

Chapter 3

Neurologic system

Just the facts



In this chapter, you'll learn:

- ◆ anatomy and physiology of the neurologic system
- ◆ assessment of the neurologic system
- ◆ diagnostic tests and procedures
- ◆ neurologic disorders and treatments.

Understanding the nervous system

The neurologic (or nervous) system is the organ system that coordinates all body functions. This complex system allows a person to adapt to changes within his body and in the environment.

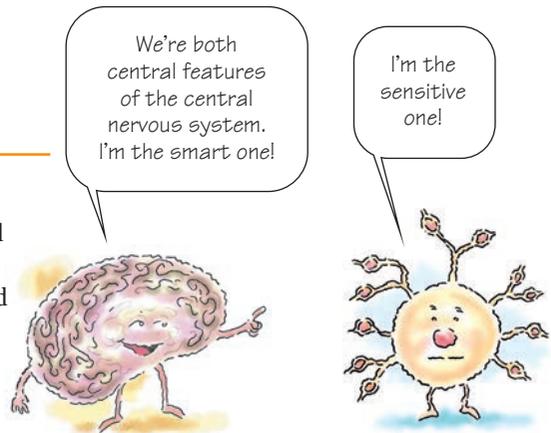
Two systems in one

The nervous system consists of:

1. the central nervous system (CNS), which includes the brain and spinal cord
2. the peripheral nervous system, which includes the cranial nerves, spinal nerves, and autonomic system.

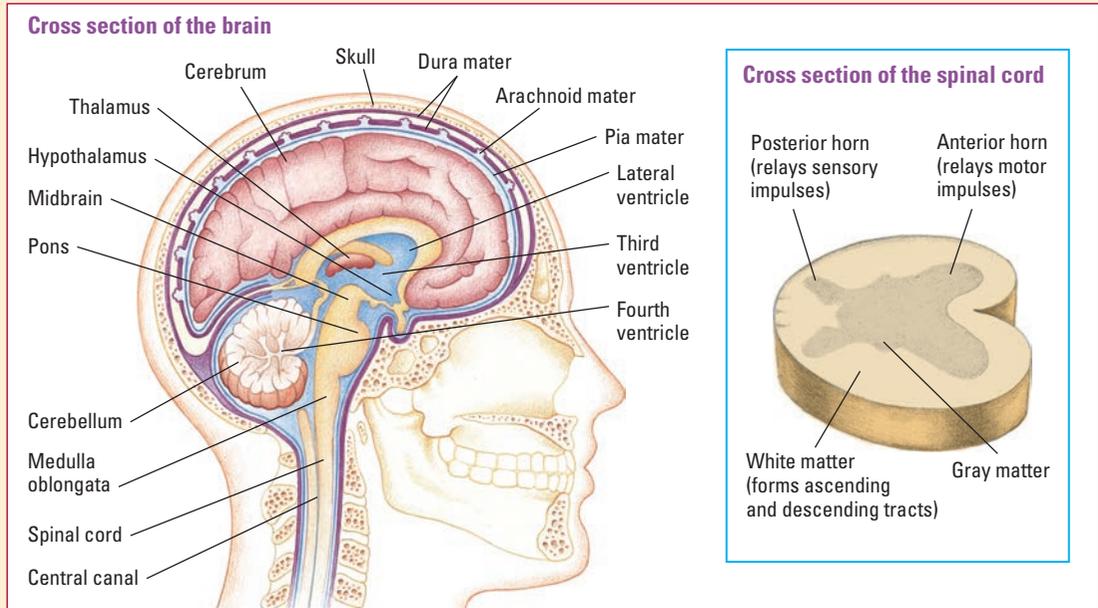
Central nervous system

The organs of the CNS—the brain and spinal cord—collect and interpret motor and sensory stimuli. In the process, voluntary and involuntary sensory impulses travel along neural pathways to the brain. (See *A close look at the CNS*, page 48.)



A close look at the CNS

This illustration depicts a cross section of the brain and spinal cord, which together make up the CNS. The brain joins the spinal cord at the base of the skull and ends near the second lumbar vertebra. Note the H-shaped mass of gray matter in the spinal cord.



Brain

The brain consists of three parts:

- cerebrum
- cerebellum
- brain stem.

Brain work

The brain collects, integrates, and interprets all stimuli and initiates and regulates voluntary and involuntary motor activity. Four major arteries supply the brain with oxygen.

Cerebrum

The cerebrum, or cerebral cortex, is the largest part of the brain.

Nerve central station

Tissues of the cerebrum make up a nerve center that controls sensory and motor activities and intelligence. It's encased by the bones of the

The cerebrum contains the nerve center that controls sensory and motor activities and intelligence.



skull and enclosed by three meninges (membrane layers): the dura mater, arachnoid mater, and pia mater.

Relay and regulate

The diencephalons, another part of the cerebrum, contain the thalamus and hypothalamus. The thalamus is a relay station for sensory and motor impulses.

The hypothalamus has many regulatory functions, such as:

- temperature control
- pituitary hormone production
- sleep and wake cycles
- water balance.

Divided in two

The cerebrum is divided into two hemispheres, left and right. The right hemisphere controls the left side of the body. The left hemisphere controls the right side of the body.

The two hemispheres of the brain are composed of four lobes. Each of the four lobes controls different functions. (See *Basic brain functions*, page 50.)

Cerebellum

The cerebellum—the brain's second largest region—lies behind and below the cerebrum. Like the cerebrum, it has two hemispheres.

Smooth moves

The cerebellum contains the major motor and sensory pathways. It enables smooth, coordinated muscle movement and helps maintain equilibrium.

Brain stem

The brain stem lies below the diencephalons and includes the:

- midbrain
- pons
- medulla.

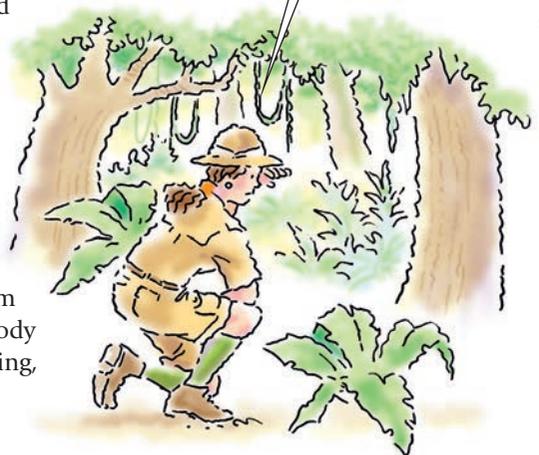
Pathway to the brain

The brain stem contains the nuclei, or cranial nerves III through XII. It's a major sensory and motor pathway for impulses running to and from the cerebral cortex. It also regulates automatic body functions, such as heart rate, breathing, swallowing, and coughing.

Two hemispheres ...
hmm ... does that mean I have an equator somewhere down the middle?

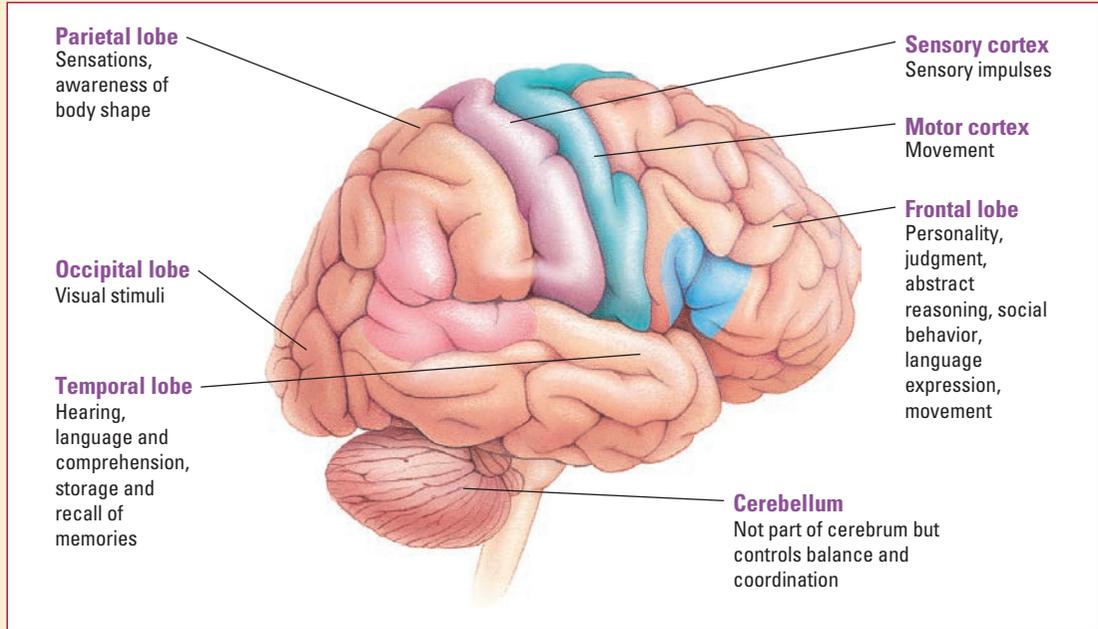


The brain stem is such a major pathway. How could I possibly be lost?



Basic brain functions

The basic structures and functions of the brain are depicted here. The cerebrum is divided into four lobes, based on location and function. The lobes—parietal, occipital, temporal, and frontal—are named for the cranial bones over them.



Circulation to the brain

The four major blood vessels of the brain include two vertebral and two carotid arteries.

Two arteries converge

The two vertebral arteries converge to become the basilar artery. The basilar artery supplies oxygenated blood to the posterior parts of the brain.

Two arteries diverge

The common carotid arteries branch into the two internal carotids, which further divide to supply oxygenated blood to the anterior and middle areas of the brain. These vessels interconnect and form the circle of Willis at the base of the brain. The circle of Willis ensures that oxygen is continuously circulated to the brain even if any of the brain's major vessels is interrupted. (See *Arteries of the brain*.)

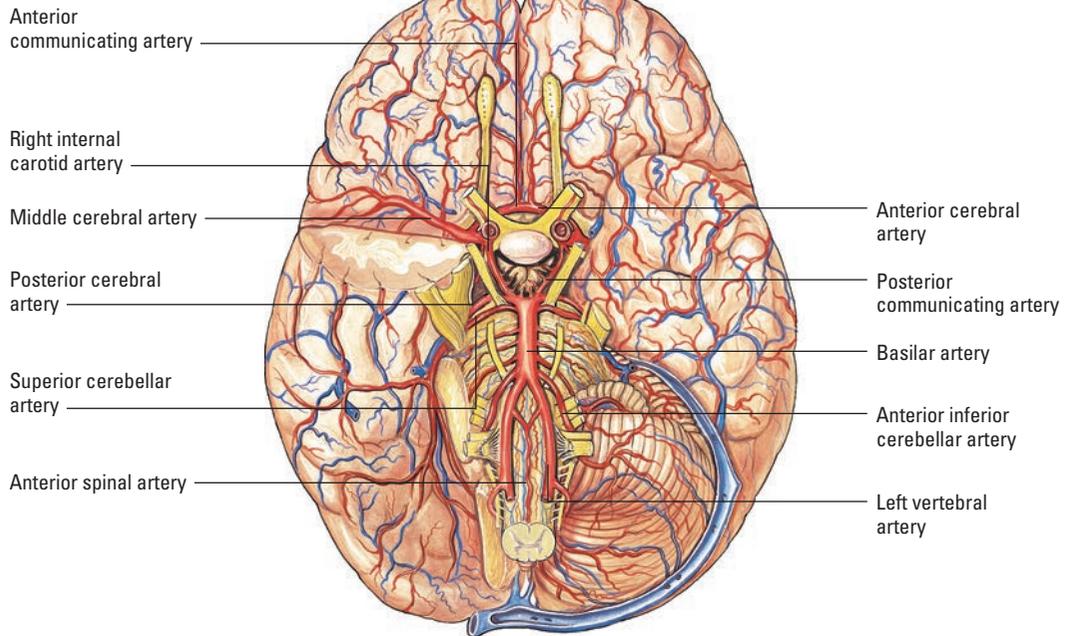
The circle of Willis ensures a constant supply of oxygen to the brain even if a major vessel is interrupted. Whew!



Arteries of the brain

Here's how the inferior surface of the brain appears. The anterior and posterior arteries join smaller arteries to form the circle of Willis.

Inferior view



Spinal cord

The spinal cord extends from the upper border of the first cervical vertebra to the lower border of the first lumbar vertebra. It's encased by the same membrane structure as the brain and is protected by the bony vertebrae of the spine.

Long paths

The spinal cord is the primary pathway for messages traveling between the peripheral parts of the body and the brain.

Short paths

The spinal cord also mediates the sensory-to-motor transmission path known as the *reflex arc*. Because the reflex arc enters and exits the spinal cord at the same level, reflex pathways don't need to travel up and down the way other stimuli do. (See *Understanding the reflex arc*, page 52.)

Understanding the reflex arc

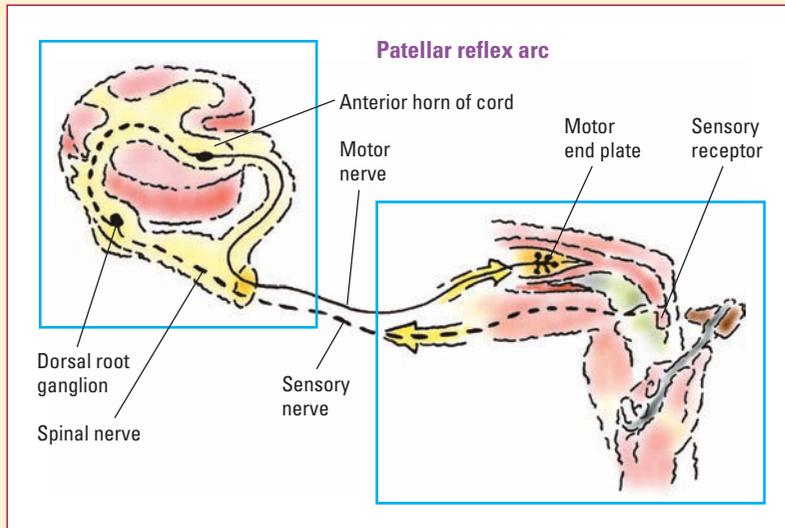
Spinal nerves—which have sensory and motor portions—control deep tendon and superficial reflexes. A simple reflex arc requires a sensory (or afferent) neuron and a motor (or efferent) neuron.

Knee jerk reaction

The knee jerk, or patellar, reflex illustrates the sequence of events in a normal reflex arc:

- First, a sensory receptor detects the mechanical stimulus produced by the reflex hammer striking the patellar tendon.

- Then the sensory neuron carries the impulse along its axon by way of the spinal nerve to the dorsal root, where it enters the spinal column.
- Next, in the anterior horn of the spinal cord, shown below, the sensory neuron joins with a motor neuron, which carries the impulse along its axon by way of the spinal nerve to the muscle. The motor neuron transmits the impulse to muscle fibers through stimulation of the motor end plate. This triggers the muscle to contract and the leg to extend.



Even sax players have to practice their spinal chords to make it in the big league.



Neural horns

The H-shaped mass of gray matter in the spinal cord is divided into four horns, which consist mainly of neuron cell bodies.

Four horns

The main function of cells in the two dorsal (posterior) horns of the spinal cord is to relay sensations; those in the two ventral (anterior) horns play a part in voluntary and reflex motor activity.

White matter matters

White matter surrounds the four horns. This white matter consists of myelinated nerve fibers grouped in vertical columns, or tracts:

- The *dorsal* white matter contains the ascending tracts, which carry impulses up the spinal cord to higher sensory centers.
- The *ventral* white matter contains the descending tracts, which transmit motor impulses down from higher motor centers to the spinal cord.

Sensory impulse pathways

Sensory impulses travel along the afferent (sensory or ascending) neural pathways to the sensory cortex in the parietal lobe of the brain. There, the impulses are interpreted. The sensory impulses travel along two major pathways:

- dorsal horn
- ganglia.

Dorsal horn

Pain and temperature sensations enter the spinal cord through the dorsal horn. After immediately crossing to the opposite side of the cord, these impulses travel to the thalamus by way of the spinothalamic tract.

