and extent of the trauma. Many of those problems with related nursing interventions are discussed in other sections of this chapter, including seizures, increased intracranial pressure, and bleeding within the brain.

Risk for Infection

The client with a skull fracture is at increased risk for infection related to access to the cranial contents through a tear in the dura. In an open, depressed fracture, the wound may be contaminated by dirt, hair, or other debris.

- Monitor for otorrhea or rhinorrhea. *Open fractures of the skull increase the possibility of leakage of CSF from the ears or nose.*
- Test drainage of clear fluid from ear and nose for glucose by using a glucose reagent strip, such as Dextrostix. *Clear drainage that tests positive for glucose indicates leakage of CSF; however, be aware that false positives may occur.*
- Observe blood-tinged fluid for "halo" sign. *CSF dries in concentric rings on gauze or tissues This sign is suggestive of CSF leakage.*
- Keep the nasopharynx and the external ear clean. Place a piece of sterile cotton in the ear, or tape a sterile cotton pad loosely under the nose; change dressings when they become wet. *Wet dressings facilitate movement of organisms.*
- Instruct client not to blow nose, cough, or inhibit sneeze; sneeze through open mouth. *Blowing the nose and coughing increase ICP. Withholding a sneeze forces bacteria backward.*
- Use aseptic technique at all times when changing head dressings or ICP monitor dressings and insertion sites. *Using aseptic technique reduces the possibility of introducing infection.*

Knowledge Deficit: Skull Fracture

The client and family need to be informed about the degree of injury that has occurred with the skull fracture. The client with a linear fracture, who may not be hospitalized, will need teaching that focuses on the need to monitor progress closely. To prevent complications, advise the client and family to go to the emergency room if the client experiences any of the following:

- Growing drowsiness or confusion
- Difficulty waking (instruct a family member to wake the client every 2 hours during the first night home)
- Vomiting (especially if projectile)
- Blurred vision
- Slurred speech
- Prolonged headache
- Blood or clear fluid leaking from the ears or nose
- Weakness in an arm or leg
- Stiff neck
- Seizure.

THE CLIENT WITH A FOCAL OR DIFFUSE TRAUMATIC BRAIN INJURY

Even when the skull and other structures overlying the brain remain intact, a blow to the head can cause significant brain injury. Closed head injuries may result in either focal or diffuse damage to the brain. They range in severity from mild to severe.

Pathophysiology

Brain injury results from both primary and secondary mechanisms. Primary injury results from the impact. A blow to the head, even with no break in the skull, can cause serious and diffuse brain injury. Injury to axons disrupts oligodendroglia, and direct mechanical disruption is caused by debris and leakage. The immediate vascular response to the injury results in increased capillary permeability to solutes.

Secondary injury is the progression of the initial injury resulting from events that affect perfusion and oxygenation of brain cells. These events include intracranial edema, hematoma, infection, hypoxia, or ischemia. Cerebral ischemia is the most common cause of secondary brain injury (Porth, 2005). Ischemia leads to cerebral hypoxia, with consequences of increased glial permeability to sodium (cytotoxic edema), an influx of calcium with changes in electrophysiology, and release of free fatty acids and lactic acidosis.

Acute brain injury affects all body systems as well as the central nervous system. Systemic effects of acute brain injury are listed in Table 44–8.

Focal Brain Injuries

Focal brain injuries are specific, grossly observable brain lesions confined to one area of the brain. They include contusions, lacerations, and intracranial hemorrhage. The force of an impact produces contusions from direct contact with the inside of the skull that in turn may cause epidural hemorrhage and subdural and intracerebral hematomas. The mechanisms of injury are coup and/or contrecoup damage to the brain at the point of the impact and the rebound effect. The damaged brain area is surrounded by edema, contributing to IICP. Infarction and necrosis, multiple hemorrhages, and edema are found within the contused areas. The maximum effects of the injury peak in 18 to 36 hours.

Intracranial hemorrhage can result directly from the trauma (e.g., beneath a fracture) or from shearing forces on cerebral arteries and veins that occur with acceleration–deceleration. Depending on the site and rate of bleeding, manifestations may appear immediately or may not become evident for hours or even weeks. Intracranial hemorrhages and the hematomas they cause place pressure on surrounding structures, causing manifestations of an expanding focal lesion. They also cause IICP, leading to altered

TABLE 44–8 **Systemic Effects of Acute Brain Injury**

CAUSE	EFFECT
■ Stimulation of the sympathetic nervous system, which stimulates the adrenal cortex and medulla to increase glucocorticoid and mineralocorticoid levels	■ Increased metabolism of carbohydrates, fats, and proteins ■ Retention of sodium and water
■ Stimulation of the sympathetic nervous system, increasing the serum catecholamine levels	■ Hypertension ■ EEG changes ■ Dysrhymias (bradycardia, sinus tachycardia)
■ Altered release of ADH from the posterior pituitary	■ Retention of water or diuresis and diabetes insipidus
■ Neurogenic pulmonary dysfunction	■ Abnormal respiratory patterns ■ Reduced residual capacity with retention of CO_2, vasodilation, and increased ICP ■ Pulmonary edema
■ Stress response to trauma	■ Hyperglycemia
■ Increased platelet, plasma fibrinogen, and thromboplastin levels	■ Decreased clotting and prothrombin times ■ Vascular occlusion ■ Disseminated intravascular coagulation ■ Anemia
■ Immunosuppression	■ Infection
■ Decreased gastric motility and increased gastric acidity	■ Gastritis ■ Gastric ulcers

TABLE 44–9 **Comparison of Intracranial Hematomas**

TYPE/FREQUENCY	LOCATION/COMMON SITE	PRECIPITATING FACTORS	MANIFESTATIONS
Epidural Hematoma 2% to 6% of all types of head injuries	Located in the space between the skull and the dura mater Common site: the temporal bone (over the middle meningeal artery)	Skull fractures Contusion	Momentary loss of consciousness followed by a lucid period lasting from a few hours to 1 to 2 days Rapid deterioration in level of consciousness (drowsiness to confusion to coma) Seizures Headache Hemiparesis (may be ipsilateral or contralateral) Fixed dilated ipsilateral pupil Rise in blood pressure with decreases in pulse and respirations indicates a rapidly increasing hematoma
Subdural Hematoma Approximately 29% of all types of head injuries	Located in the space below the dural surface (between the dura and arachnoid and pia mater layers of meninges) Common site: may occur any place in cranium	Closed head injury Acceleration–deceleration injury Cerebral atrophy (seen in older adults) Chronic alcoholism Use of anticoagulants Contusion	Acute: ■ Headache ■ Drowsiness ■ Agitation ■ Slowed thinking ■ Confusion Subacute: ■ Same as those of acute subdural hematoma but develop more slowly Chronic: ■ Manifestations may not appear until weeks to months after injury ■ Confusion, slowed thinking, drowsiness
Intracerebral Hematoma 14% to 15% of all types of head injuries	Located directly in the brain tissue Common sites: frontal or temporal region	Gunshot wounds Depressed bone fractures Stab injury Long history of systemic hypertension Contusions	Headache Deteriorating consciousness to deep coma Hemiplegia on contralateral side Dilated pupil on the side of the clot

levels of consciousness and potential herniation syndromes. Intracranial hematomas are classified by their location as epidural, subdural, or intracerebral. Table 44–9 compares the frequency, locations, common sites, precipitating factors, and manifestations of intracranial hematomas; Figure 44–6 ■ illustrates their locations.

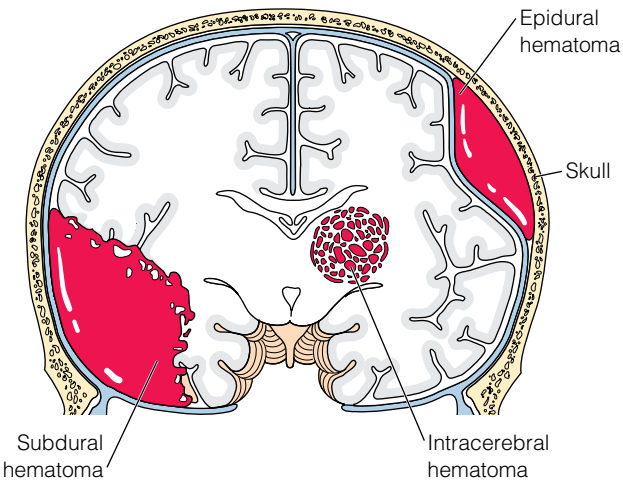


Figure 44–6 ■ Three types of hematomas: epidural hematoma, subdural hematoma, and intracerebral hematoma.

CONTUSION A *contusion* is a bruise of the surface of the brain, typically accompanied by small, diffuse venous hemorrhages. Both white and gray matter may have a bruised, discolored appearance. A decrease in pH, with accumulation of lactic acid and decreased oxygen consumption, may hinder cell function. Contusions (and other focal brain injuries) occur when the brain strikes the inner skull, often with a coup (point of impact) lesion and a contrecoup lesion on the opposite side of the brain. Contusions occur most frequently near bony prominences of the skull. Cerebral edema can follow contusion, resulting in IICP. Contusions; small, diffuse venous hemorrhages; and brain swelling are at their peak 12 to 24 hours after injury.

Manifestations of the contusion depend on the size and location of the brain injury. An initial loss of consciousness occurs; LOC may remain altered, and behavior changes such as combativeness may persist for an extended period. Full consciousness may be regained extremely slowly, and residual deficits may persist; in some clients, full LOC never really returns. Focal effects of the contusion may cause loss of reflexes, hemiparesis (muscular weakness of one-half of the body), or abnormal posturing. Manifestations of IICP may occur if cerebral edema develops. Regaining full LOC may take an extended period of time and residual deficits may persist.

EPIDURAL HEMATOMA An **epidural hematoma** (also called an *extradural hematoma*) develops in the potential space between the dura and the skull, which normally adhere to one another. As the blood collects, the expanding hematoma strips the dura away from the skull. Epidural hematomas affect young to middle-aged adults more frequently than older adults, because the dura becomes more tightly attached to the skull with aging.

Epidural hematomas usually result from a skull fracture that tears an artery, often the middle meningeal artery. Because epidural hematomas are arterial in origin, they tend to develop rapidly. The client may lose consciousness with the initial injury, and then have a brief lucid period before the LOC rapidly declines from drowsiness to coma as the hematoma expands, stripping the dura away from the skull and placing pressure on brain tissue. Other manifestations include headache; vomiting; a fixed, dilated pupil on the same side (ipsilateral) as the hematoma; contralateral (opposite side) hemiparesis or hemiplegia; and possible seizures. Because epidural hematomas usually develop rapidly, timely intervention is vital to prevent significant increases in ICP and herniation.

SUBDURAL HEMATOMA **Subdural hematomas**, in which a localized mass of blood collects between the dura mater and the arachnoid mater, are more common than epidural hematomas. Acute subdural hematomas are usually located at the top of the head, and develop within 48 hours of the initial head injury. Chronic subdural hematomas develop over weeks or months. The chronic type is seen most often in the older adult and people who have some brain atrophy with subsequent enlarged epidural space. These hematomas are often venous in origin, although they may involve bleeding from small arteries as well. Subdural hematomas may form without direct trauma or contusion; acceleration–deceleration forces may tear the bridging veins that connect veins on the surface of the cerebral cortex to the dural sinuses. As blood collects, it places direct pressure on underlying brain tissue.

Acute subdural hematomas develop rapidly following head injury. Although a lucid period may occur, the client commonly develops drowsiness, confusion, and enlargement of the ipsilateral pupil within minutes or hours of the injury. If responsive, the client may complain of a unilateral headache. Hemiparesis and respiratory pattern changes may occur.

Chronic subdural hematomas are often associated with relatively minor trauma such as a fall. Weeks to months may elapse before manifestations of the hematoma occur; the initial trauma may have been forgotten. Chronic subdural hematomas may also occur spontaneously in the older adult or in clients with bleeding disorders. Manifestations of the hematoma develop slowly and may be mistaken for the onset of dementia in the older adult. Slowed thinking, confusion, drowsiness, or lethargy are common early manifestations. Other manifestations include headache, dilation and sluggishness of the ipsilateral pupil, and possible seizures.