Ganglia

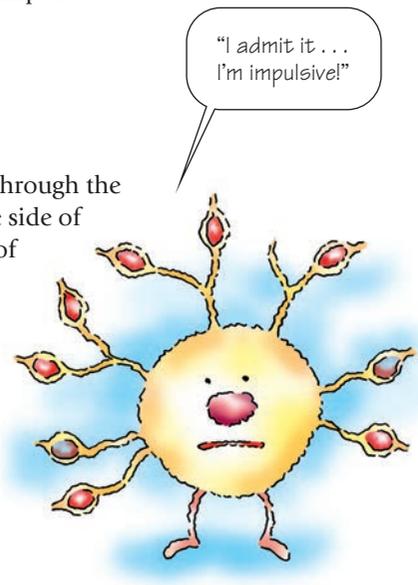
Sensations such as touch, pressure, and vibration enter the cord by way of relay stations, called *ganglia*, which are masses of nerve cell bodies on the dorsal roots of spinal nerves. Impulses travel up the dorsal column to the medulla, cross to the opposite side, and enter the thalamus. There, the sensory cortex interprets the impulses.

Motor impulse pathways

Motor impulses travel from the brain to muscles by way of the efferent (motor or descending) pathway. Motor impulses begin in the motor cortex of the frontal lobe and travel along the upper motor neurons to reach the lower motor neurons of the peripheral nervous system.

Upper motor neurons originate in the brain and form two major systems:

- the pyramidal system
- the extrapyramidal system.



Pyramidal system

The pyramidal system (corticospinal tract) is responsible for fine and skilled movements of skeletal muscle.

All the right moves

Impulses in this system travel from the motor cortex through the internal capsule to the medulla. At the medulla, they cross to the opposite side and continue down the spinal cord.

Extrapyramidal system

The extrapyramidal system (extracorticospinal tract) controls gross motor movements.

Extra, extra! Read all about it!

Impulses in this system originate in the premotor area of the frontal lobe. They then travel to the pons, where they cross to the opposite side and travel down the spinal cord to the anterior horns. They're then relayed to the lower motor neurons, which carry the impulses to muscles.

Peripheral nervous system

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

Cranial nerves

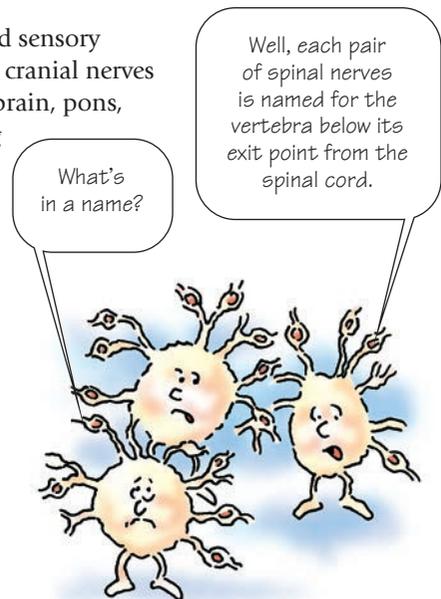
The 12 pairs of cranial nerves are the primary motor and sensory pathways between the brain and the head and neck. All cranial nerves except the olfactory and optic nerves exit from the midbrain, pons, or medulla oblongata of the brain stem. (See *Identifying cranial nerves.*)

Spinal nerves

There are 31 pairs of spinal nerves, each named for the vertebra immediately below its exit point from the spinal cord.

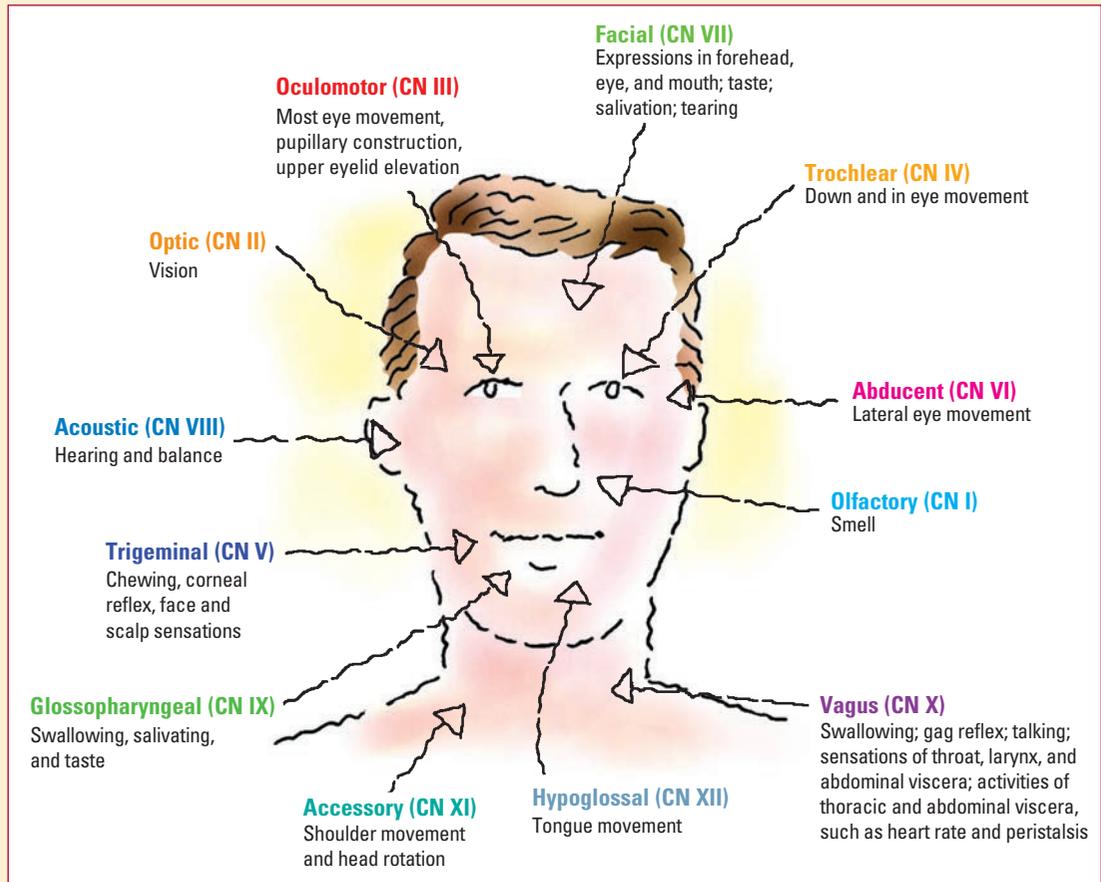
The nerve of nerves

Each spinal nerve consists of afferent (sensory) and efferent (motor) neurons, which carry messages to and from specific body regions, called *dermatomes*.



Identifying cranial nerves

The cranial nerves have either sensory or motor function or both. They're assigned Roman numerals and written this way: CN I, CN II, CN III, and so on. The locations of the cranial nerves as well as their functions are shown below.



Autonomic nervous system

The large autonomic nervous system supplies nerves to all internal organs. These visceral efferent nerves carry messages to the viscera from the brain stem and neuroendocrine system.

Two sympathetic systems

The autonomic nervous system includes two major parts:

1. the sympathetic nervous system
2. the parasympathetic nervous system.

Balancing act

When one part of the autonomic nervous system stimulates smooth muscles to contract or a gland to secrete, the other part of the system inhibits that action. Through such dual innervation, the sympathetic and parasympathetic systems counterbalance each other's activities to keep body systems running smoothly.

Sympathetic nervous system

Sympathetic nerves, called *preganglionic neurons*, exit the spinal cord between the first thoracic and second lumbar vertebrae and enter relay stations (ganglia) near the cord. These ganglia form the links of a chain that sends impulses to postganglionic neurons, which reach the organs and glands.



Enormous responses

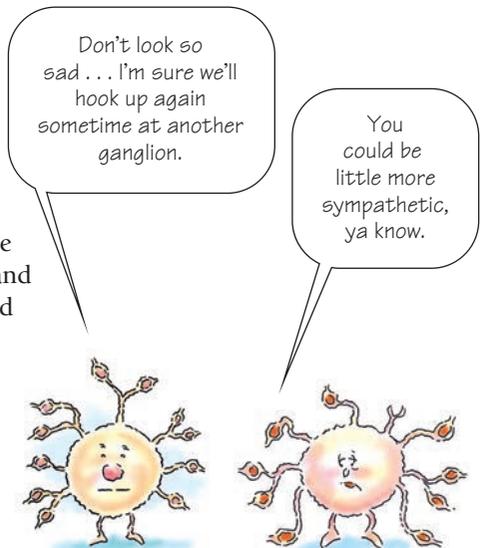
The postganglionic neurons of the sympathetic nervous system produce widespread, generalized responses, including:

- vasoconstriction
- elevated blood pressure
- enhanced blood flow to skeletal muscles
- increased heart rate and contractility
- increased respiratory rate
- smooth muscle relaxation of the bronchioles, GI tract, and urinary tract
- sphincter contraction
- pupillary dilation and ciliary muscle relaxation
- increased sweat gland secretion
- reduced pancreatic secretion.

Parasympathetic nervous system

Fibers of the parasympathetic nervous system leave the CNS by way of the cranial nerves from the midbrain and medulla and the spinal nerves between the second and fourth sacral vertebrae.

After leaving the CNS, the preganglionic fiber of each parasympathetic nerve travels to a ganglion near a specific organ or gland. The postganglionic fiber of the nerve enters that organ or gland.

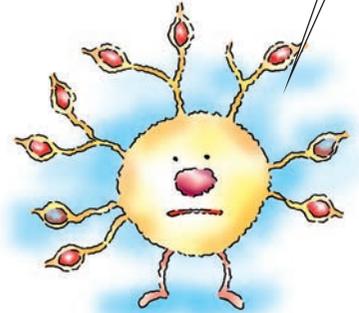


Subtle responses

The postganglionic fibers of the parasympathetic nervous system produce responses involving one specific organ or gland, such as:

- reductions in heart rate, contractility, and conduction velocity
- bronchial smooth muscle constriction
- increased GI tract tone and peristalsis, with sphincter relaxation
- increased bladder tone and urinary system sphincter relaxation
- vasodilatation of external genitalia, causing erection
- pupil constriction
- increased pancreatic, salivary, and lacrimal secretions.

The postganglionic fibers of the parasympathetic nervous system produce responses that involve one specific organ or gland.



Neurologic assessment

Assessment of subtle and elusive changes in the complex nervous system can be difficult. When you assess a patient for possible neurologic impairment, be sure to collect a thorough health history and investigate physical signs of impairment.

Check the records

If you can't interview a critically ill patient due to impairment, you may gather history information from the patient's medical record. In some cases, you may need to ask his family members or the nurse transferring the patient to the critical care unit for information.

If the patient has a neurologic impairment, gather assessment data from his medical record and interview his family and close friends to get an accurate picture of his health history.

Health history

To collect a thorough health history, gather details about the patient's current state of health, previous health status, lifestyle, and family health.

With a little help from his friends (and family)

A patient with neurologic impairment may have trouble remembering. If members of the patient's family or close friends are available, include them in the assessment process. They may be able to corroborate or correct the details of the patient's health history.

Current health

Discover the patient's chief complaint by asking such questions as, "Why did you come to the hospital?" or "What has been bothering you lately?" Use the patient's words when you document such chief complaints.



Common complaints

If your patient is suffering from a neurologic disorder, you may hear reports of headaches, motor disturbances (such as weakness, paresis, and paralysis), seizures, sensory deviations, and altered level of consciousness (LOC).

Details, please

Encourage the patient to describe details of the current condition by asking such questions as:

- Do you have headaches? How often do you have them? What precipitates them?
- Do you ever feel dizzy? How often do you feel this way? What seems to precipitate the episodes?
- Do you ever feel a tingling or prickling sensation or numbness? If so, where?
- Have you ever had seizures or tremors? Have you ever had weakness or paralysis in your arms or legs?
- Do you have trouble urinating, walking, speaking, understanding others, reading, or writing?
- How's your memory and ability to concentrate?



Previous health

Many chronic diseases affect the neurologic system, so ask questions about the patient's past health and what medications he's taking. Specifically, ask whether the patient has had any:

- major illnesses
- recurrent minor illnesses
- accidents
- injuries
- surgical procedures
- allergies.

Lifestyle

Ask questions about the patient's cultural and social background because these affect care decisions. Note the patient's education level, occupation, and hobbies. As you gather this information, also assess the patient's self-image.

Family health

Information about the patient's family may reveal a hereditary disorder. Ask if anyone in the family has had diabetes, cardiac or renal disease, high blood pressure, cancer, a bleeding disorder, a mental disorder, or a stroke.

Physical examination

A complete neurologic examination can be long and detailed. It's unlikely that you would perform one in its entirety. However, if your initial screening suggests a neurologic problem, you may need to conduct a more detailed assessment.

Top-to-bottom examination

Examine the patient's neurologic system in an orderly way. Beginning with the highest levels of neurologic function and working down to the lowest, assess these five areas:

- mental status
- cranial nerve functions
- sensory function
- motor function
- reflexes.

Mental status

Mental status assessment begins when you talk to the patient during the health history. Responses to your questions reveal clues about the patient's orientation and memory. Use such clues as a guide during the physical assessment. Also observe expression, body language, and attentiveness, which provide clues to the patient overall status.

No easy answers

Make sure that you ask questions that require more than yes-or-no answers. Otherwise, confusion or disorientation might not be apparent. If you have doubts about a patient's mental status, perform a screening examination. (See *Quick check of mental status*, page 60.)

Three-part exam

Use the mental status examination to check these three parameters:

- LOC
- speech
- cognitive function.

Level of consciousness

Watch for any change in the patient's LOC. It's the earliest and most sensitive indicator that his neurologic status has changed.

Mental status assessment begins with a health history. The patient's responses reveal clues about his mental status.





Advice from the experts

Quick check of mental status

To quickly screen your patient for disordered thought processes, ask the questions below. An incorrect answer to any question may indicate the need for a complete mental status examination. One quick tip: Make sure that you know the correct answers before asking the questions.

Question	Function screened
What's your name?	Orientation to person
What's your mother's name?	Orientation to other people
What year is it?	Orientation to time
Where are you now?	Orientation to place
How old are you?	Memory
Where were you born?	Remote memory
What did you have for breakfast?	Recent memory
Who's president of the United States now?	General knowledge
Can you count backward from 20 to 1?	Attention span and calculation skills

Let's be perfectly clear

Many terms are used to describe LOC, and definitions differ slightly among practitioners. To avoid confusion, clearly describe the patient's response to various stimuli using these definitions:

- *Alert*—Patient follows commands and responds completely and appropriately to stimuli.
- *Lethargic*—Patient is drowsy, has delayed but appropriate responses to verbal stimuli, and may drift off to sleep during the examination.
- *Stuporous*—Patient requires vigorous stimulation for a response. Responses vary in appropriateness.
- *Comatose*—Patient doesn't respond appropriately to verbal or painful stimuli and can't follow commands or communicate verbally.

Not too highly stimulating

Start by quietly observing the patient's behavior. If the patient is sleeping, try to rouse him by providing an appropriate stimulus, in this order:

1. auditory
2. tactile
3. painful.

Watch for a change in the patient's LOC—the earliest and most sensitive indicator of neurologic status change.



Always start with a minimal stimulus, increasing intensity as necessary. The Glasgow Coma Scale offers an objective way to assess the patient's LOC. (See *Using the Glasgow Coma Scale*, page 62.)

Speech

Listen to how well the patient expresses thoughts. Does he choose the correct words or seem to have problems finding or articulating words?

It's hard to say

To assess for dysarthria (difficulty forming words), ask the patient to repeat the phrase, "No ifs, ands, or buts." Assess speech comprehension by determining the patient's ability to follow instructions and cooperate with your examination.

Speaking of changes

Keep in mind that language performance tends to fluctuate with the time of day and changes in physical condition. A healthy person may have language difficulty when ill or fatigued. However, increasing speech difficulties may indicate deteriorating neurologic status, which warrants further evaluation.

Cognitive function

Assess cognitive function by testing the patient's:

- memory
- orientation
- attention span
- calculation ability
- thought content
- abstract thinking
- judgment
- insight
- emotional status.