INTRACEREBRAL HEMATOMA Intracerebral hematomas may be single or multiple, and are associated with contusions. They may occur in any location but usually are found in the frontal or temporal lobes. They may result from closed head trauma, particularly contusion or shearing of small blood vessels deep within the hemispheres. Intracerebral hematomas can also accompany other types of head trauma such as lacerations. Older adults are particularly vulnerable to intracerebral hemorrhage because cerebral blood vessels are more fragile and easily torn.

The manifestations of intracerebral hematoma vary according to the location of the hematoma. Headache may develop, along with decreasing LOC, hemiplegia, and dilation of the ipsilateral pupil. The expanding clot increases intracranial pressure, and herniation may occur.

Diffuse Brain Injury

A diffuse brain injury (DBI) affects the entire brain and is caused by a shaking motion, with twisting movement (rotational acceleration) as the primary mechanism of injury. DBIs include concussions and diffuse axonal injuries. Shearing stresses on brain tissue cause axonal damage from shearing, tearing, or stretching of nerve fibers. The most serious axonal injuries are located farthest from the brainstem, with the frontal and temporal axonal tracts being most vulnerable to injury. Physical deficits resulting from DBIs include spastic paralysis, peripheral nerve injury, swallowing disorders, visual and hearing impairments, and taste and smell disorders. Damage decreases the speed of information processing and responding and disrupts attention, resulting in serious cognitive and affective impairments. Cognitive deficits that may result include disorientation and confusion, short attention span, problems with memory and learning, perceptual problems, and poor judgment. Possible behavioral deficits include agitation, impulsivity, depression, and social withdrawal.

Initially, the damage involves tearing of axons, blood vessels, and brain tissue (visible only by electron microscope). The number of damaged axons progressively increases, with pathology involving the nuclei and axons. The damaged axons, which resemble sausage links, regress into round balls called retraction balls (visible with light microscopy). After several weeks, the retraction balls are replaced by clusters of microglia. In the final phase, astrocytosis (equivalent to scarring) occurs at the site of axonal damage, accompanied by demyelination of long axon tracts.

The categories of DBI include mild concussion, classic cerebral concussion, and diffuse axonal injury. Manifestations of a concussion are listed in the box below.

MANIFESTATIONS of Concussion

- Immediate loss of consciousness (lasting usually no longer than 5 minutes)
- Amnesia for events surrounding injury
- Headache
- Drowsiness, confusion, dizziness
- Visual disturbances
- Possible brief seizure activity with transient apnea, bradycardia, pallor, and hypotension

Postconcussion Syndrome

- Persistent headache
- Dizziness
- Irritability and insomnia
- Impaired memory and concentration, learning problems

MILD CONCUSSION The word **concussion** means violent shaking. A concussion involves temporary axonal disturbances. It is defined as a momentary interruption of brain function. A concussion is associated with an immediate, brief loss of consciousness on impact. Altered consciousness may last only seconds or persist for several hours. Amnesia for events immediately preceding (antegrade amnesia) and following (retrograde amnesia) the injury is common. Other manifestations of concussion include headache, drowsiness, confusion, dizziness, and visual disturbances such as diplopia or blurred vision.

CLASSIC CEREBRAL CONCUSSION A classic cerebral concussion involves diffuse cerebral disconnection from the brainstem RAS. An immediate loss of consciousness occurs, lasting less than 6 hours. Both retrograde and anterograde amnesia occur. Cerebral contusions may be present. In a severe concussion, a brief seizure and respiratory arrest may occur; transient pallor, bradycardia, and hypotension may accompany loss of consciousness.

Following concussion, clients may develop postconcussion syndrome with persistent headache, dizziness, irritability, insomnia, impaired memory and concentration, and learning problems. Postconcussion syndrome may last for several weeks or, rarely, up to a year.

DIFFUSE AXONAL INJURY Diffuse axonal injury (DAI) is a brain injury in which a high-speed acceleration–deceleration injury, typically associated with motor vehicle crashes, causes widespread disruption of axons in the white matter. Focal lesions may be found in the corpus callosum, midbrain, and brainstem. An immediate loss of consciousness occurs. The prognosis is poor; most clients with severe DAI either die or remain in a persistent vegetative state.

DAIs may range from mild to severe. In mild DAI, coma lasts 6 to 24 hours, and cognitive, psychologic, and sensorimotor deficits may persist. In moderate DAI, injury and impairment is spread throughout the cerebral cortex and diencephalon. There is axonal tearing, coma lasting more than 24 hours, and often incomplete recovery. In severe DAI, axonal injury occurs in both cerebral hemispheres, the diencephalon, and the brainstem. Immediate autonomic dysfunction occurs, and IICP is manifested. Profound cognitive and sensorimotor deficits occur, involving movement, verbal and written communication, ability to learn and reason, and ability to modulate behavior.

INTERDISCIPLINARY CARE

The client with a brain injury may receive medical and/or surgical treatment. Specific guidelines for the medical management of head injury have been developed by the Brain Trauma Foundation (2003) for concussion and acute TBI:

- *Concussion* Following a concussion, the client may be observed for 1 to 2 hours in the emergency department (ED), and then discharged home with instructions for further observation to detect manifestations of secondary injury. If the loss of consciousness extended more than 2 minutes, the client may be admitted to the hospital for observation.
- *Acute TBI* Recognition and management of acute TBI with transport to an ED is essential to client outcomes. Morbidity and mortality increase with hypotension (systolic pressure less than 90 mmHg) and hypoxia (Pa_{O_2} less than 60 mmHg), so fluids are given to support a mean systolic arterial blood pressure at more than 90 mmHg (Hickey, 2003). Assessment of the client's airway, breathing, and circulation (ABCs), with management of dysfunction, is necessary to decrease the secondary effects of the brain injury. The fluid of choice for intravenous fluids is hypertonic saline, because it reduces intracranial hypertension. An intracranial pressure monitor probe may be inserted to assess ICP and monitor therapy to reduce cerebral edema and maintain cerebral perfusion. Osmotic diuretics such as mannitol also may be administered to reduce cerebral edema. Adequate oxygenation is vital to maintain cerebral metabolism; carbon dioxide is a potent vasodilator, and increased levels may contribute to cerebral edema and IICP.

On admission to the ICU from the ED, the client may be placed on a special bed and connected to various monitoring devices. Invasive lines are inserted, including a central venous pressure (CVP) catheter, arterial line, pulmonary catheter, ventriculostomy, ICP monitor, and perhaps a retrograde jugular catheter. In most instances, an endotracheal tube is inserted and connected to a mechanical ventilator, cardiac monitoring is initiated, bilateral sequential pressure boots are applied, pulse oximetry is started, and a rectal temperature probe is inserted. All values are monitored for changes to ensure early detection of cerebral hypoxia and impending ischemia to prevent secondary brain injury. The values that are monitored are outlined in Table 44–10.

Diagnosis

Diagnostic testing may be done to monitor hemodynamic status and detect conditions that may contribute to cerebral edema. Radiologic examinations include skull x-rays (to identify skull fractures and assess penetrating objects) and CT scan or MRI to detect contusions and lacerations associated with diffuse axonal injury. ABGs are analyzed, with particular attention to oxygen and carbon dioxide levels.

Managing IICP

IICP is managed (as described in a previous section) to reestablish equilibrium of the intracranial contents and prevent secondary brain damage. Treatments include airway management, hyperventilation (used if signs of herniation appear), fluid resuscitation, positioning, temperature regulation, and medications. Medications other than those previously discussed include a category of drugs called neuroprotectants. These drugs are used to treat or alter some of the pathologic pathways that occur in ischemia, and must be administered within a short time of the injury to be effective. Classifications of the drugs include lipid peroxidase inhibitors, free radical scavengers, receptor antagonists, calcium channel blockers, and gangliosides.

TABLE 44–10 Monitoring Cerebral Oxygenation with Laboratory Values

ASSESSMENT	NORMAL RANGE OR VALUE	ABNORMAL FINDINGS
Partial pressure of oxygen in brain tissue ($PbtO_2$)	25–50 mmHg	<15 mmHg = ischemia
Intracranial pressure (ICP)	<10 mmHg	10–20 mmHg = mild to moderate increase in IICP >20 mmHg = severe increase in IICP
Mean arterial pressure (MAP)	70–110 mmHg	<60 mmHg = decreased cerebral perfusion pressure
Cerebral perfusion pressure (CPP)	70–100 mmHg	<50 mmHg = compromised cerebral blood flow
$PaCO_2$	35–45 mmHg	<35 mmHg may result in cerebral vasoconstriction, further decreasing oxygenation of brain tissue
PaO_2	80–100 mmHg	If decreased, can cause tissue hypoxia and decrease oxygenation of brain tissue

Source: Data from Wilensky, E., & Bloom, S. (2005). Monitoring brain tissue oxygenation after severe brain injury. *Nursing, 35*(2), 32cc1–32cc4.

Surgery

Small subdural hematomas can frequently be reabsorbed and may be treated conservatively, with close observation and supportive care. However, the treatment of choice for epidural hematomas and large acute subdural hematomas is surgical evacuation of the clot. This can often be performed through burr holes made into the skull (Figure 44–7 ■). In an epidural hematoma, the bleeding vessel can also be ligated during this procedure, preventing further bleeding. Rebleeding may occur following evacuation of an acute subdural hematoma in older adults and clients with chronic alcoholism. A craniotomy is necessary to evacuate chronic subdural hematomas because the hematoma tends to solidify, making it difficult or impossible to remove through burr holes. Surgery is less successful in treating intracerebral hematomas because of widespread tissue damage. Supportive care to manage intracranial pressure and prevent complications is provided.

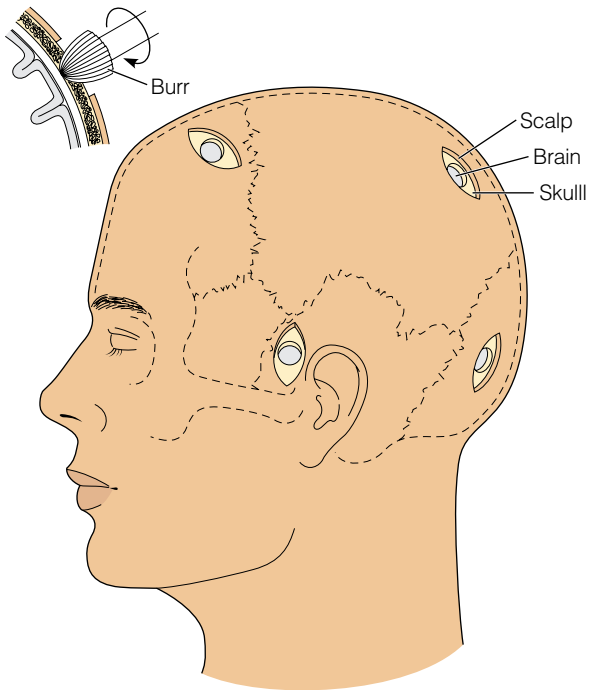


Figure 44–7 ■ Possible locations of burr holes.

NURSING CARE

In addition to the nursing care discussed in this section, a Nursing Care Plan for a client with a subdural hematoma is found on the next page.

Health Promotion

The best way to treat any injury is to prevent it from happening. Public education must continue to stress the importance of safe driving, the dangers of driving under the influence of alcohol or drugs, and the necessity of wearing seat belts and cycle helmets. Legislation has mandated such motor vehicle changes as seat belts, child safety seats, and air bags. Other behaviors that can reduce the morbidity and mortality associated with TBI are following gun safety rules, promoting farm safety, and teaching older adults about safety (such as preventing falls) in the home.

Assessment

Collect the following data through the health history and physical examination (see Chapter 43 ∞):

- *Health history:* A history of the injury is helpful in understanding the nature of the craniocerebral trauma; knowledge about loss of consciousness assists the nurse in planning care.
- *Physical examination:* Neurologic assessment, including pupils, LOC, Glasgow Coma Scale, brainstem reflexes (cornea, cough, gag, extraocular movements), vital signs; skull and face (deformity, lacerations, bruising, bleeding); movement of extremities.

Nursing Diagnoses and Interventions

Nursing care of the client in the acute care phase initially focuses on maintaining an effective airway and breathing pattern. Nursing care is also directed toward continuous assessment and monitoring of neurologic function as well as other body systems.

NURSING CARE PLAN A Client with a Subdural Hematoma

Wong Lee is a 50-year-old tug boat mechanic who is married and has three sons. Although Mr. Lee has been through rehabilitation twice for alcoholism, he has not been able to quit drinking. His physician has explained the physical consequences and the possible interaction between alcohol and the anticoagulant Mr. Lee is taking for chronic atrial fibrillation. While attending a family reunion, during which he eats a large meal and drinks several beers, Mr. Lee joins a game of softball. Mrs. Lee is concerned that Mr. Lee has consumed too much alcohol to play ball in the heat, but Mr. Lee is adamant and states that he wants to pitch. During the end of the second inning, the batter hits a ball that strikes Mr. Lee in the head. Mr. Lee stumbles and drops to the ground, holding his head. He does not lose consciousness and gets up on his own. His sons and wife try to persuade him to go to the hospital, but Mr. Lee insists he feels fine.

Two weeks later, after an evening of consuming several mixed drinks, Mr. Lee develops a headache. He attributes the headache to a hangover, but instead of improving the next day, the headache becomes steadily worse. He becomes confused and disoriented. His wife, concerned that his drinking is increasing again, calls the physician, who admits Mr. Lee to the detoxification center at the local hospital. A CT scan is performed. The diagnosis of a subdural hematoma is made, and Mr. Lee is transferred to the neurosurgical unit.

ASSESSMENT

When Saundra Knight, the nurse on the neurosurgical unit, enters the room, she notices that Mr. Lee is sitting in bed, laughing and giddy. As she begins to talk to Mr. Lee, he states, "Don't ask me anything—I can't think. My headache is getting worse." Over the next few hours, the giddiness subsides, and Mr. Lee becomes drowsy. Ms. Knight reports a Glasgow Coma Scale score of 11. An ICP monitor is inserted and reveals increased intracranial pressure. Mr. Lee is scheduled to have burr holes and hematoma evacuation that afternoon.

DIAGNOSES

- *Risk for Ineffective Breathing Pattern* related to pressure on respiratory center by intracranial hematoma
- *Ineffective Cerebral Tissue Perfusion* related to increased intracranial pressure secondary to cerebral edema

EXPECTED OUTCOMES

- Maintain a respiratory rate and rhythm within normal limits.
- Maintain adequate cerebral perfusion, as evidenced by stable vital signs, stable neurologic status, and no decrease in level of consciousness.

PLANNING AND IMPLEMENTATION

- Perform neurologic assessment every 2 hours or as needed.
- Monitor vital signs every 2 hours or as needed.
- Explain to the family the procedure for intracranial surgery.

EVALUATION

The first day postoperatively, Mr. Lee begins breathing on his own without ventilatory support. His respiratory rate and rhythm are within normal limits, with no signs of abnormal breath sounds. The ICP monitor readings are appropriate, and Mr. Lee shows significant improvement in level of consciousness, with a Glasgow Coma Scale score of 15. Mr. Lee continues to improve and is discharged to home 5 days after surgery.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe the similarities and differences between Mr. Lee's disorder and the manifestations of other types of intracranial hematomas.
2. Mr. Lee kept trying to pull out his ICP line. You know he should not be restrained, because pulling against restraints increases restlessness and increases intracranial pressure. What would you do?
3. Write a care plan for Mr. Lee for the nursing diagnosis *Acute Confusion*.

See Evaluating Your Response in Appendix C.

This close monitoring provides early recognition and treatment of problems and complications, and initiation of aggressive forms of therapy that may be needed.

Many nursing diagnoses associated with traumatic brain injury correspond with those outlined previously in the sections on the client with altered LOC and IICP. Specific nursing diagnoses discussed in this section focus on problems with intracranial adaptive capacity, airway clearance, and breathing patterns.

Decreased Intracranial Adaptive Capacity

The client with a traumatic brain injury has or is at high risk for IICP. As the mechanisms that normally compensate for changes in intracranial pressure are compromised, intracranial pressure increases in disproportional response to a variety of stimuli. (See the discussion earlier in the chapter for other nursing diagnoses and interventions for the client with IICP.)

- Monitor for manifestations of IICP, including eye opening response, motor response, and verbal response. *These responses evaluate the ability to integrate commands with conscious and involuntary movement.*
- Monitor for changes in vital signs: bradycardia or tachycardia, varying breathing patterns, hypertension, and/or widening pulse pressure. Vital signs vary depending on the site of impairment. *Cushing's triad (bradycardia, increased systolic blood pressure, and increased pulse pressure) indicates brainstem ischemia leading to cerebral herniation.*
- Monitor for vomiting, headache, lethargy, restlessness, purposeless movements, and changes in mentation. *These manifestations may be early indicators of intracranial pressure changes.*
- Monitor temperature and initiate hypothermia treatment as prescribed. *Impaired hypothalamic function can interfere with temperature regulation. Hyperthermia may increase ICP.*
- Monitor fluid status: Regularly compare intake and output, review serum osmolality, and use infusion pump to administer IV fluids (if prescribed). *Osmotic diuretics, if used to treat cerebral edema, may cause hypotension and decreased cardiac output.*

PRACTICE ALERT
Overhydration from rapid infusion of IV fluids may cause or further increase IICP.

Ineffective Airway Clearance

The primary objective in the care of any trauma client is maintaining a patent airway to prevent hypoxia. However, in the initial acute care phase, the risk of cervical vertebral fractures and spinal cord injury may complicate the process of establishing a patent airway. In addition, other multisystem injuries may complicate the interpretation of vital signs. In general, all unconscious people with a head injury should be intubated with an endotracheal tube to prevent aspiration. Clients with head trauma may also require a tracheostomy to provide an airway and be placed on a ventilator.

- Monitor neurologic manifestations on a regular schedule. *Changes in neurologic manifestations may indicate IICP, with the risk of further depression of the respiratory system and respiratory arrest.*
- Maintain head and neck in neutral alignment, immobilized until injury is determined. *Head rotation and neck flexion are associated with IICP, decreased jugular venous outflow, and localized changes in cerebral blood flow. Immobilization prevents spinal cord injury in suspected or actual fractures of the cervical spine; spinal cord injury at this level would further impair respiratory function.*
- Clear the nose and mouth of mucus and blood. *This helps maintain patency of the upper airway.*
- Suction the airway as needed, limiting suctioning time to no more than 10 seconds at one time. Do not suction the nasal passages until a dural tear has been ruled out. *Suctioning is usually necessary to maintain a patent airway.*

Ineffective Breathing Pattern

The client with a traumatic brain injury and hematoma is at high risk for ineffective breathing pattern related to IICP. If ICP increases dramatically, tentorial herniation may occur, leading to sudden respiratory arrest.

- Monitor the respiratory pattern for rate, depth, and rhythm every 2 hours or as needed if the client is not on a ventilator. Assess breath sounds, presence of cyanosis, restlessness, and use of accessory respiratory muscles. Monitor pulse oximetry and blood gas levels. *Head injuries may cause alterations in respirations. An increased respiratory rate may indicate hypoxia. A decrease in respiratory rate may be the result of depression of the medullary respiratory center.*

PRACTICE ALERT
In general, an initial increase in intracranial pressure causes respirations to slow; as the pressure continues to increase, respirations become rapid.

- Monitor ICP readings. Continuous measurement of ICP is used to diagnose and monitor increased intracranial pressure. *As ICP increases, herniation may occur, leading to respiratory arrest and death.*
- If the client is not intubated, prepare for oxygen administration and/or tracheal intubation if respiratory distress occurs. *Supplying oxygen prevents hypoxia until a hematoma can be evacuated, relieving pressure on the respiratory center.*
- Prepare for cranial surgery if deteriorating respiratory pattern and neurologic changes are noted. *Surgical intervention usually consists of placing several burr holes in the skull or performing a craniotomy to remove the hematoma. (Intracranial surgery is discussed later in the chapter.) However, the cerebral edema and increased intracranial pressure may cause death even if surgery is performed.*

Using NANDA, NIC, and NOC

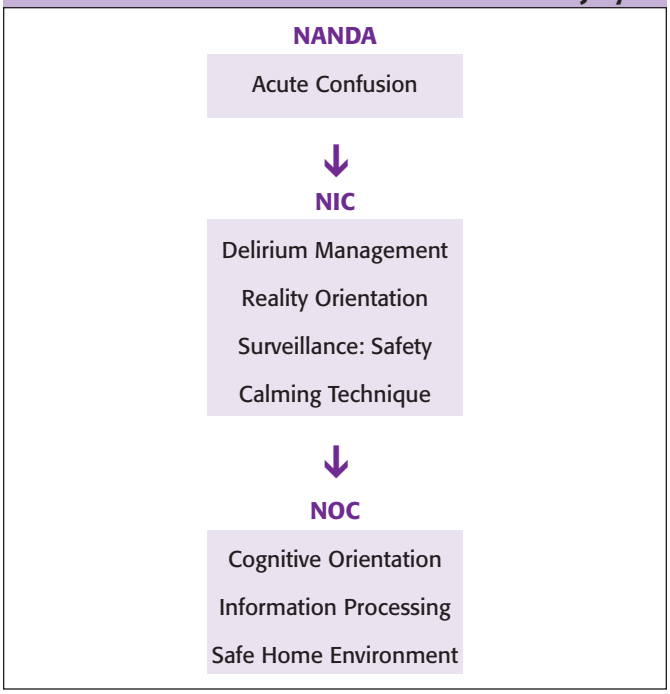
Chart 44–2 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with an acute brain injury.

Community-Based Care

Concussion

Inform the client and family that a postconcussion syndrome sometimes occurs. If the client experiences persistent headaches and dizziness, is uncharacteristically emotional, seems overly tired, or has difficulty paying attention or remembering, the healthcare provider should be notified. Explain that these manifestations may persist for some time. Rehabilitation may help the client compensate for memory impairment and attention deficits.

NANDA, NIC, AND NOC LINKAGES
CHART 44–2 **The Client with an Acute Brain Injury**



Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

Acute Brain Injury

Clients who survive an acute brain injury will require long-term physical care and rehabilitation. Although recovery is highly individualized, many clients who regain consciousness require lifelong care; others remain in a coma or vegetative state. The family often expects the client to recover fully after the coma subsides, and they need information about the real possibility of residual deficits in self-care, emotional responses, cognition, communication, and movement (see the Nursing Research box below). Topics that should be addressed for home care include:

- The need to encourage self-care and independence as much as possible
- Information to enhance recovery (CDC, 2006):
  - Get lots of rest. Don't rush back to work or school.
  - Avoid anything that could cause another blow or jolt to the head.
  - Consult with physician about when it will be safe to drive a car, ride a bike, or use heavy equipment (reaction time is often slower after a TBI).
  - Take only prescribed drugs and don't drink alcohol.
  - Write things down if you are having problems remembering.
  - If the injury was severe, therapy may be needed to learn lost skills, such as speaking, walking or reading.
- Safety issues
- Equipment needs, such as a wheelchair and hospital bed
- Vocational counseling and services
- Referral to community resources and support groups
- Helpful resources:
  - National Head Injury Foundation
  - Brain Injury Association of America
  - Brain Trauma Foundation
  - International Center for Individuals with Disabilities.

NURSING RESEARCH Evidence-Based Practice: Caregivers of Young Adults with TBI

TBI is a leading cause of death and disability across all age groups, but the age group most affected is young adults, and especially young men. This is the result of increased risk-taking behavior in the age group, including drinking alcohol and using drugs while operating a motor vehicle. This population of TBI survivors is large and caring for them is expensive. Most research of TBI survivors has focused on problems of the survivor, but few have examined the burden placed on the family. This is especially true for the experience of the survivors' mothers, who are in almost all cases the primary caregiver. A study was conducted by Kao and Stuifben (2004) to describe the meaning of the experience of the relationship between young adult TBI survivors and their mothers. Both the survivors and the mothers were interviewed and the data were analyzed to describe the experience. The survivors described having a sense of being abnormal; the mothers struggled with a balance of protecting their children and letting them become independent. The mothers also struggled to maintain relationships inside and outside of the family. All of these experiences resulted in ongoing stress for the mothers.