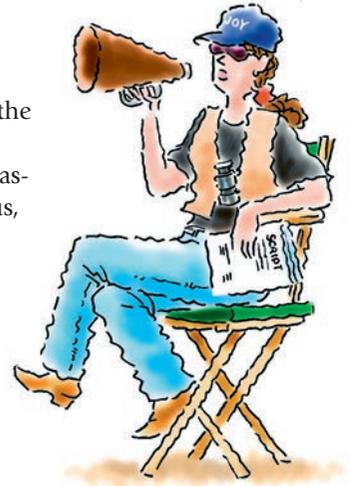
Thanks for the memories

Short-term memory is commonly affected first in a patient with neurologic disease. A patient with intact short-term memory can generally remember and repeat five to seven nonconsecutive numbers right away and again 10 minutes later.

When then who

To quickly test your patient's orientation, memory, and attention span, use the mental status screening questions. Orientation to time is usually disrupted first; orientation to person, last.

Listen up!
Increasing speech
difficulties
may indicate
deteriorating
neurologic status.





Take charge!

Using the Glasgow Coma Scale

You can use the Glasgow Coma Scale to describe the patient’s baseline mental status and detect and interpret changes in the LOC.

To use the scale, test the patient’s ability to respond to verbal, motor, and sensory stimulation and base your findings on the scale. A patient who’s alert, can follow

simple commands, and is oriented to time, place, and person receives a score of 15 points. A lower score in one or more categories may signal an impending neurologic crisis. A total score of 7 or less indicates severe neurologic damage.

Test	Score	Patient’s response
Eye-opening response		
Spontaneously	4	Opens eyes spontaneously
To speech	3	Opens eyes when told to
To pain	2	Opens eyes only on painful stimulus
None	1	Doesn’t open eyes in response to stimulus
Motor response		
Obeys	6	Shows two fingers when asked
Localizes	5	Reaches toward painful stimulus and tries to remove it
Withdraws	4	Moves away from painful stimulus
Abnormal flexion	3	Assumes a decorticate posture (shown below)
		
Abnormal extension	2	Assumes a decerebrate posture (shown below)
		
None	1	No response, just lies flaccid—an ominous sign
Verbal response		
Oriented	5	Tells current date
Confused	4	Tells incorrect year
Inappropriate words	3	Replies randomly with incorrect word
Incomprehensible	2	Moans or screams
None	1	No response
Total score	<input type="text"/>	

Always consider the patient's environment and physical condition when assessing orientation. For example, a patient admitted to the critical care unit for several days may not be oriented to time because of the constant activity and noise of the monitoring equipment.

Attention and calculation

When testing attention span and calculation skills, keep in mind that lack of mathematical ability and anxiety can affect the patient's performance. If he has difficulty with numerical computation, ask him to spell the word "world" backwards. While he's performing these functions, note his ability to pay attention.

Thought content

Disordered thought patterns may indicate delirium or psychosis. Assess thought pattern by evaluating the clarity and cohesiveness of the patient's ideas. Is his conversation smooth, with logical transitions between ideas? Does he have hallucinations (sensory perceptions that lack appropriate stimuli) or delusions (beliefs not supported by reality)?

Hypothetically speaking . . .

Test the patient's judgment by asking him how he would respond to a hypothetical situation. For example, what would he do if he were in a public building and the fire alarm sounded? Evaluate the appropriateness of his answer.

Insight on insight

Test your patient's insight by finding out:

- whether the patient has a realistic view of himself
- whether he's aware of his illness and circumstances.

Assess insight by asking, for example, "What do you think caused your chest pain?" Expect different patients to have different degrees of insight. For instance, a patient may attribute chest discomfort to indigestion rather than acknowledge that he has had a heart attack.

Lost in emotion

Throughout the interview, assess your patient's emotional status. Note his mood, emotional lability or stability, and the appropriateness of his emotional responses. Also, assess the patient's mood by asking how he feels about himself and his future. Keep in mind that signs and symptoms of depression in an elderly patient may be atypical. (See *Depression and elderly patients*, page 64.)

Orientation to time is usually disrupted first; orientation to person, last.



Test the patient's judgment: Ask how he would respond to a hypothetical situation—like what he would do if he were in a public building and the fire alarm sounded—and then evaluate his answer.



Cranial nerve function

Cranial nerve assessment reveals valuable information about the condition of the CNS, especially the brain stem.

Under pressure

Because of their location, some cranial nerves are more vulnerable to the effects of increasing intracranial pressure (ICP). Therefore, a neurologic screening assessment of the CNS focuses on these key nerves:

- optic (II)
- oculomotor (III)
- trochlear (IV)
- abducens (VI).

Go on

Also evaluate other nerves if the patient's history or symptoms indicate a potential CNS disorder or when performing a complete nervous system assessment. (See *Checking brain stem function*.)

Be nosey

Assess the olfactory nerve (cranial nerve [CN] I) first. Check the patency of each nostril. Then instruct the patient to close his eyes. Occlude one nostril and hold a familiar, pungent-smelling substance under the patient's nose and ask him to identify it. Repeat this with the other nostril.

See about sight

Next, assess the optic (CN II) and oculomotor (CN III) nerves:

- To assess the optic nerve, check visual acuity, visual fields, and retinal structures. Do this by asking the patient to read a newspaper, starting with large headlines and moving to small print.
- To assess the oculomotor nerve, check pupil size, pupil shape, and pupillary response to light. When assessing pupil size, look for trends, such as a gradual increase in the size of one pupil or appearance of unequal pupils. (See *Recognizing pupillary changes*, page 66.)

Check three nerves at once

Assess the coordinated function of the oculomotor (CN III), trochlear (CN IV), and abducens (CN VI) nerves simultaneously. Here's how these nerves normally work:

- The oculomotor nerve controls extraocular movement, pupillary constriction, and raising of the eyelid.
- The trochlear nerve controls downward and inward eye movement.
- The abducens nerve controls lateral eye movement.



Depression and elderly patients

Symptoms of depression in elderly patients may be different from those found in other patients. For example, rather than the usual sad affect seen in patients with depression, your elderly patient may exhibit such atypical signs as decreased function and increased agitation.

"Ah . . . the pungent aroma of hospital coffee—who could mistake it for anything else?"



Checking brain stem function

In an unconscious patient, assist the practitioner in assessing brain stem function by testing for the oculocephalic (doll's eye) reflex and the oculovestibular reflex. If the patient has a cervical spine injury, expect to use the oculovestibular reflex test as an alternative. The oculovestibular reflex test may also be used to determine the status of the vestibular portion of the acoustic nerve (CN VIII).

Oculocephalic reflex

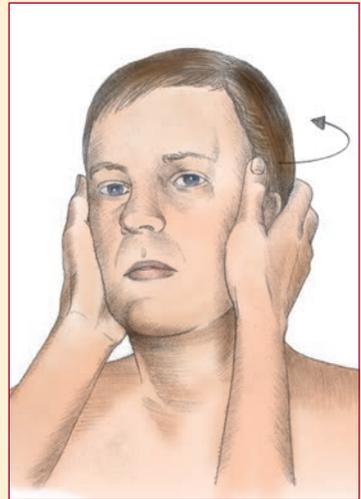
Before beginning, examine the patient's cervical spine. Don't perform this procedure if you suspect the patient has a cervical spine injury. If the patient has no cervical spine injury, proceed as follows:

- Place both hands on either side of his head and use your thumbs to gently hold his eyelids open.
- While watching the patient's eyes, briskly rotate the head from side to side (as shown at right) or briskly flex and extend the patient's neck.
- Observe how the patient's eyes move in relation to head movement.

In a normal response, which indicates an intact brain stem, the eyes appear to move opposite to the movement of the head. For example, if the neck is flexed, the eyes appear to look upward. If the neck is extended, the eyes gaze downward.

Abnormal response

With an abnormal (doll's eye) response, the eyes appear to move passively in the same direction as the head, indicating the absence of oculocephalic reflex. Such a response suggests a severe brain stem damage at the level of the pons or midbrain.



Oculovestibular reflex

To assess the oculovestibular reflex, the practitioner first determines that the patient has an intact tympanic membrane and a clear external ear canal. Then follow these steps:

- Elevate the head of the bed 30 degrees.
- Using a large syringe with a small catheter on the tip, slowly irrigate the external auditory canal with 20 to 200 ml of cold water or ice water (as shown at right).
- During irrigation, watch the patient's eye movements. In a patient with an intact oculovestibular reflex, the eyes deviate toward the side being irrigated with cold water.

Abnormal responses

If the patient is conscious to some degree, there may be nystagmus (involuntary, rapid movement of the eyeball) with rapid jerking of the eyes away from the side being irrigated. In an abnormal conscious individual, as little as 10 ml of ice water may produce such a response and may also cause nausea. In a comatose patient with an intact brain stem, the eyes tonically deviate toward the stimulated ear. Absence of eye movement suggests a brain stem lesion.



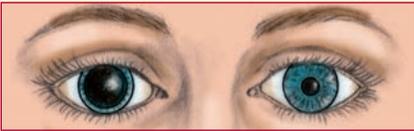
Recognizing pupillary changes

Use this table as a guide to recognize pupillary changes and identify possible causes.

Pupillary change

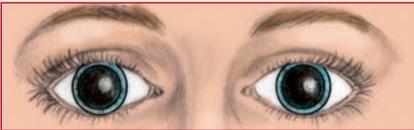
Possible causes

Unilateral, dilated (4 mm), fixed, and nonreactive



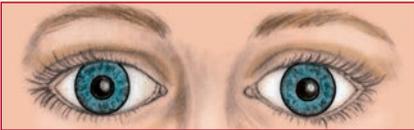
- Uncal herniation with oculomotor nerve damage
- Brain stem compression
- Increased ICP
- Tentorial herniation
- Head trauma with subdural or epidural hematoma
- May be normal in some people

Bilateral, dilated (4 mm), fixed, and nonreactive



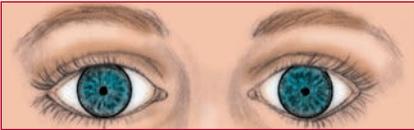
- Severe midbrain damage
- Cardiopulmonary arrest (hypoxia)
- Anticholinergic poisoning

Bilateral, midsize (2 mm), fixed, and nonreactive



- Midbrain involvement caused by edema, hemorrhage, infarctions, lacerations, or contusions

Bilateral, pinpoint (<1 mm), and usually nonreactive



- Lesions of pons, usually after hemorrhage

Unilateral, small (1.5 mm), and nonreactive



- Disruption of sympathetic nerve supply to the head caused by spinal cord lesion above the first thoracic vertebra

The cardinal rules

Make sure that the patient's pupils constrict when exposed to light and that his eyes adapt to seeing objects at various distances. Ask the patient to follow your finger through six cardinal positions of gaze:

1. left superior
2. left lateral
3. left inferior
4. right superior
5. right lateral
6. right inferior.

Pause slightly before moving from one position to the next, to assess the patient for nystagmus, or involuntary eye movement, and the ability to hold gaze in that particular position.

No need for safety pins. I can sense every line and wrinkle on my face. Pass the moisturizer, please!



Focus on the face

To assess the sensory portion of the trigeminal nerve (CN V), gently touch the right and left sides of the patient's forehead with a cotton ball while his eyes are closed. Instruct him to tell you the moment the cotton touches each area. Compare the patient's responses on both sides.

Repeat the technique on the right and left cheek and on the right and left jaw. Next, repeat the entire procedure using a sharp object, such as the tip of a safety pin. Ask the patient to describe and compare both sensations.

To assess the motor function of the trigeminal nerve, ask the patient to clench his teeth while you palpate his temporal and masseter muscles.

Make someone smile

To test the motor portion of the facial nerve (CN VII), ask the patient to:

- wrinkle his forehead
- raise and lower his eyebrows
- smile to show his teeth
- puff out his cheeks.

Also, with the patient's eyes tightly closed, attempt to open his eyelids. As you conduct each part of this test, look for symmetry.

I'm still stuck on sweet. I'll get to salty, sour, and bitter after this cake. Could be a while, though.



Keep it tasteful

The sensory portion of the facial nerve (CN VII) supplies taste sensation to the anterior two-thirds of the tongue. Test the taste sensation by placing items with various flavors on the patient's tongue. Use items such as sugar (sweet), salt, lemon juice (sour), and quinine (bitter). Between items, have the patient wash away each substance with a sip of water.

Now hear this

To assess the acoustic nerve (CN VIII), first test the patient's hearing. Ask the patient to cover one ear. Then stand on the opposite side and whisper a few words. Find out whether the patient can repeat what you said. Test the other ear in the same way.

To test the vestibular portion of the acoustic nerve, observe the patient for nystagmus and disturbed balance. Note reports of the room spinning or dizziness.

Check the pipes

Test the glossopharyngeal nerve (CN IX) and vagus nerve (CN X) together because their innervation overlaps in the pharynx:

- The glossopharyngeal nerve is responsible for swallowing, salivating, and taste perception on the posterior one-third of the tongue.
- The vagus nerve controls swallowing and is responsible for voice quality.

Assess these nerves, first, by listening to the patient's voice. Then check the gag reflex by touching the tip of a tongue blade against the posterior pharynx and asking the patient to open wide and say "ah." Watch for the symmetrical upward movement of the soft palate and uvula and for the midline position of the uvula.



Shrug it off

To assess the spinal accessory nerve (CN XI), which controls the sternocleidomastoid muscles and the upper portion of the trapezius muscles, press down on the patient's shoulders while he attempts to shrug against this resistance. Note shoulder strength and symmetry while inspecting and palpating the trapezius muscles.

To further test the trapezius muscles, apply resistance from one side while the patient tries to return his head to midline position. Look for neck strength. Repeat on the other side.

Test tongue toughness

To assess the hypoglossal nerve (CN XII), follow these steps:

1. Ask the patient to stick out his tongue. Look for any deviation from the midline, atrophy, or fasciculations.
2. Test tongue strength by asking the patient to push his tongue against his cheek as you apply resistance. Observe the tongue for symmetry.
3. Test the patient's speech by asking him to repeat the sentence, "Round the rugged rock that ragged rascal ran."

Sensory function

Assess the sensory system to evaluate:

- ability of the sensory receptors to detect stimuli
- ability of the afferent nerves to carry sensory nerve impulses to the spinal cord
- ability of the sensory tracts in the spinal cord to carry sensory messages to the brain.

Five sensations

During your assessment, check five types of sensation, including pain, light touch, vibration, position, and discrimination.

This is gonna hurt

To test for pain sensation, have the patient close his eyes; then touch all the major dermatomes, first with the sharp end of a safety pin and then with the dull end. Proceed in this order:

- fingers
- shoulders
- toes
- thighs
- trunk.

While testing, occasionally alternate sharp and dull ends. Ask the patient to tell you when he feels the sharp stimulus. If the patient has known deficits, start in the area with the least sensation and move toward the area with the most sensation.



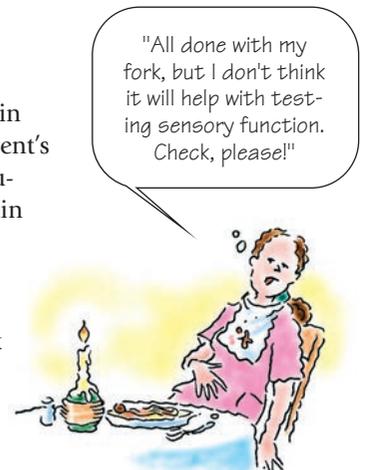
Use a light touch

To test for the sense of light touch, follow the instructions for pain sensation, using a wisp of cotton or tissue. Lightly touch the patient's skin; don't swab or sweep the skin. A patient with peripheral neuropathy might retain the sensation for light touch after losing pain sensation.

Find the right vibe

To check for response to vibration, tap a low-pitched tuning fork on the heel of your hand and then place the base of the fork firmly over the distal interphalangeal joint of the index finger. Then move proximally until the patient feels the vibration; everything above that level is intact.

If the patient's vibratory sense is intact, further testing for position sense isn't necessary because they follow the same pathway.



Where the toes are

To experience position sense, the patient needs intact vestibular and cerebellar function. To assess for position sense, have the patient

close his eyes. Then, grasp the sides of his big toe and move it up and down. Ask the patient what position the toe is in.