IMPLICATIONS FOR NURSING

The findings of this study have implications for community-based teaching and interventions for TBI survivors and their caregivers. Each year a significant number of TBI survivors leave the acute care setting and return to their homes, where caregivers are often unprepared for the lifelong multiple disabilities and problems they will face.

Developing an appropriate tool to measure family caregiver burden for those with disabilities from TBI is recommended, so as to be able to design and implement individualized interventions and teaching. The teaching and interventions should be developed as part of a systematic and integrated discharge plan, a community-based program, and a follow-up approach to assist TBI survivors and their families to effectively decrease the stressors that are a part of caregiver load.

CRITICAL THINKING IN CLIENT CARE

1. You are making a home visit to a 22-year-old woman who has recently been discharged from acute care following a serious automobile crash resulting in mild diffuse axonal injury. She says to you "I had my accident on the way home from spring break. This is my third year in nursing school. Will I be able to be a nurse?" How would you respond?
2. On discharge from the hospital, a nursing diagnosis for a young adult TBI survivor who would be cared for at home by his single mother was *Risk for Parental Role Conflict.* Do you think this is an appropriate diagnosis? Why or why not?
3. If you were to outline topics for a program to reduce the incidence of TBI in a rural community, how would it differ from one for residents of a large urban city?

Source: Data from Kao, H., & Stuifbergen, A. (2004). Love and load: The lived experience of the mother–child relationship among young adult traumatic brain-injured survivors. *Journal of Neuroscience Nursing, 36*(2), 73–81.

CENTRAL NERVOUS SYSTEM INFECTIONS

THE CLIENT WITH A CENTRAL NERVOUS SYSTEM INFECTION

The central nervous system, including the meninges, neural tissues, and blood vessels, may be directly affected by bacteria, viruses, fungi, protozoans, and rickettsiae. The CNS may also be affected by toxins from bacterial infections. In general, organisms enter the brain in two ways: through the bloodstream by crossing the blood–brain barrier or by direct invasion through a skull fracture or bullet hole. Very rarely, a CNS infection occurs as a result of contamination of a surgical field or lumbar puncture. The major CNS infections include meningitis, encephalitis, and brain abscesses.

The incidence of pathogenic infections of the CNS increases with the onset of AIDS. Clients who are HIV positive may have CNS infections caused by toxoplasmosis, cryptococcus, tuberculosis, herpes simplex, cytomegalovirus (CMV), or a polyoma virus (resulting in progressive multifocal leukoencephalopathy).

Pathophysiology

When pathogens enter the CNS and the meninges, an inflammatory process results. The pathology of CNS infections includes the invading pathogens, the subsequent inflammation, and the increase in intracranial pressure that may result from the inflammatory processes. Both the pathogenic damage and the IICP may result in brain damage and life-threatening complications.

Meningitis

Meningitis is an inflammation of the pia mater, the arachnoid, and the subarachnoid space. Inflammation spreads rapidly throughout the CNS because of the circulation of CSF around the brain and spinal cord. Meningitis may be acute or chronic, and it may be bacterial, viral, fungal, or parasitic in origin. Meningococcal meningitis may occur in epidemics among people who are in close contact with one another, such as military recruits and students living in dormitories. Pneumococcal meningitis, in contrast, primarily affects the very young and very old.

The organism responsible for meningitis must overcome nonspecific and specific host defense mechanisms to invade and replicate in the CSF. These defenses include the skin barrier, the blood–brain barrier, the nonspecific inflammatory response, and the immune response. Host response to the particular pathogen is responsible for the manifestations of clinical meningitis. The organisms that initiate the host response in meningitis demonstrate an affinity for the nervous system. They colonize and invade the nasopharyngeal mucosa, survive intravascularly, and penetrate the CNS if the blood–brain barrier is damaged, as can happen during surgery, the inflammatory response, or cerebral edema.

Infection of the CSF and meninges causes an inflammatory response in the pia, arachnoid, and CSF. Because the meninges and subarachnoid space are continuous around the brain, spinal cord, and optic nerves, the infection and inflammatory response are always cerebrospinal, involving both the brain and the spinal cord. Inflamed blood vessels in the area leak fluids as cell permeability increases. Purulent exudate infiltrates cranial nerve sheaths and blocks the choroid plexus and subarachnoid villi. IICP occurs as brain tissue responds to the pathogen. With an increase in ICP, cerebral perfusion decreases and cerebral autoregulation is lost.

BACTERIAL MENINGITIS The causative organisms of bacterial meningitis include *Neisseria meningitis*, meningococcus, *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Escherichia coli*. Risk factors include head trauma with a basal skull fracture, otitis media, mastoiditis, sinusitis, neurosurgery, systemic sepsis, or immunocompromise (Porth, 2005). Even when appropriate antibiotics are used, the mortality rate for adults remains at approximately 25%.

Once the pathogen enters the central nervous system, it or its toxic products (free radicals) initiate an inflammatory response in the meninges, CSF, and ventricles. Meningeal vessels become engorged, and their permeability increases. Phagocytic white blood cells migrate into the subarachnoid space, forming a purulent exudate that thickens and clouds the CSF and interferes with its flow. Rapid exudate formation causes further inflammation and edema of meningeal cells. Blood vessel engorgement, exudate formation, impaired CSF flow, and cellular edema cause the intracranial pressure to increase.

Manifestations The client with bacterial meningitis typically presents with fever and chills, headache, back and abdominal pain, and nausea and vomiting. (The older adult may not have a high fever, but may instead exhibit confusion.) Meningeal irritation causes nuchal rigidity (stiff neck) and positive Brudzinski's sign (flexion of the neck that causes the hip and knee to flex) and positive Kernig's sign (inability to extend the knee while the hip is flexed at a 90-degree angle). Photophobia is present; the client may also experience diplopia. With meningococcal meningitis, a rapidly spreading petechial rash involving the skin and mucous membranes may be noted. The client may also have IICP, manifested by decreased LOC, seizures, changes in vital signs and respiratory pattern, and papilledema. The manifestations of bacterial meningitis are listed in the box below.

Complications Complications of bacterial meningitis include arthritis, cranial nerve damage, and hydrocephalus. Cranial nerve VIII, the auditory nerve, is frequently affected, with resulting nerve deafness. Thrombophlebitis may develop in cerebral vessels, with infarction of surrounding tissues (Porth, 2005).

VIRAL MENINGITIS Acute viral meningitis, also called *aseptic meningitis*, is a less severe disease than bacterial meningitis. It can be caused by numerous viruses, such as herpes simplex, herpes zoster, Epstein-Barr virus, or cytomegalovirus (CMV). Viral meningitis most often appears after a case of mumps. Although viral infection also triggers the inflammatory response, the course of the disease is benign and of short duration. Recovery is uneventful.

Manifestations The manifestations of viral meningitis are similar to those of bacterial meningitis, although usually milder. The client may have a mild flulike illness prior to the onset of meningitis. Headache is intense and is accompanied by malaise, nausea, vomiting, and lethargy. Photophobia may be present. The

MANIFESTATIONS of Bacterial Meningitis

- Restlessness, agitation, and irritability
- Severe headache
- Signs of meningeal irritation:
  a. Nuchal rigidity
  b. Positive Brudzinski's sign
  c. Positive Kernig's sign
- Chills and high fever
- Confusion, altered LOC
- Photophobia (aversion to light), diplopia
- Seizures
- Signs of increased ICP (widened pulse pressure and bradycardia, respiratory irregularity, decreased LOC, headache, and vomiting)
- Petechial rash (in meningococcal meningitis)

MEDIALINK Case Study: Bacterial Meningitis

client generally remains oriented, although possibly drowsy. Temperature is mildly elevated. Neck stiffness, positive Brudzinski's sign, and positive Kernig's sign are usually present.

Encephalitis

Encephalitis is an acute inflammation of the parenchyma of the brain or spinal cord. It is almost always caused by a virus, but it may also be caused by bacteria, fungi, and other organisms. Other less common causes include ingested lead; postvaccination encephalitis (from vaccines for measles, mumps, and rabies), and HIV (Porth, 2005). See Table 44–11 for a list of the most common causes of encephalitis.

VIRAL ENCEPHALITIS Viruses depend on living tissue for reproduction and become highly destructive when they invade brain tissue. The inflammatory response extends over the cerebral cortex, the white matter, and the meninges, with degeneration of the neurons. The pathology of encephalitis includes local necrotizing hemorrhage, which ultimately becomes generalized, with prominent edema. There is progressive degeneration of nerve cell bodies. The inflammatory response in encephalitis does not cause exudate formation as it does in meningitis. Certain viruses show a propensity for specific areas of the brain (e.g., herpes simplex virus involves frontal and temporal lobes). The virus gains access to the CNS via the bloodstream or along peripheral or cranial nerves, or it may already be present in the meninges in the client with meningitis.

The manifestations of viral encephalitis vary, depending on the organism and area of the brain affected. Usual manifestations are similar to those of meningitis, including fever, headache, seizures, stiff neck, and altered LOC. The client may be disoriented, agitated and restless, or lethargic and drowsy. As the disease progresses, the LOC deteriorates, and the client may become comatose.

ARBOVIRUS ENCEPHALITIS The arboviruses are arthropod (mosquito or tick)-borne agents that infect humans. They include many different types, including Western equine encephalitis, St. Louis encephalitis, and Rift Valley fever. Adults are most often infected with St. Louis encephalitis, with older adults affected more often. The arthropods may live in small mammals and birds, or may be carried by horses and deer. The newest arboviral encephalitis in the United States is West Nile encephalitis.

The arthropod-borne agents cause widespread degeneration of nerve cells, and edema and necrosis with or without hemorrhage occur. IICP may develop. Manifestations include fever, malaise, sore throat, nausea and vomiting, stiff neck, tremors, paralysis of extremities, exaggerated deep tendon reflexes, seizures, and altered LOC.

Brain Abscess

A brain abscess is an infection with a collection of purulent material within the brain tissue. Approximately 80% are found in the cerebrum and 20% are cerebellar.

The causes of a brain abscess include open trauma and neurosurgery; infections of the mastoid, middle ear, nasal cavity, or nasal sinuses; metastatic spread from distant foci (such as heart, lungs, skin, abscessed teeth, and dirty needles); and arising from other associated areas of infection. The immunocompromised are at increased risk for abscesses. The most common

TABLE 44–11 **Causes of Encephalitis**

CAUSE	COMMENTS
Arboviruses	Transmitted by bites from ticks and mosquitoes. Bites from ticks occur more frequently in spring. Bites from mosquitoes occur in middle to late summer. Most common types are St. Louis and Eastern and Western equine encephalitis. May destroy major parts of the lobe or hemisphere. Two-thirds of clients who develop Eastern equine encephalitis either die or develop severe residual disabilities (e.g., seizures, blindness, deafness, speech disorders, or mental retardation). The incubation is 5 to 15 days. Mortality rates associated with arboviruses are higher than those associated with enteroviruses.
Enteroviruses, such as echovirus, coxsackievirus, poliovirus, paramyxovirus (the virus that causes mumps), and varicella-zoster (the virus that causes chickenpox)	Infection occurs more frequently in summer (except infection by the mumps virus, which occurs more frequently in early winter). Some degree of protection can be afforded by immunization against measles, mumps, and poliomyelitis. Mortality rates are lower than those associated with herpes simplex type 1 virus.
Herpes simplex type 1 virus	Most common nonepidemic encephalitis in North America. Can occur any time of year and throughout the world. Has an affinity for the frontal and temporal lobes. Prognosis is grave but not hopeless: Mortality rate can be as high as 40%, and client may die within 2 weeks.
Amebic meningoencephalitis due to infection by *Naegleria* and *Acanthamoeba* protozoa	Both protozoa are found in warm freshwater. Enter the nasal mucosa of people swimming in ponds or lakes. May also be found in soil and decaying vegetation. Incidence of infection is increasing in North America.
Exogenous poisoning	May occur after ingestion of lead or arsenic or inhalation of carbon monoxide.

pathogens causing the abscess are streptococci, staphylococci, and bacteroids. Yeast and fungi may also cause brain abscess.