To perform the same test on the patient's upper extremities, grasp the sides of his index finger and move it back and forth. Ask the patient what position the finger is in.

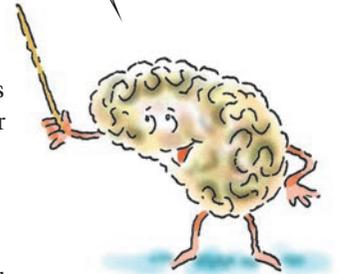
Discrimination, integration, and extinction

Discrimination is the cortex's ability to integrate sensory input. Stereognosis is the ability to discriminate the shape, size, weight, texture, and form of an object by touching and manipulating it.

To test stereognosis, ask the patient to close both eyes and open one hand. Then place a common object, such as a key, in the hand and ask the patient to identify it. If the patient can't identify the object, test graphesthesia (ability to identify something by tactile sense). Here's how to do this: While the patient's eyes are closed, draw a large number on the palm of one hand and ask the patient to identify the number.

Extinction is the failure to perceive touch on one side. To test point localization, have the patient close his eyes, touch one of his limbs, and then ask where you touched him. To test two-point discrimination, touch the patient simultaneously in two contralateral areas and note whether he can identify touch on both sides.

Discrimination is the ability to integrate sensory input to determine the shape, size, weight, texture, and form of an object.



Motor function

Assess motor function to aid evaluation of these structures and functions:

- the cerebral cortex and its initiation of motor activity by way of the pyramidal pathways
- the corticospinal tracts and their capacity to carry motor messages down the spinal cord
- the lower motor neurons and their ability to carry efferent impulses to the muscles
- the muscles and their capacity to carry out motor commands
- the cerebellum and basal ganglia and their capacity to coordinate and fine-tune movement.

A careful motor function assessment reveals a lot of information about neurologic structures and functions.



Tone test

Muscle tone represents muscular resistance to passive stretching. To test muscle tone of the arm, move the patient's shoulder through its passive range of motion (ROM); you should feel a slight resistance. When you let the patient's arm drop to his side, it should fall easily.

To test leg muscle tone, guide the patient's hip through its passive ROM and then let his leg fall to the bed. If it falls in an externally rotated position, note this abnormal finding.

Feats of strength

To assess arm muscle strength, ask the patient to push you away as you apply resistance. To assess hand strength, ask the patient to grip your hand. Then ask the patient to extend both arms, palms up. Have him close his eyes and maintain this position for 20 to 30 seconds. Observe the arm for downward drifting and pronation.

Strength of feet

Assess leg movement by first asking the patient to move each leg and foot with and without applying resistance. If he fails to move the leg on command, watch for spontaneous movement.

Grace and gait

Assess the patient's coordination and balance through cerebellar testing. Note whether the patient can sit and stand without support. If appropriate, observe as the patient walks across the room, turns, and walks back.

While observing the patient, note imbalances and abnormalities. When cerebellar dysfunction is present, the patient has a wide-based, unsteady gait. Deviation to one side may indicate a cerebellar lesion on the side.

Synchronized standing

Perform Romberg's test to evaluate cerebellar synchronization of movement with balance. Have the patient stand with his feet together, arms at his sides, and without support. Note his ability to maintain balance with both eyes open and then closed. (Stand nearby in case the patient loses his balance.)

A small amount of swaying normally occurs when the eyes are closed. If the patient has trouble maintaining a steady position with eyes open or closed, cerebellar ataxia may be present.

Extreme coordination

Test the extremities for coordination by having the patient touch nose and then your outstretched finger as you move it. Have him do this faster and faster. His movements should be accurate and smooth.

Test cerebellar function further by assessing rapid alternating movements. Tell the patient to use the thumb of one hand to touch each finger of the same hand in rapid sequence. Repeat with the other hand.

To assess the legs, have the patient rapidly tap the floor with the ball of one foot. Test each leg separately. Note any slowness or awkwardness. Abnormalities can indicate cerebellar disease or motor weakness associated with extrapyramidal or pyramidal disease.

When assessing coordination and balance, note any imbalance or abnormality.



Present and absent actions

Motor responses in an unconscious patient may be appropriate, inappropriate, or absent. Appropriate responses, such as localization or withdrawal, mean that the sensory and corticospinal pathways are functioning. Inappropriate responses, such as decorticate or decerebrate posturing, indicate a dysfunction.

It can be challenging to assess motor responses in a patient who can't follow commands or is unresponsive. Make sure that you note whether any stimulus produces a response, what that response is, and the stimulus that was used.

Reflexes

Assess deep tendon and superficial reflexes to learn about the integrity of the sensory receptor organ. You can also evaluate how well afferent nerves relay sensory messages to the spinal cord or brain stem segment to mediate reflexes.

How deep is your reflex?

Test deep tendon reflexes by checking the responses of the biceps, triceps, brachioradialis, patellar, and Achilles tendons:

- The biceps reflex contracts the biceps muscle and forces flexion of the forearm.
- The triceps reflex contracts the triceps muscle and forces extension of the forearm.
- The brachioradialis reflex causes supination of the hand and flexion of the forearm at the elbow.
- The patellar reflex forces contraction of the quadriceps muscle in the thigh with extension of the leg.
- The Achilles reflex forces plantar flexion of the foot at the ankle.

Test deep tendon reflexes by checking responses of the biceps, triceps, brachioradialis, patellar, and Achilles tendons. Check superficial reflexes using light, tactile stimulation.

Superficially speaking

You can elicit superficial reflexes using light, tactile stimulation, such as stroking or scratching the skin.

Because these are cutaneous reflexes, the more you try to elicit them in succession, the less response you'll get. Therefore, observe carefully the first time you stimulate these reflexes.

Superficial reflexes include the pharyngeal, abdominal, and cremasteric reflexes. Here's how to test them:

- Pharyngeal reflex: To test CN IX and CN X, have the patient open his mouth wide. Then, touch the back of the pharynx with a tongue blade. Normally, this causes the patient to gag.
- Abdominal reflex: To test the intactness of thoracic spinal segments T8, T9, and T10, use the tip of the handle on the reflex hammer to stroke one side, and then the opposite side, of the patient's abdomen above the umbilicus. Repeat on the lower abdomen. Normally, the abdominal muscles contract and the umbilicus deviates toward the stimulated side.



- Cremasteric reflex: To test the intactness of lumbar spinal segments L1 and L2 in a male patient, use a tongue blade to scratch the inner aspects of each thigh gently. Normally, this action causes the testicles to lift.

Write it down

After you examine the patient, document your findings using a grading scale to rate each reflex. Document the rating for each reflex at the appropriate site on a stick figure. (See *Documenting reflex findings*.)

Documenting reflex findings

Use these grading scales to rate the strength of each reflex in a deep tendon and superficial reflex assessment.

Deep tendon reflex grades

0 absent

+ present but diminished

++ normal

+++ increased but not necessarily abnormal

++++ hyperactive or clonic (involuntary contraction and relaxation of skeletal muscle)

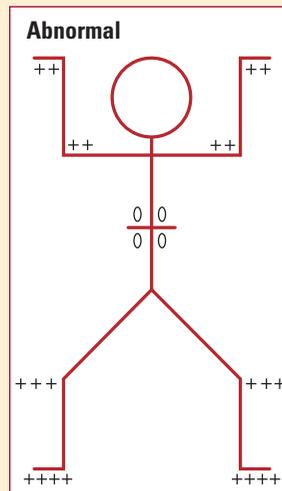
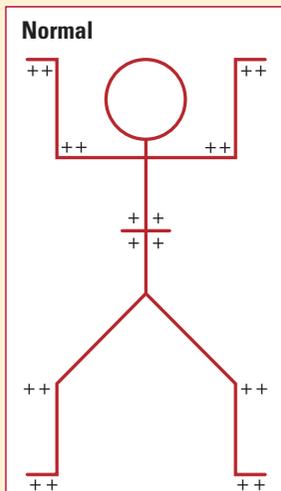
Superficial reflex grades

0 absent

+ present

Findings

Record the patient's reflex ratings on a drawing of a stick figure. The figures depict documentation of normal and abnormal reflex responses.



Diagnostic tests

Diagnostic testing to evaluate the nervous system typically includes imaging studies, angiography, and electrophysiologic studies. Other tests, such as lumbar puncture and transcranial Doppler studies, may also be used.

Tell it like it is

Diagnostic testing may be routine for you, but it can be frightening for the patient. Make sure that you carefully prepare the patient and his family for each test and follow-up monitoring procedure. Some tests can be performed at the patient's bedside, but many require transportation to the imaging department.

Diagnostic testing can be frightening. Make sure that you prepare the patient and his family for each test.



Imaging studies

The most common imaging studies used to detect neurologic disorders include computed tomography (CT) scan, magnetic resonance imaging (MRI), positron emission tomography (PET) scan, and skull and spinal X-rays. Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) are also available as diagnostic tools for cerebrovascular disease. (See *Understanding CTA and MRA*, page 76.)

Computed tomography scan

CT scanning of intracranial structures combines radiology and computer analysis of tissue density. CT angiography shows blood vessels, and it carries less risk of complications than cerebral angiography. CTA is becoming the new standard.

Before CT scanning of the brain, make sure that the patient isn't allergic to iodine or shellfish, which may foretell an adverse reaction to the contrast medium. Thankfully, we aren't allergic!

Spine scanning

CT scanning of the spine can be used to assess such disorders as herniated disk, spinal cord tumors, and spinal stenosis for patients that cannot have an MRI. It is best used to look at the bony architecture of the vertebral column.

Brain scanning

CT scanning of the brain can be done with or without contrast and is used to detect brain contusion, brain calcifications, cerebral atrophy, hydrocephalus, inflammation, space-occupying lesions (tumors, hematomas, edemas, and abscesses), and vascular anomalies (arteriovenous malformation [AVM], infarctions, blood clots, and hemorrhage).



Nursing concerns

- If a contrast medium is ordered, confirm that the patient isn't allergic to iodine or shellfish to avoid an adverse reaction.
- If the test calls for a contrast medium, tell the patient that it's injected into an existing I.V. line or that a new line may be inserted.
- Preprocedure testing should include evaluation of renal function (serum creatinine and blood urea nitrogen [BUN] levels) because the contrast medium can cause acute renal failure.
- Warn the patient that he may feel flushed or notice a metallic taste in his mouth when the contrast medium is injected.
- Tell him that the CT scanner circles around him for 10 to 30 minutes, depending on the procedure and type of equipment.
- Explain that he must lie still during the test.
- Tell him that the contrast medium may discolor his urine for 24 hours. Suggest that he drink more fluids to flush the medium out of his body, unless this is contraindicated or he has oral intake restrictions; otherwise, the practitioner may write an order to increase the I.V. flow rate.

Magnetic resonance imaging

MRI generates detailed pictures of body structures. It is best for imaging soft tissue. The test may involve the use of a contrast medium such as gadolinium.

Sharper images

Compared with conventional X-rays and CT scans, MRI provides superior contrast of soft tissues, sharply differentiating healthy, benign, and cancerous tissue and clearly revealing blood vessels. In addition, MRI permits imaging in multiple planes, including sagittal and coronal views in regions where bones normally hamper visualization.

MRI is especially useful for studying the CNS because it can reveal structural and biochemical abnormalities associated with such conditions as transient ischemic attack (TIA), tumors, multiple sclerosis (MS), cerebral edema, and hydrocephalus.

Nursing concerns

- Confirm that the patient isn't allergic to the contrast medium (usually gadolinium).
- If the test calls for a contrast medium, tell the patient that it's injected into an existing I.V. line or that a new line may be inserted.
- Explain that the procedure can take up to 1½ hours; tell the patient that he must remain still for intervals of 5 to 20 minutes.
- Instruct the patient to remove all metallic items, such as hair clips, bobby pins, jewelry (including body-piercing jewelry), watches, eyeglasses, hearing aids, and dentures.

Understanding CTA and MRA

Because of the less invasive nature of CTA and MRA compared with conventional angiography, these two tests are becoming more readily available and being used more frequently. In addition, the lack of arterial access in CTA and MRA usually means fewer complications. Both scans typically take less time to perform than conventional angiography, which is especially helpful when your patient is critically ill.

CTA

CTA is a type of CT scan that uses a computer to produce images that are taken via X-ray. The type of CT scanner used must have a multidetector to be capable of performing a CTA. The multidetector functions by allowing the CT scanner to take high-quality pictures of the brain quickly. I.V. contrast media is used to help produce a clear picture of the cerebral arteries. With this type of scan, the doctor can see an aneurysm, even if it's ruptured.

Before a CTA

To prepare your patient for CTA, you'll need to establish vascular access for the contrast injection. Also, explain that it's important for the patient to lie still during the procedure to ensure good-quality images. Let the patient know that it will be possible to communicate with the technician and any other health care personnel that are present during the scan. Ask the patient about his history of adverse reactions to contrast medium and document what happened during that reaction. Be sure to communicate this information to the doctor and radiologist and follow your facility's policy on using contrast in these patients.

Intravascular contrast can make patients feel warm, so explain to the patient the difference between this type of expected reaction and unanticipated adverse reactions. Mild adverse reactions include nausea, vomiting, local urticaria, or pruritus; moderate adverse reactions include vasovagal reactions, bronchospasm, or mild laryngeal edema; and severe adverse reactions (rare) include seizures or cardiac arrest.

You may accompany a critically ill patient to the CTA scan; make sure that you use the necessary monitoring equipment and have resuscitation equipment available.

Because the patient must be supine for the duration of the scan, you may need to administer a diuretic before the procedure if the patient has ICP compromise.

After a CTA

Unless contraindicated, encourage oral fluid intake or administer I.V. fluids after the scan is complete to enhance urinary excretion of the contrast medium. Monitor the patient's renal function, assess for signs and symptoms of adverse reactions, and monitor ICP and neurologic status for signs of compromise.

MRA

MRA is a type of MRI. Magnetic resonance works by manipulating hydrogen, the most common element in human tissue. Hydrogen creates a radiofrequency signal when exposed to a magnetic field. A computer gathers the radiofrequency signals into a readable picture. In MRA, the radiofrequency signals created by hydrogen traveling in the arteries create an image. The computer removes the images of other structures and provides a clear image of the cerebral arteries and pathology. An I.V. contrast medium, gadolinium, is also injected to highlight the arteries in MRA. An MRA is valuable for evaluating intracranial or extracranial atherosclerosis, AVM, intact aneurysms, or other cerebrovascular disease.

Before an MRA

You should explain to your patient that it will be necessary for him to lie still during the procedure so that clear images can be obtained. Warn him that the scanner produces a loud jackhammer-like noise but that ear protection will be available. If not already present, you'll need to establish vascular access for the contrast medium injection.

Understanding CTA and MRA *(continued)*

Before the procedure, ask about any metal implants in his body and document your findings. Most facilities have an MRI/MRA screening form. You may want to also screen your patient for claustrophobia and obtain an order for anxiolytic if necessary. If you accompany a critically ill patient to the scan, make sure that you use the necessary monitoring equipment and have resuscitation equipment available. Assure the patient that he will be able to communicate with

you and other health care personnel throughout the scan.

After an MRA

After the scan, monitor the patient's renal function. If you administered sedation, continue to monitor the patient's cardiopulmonary status. Encourage the patient to drink fluids or administer I.V. fluids for 24 to 48 hours after the MRA, unless contraindicated.

- Explain that the test is painless but that the machinery may seem loud and frightening and the tunnel confining. Tell the patient that he'll receive earplugs to reduce the noise.
- Provide sedation, as ordered, to promote relaxation during the test.
- After the procedure, increase the I.V. flow rate, as ordered, or encourage the patient to increase his fluid intake to flush the contrast medium from his system.

Positron emission tomography scan

PET scanning provides colorimetric information about the brain's metabolic activity. It works by detecting how quickly tissues consume radioactive isotopes.

PET scanning is used to reveal cerebral dysfunction associated with tumors, seizures, TIA, head trauma, Alzheimer's disease, Parkinson's disease, MS, and some mental illnesses. In addition, a PET scan can be used to evaluate the effect of treatment.