A brain abscess results from the presence of microorganisms in the brain tissue. If the abscess is encapsulated, it has the ability to enlarge and, therefore, behave as a space-occupying lesion within the cranium. This predisposes the client not only to the systemic effects of the inflammatory process but also to the serious consequences of increased intracranial pressure. Occasionally, the abscess does not become encapsulated; instead, it spreads through the brain tissue to the subarachnoid space and ventricular system.

Initially, the client exhibits the general symptoms associated with an acute infectious process, such as chills, fever, malaise, and anorexia. Because brain abscess generally forms after infection, the client may consider these signs to be an exacerbation of that illness. The client may experience seizures, altered LOC, and manifestations of IICP. As the abscess enlarges, specific symptoms are related to location; for example, the client with a frontal lobe abscess may have contralateral hemiparesis, expressive aphasia, focal seizures, and frontal headache.

INTERDISCIPLINARY CARE

Bacterial meningitis is a medical emergency that, if not treated immediately, can be fatal within days. Successful management depends on rapid diagnosis and aggressive treatment with antibiotics and corticosteroids to eradicate the infecting organism and support vital functions. The client may be placed in strict or respiratory isolation until the organism has been identified, depending on hospital policy. Universal precautions apply to CSF as well as blood.

Treatment for viral meningitis focuses on managing client symptoms and is supportive. Antipyretics and analgesics may provide relief. Antibiotic therapy is not indicated, and isolation precautions are not required.

Treatment of the client with a brain abscess focuses on prompt initiation of antibiotic therapy. Other manifestations are treated symptomatically, as with the client diagnosed with meningitis or encephalitis. If pharmacologic management is not effective, the abscess may be drained or, if it is encapsulated, removed.

Diagnosis

The diagnosis of meningitis is based on manifestations and diagnostic tests results. Gram stain and culture of the CSF are used to determine if a bacterial infection is present and to determine the specific infectious agent. Counterimmunoelectrophoresis (CIE) is a laboratory test that may be ordered to determine the presence of viruses or protozoa. Polymerase chain reaction techniques may be used to detect viral DNA or RNA in spinal fluid. CT scan will show an area of increased contrast surrounding a low-density core with brain abscess.

Lumbar puncture with examination of the CSF is the definitive diagnostic measure for bacterial meningitis. Data that indicate bacterial meningitis include turbid, cloudy fluid; a markedly increased white blood cell (WBC) count and protein content; and a decreased glucose content. The opening pressure on the lumbar puncture is elevated. In contrast, the client with encephalitis may have a normal CSF analysis and pressure or may have some lymphocytes. The client with a brain abscess will have a markedly elevated pressure with elevated protein content and elevated WBC count. Glucose content is normal. (Because a lumbar puncture in the presence of a space-occupying lesion can result in brain herniation and death, a CT scan is performed first if neurologic findings support such a lesion.)

Medications

Immediate intravenous administration of a broad-spectrum antibiotic that crosses the blood–brain barrier into the subarachnoid space is instituted in cases of bacterial meningitis. Once culture reports identify the causative organism, drug therapy is continued from 7 to 21 days, using the most effective drug or drugs specific to that bacterium. The cephalosporin antibiotics are preferred. A major concern in the treatment of CNS infections is penicillin-resistant streptococci. Recommendations for treatment are for a broad-spectrum cephalosporin, such as rifampin (Rifadin), cefotaxime (Claforan), or vancomycin (Vancocin). However, as the bacteria are killed, the toxins they release increase production of inflammatory cytokines, which are potentially lethal. Steroids such as dexamethasone (Decadron) are often given with the antibiotics to suppress inflammation. The CDC recommends that the client remain on isolation for 24 hours after the start of antibiotic therapy.

Treatment for encephalitis consists of administering specific medications and preventing complications. Fungal meningitis is treated with antifungal agents, such as amphotericin-B (Amphotec), flucytosine (Ancobon), and fluconazole (Diflucan). Viral encephalitis is treated with intravenous acyclovir (Zovirax) or vidarabine (Vira-A).

Antibiotic therapy is the primary treatment for brain abscess. A combination of broad-spectrum antibiotics is used if the infecting organism is unknown.

Anticonvulsant medications such as phenytoin (Dilantin) are often prescribed to prevent or control seizure activity. Antipyretic and analgesic medications may provide symptomatic relief; however, analgesics that have a depressant effect on the CNS (such as opiates) are avoided to prevent masking of early manifestations of deteriorating LOC. The client initially may require antiemetics to control nausea and vomiting. Fluid and electrolyte status is maintained through intravenous fluid replacement until the client is able to resume oral intake.

Surgery

Surgical drainage of an encapsulated abscess may be necessary. The decision to perform surgery is based on the client's general condition, the stage of abscess development, and the site of the abscess.

NURSING CARE

Central nervous system infections are serious illnesses, with potentially life-threatening effects and complications. Nursing assessments and interventions are critical in identifying changes in the client's neurologic status and preventing complications from IICP. In addition to the nursing care described in this section, a Nursing Care Plan for a client with bacterial meningitis is found on the next page.

NURSING CARE PLAN A Client with Bacterial Meningitis

Monty Cook is a 22-year-old musician who plays in a local rock band. He is unmarried and lives with his parents. He is known by everyone in the community as a quiet, low-key, easygoing person and an excellent guitar player. During a performance 2 days ago, he had difficulty playing his guitar, complaining of bright stage lights blazing in his eyes. When he tried to keep his head down to prevent the lights from hurting his eyes, he noticed his neck was very stiff. After the performance, one of the newest members of the band remarked that it certainly was not their best performance. Monty responded angrily that maybe the new members of the group needed more practice. Then he stomped out and went home to bed.

He wakes at 4:00 A.M. with a severe headache, sweating, and chills; his temperature is 102°F, and he cannot bend his neck without severe pain. His mother recognizes that he is agitated and irritable, which is uncharacteristic. Frightened, she rushes him to the hospital emergency room. A lumbar puncture performed in the emergency room reveals turbid, cloudly fluid; a markedly increased WBC count; and protein with a decreased glucose content. Bacterial meningitis is the medical diagnosis. Mr. Cook is admitted to the hospital for treatment and care.

ASSESSMENT

When the nurse, Aisha Aldi, enters Mr. Cook's isolation room, she sees him thrashing about in the bed, talking incoherently, and becoming more agitated. On assessment, Ms. Aldi notes dry mucous membranes, cracked lips, and small petechiae over the upper torso and abdomen. Mr. Cook's temperature is 104°F. Kernig's sign is positive. Intravenous broad-spectrum antibiotics are prescribed and initiated. After the first 2 hours of care, Ms. Aldi notes a decrease in Mr. Cook's level of consciousness.

DIAGNOSES

- *Hyperthermia* related to infection and abnormal temperature regulation by hypothalamus
- *Disturbed Thought Processes* related to intracranial infection
- *Ineffective Protection* related to progression of illness

EXPECTED OUTCOMES

- Have a decrease in body temperature.
- Become less restless and agitated.
- Remain free of injury.

PLANNING AND IMPLEMENTATION

- Monitor vital signs every 2 hours.
- Provide sponge baths if temperature continues to rise.
- Provide a quiet, nonstimulating environment with the shades drawn.
- Provide oral care every 4 hours.
- Measure and compare intake and output every 2 hours.
- Perform neurologic assessments every 2 to 4 hours.
- Monitor for and report seizure activity and decreasing level of consciousness.
- Keep bed in low position with side rails elevated.
- Administer prescribed intravenous antibiotics.

EVALUATION

After 4 days of antibiotic therapy, Mr. Cook's temperature has returned to near normal. Ms. Aldi notes that he has begun opening his eyes and visually tracking her as she moves about the room. Mr. Cook responds to a request to squeeze Ms. Aldi's fingers and after several hours asks her what had happened. On day 5, Mr. Cook states that he feels better and his headache is gone. He ask for sips of juice and begins urinating regularly. Seven days after admission, Mr. Cook is discharged and is able to go home with his mother. He has some weakness in his legs, but otherwise has no evidence of neurologic deficits.

CRITICAL THINKING IN THE NURSING PROCESS

1. What strategies should the nurse use to decrease the environmental stimuli for Mr. Cook, and what is the rationale for doing these?
2. If you were caring for Mr. Cook in the initial phase of the illness and he became combative, what would you do?
3. Develop a plan of care for Mr. Cook for the nursing diagnosis *Acute Pain*. Consider the effect of narcotics on respiratory function in designing the plan.

See Evaluating Your Response in Appendix C.

Health Promotion

As with many other intracranial injuries and disorders, educational activities to promote health by preventing CNS infections are important nursing interventions. The following information should be provided:.

- Vaccinations for meningococcal meningitis are recommended or required for military recruits and college students (groups at increased risk for invasive meningococcal meningitis).
- Administration of prophylactic rifampin (Rifadin) is recommended for people exposed to meningococcal meningitis.
- Mosquito control with repellants, insecticides, and protective clothing.
- Destruction of the insect larvae and elimination of breeding places, such as pools of stagnant water.
- Vaccination against Japanese B encephalitis (recommended for summer travelers to rural East Asia).
- Prompt diagnosis and treatment of infections of the head, neck, and respiratory system.

Assessment

Collect the following data through the health history and physical examination (see Chapter 43 ∞). Further focused assessments are described with nursing interventions below.

- *Health history:* Risk factors (concurrent infections, other illnesses, travel), when manifestations began, severity of manifestations, current nausea and headache, seizures.
- *Physical examination:* Glasgow Coma Scale, level of consciousness, vital signs, motor function, pupillary check, cranial nerves, neck ROM, Brudzinski's sign, Kernig's sign, skin (rash, petechiae, purpura), muscle movement and strength, speech.

Nursing Diagnoses and Interventions

In planning and implementing nursing care for the client with a CNS infection, the prognosis may depend on the supportive care given. The client is often very ill, and the combination of fever, dehydration, and cerebral edema may predispose the client to

seizures. Airway obstruction, respiratory arrest, or cardiac dysrhythmias may occur. Nursing diagnoses and interventions previously discussed for the client with an altered LOC, IICP, and seizures are also appropriate for the client with a CNS infection. Nursing interventions in this section focus on ineffective protection and risk for fluid volume deficit.

Ineffective Protection

Clients with CNS infections are less able to protect themselves against insults from both internal and external sources. The effects of the inflammation and resulting pathophysiologic processes may include pain, fever, altered LOC, seizures, IICP, and cranial nerve dysfunction. In addition, pathophysiologic effects on the brain from toxins or thrombosis of a cerebral vessel may lead to permanent neurologic deficits, such as loss of motor function or dementia.

- Monitor neurologic status on a regular basis. *Many complications are evidenced by changes in neurologic manifestations.*
- Monitor vital signs, including temperature, on a regular basis. *The client often has a high temperature throughout the illness, ranging from 101°F (38°C) to 105°F (40.5°C).*

PRACTICE ALERT
Hyperthermia may result from increased intracranial pressure, while an increased temperature can also increase ICP.

- Monitor levels of consciousness. Assess levels of orientation, memory, attention span, and response to stimuli. *Early in the infection, the client often has problems with memory and orientation. There may be problems with following commands, restlessness, irritability, and combativeness. As the illness progresses, the LOC decreases to lethargy and finally into deep coma.*
- Monitor for manifestations of seizure activity, and institute seizure precautions:
  - Monitor twitching of hands or face and tonic-clonic movements.
  - Have an oral airway and suction equipment readily available.
  - Pad side rails, maintain bed in low position, and keep side rails up.

  Irritation of the cerebral cortex secondary to meningeal inflammation may cause seizures. Careful monitoring and seizure precautions are necessary to prevent injury.
- Monitor for manifestations of cranial nerve damage; monitor extraocular movements, facial movement, dizziness, ability to hear, double vision, drooping upper eyelids (ptosis), and pupillary changes. *Cranial nerve dysfunction may result from inflammation or vascular changes in the brain.*
- Monitor for manifestations of increased intracranial pressure: decreased pulse, increased blood pressure, widening pulse pressure, respiratory changes, and vomiting. *Increased intracranial pressure results from infectious or inflammatory exudate, cerebral edema, and hydrocephalus.*
- Administer prescribed medications and maintain prescribed fluid restrictions. *Diuretics are often prescribed to prevent increases in ICP, anticonvulsants are prescribed to prevent or control seizures, and antibiotics are prescribed to eradicate the bacteria. Fluids may be restricted to help prevent IICP.*

Risk for Deficient Fluid Volume

The client is at risk for fluid volume deficit related to increased metabolic rate, diaphoresis, and fluid restrictions.