PET project

Here's how PET scanning works:

- A technician administers a radioactive gas or an I.V. injection of glucose or other biochemical substance tagged with isotopes, which act as tracers.
- The isotopes emit positrons that combine with negatively charged electrons in tissue cells to create gamma rays.
- The PET scanner registers the emitted gamma rays, and a computer translates the information into patterns that reflect cerebral blood flow, blood volume, and neuron and neurotransmitter metabolism.

PET scanning is used to gather clues about cerebral blood flow, blood volume, and neuron and neurotransmitter metabolism. Don't worry, little buddy, it has nothing to do with you.



Nursing concerns

- Provide reassurance that PET scanning doesn't expose the patient to dangerous levels of radiation.
- Explain that insertion of an I.V. catheter may be required.
- Instruct the patient to lie still during the test.

Skull and spinal X-rays

Skull X-rays are typically taken from two angles: anteroposterior and lateral. The practitioner may order other angles, including Waters view, or occipitomeatal projection, to examine the frontal and maxillary sinuses, facial bones, and eye orbits.

Having one's head examined

Skull X-rays are used to detect fractures; bony tumors or unusual calcifications; pineal displacement or skull or sella turcica erosion, which indicates a space-occupying lesion; and vascular abnormalities.

Spinal findings

The practitioner may order anteroposterior and lateral spinal X-rays when:

- spinal disease is suspected
- injury to the cervical, thoracic, lumbar, or sacral vertebral segments exists.

Depending on the patient's condition, other X-ray images may be taken from special angles, such as the open-mouth view (to confirm odontoid fracture).

Spinal X-rays are used to detect spinal fracture; displacement and subluxation due to partial dislocation; destructive lesions, such as primary and metastatic bone tumors; arthritic changes or spondylolisthesis; structural abnormalities, such as kyphosis, scoliosis, and lordosis; and congenital abnormalities.

Nursing concerns

- Reassure the patient that X-rays are painless.
- As ordered, administer an analgesic before the procedure if the patient has existing pain, so he'll be more comfortable.
- Remove the patient's cervical collar if cervical X-rays reveal no fracture and the practitioner permits it.

Skull X-rays are typically taken from the anteroposterior and lateral angles.



Critical care nursing may seem like backbreaking work, but an X-ray is needed to confirm the diagnosis.



Angiography

Cerebral angiography

During cerebral angiography, the doctor injects a radiopaque contrast medium, usually into the brachial artery (through retrograde brachial injection) or femoral artery (through catheterization).

Why it's done

This procedure highlights cerebral vessels, making it easier to:

- detect stenosis or occlusion associated with thrombus or spasm
- identify aneurysms and AVMs
- locate vessel displacement associated with tumors, abscesses, cerebral edema, hematoma, or herniation
- assess collateral circulation
- treat vasospasm.

Nursing concerns

- Explain the procedure to the patient and answer all questions honestly.
- Confirm that the patient isn't allergic to iodine or shellfish because a person with such allergies may have an adverse reaction to the contrast medium.
- Preprocedure testing should include evaluation of renal function (serum creatinine and BUN levels) and potential risk of bleeding (prothrombin time [PT], partial thromboplastin time [PTT], and platelet count). Notify the practitioner of abnormal results.
- Instruct the patient to lie still during the procedure.
- Explain that he'll probably feel a flushed sensation in his face as the dye is injected.
- Maintain bed rest, as ordered, and monitor the patient's vital signs.
- Monitor the catheter injection site for signs of bleeding.
- As ordered, keep a sandbag over the injection site.
- Monitor the patient's peripheral pulse in the arm or leg used for catheter insertion and mark the site of the pulse for reference.
- Unless contraindicated, encourage the patient to drink more fluids to flush the dye from the body; alternatively, increase the I.V. flow rate as ordered.
- Monitor the patient for neurologic changes and such complications as hemiparesis, hemiplegia, aphasia, and impaired LOC.
- Monitor for adverse reactions to the contrast medium, which may include restlessness, tachypnea and respiratory distress, tachycardia, facial flushing, urticaria, and nausea and vomiting.

Cerebral angiography is used to detect and identify problems affecting the cerebral vessels.



Encourage fluid consumption to flush out the contrast dye after testing, unless it's contraindicated.



Electrophysiologic studies

Common electrophysiologic studies include EEG and evoked potential studies.

Electroencephalography

During EEG, the brain's continuous electrical activity is recorded.

The results are used to identify seizure disorders; metabolic encephalopathy; other multifocal brain lesions, such as those caused by dementia or herpes; and brain death.

Nursing concerns

- Explain that a technician applies paste and attaches electrodes to areas of skin on the patient's head and neck after these areas have been lightly abraded to ensure good contact.
- Instruct the patient to remain still during the test.
- Discuss what the patient may be asked to do during the test, such as hyperventilating for 3 minutes or sleeping, depending on the purpose of the EEG.
- After the test, use acetone to remove any remaining paste from the patient's skin.

Evoked potential studies

Evoked potential studies are used to measure the nervous system's electrical response to a visual, auditory, or sensory stimulus. The results are used to detect subclinical lesions such as tumors of CN VIII and complicating lesions in a patient with MS.

Showing potential

Evoked potential studies are also useful in diagnosing blindness and deafness in infants.

Nursing concerns

- Explain to the patient that he must remain still during the test.
- Describe how a technician applies paste and electrodes to the head and neck before testing.
- Describe activities—such as gazing at a checkerboard pattern or a strobe light or listening with headphones to a series of clicks—performed during testing. The patient may have electrodes placed on an arm and leg and be asked to respond to a tapping sensation.
- Explain that the test equipment may emit noises.



EEG is used to identify problems that affect the brain's electrical activity.

Other tests

Other neurologic tests include lumbar puncture and transcranial Doppler studies.

Lumbar puncture

During lumbar puncture, a sterile needle is inserted into the subarachnoid space of the spinal canal, usually between the third and fourth lumbar vertebrae. A doctor does the lumbar puncture, with a nurse assisting. It requires sterile technique and careful patient positioning.

Be careful! If the patient has increased ICP, the rapid drop in ICP after CSF is withdrawn can lead to serious problems.

Why do it?

Lumbar puncture is used to:

- detect blood in cerebrospinal fluid (CSF)
- obtain CSF specimens for laboratory analysis
- inject dyes or gases for contrast in radiologic studies.

It's also used to administer drugs or anesthetics.

Contraindications and cautions

Lumbar puncture is contraindicated in patients with lumbar deformity or infection at the puncture site. It's not performed in patients with increased ICP because the rapid decrease of pressure that follows withdrawal of CSF can cause tonsillar herniation and medullary compression.



Nursing concerns

- Calmly describe lumbar puncture to the patient, explaining that the procedure may cause some discomfort.
- Reassure the patient that a local anesthetic is administered before the test. Tell him to report any tingling or sharp pain he feels as the anesthetic is injected.
- To prevent headache after the test, instruct the patient to lie flat for 4 to 6 hours after the procedure.
- Monitor the patient for neurologic deficits and complications, such as headache, fever, back spasms, or seizures, according to facility policy.
- Administer analgesics as needed.
- Monitor the puncture site for signs of infection.

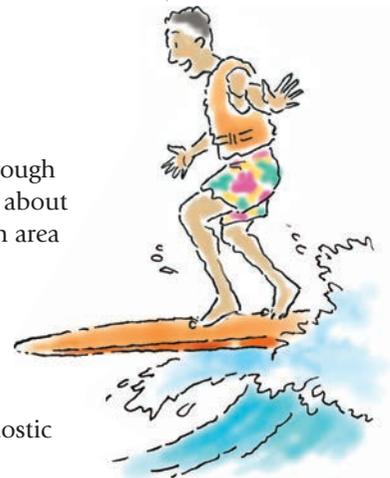
In transcranial Doppler studies, waveforms and velocities of blood flow are measured. High velocities are typically abnormal.

Transcranial Doppler studies

In transcranial Doppler studies, the velocity of blood flow through cerebral arteries is measured. The results provide information about the presence, quality, and changing nature of blood flow to an area of the brain.

What blood flow tells you

The types of waveforms and velocities obtained by testing indicate whether disease exists. Test results commonly aren't definitive, but this is a noninvasive way to obtain diagnostic information.



High velocities are typically abnormal, suggesting that blood flow is too turbulent or the vessel is too narrow. They may also indicate stenosis or vasospasm. High velocities may also indicate AVM due to the extra blood flow associated with stenosis or vasospasm.

Nursing concerns

- Tell the patient that the study usually takes less than 1 hour, depending on the number of vessels examined and on any interfering factors.
- Explain that a small amount of gel is applied to the skin and that a probe is then used to transmit a signal to the artery being studied.

Treatments

Treatments for patients with neurologic dysfunction may include medication therapy, surgery, and other forms of treatment.

Medication therapy

For many of your patients with neurologic disorders, medication or drug therapy is essential. For example:

- thrombolytics are used to treat patients with acute ischemic stroke
- anticonvulsants are used to control seizures
- corticosteroids are used to reduce inflammation.

Typical types

Types of drugs commonly used to treat patients with neurologic disorders include:

- analgesics
- anticonvulsants
- anticoagulants and antiplatelets
- barbiturates
- benzodiazepines
- calcium channel blockers
- corticosteroids
- diuretics
- thrombolytics.

Heads up!

When caring for a patient undergoing medication therapy, stay alert for severe adverse reactions and interactions with other drugs. Some drugs such as barbiturates also carry a high risk of toxicity.

Watch for severe adverse reactions and interactions with other drugs.



Stay the course

Successful therapy hinges on strict adherence to the medication schedule. Compliance is especially critical for drugs that require steady blood levels for therapeutic effectiveness such as anticonvulsants. (See *Common neurologic drugs*, pages 84 to 87.)

Surgery

Life-threatening neurologic disorders usually call for emergency surgery.

Surgery commonly involves craniotomy, a procedure to open the skull and expose the brain.

Be ready before and after

You may be responsible for the patient's care before and after surgery.

Here are some general preoperative and postoperative pointers:

- The prospect of surgery usually causes fear and anxiety, so give ongoing emotional support to the patient and his family. Make sure that you're ready to answer their questions.
- Postoperative care may include teaching about diverse topics, such as ventricular shunt care and tips about cosmetic care after craniotomy. Be ready to give good advice after surgery.



I feel so exposed. Life-threatening neurologic disorders may call for emergency surgery, beginning with craniotomy.

Craniotomy

During craniotomy, a surgical opening into the skull exposes the brain. This procedure allows various treatments, such as ventricular shunting, excision of a tumor or abscess, hematoma aspiration, and aneurysm clipping (placing one or more surgical clips on the neck of an aneurysm to destroy it).

Condition and complexity count

The degree of risk depends on your patient's condition and the complexity of the surgery. Craniotomy raises the risk of having various complications, such as:

- infection
- hemorrhage
- respiratory compromise
- increased ICP.

Nursing concerns

- Encourage the patient and his family to ask questions about the procedure. Provide clear, honest answers to reduce their confusion and anxiety and to enhance effective coping.
- Explain that the patient's head will be shaved before surgery in the area where the incision will be made.

After craniotomy, provide emotional support to the patient and his family to help them cope with any remaining deficits.



(Text continues on page 86.)

Common neurologic drugs

Use this table to find out about common neurologic drugs, their indications and adverse effects, and related monitoring measures.

Drugs	Indications	Adverse effects
Nonopioid analgesics		
Acetaminophen (Tylenol)	<ul style="list-style-type: none"> Mild pain, headache 	<ul style="list-style-type: none"> Severe liver damage, neutropenia, thrombocytopenia
Opioid analgesics		
Morphine (MS Contin)	<ul style="list-style-type: none"> Severe pain 	<ul style="list-style-type: none"> Respiratory depression, apnea, bradycardia, seizures, sedation
Codeine	<ul style="list-style-type: none"> Mild to moderate pain 	<ul style="list-style-type: none"> Respiratory depression, bradycardia, sedation, constipation
Anticonvulsants		
Carbamazepine (Tegretol)	<ul style="list-style-type: none"> Generalized tonic-clonic seizures, complex partial seizures, mixed seizures 	<ul style="list-style-type: none"> Heart failure, hepatitis, Stevens-Johnson syndrome, aplastic anemia
Fosphenytoin (Cerebyx)	<ul style="list-style-type: none"> Status epilepticus, seizures during neurosurgery 	<ul style="list-style-type: none"> Increased ICP, cerebral edema, somnolence, ventricular fibrillation, hepatotoxicity
Levetiracetam (Keppra)	<ul style="list-style-type: none"> Generalized tonic-clonic seizures, partial-onset seizures, juvenile myoclonic epilepsy 	<ul style="list-style-type: none"> Leukopenia, neutropenia, somnolence, asthenia, dizziness
Phenytoin (Dilantin)	<ul style="list-style-type: none"> Generalized tonic-clonic seizures, status epilepticus, nonepileptic seizures after head trauma 	<ul style="list-style-type: none"> Agranulocytosis, thrombocytopenia, toxic hepatitis, slurred speech, Stevens-Johnson syndrome, ataxia
Primidone (Mysoline)	<ul style="list-style-type: none"> Generalized tonic-clonic seizures, focal seizures, and complex partial seizures 	<ul style="list-style-type: none"> Thrombocytopenia, drowsiness, ataxia
Valproic acid, valproate (Depakene)	<ul style="list-style-type: none"> Complex partial seizures, simple and complex absence seizures 	<ul style="list-style-type: none"> Thrombocytopenia, pancreatitis, toxic hepatitis, sedation, Stevens-Johnson syndrome
Anticoagulants		
Heparin	<ul style="list-style-type: none"> Embolism prophylaxis 	<ul style="list-style-type: none"> Hemorrhage, thrombocytopenia, prolonged clotting time
Antiplatelets		
Aspirin (Ecotrin)	<ul style="list-style-type: none"> TIAs, prophylaxis for TIAs 	<ul style="list-style-type: none"> GI bleeding, acute renal insufficiency, thrombocytopenia, liver dysfunction, angioedema
Clopidogrel (Plavix)	<ul style="list-style-type: none"> Thrombotic stroke prophylaxis 	<ul style="list-style-type: none"> Purpura, dizziness, rash, epistaxis
Ticlopidine (Ticlid)	<ul style="list-style-type: none"> Thrombotic stroke prophylaxis 	<ul style="list-style-type: none"> Thrombocytopenia, agranulocytosis, intracranial bleeding, hepatitis

Practice pointers

- Monitor total daily intake of acetaminophen because of risk of liver toxicity. Use with caution in elderly patients and those with liver disease.
- Monitor for respiratory depression. Use with caution in elderly patients and those with head injury, seizures, or increased ICP. Contraindicated in patients with acute bronchial asthma.
- Monitor for respiratory depression. Use with caution in elderly patients and those with head injury, seizures, or increased ICP.
- Use cautiously in patients with mixed seizure disorders because it can increase the risk of seizure. Use cautiously in patients with hepatic dysfunction. Obtain baseline liver function studies, complete blood count (CBC), and BUN level. Monitor blood levels of the drug; therapeutic level is 4 to 12 mcg/ml.
- Stop drug with acute hepatotoxicity. May cause hyperglycemia; monitor blood glucose in diabetic patients. Fosphenytoin should be prescribed and dispensed in phenytoin sodium equivalent units. Monitor for cardiac arrhythmias and QT prolongation.
- Use cautiously in immunocompromised patients and patients with a history of psychotic symptoms and behaviors. Use only with other anticonvulsants. Monitor renal function.
- Abrupt withdrawal can trigger status epilepticus. Contraindicated in patients with heart block. Use cautiously in patients with hepatic disease and myocardial insufficiency. Monitor blood levels of the drug; therapeutic range is 10 to 20 mcg/ml. If rash appears, stop the drug.
- Abrupt withdrawal can cause status epilepticus. Reduce dosage in elderly patients.
- Obtain baseline liver function tests. Avoid use in patients at high risk for hepatotoxicity. Abrupt withdrawal may worsen seizures. Monitor blood levels of the drug; therapeutic range is 50 to 100 mcg/ml.
- Monitor for bleeding. Obtain baseline prothrombin time/International Normalized Ratio (PT/INR) and PTT. Monitor PTT at regular intervals. Protamine sulfate reverses the effects of heparin.
- Monitor for bleeding. Avoid use in patients with active peptic ulcer and GI inflammation.
- Monitor for signs of thrombotic thrombocytopenia purpura.
- Monitor for bleeding. Avoid use in patients with hepatic impairment and peptic ulcer disease.

(continued)

Common neurologic drugs *(continued)*

Drugs	Indications	Adverse effects
Barbiturates		
Phenobarbital (Solfoton)	<ul style="list-style-type: none"> All types of seizures except absence seizures, febrile seizures in children; also used for status epilepticus, sedation, and drug withdrawal 	<ul style="list-style-type: none"> Respiratory depression, apnea, bradycardia, angioedema, Stevens-Johnson syndrome
Benzodiazepines		
Clonazepam (Klonopin)	<ul style="list-style-type: none"> Absence and atypical seizures, status epilepticus, panic disorders 	<ul style="list-style-type: none"> Respiratory depression, thrombocytopenia, leukopenia, drowsiness, ataxia
Diazepam (Valium)	<ul style="list-style-type: none"> Status epilepticus, anxiety, acute alcohol withdrawal, muscle spasm, repetitive seizure activity 	<ul style="list-style-type: none"> Bradycardia, cardiovascular collapse, drowsiness, acute withdrawal syndrome, ataxia
Lorazepam (Ativan)	<ul style="list-style-type: none"> Status epilepticus, anxiety, agitation 	<ul style="list-style-type: none"> Drowsiness, acute withdrawal syndrome
Corticosteroids		
Dexamethasone, methylprednisolone (Medrol)	<ul style="list-style-type: none"> Cerebral edema associated with brain tumors, severe inflammation 	<ul style="list-style-type: none"> Heart failure, cardiac arrhythmias, circulatory collapse, thromboembolism, pancreatitis, peptic ulceration
Diuretics		
Furosemide (Lasix) (loop)	<ul style="list-style-type: none"> Edema, hypertension 	<ul style="list-style-type: none"> Renal failure, thrombocytopenia, agranulocytosis, volume depletion, dehydration, hypokalemia
Mannitol (Osmitrol)	<ul style="list-style-type: none"> Cerebral edema, increased ICP 	<ul style="list-style-type: none"> Seizures, fluid and electrolyte imbalance, diarrhea
Thrombolytics		
Alteplase (Activase) (recombinant tissue plasminogen activator)	<ul style="list-style-type: none"> Acute ischemic stroke 	<ul style="list-style-type: none"> Cerebral hemorrhage, spontaneous bleeding, allergic reaction

- Discuss the recovery period so the patient understands what to expect. Explain that he'll awaken with a dressing on his head to protect the incision and may have a surgical drain.
- Tell him to expect a headache and facial swelling for 2 to 3 days after surgery and reassure him that he'll receive pain medication.
- Monitor the patient's neurologic status and vital signs and report any acute change immediately. Watch for signs of increased ICP, such as pupil changes, weakness in extremities, headache, and change in LOC.

Practice pointers

- Monitor for respiratory depression and bradycardia. Keep resuscitation equipment on hand when administering I.V. dose; monitor respirations.
- Abrupt withdrawal may precipitate status epilepticus. Withdrawal of drug may cause insomnia, tremors, and hallucinations.
- Monitor for respiratory depression and cardiac arrhythmia. Don't stop suddenly; can cause acute withdrawal in physically dependent persons.
- Don't stop abruptly; can cause withdrawal. Monitor for CNS depressant effects in elderly patients.
- Use cautiously in patients with recent myocardial infarction, hypertension, renal disease, and GI ulcer. Monitor blood pressure and blood glucose levels. Monitor the patient's potassium level (can cause hypokalemia).
- Monitor blood pressure, pulse, and intake and output. Monitor serum electrolyte levels, especially potassium levels. Monitor for cardiac arrhythmias.
- Contraindicated in severe pulmonary congestion and heart failure. Monitor blood pressure, heart rate, and intake and output. Monitor serum electrolyte levels. Use with caution in patients with renal dysfunction.
- Contraindicated in patients with intracranial or subarachnoid hemorrhage. The patient must meet criteria for thrombolytic therapy before initiation of therapy. Monitor baseline laboratory values: hemoglobin (Hb) level, hematocrit (HCT), PTT, PT/INR. Monitor vital signs. Monitor for signs of bleeding. Monitor puncture sites for bleeding.
- Monitor the incision site for signs of infection or drainage.
- Provide emotional support to the patient and his family as they cope with remaining neurologic deficits.

Cerebral aneurysm repair

Surgical intervention is the only sure way to prevent rupture or rebleeding of a cerebral aneurysm.

Endovascular aneurysm repair

An alternative to craniotomy and clipping for intracranial aneurysm repair, an endovascular treatment method called *embolization*, may be used. Embolization is most successful in aneurysms with small necks and those without significant intraluminal thrombus.

Embolization

Here's what happens in embolization:

- A microcatheter with a coil attached is introduced through the initial catheter.
- After the coil is positioned within the fundus of the aneurysm, it is detached from the catheter using an electrical current.
- The delivery catheter is removed, leaving the platinum coil in place, and another coil is introduced into the fundus.

- The process is continued until the aneurysm is densely packed with platinum and no longer opacifies during diagnostic contrast injections.

How it works

The positively charged platinum left in the aneurysm theoretically attracts negatively charged blood elements, such as white and red blood cells, platelets, and fibrinogen. This induces intra-aneurysmal thrombosis.

The coils provide immediate protection against further hemorrhage by reducing blood pulsations in the fundus and sealing the hole or weak portion of the artery wall. Eventually, clots form, and the aneurysm is separated from the parent vessel by the formation of new connective tissue.

Northern exposure

In cerebral aneurysm repair, a craniotomy is performed to expose the aneurysm. Depending on the shape and location of the aneurysm, the surgeon then uses one of several corrective techniques, such as:

- clamping the affected artery
- wrapping the aneurysm wall with a biologic or synthetic material
- clipping or ligating the aneurysm.

New and improved

Other techniques for surgery include *interventional radiology* in conjunction with endovascular balloon therapy. This technique occludes the aneurysm or vessel and uses cerebral angiography to treat arterial vasospasm.

In some cases, a type of nonsurgical repair called *embolization* is used. (See *Endovascular aneurysm repair*.)

Nursing concerns

- Tell the patient and his family that monitoring is done in the critical care unit before and after surgery. Explain that several I.V. lines, intubation, and mechanical ventilation may be needed.
- Monitor the incision site for signs of infection or drainage.
- Monitor the patient's neurologic status and vital signs and report acute changes immediately. Watch for signs of increased ICP, such as pupil changes, weakness in extremities, headache, and a change in LOC.
- Give emotional support to the patient and his family to help them cope with remaining neurologic deficits.

Other treatments

Other treatments include barbiturate coma, CSF drainage, ICP monitoring, and plasmapheresis.

Barbiturate coma

The practitioner may order barbiturate coma when conventional treatments, such as fluid restriction, diuretic or corticosteroid therapy, or ventricular shunting, don't correct sustained or acute episodes of increased ICP.

A barbiturate coma produces a drug-induced state that reduces the patient's metabolic rate and cerebral blood flow. I suddenly feel very sleepy . . .

High I.V.

During barbiturate coma, the patient receives high I.V. doses of a short-acting barbiturate (such as pentobarbital [Nembutal]) to produce a comatose state. The drug reduces the patient's metabolic rate and cerebral blood flow.



Last resort

The goal of barbiturate coma is to relieve increased ICP and protect cerebral tissue. It's a last resort for patients with:

- acute ICP elevation (over 40 mm Hg)
- persistent ICP elevation (over 20 mm Hg)
- rapidly deteriorating neurologic status that's unresponsive to other treatments.

If barbiturate coma doesn't reduce ICP, the patient's prognosis for recovery is poor.

Nursing concerns

- Focus your attention on the patient's family. The patient's condition and apprehension about the treatment is likely to frighten them. Provide clear explanations of the procedure and its effects and encourage them to ask questions. Convey a sense of optimism but provide no guarantees of the treatment's success.
- Prepare the family for expected changes in the patient during therapy, such as decreased respirations, hypotension, and loss of muscle tone and reflexes.
- Closely monitor the patient's ICP, electrocardiogram (ECG), bispectral index, and vital signs. Notify the practitioner of increased ICP, arrhythmias, or hypotension.
- Because the patient is in a drug-induced coma, devote special care to safety measures to prevent injury.

Devote special care to pressure ulcer prevention when the patient is in a drug-induced coma.



Cerebrospinal fluid drainage

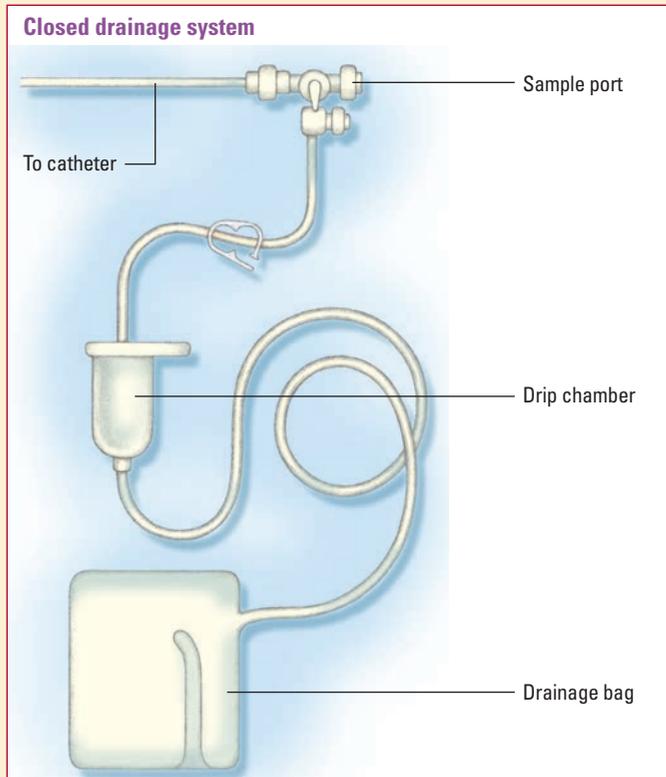
The goal of CSF drainage is to reduce ICP to the desired level and keep it at that level. Fluid is withdrawn from the lateral ventricle through an external ventricular catheter (EVD).

CSF closed drainage system

The goal of CSF drainage is to control ICP during treatment for traumatic injury or other conditions that cause increased ICP. Here's one common procedure.

External ventricular drain

For a ventricular drain, the neurosurgeon makes a burr hole in the patient's skull and inserts the catheter into the ventricle. The distal end of the catheter is connected to a closed drainage system.



Observe for complications such as rapid or excessive CSF drainage—an emergency! Signs and symptoms include headache, tachycardia, diaphoresis, and nausea.

To place the ventricular drain, a neurosurgeon inserts a ventricular catheter through a burr hole in the patient's skull. This is usually done in the operating room but can be done in the emergency department (ED) or at the bedside in the intensive care unit. (See *CSF closed drainage system*.)

Nursing concerns

- For continuous drainage of CSF by maintaining the drainage system, drip chamber at the desired level.



- To intermittently drain the CSF, put on gloves and turn the main stopcock on to drainage and allow the CSF to collect in the drip chamber according to ordered parameters.
- To stop drainage, turn off the stopcock to drainage. Record the time and the amount of collected CSF as well as its color.
- Check the patient's dressing frequently for drainage or bleeding.
- Check the tubing for patency by watching the CSF drops in the drip chamber.
- Observe the CSF for color, clarity, amount, blood, and sediment.
- Maintain the patient on bed rest with the head of the bed at 30 degrees to promote venous drainage that will also help control ICP.
- Observe for complications, such as excessive CSF drainage, characterized by headache, tachycardia, diaphoresis, and nausea. Overly rapid accumulation of drainage can be a neurosurgical emergency by causing a subdural hematoma. Cessation of drainage may indicate clot formation or collapse of ventricles.

Intracranial pressure monitoring

In ICP monitoring, pressure exerted by the brain, blood, and CSF against the inside of the skull is measured. ICP monitoring enables prompt intervention, which can avert damage caused by cerebral hypoxia and shifts of brain mass.

Indications for ICP monitoring include:

- closed head injury with severe neurologic deficit
- overproduction or insufficient absorption of CSF (hydrocephalus)
- cerebral hemorrhage
- space-occupying lesions.

Four similar systems

There are four basic types of ICP monitoring systems. (See *Monitoring ICP*, page 92.) Regardless of which system is used, the insertion procedure is always performed by a neurosurgeon in the operating room, ED, or critical care unit. Insertion of an ICP monitoring device requires sterile technique to reduce the risk of CNS infection.

Doctors do this

The neurosurgeon inserts a ventricular catheter or subarachnoid screw through a twist-drill hole created in the skull. The device is attached to a transducer that converts ICP to electrical impulses displayed as waveforms, allowing constant monitoring.

Nursing concerns

- Observe digital ICP readings and waveforms. (See *Interpreting ICP waveforms*, page 93.)

Pressure exerted by the brain, blood, and CSF against the inside of my skull can cause cerebral hypoxia and shifts in brain mass. That can hurt!



Monitoring ICP

ICP can be monitored using one of four systems.

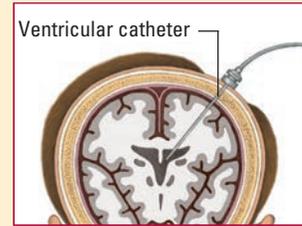
1 Intraventricular catheter monitoring

In intraventricular catheter monitoring, used to monitor ICP directly, the doctor inserts a small polyethylene or silicone rubber catheter into the lateral ventricles through a burr hole.

Although this method is most accurate for measuring ICP, it carries the

greatest risk of infection. This is the only type of ICP monitoring that allows evaluation of brain compliance and significant drainage of CSF.

Contraindications usually include stenotic cerebral ventricles, cerebral aneurysms in the path of catheter placement, and suspected vascular lesions.

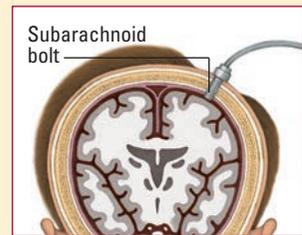


2 Subarachnoid bolt monitoring

Subarachnoid bolt monitoring involves insertion of a special bolt into the subarachnoid space through a twist-drill burr hole in the front of the skull, behind the hairline.

Placing the bolt is easier than placing an intraventricular catheter, especially

if a CT scan reveals that the cerebrum has shifted or the ventricles have collapsed. This type of ICP monitoring carries less risk of infection and parenchymal damage because the bolt doesn't penetrate the cerebrum.

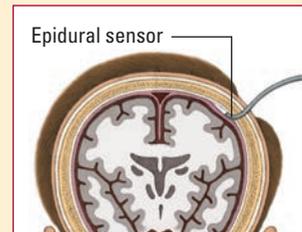


3 Epidural or subdural sensor monitoring

ICP can also be monitored from the epidural or subdural space. For epidural monitoring, a fiber-optic sensor is inserted into the epidural space through a burr hole. This system's main drawback is its questionable accuracy because

ICP isn't being measured directly from a CSF-filled space.

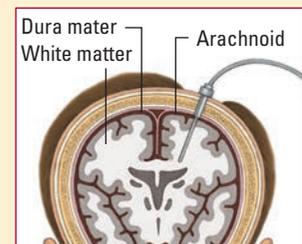
For subdural monitoring, a fiber-optic transducer-tipped catheter is tunneled through a burr hole and is placed on brain tissue under the dura mater. The main drawback to this method is its inability to drain CSF.



4 Intraparenchymal monitoring

In intraparenchymal monitoring, the doctor inserts a catheter through a small subarachnoid bolt and, after punctuating the dura mater, advances the catheter a few centimeters into the brain's white matter. There's no need to balance or calibrate the equipment after insertion.

This method doesn't provide direct access to CSF, but measurements are accurate because brain tissue pressure correlates well with ventricular pressures. Intraparenchymal monitoring may be used to obtain ICP measurements in patients with compressed or dislocated ventricles.