- Monitor for presence, or worsening, of fluid volume deficit.
  - Measure and compare intake and output every 2 to 4 hours.
  - Monitor daily body weights.
  - Monitor skin turgor.
  - Monitor condition of mucous membranes.
  - Monitor urine amount, color, and odor.
  - Monitor BUN:creatinine ratio.

  The elastic property of the skin depends partially on interstitial fluid volume. If there is a fluid volume deficit, skin flattens more slowly after a pinch is released. Mucous membranes are dry. In fluid volume deficit, urine output is decreased, urine is dark in color and concentrated with a strong odor, and urine specific gravity is greater than 1.020, and BUN will rise out of proportion to serum creatinine.

PRACTICE ALERT
A weight loss of 1 lb represents a fluid loss of approximately 500 mL.

- When administering fluids, either orally or parenterally, consider concurrent illnesses. *Clients with increased intracranial pressure or renal failure require complex management. See Chapter 10 for a further discussion of fluid volume deficit.*

Using NANDA, NIC, and NOC

Chart 44–3 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with a CNS infection.

NANDA, NIC, AND NOC LINKAGES
CHART 44–3 **The Client with a CNS Infection**

NANDA

Risk for Imbalanced Body Temperature

↓

NIC

Vital Signs Monitoring

Cerebral Edema Management

Temperature Regulation

↓

NOC

Infection Severity

Neurologic Status: Autonomic

Thermoregulation

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

Community-Based Care

The importance of preventive measures, such as recognizing predisposing conditions, is a major focus for client education. People who have had close contact with the client with meningitis should be monitored for fever, headache, or neck stiffness. Some physicians believe that those closest to the client are candidates for antimicrobial prophylaxis. Also address the following topics:

- The need to report any manifestations of ear infection, sore throat, or upper respiratory infection
- The names and purposes of all medications that may be prescribed
- The importance of taking all medication until completely gone, because some clients may think it is acceptable to stop the medication as soon as they feel better.

TUMORS OF THE BRAIN

THE CLIENT WITH A BRAIN TUMOR

Brain tumors are growths within the cranium, including tumors in brain tissue, meninges, the pituitary gland, or blood vessels. Brain tumors may be benign or malignant, primary or metastatic, and intracerebral or extracerebral. Regardless of type or location, brain tumors are potentially lethal as they grow within a closed cranial vault and displace or impinge on CNS structures.

Incidence and Prevalence

An estimated 19,000 new cases of malignant brain tumors are diagnosed annually in the United States, and approximately 13,000 people die each year (American Cancer Society, 2006). Although brain tumors can occur in any age group, the highest incidence in adults is in those ages 50 to 70. In the adult population, the most common tumor is glioblastoma multiforme, followed by meningioma and cytoma. Glioblastomas represent more than 50% of all primary intracranial lesions.

The cause of many brain tumors is unknown. Although a number of chemical and viral agents can cause brain tumors in laboratory animals, there is no evidence that these agents cause tumors in humans. Other factors associated with brain tumors include heredity, cranial irradiation, and exposure to some chemicals (Porth, 2005).

Pathophysiology

Brain tumors may be classified as benign or malignant, based on the tissue type and characteristics of the cells. The use of the term *benign* may be misleading. A tumor that is benign by histologic examination but is surgically inaccessible may continue to grow and expand, increasing intracranial pressure and causing neurologic deficits, herniation, and finally death. In discussions of brain tumors, the term *malignant* is used to describe the lack of cell differentiation, the invasive nature of the tumor, and its ability to metastasize.

Brain tumors also may be classified as primary or metastatic, depending on their origin (Table 44–12). Primary tumors of CNS tissue arise from the cells and structures that are found within the brain, for example, neurons and neuroglia. The primary intracranial tumors that originate in the skull cavity but not from brain tissue itself arise from the supporting structures, including the meninges, pituitary gland, and pineal gland. Primary brain tumors rarely metastasize outside the CNS. Metastatic brain tumors originate from structures outside the brain, such as the breasts, lungs, and prostate gland.

Focal disturbances take place when there is compression of brain tissue and infiltration or direct invasion of brain parenchyma with destruction of neural tissue. As the tumor grows, edema develops in adjacent tissues. The mechanism is not completely understood, but it is thought that an osmotic gradient causes the tumor to absorb fluid. Some tumors may cause hemorrhage. Venous obstruction and edema due to breakdown of the blood–brain barrier increase intracranial volume and intracranial pressure. Obstruction of the circulation of CSF from the lateral ventricles to the subarachnoid space causes hydrocephalus.

An estimated 25% of people with cancer develop brain metastasis. Metastatic brain tumors present in the same way as primary brain tumors, with IICP, and focal and/or diffuse cerebral dysfunction. The most common source of intracranial metastasis is cancer of the lung. Other common primary sites are the breast, kidney, and gastrointestinal tract. The metastasis reaches the brain through the circulation. In most cases, the tumors are multiple and are scattered through the cerebellum and cerebrum.

Manifestations

Multiple manifestations can develop as a result of the growth of the tumor, while others are related to the location of the lesion (see the box below). Some of the more common mani-

MANIFESTATIONS of Brain Tumors

Frontal Lobe Tumors
- Inappropriate behavior
- Personality changes
- Inability to concentrate
- Impaired judgment
- Recent memory loss
- Headache
- Expressive aphasia
- Motor dysfunctions

Parietal Lobe Tumors
- Sensory deficits: paresthesia, loss of two-point discrimination, visual field deficits

Temporal Lobe Tumors
- Psychomotor seizures

Occipital Lobe Tumors
- Visual disturbances

Cerebellum Tumors
- Disturbances in coordination and equilibrium

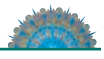
Pituitary Tumors
- Endocrine dysfunction
- Visual deficits
- Headache

TABLE 44–12 **Classification on Brain Tumors**

TUMOR TYPE	TUMOR NAME	CHARACTERISTICS
Primary Tumors		
Intracerebral tumors	*Glioma*	
Account for 40%–50% of all brain tumors	■ Astrocytoma	Most common glioma Graded I to IV according to degree of cell differentiation
Originate from neuroglia and invade brain tissue	■ Glioblastoma multiforme	Most malignant form Fast growing
Most common type of brain tumor	■ Ependymoma	Tumor that develops from lining of ventricles Graded I to IV according to degree of cell differentiation Slow growing
	■ Oligodendroglioma	Rare, slow growing May be encapsulated
	■ Astroblastoma	Benign
Extracerebral tumors		
Tumors arising from the supporting structures of the nervous system Account for 10% to 15% of all brain tumors	Medulloblastoma	Fast growing and malignant Occurs primarily in children; can occur in adults Found in cerebellum
	Meningioma	Slow growing Develops in meninges (especially dura) Firm and encapsulated
	Acoustic neuroma	Slow growing Benign Originates from Schwann cells of the cranial nerve XIII May also affect cranial nerves V, VII, IX, and X Also called neurofibromatosis Genetic origin due to autosomal dominant mendelian trait Firm, encapsulated lesions attached to nerve
Congenital (developmental) tumors	Hemangioblastoma	Vascular tumor Slow growing
Account for 4% to 8% of all brain tumors	Craniopharyngioma	Originates from Rathke's pouch Solid or cystic tumor Compresses pituitary gland Presses on the third ventricle and may cause blockage of cerebrospinal fluid (CSF)
Pituitary adenomas Account for 8% to 12% of all brain tumors	Chromophobic	Account for 90% pituitary tumors Nonsecreting tumor Slow growing
	Eosinophilic	Secreting tumors that produce growth hormone
	Basophilic	Secreting tumors that produce adrenocorticotropic hormone Fast growing
Secondary Tumors		
Metastatic brain tumors Account for 10% of all brain tumors		Slow-growing tumors that arise from other parts of the body Usually well differentiated from the brain Spread from tumors of the lung, breast, lower gastrointestinal tract, pancreas, kidney, skin

festations include changes in cognition or consciousness, a headache that is usually worse in the morning, seizures, and vomiting. Compression of brain tissue and the invasion of the brain tumor into the cerebral tissue may lead to changes typically seen with cerebral edema and IICP. Cerebral blood supply may diminish as the tumor compresses blood vessels. Shifts in brain tissue can occur, leading to brain herniation syndromes and, if untreated, death.

INTERDISCIPLINARY CARE



Treatment for a brain tumor may involve chemotherapy, radiation therapy, surgery, or any combination of these. Several variables are considered when selecting the appropriate treatment modality: the size and location of the tumor, the type of tumor, related symptoms (such as neurologic deficits), and the client's overall condition.

Diagnosis

The following diagnostic tests may be ordered.

- A CT scan or an MRI with gadolinium enhancement can locate the tumor and define its size, shape, extent to which normal anatomy is distorted, and the degree of any associated cerebral edema.
- Arteriography may show stretching or displacement of cerebral vessels by the tumor, as well as the presence of tumor vascularity.
- EEG provides information about cerebral function, may demonstrate focal or diffuse changes, and is useful if seizures are present.
- Endocrine studies are conducted if a pituitary tumor is suspected.

Medications

The choice of drug for treatment is based on the type of tumor, its location, and the client's response to therapy. The use of chemotherapy to treat brain tumors is still emerging. An intraventricular method of medication administration uses an Ommaya reservoir that has been surgically implanted into a lateral ventricle of the brain (Figure 44–8 ■). Other medications that may be prescribed include corticosteroids and anticonvulsants.

Surgery

Surgery is used to remove tumors, to reduce the size of the tumor, or for symptom relief (palliation). The type of procedure, the surgical approach, and the timing of surgery (emergency versus planned procedure) influence the overall nursing management of the client having intracranial surgery.

Some of the more common intracranial neurosurgical procedures follow:

- *Burr hole.* A hole made in the skull with a special drill. The hole may facilitate the evacuation of an extracerebral clot, or a series of holes may be made in preparation for craniotomy (see Figure 44–7).
- *Craniotomy.* A surgical opening into the cranial cavity (Figure 44–9 ■). For a craniotomy, a series of burr holes are made. The bone between the holes is then cut with a special saw called a craniotome. The tumor is excised, and the bone flap is returned to the opening. A craniotomy may also be performed to repair defects associated with traumatic head injuries or to repair a cerebral aneurysm.

  A supratentorial craniotomy refers to surgery above the tentorium. It provides access to the frontal, temporal, parietal, and occipital lobes. The incision for this procedure is usually within the hairline over the area involved.

  An infratentorial craniotomy refers to surgery below the tentorium. Access is provided to lesions in the cerebellum and the brainstem. The incision is made at the nape of the neck, around the occipital lobe.
- *Craniectomy.* An excision of a portion of the skull and complete removal of the bone flap. This procedure may be done to provide decompression after cerebral edema. Pressure on the brain structures is lessened by providing space for expansion.
- *Cranioplasty.* Plastic repair to the skull in which synthetic material is inserted to replace the cranial bone that was removed. This procedure may be performed after a large craniectomy. The plastic repair restores the contour and integrity of the cranium.

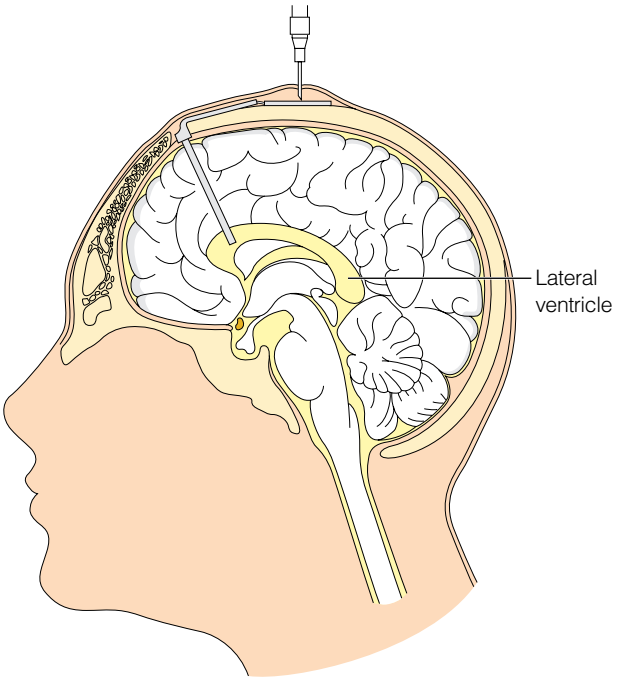


Figure 44–8 ■ Ommaya reservoir for medication administration.

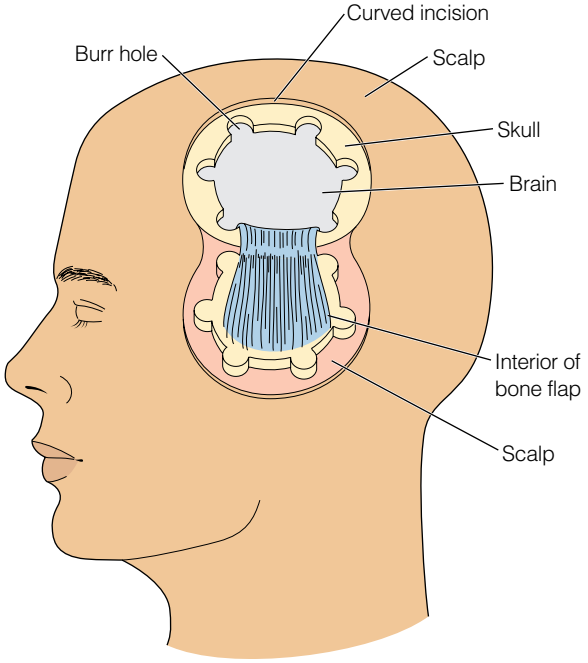


Figure 44–9 ■ In a craniotomy, a portion of the skull and overlying scalp is removed to allow access to the brain.

Radiation Therapy

Radiation therapy may be administered alone or as adjunctive therapy with surgery. Radiation is often the treatment of choice for surgically inaccessible tumors; it may also be used to decrease the size of a tumor prior to surgery. Tumors that were not completely excised by surgery may also be treated with radiation.

Specialty Procedures

Technologic advances—including the development of special instruments, the use of stereotaxic techniques for localizing a specific target, and the use of the laser beam—have greatly advanced neurosurgical practice. Microsurgery involves an operating microscope with microinstruments and supportive illumination equipment. Using stereotaxic techniques to precisely locate a specific target point allows for location of discrete areas of the brain that control specific functions and exact locations of deep brain lesions. The use of a laser beam for excision of a tumor results in less damage to surrounding tissue and less postoperative swelling. The gamma knife, which is not actually a knife but a gamma unit, consists of a heavily shielded helmet containing 201 sources of cobalt-60, which is capable of destroying deep and otherwise inaccessible lesions in a single treatment session. A new area of chemotherapy is that of biodegradable anhydrous wafers, which are impregnated with the chemotherapy drug and implanted into the tumor at the time of surgery. The wafers are made so they slowly release the drug over a period of many months (Porth, 2005).

NURSING CARE

The nursing care of the client with a brain tumor includes support during the diagnostic period and specific management as directed by the selected treatment. The foundation for care is data from the health history and physical assessment, which includes identifying neurologic deficits. This information directs planning and implementing care. Many of the alterations in health commonly experienced by the client with a brain tumor have been discussed throughout this chapter, including altered level of consciousness, increased intracranial pressure, and seizures. The client will require intensive care in the immediate postoperative period. In addition to the nursing care described in this section, a Nursing Care Plan for a client with a brain tumor is found on page 1573.

Nursing Diagnoses and Interventions

This section of the chapter focuses on nursing interventions for the client who has intracranial surgery. The nursing diagnoses discussed are anxiety, risk for infection, ineffective protection, acute pain, and low self-esteem.

Anxiety

The diagnosis of a brain tumor brings anxiety and feelings of uncertainty about the future. Both the client and family members are likely to be apprehensive and require education and emotional support.

- Explain routine medical procedures, including blood work and radiologic studies. *Baseline laboratory and radiologic studies are needed to ensure that the client has no other preexisting medical condition. Explaining the procedures and assisting the client through this process help decrease anxiety.*
- Reinforce, clarify, and repeat information. *Both client and family may have limited understanding of the scheduled diagnostic tests, procedures, and treatment modalities. The client may be confused or have altered thought processes as a result of the tumor. Information may need to be repeated or reexplained.*
- Encourage client and family to verbalize feelings, questions, and fears; provide realistic information appropriate to their level of understanding. *Verbalization helps reduce anxiety and fear.*
- Review client and family strengths and effective coping skills. *Personal strengths, support systems, and coping skills can aid in the development of appropriate strategies to reduce anxiety.*
- Arrange for a member of the clergy to visit if desired. *Faith in a higher being is often a strong source of strength and support.*
- Provide preoperative teaching, including the following information.
  - Type of anesthesia and surgery
  - Time surgery will begin and expected length of procedure and recovery room stay
  - Where the client will be taken after surgery (CCU, ICU) (If possible, show the client and family the CCU or ICU and introduce them to the nurse who will be in charge of care after the surgery.)
  - Where family can wait during and following surgery
  - Appearance of the client after surgery, which may include swollen, bruised eyelids and other facial features; a large dressing covering the head; a partially or fully shaved head; and a tracheostomy or endotracheal tube
  - Behavior of the client after surgery, which will differ depending on the site of surgery, although cognitive and behavioral changes are common.

  Information about what to expect reduces anxiety.
- Allow time for client and family to be together. *Clients and families need quiet time together to support each other and prepare emotionally for surgery.*

Risk for Infection

The client who has had intracranial surgery is at risk for infection from multiple invasive lines, the scalp wound, and the risk of introduction of bacteria into the operative area. The nurse provides interventions to monitor for and prevent infection.

- Monitor for leakage of CSF:
  - Presence of glucose in clear drainage from ears, nose, or wound
  - Complaints of "something dripping down the back of the throat"
  - Constant swallowing.

  These manifestations indicate an opening in the dura, which provides an avenue for an ascending infection.
- Provide interventions to prevent contamination of area leaking CSF:

NURSING CARE PLAN A Client with a Brain Tumor

Claire Lange is a 44-year-old television announcer. During one night's broadcast, she confuses several major news items so badly that her co-anchor tries to correct her. Ms. Lange responds angrily that she does not need any help and then rises and storms off the set. As she leaves the camera area, she limps noticeably and appears to drag her left leg. The show's producer asks her what is wrong; she screams that nothing is wrong—she simply has another headache. He follows her to her dressing room and inquires about her headaches. She tells him that they come and go but have been getting worse lately. He then asks her if she has injured her left leg; she responds that the leg was weak because she was tired. As the producer leaves the dressing room, Ms. Lange begins to shake and collapses on the floor. The producer recognizes that she is having a seizure and calls for an ambulance.

Ms. Lange is admitted to the neurology floor of the local hospital for evaluation. A CT scan, MRI, and EEG are completed and identify an intracranial mass. A biopsy of the mass is positive for malignant cells. A glioma in the frontal lobe is identified, and surgery is scheduled for that week.

ASSESSMENT

When Clara Rosetti, RN, enters Ms. Lange's room, she sees Ms. Lange looking at her shoulder-length hair in the mirror. Ms. Lange tells Ms. Rosetti that she has never in her life worn her hair any shorter, and "Now you're going to cut it all off!" She paces the room and makes the statement, "I guess the hair isn't really important if I survive this situation." She also says that she has a headache.

DIAGNOSES

- *Acute Pain* (headache) related to tumor and increase in intracranial pressure
- *Disturbed Body Image* related to upcoming hair loss and cranial incision
- *Anxiety* related to unknown future following surgery

EXPECTED OUTCOMES

- Verbalize the causes of pain.
- Verbalize an understanding of the changes in body appearance that are associated with the scheduled intracranial surgery (e.g., shaving of the head prior to surgery, cranial incision, facial swelling postoperatively).
- Identify measures that will help minimize the effect of the hair loss.
- Verbalize a reduction in anxiety.

PLANNING AND IMPLEMENTATION

- Assess level of discomfort using a rating scale of 0 to 10.
- Provide a quiet, nonstimulating environment.
- Position the client for comfort, keeping the head of the bed elevated to promote venous drainage.
- Assess level of consciousness for potential increases in ICP.
- Encourage to verbalize feelings about the surgery.
- Suggest measures that may help minimize the hair loss, such as the use of turbans, scarves, hats, and wigs.
- Suggest relaxation techniques to decrease anxiety.

EVALUATION

By the time of surgery, Ms. Lange has recognized the relationship between the brain tumor and the headache. She states that lying in a flat position and coughing increase the headache. The head of the bed is kept at a 30- to 45-degree angle. Daily activities are spaced to provide periods of rest. Ms. Lange demonstrates no significant changes in level of consciousness. She has talked about the effect of the hair loss and her television responsibilities. Ms. Lange has learned that the hair preparation would be done in surgery and that the hair would be saved for her. She states she has already consulted her hair stylist and that "scarves and turbans are on the way."

CRITICAL THINKING IN THE NURSING PROCESS

1. Outline interventions to decrease intracranial pressure both before and after surgery.
2. When making your initial assessments on the morning of surgery, you find that Ms. Lange has a decreased pulse and increased blood pressure. She tells you her headache is worse and suddenly vomits. What do you do now?
3. Ms. Lange asks you to be sure that she has absolutely no visitors after surgery, because she knows how ugly she will look. How would you respond?
4. Design a plan of care for Ms. Lange for the nursing diagnosis of *Powerlessness*.

See Evaluating Your Response in Appendix C.

- If leaking from the nose: Keep head of bed elevated 20 degrees unless contraindicated; do not suction nasally; do not clean nose; tell client not to put finger in nose; do not insert packing.
- If leaking from the ear: Position client on side of leakage unless contraindicated; do not clean ear; tell client not to put finger in ear; do not insert packing.
- Place a sterile dressing over the area of drainage and change as soon as it becomes damp.

Leakage of CSF indicates a break in the dura and increases the risk of an ascending infection. Surgery may be necessary to repair the break; however, the leak usually heals spontaneously in about 1 week.

- Monitor for and report manifestations of infection:
  - Take and record temperature on a regular basis.
  - Assess IV insertion sites for redness, swelling, drainage, and pain.
  - Assess scalp wound for redness, swelling, bulging, drainage, and pain.
  - Assess for manifestations of meningitis: fever and chills, increasing headache, neck stiffness, positive Kernig's or Brudzinski's sign, photophobia.
  - Monitor laboratory reports for increased WBC count.

Intact skin is the first line of defense against infection. Any break in the skin increases the risk of infection. Intracranial surgery increases the risk of meningitis, with infectious agents ascending into the brain.

- Implement interventions to prevent infection:
  - Use strict aseptic technique when changing dressings and when caring for wound drains and ICP monitor lines.

- Keep the client's hands away from drains and dressings; use mitten restraints if necessary.
- Administer prescribed antibiotics.

Sterile technique decreases the risk of introducing infection into a wound. Antibiotics are usually prescribed prophylactically to prevent infection.

Ineffective Protection

The client who has intracranial surgery does not have normal human defenses against changes in intracranial pressure and is also at risk from cerebral edema and a shift of intracerebral contents. In addition, the surgery may cause cerebral bleeding or hematoma formation.