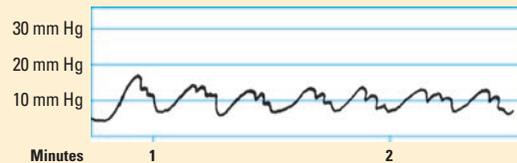


Interpreting ICP waveforms

Three waveforms—A, B, and C—are used to monitor ICP. A waves are an ominous sign of intracranial decompensation and poor compliance, B waves correlate with changes in respiration, and C waves correlate with changes in arterial pressure.

Normal waveform

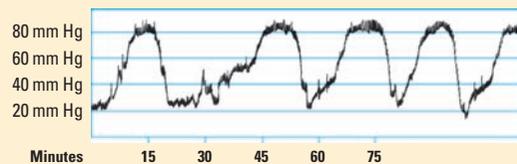
A normal ICP waveform typically shows a steep upward systolic slope followed by a downward diastolic slope with a dicrotic notch. In most cases, this waveform is continuous and indicates an ICP between 0 and 15 mm Hg (normal blood pressure).



A waves

The most clinically significant ICP waveforms are A waves, which may reach elevations of 50 to 200 mm Hg, persist for 5 to 20 minutes, then drop sharply, signaling exhaustion of the brain's compliance mechanisms.

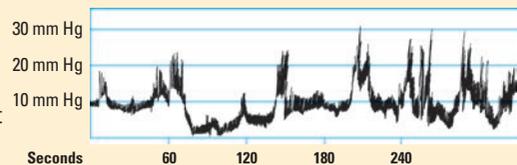
A waves may come and go, spiking from temporary increases in thoracic pressure or from any condition that increases ICP beyond the brain's compliance limits. A waves are commonly associated with a temporary decrease in neurologic status.



B waves

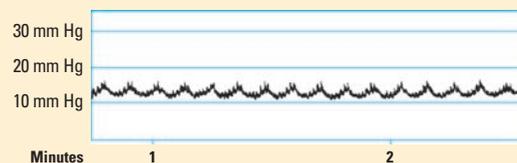
B waves, which appear sharp and rhythmic with a sawtooth pattern, occur every 30 seconds to 2 minutes and may reach an elevation of 50 mm Hg. The clinical significance of B waves isn't clear; however, the waves correlate with respiratory changes and may occur more frequently with decreasing compensation.

Because B waves sometimes precede A waves, notify the practitioner if B waves occur frequently.



C waves

Like B waves, C waves are rapid and rhythmic, but they aren't as sharp. They may reach an elevation of 20 mm Hg and occur up to six times per minute. They're clinically insignificant and may fluctuate with respirations or systemic blood pressure changes.



Waveform showing equipment problem

A waveform like the one shown at right signals a problem with the transducer or monitor. Check for line obstruction and determine whether the transducer needs rebalancing.



- Assess the patient's clinical status and monitor routine and neurologic vital signs every hour or as ordered.
- Calculate cerebral perfusion pressure (CPP) hourly. To calculate CPP, subtract ICP from mean arterial pressure (MAP).
- Inspect the insertion site at least every 24 hours for redness, swelling, and drainage.

Plasmapheresis

Symptoms of several neurologic disorders are reduced through plasma exchange or plasmapheresis.

Out with the bad

In plasmapheresis, blood from the patient flows into a cell separator, which separates plasma from formed elements. The plasma is then filtered to remove toxins and disease mediators, such as immune complexes and autoantibodies, from the patient's blood.

In with the good

The cellular components are then transfused back into the patient using fresh frozen plasma or albumin in place of the plasma removed.

Who benefits?

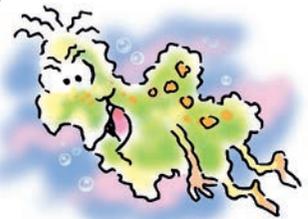
Plasmapheresis benefits patients with neurologic disorders such as Guillain-Barré syndrome and, especially, myasthenia gravis. In myasthenia gravis, plasmapheresis is used to remove circulating antiacetylcholine receptor antibodies.

Plasmapheresis is used most commonly for patients with long-standing neuromuscular disease, but it can also be used to treat patients with acute exacerbations. Some acutely ill patients require treatment up to four times per week; others about once every 2 weeks. When it's successful, treatment may relieve symptoms for months, but results vary.

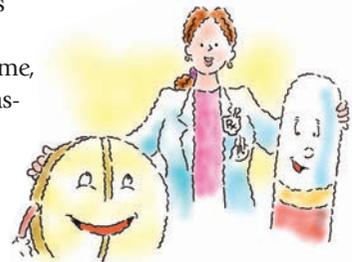
Nursing concerns

- Discuss the treatment and its purpose with the patient and his family.
- Explain that the procedure can take up to 5 hours. During that time, blood samples are taken frequently to monitor calcium and potassium levels. Blood pressure and heart rate are checked regularly. Tell the patient to report any paresthesia (numbness, burning, tingling, prickling, or increased sensitivity) during treatment.
- If possible, give the patient prescribed medications after treatment because they're removed from the blood during treatment.
- Monitor the patient's vital signs according to your facility's policy.
- Check puncture sites for signs of bleeding or extravasation.

Looks like I'm movin' out!
In plasmapheresis, plasma is filtered to remove toxins and disease mediators from the patient's blood.



Administer prescribed medications after plasmapheresis; otherwise, they're removed from the blood during treatment.



Neurologic system disorders

In the critical care unit, you're likely to encounter patients with common neurologic disorders, especially acute spinal cord injury, AVM, cerebral aneurysm, encephalitis, Guillain-Barré syndrome, head injury, meningitis, seizure disorders, and stroke.

Acute spinal cord injury

Spinal injuries include fractures, contusions, and compressions of the vertebral column. They usually result from trauma to the head or neck. Fractures of the 5th, 6th, or 7th cervical; 12th thoracic; and 1st lumbar vertebrae are most common.

Dangerous damage

The real danger with spinal injury is spinal cord damage due to cutting, pulling, twisting, and compression. Spinal cord injury can occur at any level, and the damage it causes may be partial or involve the entire cord.

Complications of spinal cord injury include neurogenic shock and spinal shock. (See *Complications of spinal cord injury*, page 96.)

What causes it

The most serious spinal cord trauma typically results from motor vehicle accidents, falls, sports injuries, diving into shallow water, and gunshot or stab wounds. Less serious injuries commonly occur from lifting heavy objects and minor falls.

Spinal dysfunction may also result from hyperparathyroidism and neoplastic lesions.

How it happens

Spinal cord trauma results from acceleration, deceleration, or other deforming forces. Types of trauma include:

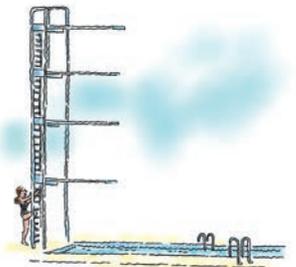
- hyperextension due to acceleration–deceleration forces
- hyperflexion from sudden and excessive force
- vertebral compression from downward force from the top of the cranium, along the vertical axis, and through the vertebra
- rotational twisting, which adds shearing forces.

Trauma trail

Here's what happens during spinal cord trauma:

- An injury causes microscopic hemorrhages in the gray matter and pia–arachnoid.
- The hemorrhages gradually increase in size until all of the gray matter is filled with blood, which causes necrosis.

“Diving injuries can cause the most serious spinal cord trauma.”





Take charge!

Complications of spinal cord injury

When you assess your patient, watch for these two complications related to spinal cord injuries.

Neurogenic shock

Neurogenic shock is an abnormal vasomotor response that occurs secondary to disruption of sympathetic impulses from the brain stem to the thoracolumbar area. It's most common in patients with cervical cord injury. It causes temporary loss of autonomic function below the level of injury and leads to cardiovascular changes.

Signs of neurogenic shock include:

- orthostatic hypotension
- bradycardia
- inability to sweat below the level of the injury.

Spinal shock

Spinal shock is the loss of autonomic, reflex, motor, and sensory activity below the level of the cord lesion. It occurs secondary to damage of the spinal cord.

Signs of spinal shock include:

- flaccid paralysis
- loss of deep tendon and perianal reflexes
- loss of motor and sensory function.

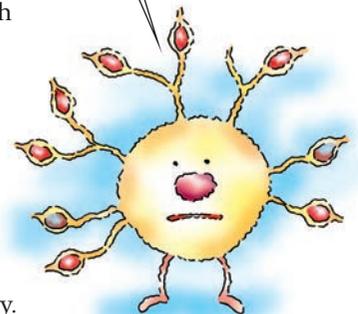
- From the gray matter, the blood enters the white matter, where it impedes circulation within the spinal cord.
- Resulting edema causes compression and decreases the blood supply.
- The spinal cord loses perfusion and becomes ischemic. The edema and hemorrhage are usually greatest in the two segments above and below the injury.
- The edema temporarily adds to the patient's dysfunction by increasing pressure and compressing the nerves. For example, edema near the 3rd to 5th cervical vertebrae may interfere with respiration.

After acute injury

Here's what happens following acute trauma:

- In the white matter, circulation usually returns to normal within 24 hours after injury.
- In the gray matter, an inflammatory reaction prevents restoration of circulation.
- Phagocytes appear at the site within 35 to 48 hours after injury.
- Macrophages engulf degenerating axons, and collagen replaces the normal tissue.

After acute spinal injury, scarring and meningeal thickening leave the nerves in the area blocked or tangled.



- Scarring and meningeal thickening leave the nerves in the area blocked or tangled.

What to look for

In your assessment, look for:

- history of trauma, a neoplastic lesion, an infection that could produce a spinal abscess, or an endocrine disorder
- muscle spasm and back or neck pain that worsens with movement; in cervical fractures, pain that causes point tenderness; in dorsal and lumbar fractures, pain that may radiate to other areas, such as the legs
- mild paresthesia to quadriplegia and shock, if the injury damages the spinal cord.

Speaking specifically

Specific signs and symptoms depend on the type and degree of injury. (See *Types of spinal cord injury*, page 98.)

What tests tell you

Diagnoses of acute spinal cord injuries are based on the results of these tests:

- Spinal X-rays reveal fracture.
- CT scan and MRI show the location of fracture and the site of compression and reveal spinal cord edema and a possible spinal cord mass.
- Neurologic assessment is essential to determine the level of injury and detect cord damage.

How it's treated

The primary treatment after spinal injury is immediate immobilization to stabilize the spine and prevent further damage. Other treatment is supportive.

Cervical injuries require immobilization, using sandbags on both sides of the patient's head, a hard cervical collar, or skeletal traction with skull tongs or a halo device.

What to do

Here's what you should do for patients with spinal cord injuries:

- Immediately stabilize the patient's spine. As with all spinal injuries, suspect cord damage until proven otherwise.
- Perform a neurologic assessment to establish a baseline and continually reassess neurologic status for changes.
- Assess respiratory status closely at least every hour, initially. Obtain baseline tidal volume, vital capacity, negative inspiratory forces, and minute volume.
- Auscultate breath sounds and check secretions as necessary.

I think the correct treatment option is sandbags on both sides of the head. Can anyone reach that shovel?



Types of spinal cord injury

A patient with an acute spinal cord injury (SCI) typically has pain at the site of the spinal fracture. However, it's important to remember that these patients also often have associated brain and systemic injuries that may limit the patient's ability to report localized pain.

Immediately after an SCI, there may be a physiologic loss of all spinal cord function caudal to the level of the injury, with flaccid paralysis, anesthesia, absent bowel and bladder control, and loss of reflex activity. In males, especially those with a cervical cord injury, priapism may develop. This altered physiologic state may last several hours to several weeks and is sometimes referred to as *spinal shock*.

SCI may be complete or incomplete.

Complete injury

Description

- All tracts below the level of the injury are disrupted.
- Loss of motor and sensory function below the level of the injury
 - Cervical cord level—quadriplegia
 - Thoracic level—paraplegia
- Loss is complete and permanent.

Signs and symptoms

- Muscle flaccidity
- Loss of all reflexes and sensory function
- Bowel and bladder atony
- Spinal shock—loss of vasomotor tone
- May have respiratory impairment if the level is at or above C4

Incomplete injury

There are varying degrees of preserved neurologic function depending on the spinal tracts involved.

Description: central cord syndrome

- Injury involves the more centrally located motor fibers.
- Common in elderly after a fall—related to the degenerative changes
- Often a hyperextension/flexion injury

Signs and symptoms

- Motor weakness greater in the upper extremities than the lower extremities

- Often accompanied by a burning dysesthesia in the hands

Description: anterior cord syndrome

- Injury involves the more anterior fibers both motor and sensory.
- Can be the result of a vascular compromise

Signs and symptoms

- Loss of motor function below the level of the injury
- Loss of pain and temperature below the level of the injury
- Maintenance of light touch, pressure, position, and vibratory sensation

Description: Brown-Séquard syndrome

- Damage to only one side of the cord
- Common with gunshot wounds or stabbing

Signs and symptoms

- Ipsilateral loss of motor function below the level of the injury
- Ipsilateral loss of light touch, pressure, position, and vibratory sensation below the level of the injury
- Contralateral loss of pain and temperature below the level of the injury

- Monitor oxygen saturation levels as ordered. Administer supplemental oxygen as indicated.
- Assess cardiac status frequently, at least every hour initially. Begin cardiac monitoring. Monitor blood pressure and hemodynamic status frequently. If a pulmonary artery catheter is in place, inform the practitioner if there's a decrease in right atrial pressure, pulmonary artery pressure, pulmonary wedge pressure, and systemic vascular resistance that indicates neurogenic shock.

- If your patient becomes hypotensive, prepare to administer vasopressors.
- Prepare the patient for surgical stabilization, if necessary.
- Assess GI status closely for signs of ulceration or bleeding. Anticipate nasogastric (NG) tube insertion and low intermittent suctioning. Assess the abdomen for distention, auscultate bowel sounds, and report any decrease or absence. Be alert for paralytic ileus, which usually occurs 72 hours after injury.
- Monitor intake and output for fluid imbalance.
- Insert an indwelling urinary catheter as ordered.
- Begin measures to prevent skin breakdown due to immobilization, including repositioning, padding, and care of equipment such as halo or traction devices.
- Monitor laboratory and diagnostic test results, including BUN and creatinine levels, CBC, and urine culture (if indicated).
- Monitor the patient for deep vein thrombosis and pulmonary embolism. Apply antiembolism stockings or intermittent sequential compression devices as ordered.
- Provide emotional support to the patient and his family.
- Begin rehabilitation as soon as possible. An obese patient with spinal cord injury may have additional needs. (See *Obesity and acute spinal cord injury*.)

Whew! This is quite a list. Spinal injuries are very serious and require careful assessment and monitoring.



Weighing the evidence

Obesity and acute spinal cord injury

In people with SCI, obesity is commonly cited as one of the major risk factors for the higher prevalence of cardiovascular disease (CVD). Studies on SCI have shown relationships between measures of adiposity and CVD risk factors, such as abnormalities in carbohydrate metabolism and serum lipid levels and hypertension. For example, body mass index (BMI) or upper body obesity is positively correlated with serum triglycerides, insulin, glucose, and C-reactive protein levels.

A recent study using a combination of self-reported and measured heights and weights to determine BMI and a blood pressure level of <120/80 mm Hg as the reference showed that borderline and high blood pressure levels were positively related to being overweight or obese in veterans with SCI. However, data on smoking, a variable related to BMI and blood pressure, were lacking in this study. Findings from these studies on obesity and health outcomes must be interpreted cautiously, keeping in mind that cross-sectional associations don't prove causality.

Source: Rajan, S., McNeely, M. J., Warms, C., & Goldstein, B. (2008). Clinical assessment and management of obesity in individuals with spinal cord injury: A review. *Journal of Spinal Cord Medicine, 31*(4), 361–372.

Arteriovenous malformation

AVMs are tangled masses of thin-walled, dilated blood vessels between arteries and veins that aren't connected by capillaries. Abnormal channels between the arterial and venous system mix oxygenated and unoxygenated blood. This prevents adequate perfusion of brain tissue.

Looking for AVMs

AVMs are common in the brain, especially in the posterior parts of the cerebral hemispheres. They range in size from a few millimeters to large malformations extending from the cerebral cortex to the ventricles. More than one AVM is commonly found.

Males and females are affected equally, and some evidence exists that AVMs occur in families. Most AVMs are present at birth, but symptoms typically occur later, when the person is 10 to 20 years old.