- Monitor for manifestations of increased intracranial pressure:
  - Restlessness, agitation, and decreasing level of consciousness
  - Headache
  - Vomiting
  - Seizures
  - Decreasing sensory and motor function
  - Changes in pupil size and reaction
  - Changes in vital signs: altered respiratory rate or depth, increasing pulse pressure, decreasing pulse rate, increasing blood pressure
  - Abnormal posturing

  Increasing intracranial pressure is manifested by alterations in the functions and centers controlled by the brain.
- Implement interventions to decrease the risk of increased intracranial pressure:
  - Elevate the head of the bed 15 to 30 degrees as prescribed (unless contraindicated).
  - Avoid neck flexion or rotation; keep head in midline position unless a large bone flap or mass was removed; then position the client on unoperated side to decrease venous congestion in the operative area.
  - Do not take rectal temperatures.
  - Avoid clustering activities that increase intracranial pressure: suctioning, turning, bathing.
  - Administer medications to prevent vomiting.
  - Do not suction for more than 10 seconds at one time.
  - Teach the client (if possible) to avoid coughing, sneezing, and straining to have a bowel movement.
  - Maintain fluid restrictions as prescribed.
  - For internal shunts: Avoid pressure on the shunt, reservoir, or tubing. Pump the shunt only if prescribed.
  - For external shunts: Avoid kinks in tubing, and maintain the drainage collecting device and client's head at the prescribed levels.

  Keeping the head of the bed slightly elevated facilitates venous drainage from the brain. Neck flexion or rotation disrupts circulation to and from the brain. Rectal stimulation, suctioning, turning, bathing, coughing, sneezing, and straining to have a bowel movement all initiate Valsalva's maneuver, which constricts the jugular veins and impairs venous return from the brain. Fluid restriction may be prescribed to dehydrate the client slightly and lessen ICP.
- Maintain (as much as possible) a quiet, calm, softly lighted environment. Avoid excessive sensory stimulation. *These interventions promote rest and decrease stimulation, thereby reducing ICP.*
- Implement interventions to prevent seizures or, if they occur, to prevent injury to the client:
  - Pad side rails of the bed.
  - Place bed in lowest position, and keep side rails up.
  - Carry out interventions to prevent and treat increased intracranial pressure.
  - Have an oral airway and suction equipment immediately available.
  - Administer prescribed anticonvulsants.
  - If a seizure occurs: Maintain a patent airway; do not restrain client; do not force anything into the client's mouth; provide physical and emotional support.

  These interventions promote safety and help prevent injury. Anticonvulsants are often prescribed prophylactically to prevent seizures after intracranial surgery.
- Carefully monitor hydration status. Compare trends in intake and output, laboratory results of serum osmolality, and urine specific gravity and osmolality. *Changes in fluid balance and osmolality may result from excess intravenous fluids, osmotic diuretics, surgically induced diabetes insipidus or syndrome of inappropriate antidiuretic hormone secretion, fever, diarrhea, tube feedings, or hyperglycemia.*

Acute Pain

The client who has intracranial surgery has pain, manifested as a headache, as a result of either compression or displacement of brain tissue or from increased intracranial pressure. A headache may also be a manifestation of meningitis.

- Assess the location, duration, and intensity of the pain, using a scale from 0 (no pain) to 10 (worst pain) in the client who can verbally communicate. *The client is the best source of information about pain.*
- Implement interventions to reduce the pain:
  - Raise the head of the bed slightly.
  - Reduce noise and bright lights in the room.
  - If allowed, loosen head dressing.
  - Administer narcotic analgesics with caution.

  Nonpharmacologic measures may be used to reduce increased intracranial pressure and headache.

PRACTICE ALERT

Narcotic analgesics mask changes in eye signs and depress respirations.

Situational Low Self-Esteem

The client who has intracranial surgery has many alterations that affect self-esteem and body image. Physical changes include a loss of hair on the scalp, swelling and bruising in the eyelids and face, and perhaps an indentation in the skull. The client is no longer independent in self-care, but must depend on others to meet basic needs. There are often long-term neuro-

logic deficits, affecting areas such as speech, vision, and motor abilities, which require changes in roles and relationships.

- Assess for verbal and nonverbal manifestations of negative self-esteem:
  - Denial of changes
  - Preoccupation with changes
  - Refusal to look in the mirror
  - Withdrawal from family and friends
  - Expressions of grief and loss (see Chapter 5 ∞).

  Low self-esteem can be initiated by stressful situations and changes in body image.
- Provide interventions to improve self-concept:
  - Limit negative self-assessment.
  - Help focus on positive areas of life.
  - Help identify sources of support and strength.
  - Help identify and use helpful coping methods.
  - Encourage significant others to visit.
  - Encourage independence in self-care.

  Self-esteem is derived from one's own perceptions of competence and from the responses of others. When one's self-concept and self-ideal are congruent, self-esteem is enhanced.

Using NANDA, NIC, and NOC

Chart 44–4 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with a brain tumor.

NANDA, NIC, AND NOC LINKAGES

CHART 44–4 The Client with a Brain Tumor

NANDA

Powerlessness

↓

NIC

Self-Esteem Enhancement

Self-Responsibility Facilitation

Emotional Support

Environmental Management

Self-Care Assistance

↓

NOC

Self-Esteem

Health Beliefs: Perceived Control

Health Beliefs: Perceived Resources

Social Support

Hope

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

Community-Based Care

The effect of the possible outcomes following the surgery produces fear in both client and family, interfering with their ability to retain information. The client may have cognitive or neurologic deficits that interfere with learning. Family members also must be assessed for their ability to cope with the stress of the surgery. Information may have to be repeated several times.

Clients and their families who have experienced intracranial surgery require emotional support. The process of recovery is often extended and may involve adaptation to changes in body image and management of any motor or sensory deficits. The family should be involved in the care of the client. If family members are willing, they may begin to assist with ADLs while the client is in the hospital, such as assisting with personal hygiene and meals. Clients should also be encouraged to take an active role in their own care. Discharge planning includes a discussion of the following topics: medication information; wound care; the use of wigs, turbans, hats, or colorful scarves; and the importance of follow-up visits. In addition, emphasize the importance of reporting manifestations such as stiff neck, increasing headache, elevated temperature, new motor or sensory deficits, vision changes, or seizures.

Provide information about the overall treatment plan, management of deficits and/or disabilities, and future needs. Specific teaching topics are as follows:

- Safety measures for motor deficits, sensory deficits, lack of coordination, seizures, and cognitive deficits
- Comfort measures for nausea, vomiting, and pain
- Measures for communication if aphasia is present
- Measures to improve vision if visual deficits are present
- How to buy wigs and hairpieces
- Referrals to support groups and community resources
- Helpful resources:
  - American Cancer Society
  - American Brain Tumor Association
  - National Brain Tumor Foundation
  - Brain Tumor Society.

EXPLORE MEDIALINK

Prentice Hall Nursing MediaLink DVD-ROM

Audio Glossary
NCLEX-RN® Review

Animations

Complex Seizure
Coup-Contrecoup Injury
Epilepsy
Grand Mal Seizure

COMPANION WEBSITE www. prenhall.com/lemone

Audio Glossary
NCLEX-RN® Review
Care Plan Activity: Subdural Hematoma
Case Study: Bacterial Meningitis
MediaLink Applications
Meningitis Prevention
Paralysis
Links to Resources

CHAPTER HIGHLIGHTS

- Altered level of consciousness (LOC) is a common response to intracranial disorders, and is an early manifestation of deterioration of the function of the cerebral hemispheres. The alteration in cerebral function occurs in a sequential pattern, with characteristic changes in LOC, respiratory patterns, pupillary and oculomotor responses, and motor function. Coma states include persistent vegetative state and locked-in syndrome.
- Increased intracranial pressure (IICP) is a sustained elevated pressure (≥10 mmHg) within the cranial cavity. IICP may result from cerebral edema, hydrocephalus, head trauma, tumors, abscesses, inflammation, hemorrhage, or stroke.
- The manifestations of IICP include a decreasing LOC, abnormal motor weakness and responses, altered vision, altered vital signs, headache, papilledema, and projectile vomiting. If untreated, IICP causes a displacement (herniation) of cerebral tissue, herniation of the cerebellum through the tentorium, followed by herniation of the brainstem through the foramen magnum. This is a lethal complication of IICP because it puts pressure on the vital centers in the medulla. IICP is primarily managed with osmotic diuretics and monitored with continuous intracranial pressure monitors.
- Headaches, a common type of intracranial pain, are categorized as tension, migraine, and cluster. A classic migraine is characterized by an aura; a common migraine does not have an aura.
- Epilepsy is a chronic disorder of abnormal, recurring, excessive, and self-terminating electrical discharges from neurons. A seizure is a single event of abnormal electrical discharge. Seizures are categorized into those that affect only a part of the brain (partial seizures) and those that affect all of the brain (generalized). The most common type of seizure in adults is a tonic-clonic generalized seizure. Seizures are treated medically with antiepileptic drugs (AEDs), surgery, and/or vagal nerve stimulation therapy.
- Traumatic brain injury (TBI) refers to any injury of the scalp, skull, or brain, and is a leading cause of death and disability. TBIs include skull fractures and focal or diffuse brain injury. An acute brain injury affects all body systems, and carries the risk of secondary injury to the brain from hypoxia and ischemia.
- An epidural hematoma develops in the potential space between the dura and the skull. A subdural hematoma collects between the dura mater and the arachnoid mater. Diffuse brain injuries include contusions, concussions, and diffuse axonal injury. Clients with an acute TBI must have immediate transport and treatment in an ED, followed by care in an ICU. They will require long-term physical care and rehabilitation.
- Central nervous system infections may be caused by bacteria, bacterial toxins, viruses, fungi, protozoans, and rickettsiae. Organisms may enter the brain through the bloodstream or by direct invasion. The major CNS infections are meningitis, encephalitis, and brain abscess. CNS infections are treated with broad-spectrum antibiotics and antifungal agents.
- Brain tumors are growths within the cranium, including on or in brain tissue, the meninges, the pituitary gland, or blood vessels. Brain tumors may be benign or malignant, primary or metastatic, and intracerebral or extracerebral. Regardless of the type or location, brain tumors are potentially lethal because they displace or impinge on CNS structures within a closed bony system.

TEST YOURSELF NCLEX-RN® REVIEW

1 Which of the following pathophysiologic events results in irregular respiratory patterns as LOC decreases?
1. pressure on the meninges
2. reflexive motor responses
3. loss of the oculocephalic reflex
4. brainstem responses to changes in $Paco_2$

2 The unconscious client has depressed or absent gag and swallowing reflexes. Which nursing diagnosis would be appropriate?
1. *Decreased Intracranial Adaptive Capacity*
2. *Risk for Aspiration*
3. *Imbalanced Nutrition: Less than Body Requirements*
4. *Ineffective Breathing Pattern*

3 What is the rationale for the use of osmotic diuretics to treat IICP?
1. Hyperthermia increases the cerebral metabolic rate and exacerbates IICP.
2. Increased blood osmolality draws edematous fluid into the vascular system.
3. Clients with ICP are at increased risk for gastrointestinal hemorrhage.
4. Brain injury and IICP often cause seizures.

4 You are monitoring the neurologic status of a client in a coma. Which of the following commands would be most accurate in identifying changes in mental status?
1. "Tell me your name."
2. "Look at this light when I shine it in your eyes."
3. "Squeeze my hand."
4. "Are you having trouble breathing?"

5 On admission to the ED, a client who has altered LOC has a variety of laboratory tests to facilitate the diagnosis of the etiology of the condition. Which tests would likely be performed? (Select all that apply.)
1. blood glucose
2. serum electrolytes
3. blood and urine toxicology
4. urine for WBCs
5. spinal fluid osmolarity

6 Of the following diagnostic tests, which one is the most accurate indicator of hydration status in the client with altered LOC?
1. CBC
2. urinalysis
3. serum osmolality
4. blood culture

7 What manifestation is consistently assessed in clients with generalized seizures?
1. loss of consciousness
2. repetitive nonpurposeful activity
3. tonic movements
4. clonic movements

8 When assessing a client with a head injury, you test fluid dripping from one ear for glucose. What are you assessing for?
1. infection
2. blood
3. CSF
4. serum

9 You are administering an antiepileptic drug to a newly diagnosed seizure client. The client says, "Will this cure my convulsions?" What would you say?
1. "No, but it will relieve your headache."
2. "No, but it will help decrease the aura you experience."
3. "No, not for the first year."
4. "No, but it may reduce or control them."

10 Which of the following statements is true of brain tumors?
1. All brain tumors are potentially lethal.
2. Only malignant tumors are lethal.
3. Metastatic brain tumors are benign tumors.
4. Benign brain tumors rarely require treatment.

See Test Yourself answers in Appendix C.

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