Uh-oh

Complications depend on the severity (location and size) of the AVM and include:

- aneurysm development and subsequent rupture
- hemorrhage (intracerebral, subarachnoid, or subdural, depending on the location of the AVM)
- hydrocephalus.

What causes it

Causes of AVMs may be either:

- congenital—due to a hereditary defect
- acquired—due to penetrating injuries such as trauma.

How it happens

AVMs lack the typical structural characteristics of the blood vessels; the vessels of an AVM are very thin.

Blood pressure, aneurysm, and hemorrhage

One or more arteries feed into the AVM; the typically high-pressured arterial blood flow moves into the venous system through connecting channels. This increases venous pressure, engorging and dilating the venous structures, which may result in the development of an aneurysm.

If the AVM is large enough, the shunting can deprive the surrounding tissue of adequate blood flow. Additionally, the thin-walled vessels may ooze small amounts of blood or rupture, causing hemorrhage into the brain or subarachnoid space.

Most patients with AVM exhibit few, if any, signs or symptoms—unless the AVM is large or it leaks or ruptures.



What to look for

Typically, patients exhibit few, if any, signs and symptoms unless the AVM is large or it leaks or ruptures.

Some signs and symptoms

In some patients, signs and symptoms include:

- chronic mild headache and confusion from AVM dilation, vessel engorgement, and increased pressure
- seizures secondary to compression of the surrounding tissues by the engorged vessels
- systolic bruit over the carotid artery, mastoid process, or orbit, indicating turbulent blood flow
- focal neurologic deficits (depending on the location of the AVM) resulting from compression and diminished perfusion
- symptoms of intracranial (intracerebral, subarachnoid, or subdural) hemorrhage, including sudden severe headache, seizures, confusion, lethargy, and meningeal irritation from bleeding into the brain tissue or subarachnoid space
- hydrocephalus from AVM extension into the ventricular lining.

Loss of blood through thin-walled vessels can lead to rupture and hemorrhage.



What tests tell you

The following tests are used to diagnose AVM:

- Cerebral angiography yields the most definitive diagnostic information. It's used to localize the AVM and allow visualization of large feeding arteries and drainage veins.
- MRI/MRA is used to determine the location and size of AVM.

How it's treated

The choice of treatment depends on the:

- size and location of the AVM
- feeder vessels supplying it
- age and condition of the patient.

Supportive, corrective, or both

Treatment can be supportive, corrective, or both, including:

- support measures, such as aneurysm precautions to prevent possible rupture
- surgery—block dissection, laser, or ligation—to repair the communicating channels and remove the feeding vessels
- embolization or stereotactic radiosurgery if surgery isn't possible, to close the communicating channels and feeder vessels and thus reduce blood flow to the AVM.

What to do

If hemorrhage hasn't occurred in your patient with AVM, focus your efforts on bleeding prevention.

Steps to take

To prevent bleeding, follow these steps to control hypertension and seizure activity:

- Maintain a quiet therapeutic environment.
- Monitor and control associated hypertension with drug therapy as ordered.
- Conduct ongoing neurologic assessments.
- Monitor vital signs frequently.
- Assess and monitor characteristics of headache, seizure activity, or bruit as needed.
- Provide emotional support.

Rupture measures

If the AVM ruptures, work to control elevated ICP and intracranial hemorrhage. Follow the previously described steps as well as those listed here:

- Provide appropriate preoperative teaching.
- After surgery, monitor neurologic status and vital signs frequently.
- Monitor the wound for signs of infection.
- Monitor fluid balance and electrolyte levels.

Listen up! To prevent bleeding, maintain a quiet, therapeutic environment and take the necessary steps to control hypertension and seizure activity.



Cerebral aneurysm

In intracranial or cerebral aneurysm, a weakness in the wall of a cerebral artery causes that area of the artery to dilate or bulge. The most common form is the berry aneurysm, a saclike outpouching in a cerebral artery.

The usual place

Cerebral aneurysms usually arise at an arterial junction in the circle of Willis, the circular anastomosis forming the major cerebral arteries at the base of the brain. Cerebral aneurysms commonly rupture and cause subarachnoid hemorrhage.

The usual patient

The incidence of cerebral aneurysm is slightly higher in women than in men, especially those in their late 40s or early to mid-50s, but a cerebral aneurysm can occur at any age in either sex.

The usual prognosis

The prognosis for any patient with cerebral aneurysm is guarded. About 50% of all patients who suffer a subarachnoid hemorrhage die immediately.

In cerebral aneurysm, blood pressure against a weak arterial wall stretches the vessel like a balloon, making it likely to rupture.





Take charge!

Signs of increased ICP

The earlier you spot signs of increased ICP, the quicker you can intervene and the better your patient's chance of recovery. By the time late signs appear, interventions may be useless.

Assessment area	Early signs	Late signs
LOC	<ul style="list-style-type: none"> • Requires increased stimulation • Subtle orientation loss • Restlessness and anxiety • Sudden quietness 	<ul style="list-style-type: none"> • Unarousable
Pupils	<ul style="list-style-type: none"> • Pupil changes on side of lesion • One pupil constricts but then dilates (unilateral hippus). • Sluggish reaction of both pupils • Unequal pupils 	<ul style="list-style-type: none"> • Pupils fixed and dilated or "blown"
Motor response	<ul style="list-style-type: none"> • Sudden weakness • Motor changes on side opposite the lesion • Positive pronator drift: With palms up, one hand pronates. 	<ul style="list-style-type: none"> • Profound weakness • Decorticate or decerebrate posturing
Vital signs	<ul style="list-style-type: none"> • Intermittent increases in blood pressure 	<ul style="list-style-type: none"> • Increased systolic blood pressure with widening pulse pressure, bradycardia, and abnormal respirations (Cushing's triad)

Of those who survive untreated, 40% die from the effects of hemorrhage and another 20% die later from recurring hemorrhage. New treatments are improving the prognosis.

The major complications of cerebral aneurysm include death from increased ICP and brain herniation, rebleeding, and vasospasm. (See *Signs of increased ICP*.)

What causes it

Causes of cerebral aneurysm include:

- congenital defect
- degenerative process
- combination of congenital defect and degenerative process
- trauma.

How it happens

Blood flow exerts pressure against a weak arterial wall, stretching it like an overblown balloon and making it likely to rupture.

Right after rupture

Rupture causes a subarachnoid hemorrhage, in which blood spills into the space normally occupied by CSF. Sometimes, blood also spills into brain tissue, where a clot can cause potentially fatal increased ICP and brain tissue damage.

What to look for

Occasionally, your patient may exhibit signs and symptoms due to blood oozing into the subarachnoid space. The symptoms, which may persist for several days, include:

- headache
- intermittent nausea
- nuchal rigidity
- stiff back and legs.

Rupture without warning

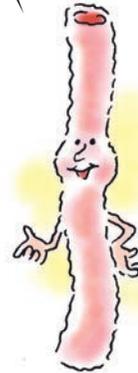
Aneurysm rupture usually occurs abruptly and without warning, causing:

- sudden severe headache caused by increased pressure from bleeding into a closed space
- nausea and projectile vomiting related to increased ICP
- altered LOC, possibly including deep coma, depending on the severity and location of bleeding, due to increased pressure caused by increased cerebral blood volume
- meningeal irritation due to bleeding into the meninges and resulting in nuchal rigidity, back and leg pain, fever, restlessness, irritability, occasional seizures, photophobia, and blurred vision
- hemiparesis, hemisensory defects, dysphagia, and visual defects due to bleeding into the brain tissues
- diplopia, ptosis, dilated pupil, and inability to rotate the eye caused by compression on the oculomotor nerve if the aneurysm is near the internal carotid artery.

Grading severity

Typically, the severity of a ruptured cerebral aneurysm is graded according to the patient's signs and symptoms. (See *Grading cerebral aneurysm rupture*.)

Reports of headache, nausea, and back and leg stiffness lasting several days may signal an impending aneurysm rupture.



A cerebral aneurysm typically ruptures before symptoms are seen. Signs after rupture are sudden and severe.



Grading cerebral aneurysm rupture

The severity of symptoms varies from patient to patient, depending on the site and amount of bleeding. Five grades characterize ruptured cerebral aneurysm:

- *Grade I: minimal bleeding*—The patient is alert, with no neurologic deficit; he may have a slight headache and nuchal rigidity.
- *Grade II: mild bleeding*—The patient is alert, with a mild to severe headache and nuchal rigidity; he may have third nerve palsy.
- *Grade III: moderate bleeding*—The patient is confused or drowsy, with nuchal rigidity and, possibly, a mild focal deficit.
- *Grade IV: severe bleeding*—The patient is stuporous, with nuchal rigidity and, possibly, mild to severe hemiparesis.
- *Grade V: moribund (usually fatal)*—If the rupture is nonfatal, the patient is in a deep coma or decerebrate.

What tests tell you

The following tests help diagnose cerebral aneurysm:

- Cerebral angiography confirms a cerebral aneurysm that isn't ruptured and reveals altered cerebral blood flow, vessel lumen dilation, and differences in arterial filling.
- CT scan, CTA, or MRA reveals evidence of aneurysm and possible hemorrhage.
- Transcranial Doppler sonography is used to detect vasospasm.

How it's treated

Emergency treatment begins with oxygenation and ventilation. Then, to reduce the risk of rebleeding, the doctor may attempt to repair the aneurysm. Surgical repair usually includes clipping, ligating, or wrapping the aneurysm neck with muscle, or embolization.

Conservative whys

The patient may receive conservative treatment when surgical correction poses too much risk, such as when:

- the patient is elderly
- the patient has heart, lung, or other serious disease
- the aneurysm is in a dangerous location
- the vasospasm necessitates a delay in surgery.

Think fast!
Emergency
treatment begins
with oxygenation and
ventilation before the
doctor attempts
aneurysm repair.



Conservative ways

Conservative treatment methods include:

- bed rest in a quiet, darkened room with the head of bed flat or raised less than 30 degrees, which may continue for 4 to 6 weeks
- avoidance of coffee, other stimulants, and aspirin to reduce the risk of rupture and elevation of blood pressure
- possible administration of codeine or another analgesic
- administration of hydralazine (Apresoline) or another antihypertensive, if the patient is hypertensive
- administration of corticosteroids to reduce inflammation
- administration of phenobarbital (Solfoton) or another sedative
- administration of a vasoconstrictor to maintain an optimum blood pressure level (20 to 40 mm Hg above normal), if necessary
- administration of nimodipine to reduce vasospasm.

When surgical correction is too risky for your patient, implement these conservative treatment methods.



What to do

When caring for a patient with an intact cerebral aneurysm, an accurate neurologic assessment, good patient care, patient and family teaching, and psychological support can speed recovery and reduce complications.

Your next step

During the initial treatment after hemorrhage, follow these steps:

- Establish and maintain a patent airway and anticipate the need for supplementary oxygen or mechanical ventilatory support. Monitor arterial blood gas (ABG) levels.
- Position the patient to promote pulmonary drainage and prevent upper airway obstruction. If intubated, preoxygenate with 100% oxygen before suctioning.
- Impose aneurysm precautions (such as bed rest, limited visitors, and avoidance of coffee and physical activity) to minimize the risk of rebleeding and avoid increased ICP.
- Administer a stool softener, as ordered, to prevent straining.
- Monitor LOC and vital signs frequently. Avoid rectal temperatures.
- Determine the CPP. Institute cerebral blood flow monitoring as ordered to determine CPP. If not available, calculate CPP by subtracting the patient's ICP from the MAP (systolic blood pressure plus twice the diastolic blood pressure divided by 3).
- Accurately measure intake and output.

- Be alert for danger signs that may indicate an enlarging aneurysm, rebleeding, intracranial clot, increased ICP, or vasospasm, including decreased LOC, unilateral enlarged pupil, onset or worsening of hemiparesis or motor deficit, increased blood pressure, slowed pulse rate, worsening of headache or sudden onset of a headache, renewed or worsened nuchal rigidity, and renewed or persistent vomiting.
- If the patient develops vasospasm—evidenced by focal motor deficits, increasing confusion, and worsening headache—initiate hypervolemic-hemodilution therapy, as ordered, such as the administration of normal saline, whole blood, packed red blood cells, albumin plasma protein fraction, and crystalloid solution. A calcium channel blocker may reduce smooth muscle spasm and maximize perfusion during spasm. During therapy, assess the patient for fluid overload.
- Turn the patient often, apply antiembolism stockings or intermittent sequential compression devices to the patient's legs, and begin measures to prevent skin breakdown.
- If the patient has facial weakness, assist during meals. If he can't swallow, insert an NG tube, as ordered. Give all tube feedings slowly.
- Prepare the patient for surgery, as appropriate, and provide preoperative teaching if the patient's condition permits.
- Teach the patient and his family about the condition and how to recognize and report signs of rebleeding.

Encephalitis is usually caused by a mosquito-borne virus, but it can be acquired in other ways, too. Think I'll stock up on some new netting as a precaution, though.

Encephalitis

Encephalitis is severe inflammation of the brain, usually caused by a mosquito-borne or, in some areas, a tick-borne virus. Other means of transmission include ingestion of infected goat's milk and accidental injection or inhalation of the virus.

What causes it

Encephalitis results from infection with arboviruses specific to rural areas. In urban areas, it's most commonly caused by enteroviruses (coxsackievirus, poliovirus, and echovirus).

Other causes include herpesvirus, mumps virus, human immunodeficiency virus, adenoviruses, and demyelinating diseases following measles, varicella, rubella, or vaccination.



How it happens

With encephalitis, intense lymphocytic infiltration of brain tissues and the leptomeninges causes cerebral edema, degeneration of the brain's glial cells, and diffuse nerve cell destruction.

What to look for

Watch for the signs and symptoms that signal the beginning of acute illness, including:

- fever (102° to 105° F [38.9° to 40.6° C])
- headache
- vomiting.

Negative progression

The illness can progress to include signs and symptoms of meningeal irritation, such as stiff neck and back. Be alert for signs of neuron damage, such as:

- drowsiness
- coma
- paralysis
- seizures
- ataxia
- organic psychoses.

What tests tell you

These tests help diagnose encephalitis:

- CSF or blood analysis used to identify the causative virus confirms the diagnosis.
- Technetium-99 scan results may show localized abnormalities.
- CT scan may disclose localized abnormalities.

How it's treated

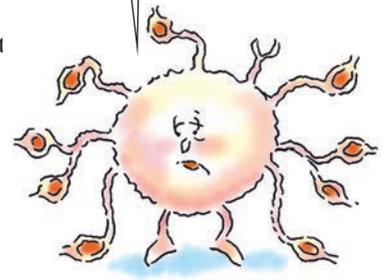
Most of the treatments for patients with encephalitis are entirely supportive:

- The antiviral agent acyclovir (Zovirax) is effective in treatment herpes encephalitis.
- Anticonvulsants to prevent or control seizures
- Furosemide (Lasix) or mannitol reduces cerebral swelling.
- Sedatives are given to alleviate restlessness.
- Aspirin (Ecotrin) or acetaminophen (Tylenol) relieves headache and reduces fever.

When a patient presents with fever, headache, vomiting, and stiff neck and back, suspect encephalitis.



Stay alert for changes! Symptoms can escalate to include drowsiness, coma, paralysis, and other signs of neuron damage.



- Fluids and electrolytes prevent dehydration and electrolyte imbalance.
- Antibiotics are used to fight an associated infection such as pneumonia.

What to do

During the acute phase of the illness, follow these guidelines:

- Assess neurologic function frequently. Check for changes in LOC and signs of increased ICP. Watch for signs and symptoms of cranial nerve involvement, such as ptosis, strabismus, diplopia, abnormal sleep patterns, and behavior changes.
- Monitor intake and output carefully to maintain fluid balance. Be aware that fluid overload can increase cerebral edema.
- Position the patient carefully and turn him often to prevent joint stiffness and neck pain.
- Perform ROM exercises to prevent contractures.
- Provide a quiet, darkened room to ease headache and photophobia.
- Maintain adequate nutrition by giving small, frequent meals and NG tube or parenteral feedings, as ordered.
- Reassure the patient and his family that behavior changes caused by encephalitis usually disappear.
- If the patient is disoriented or confused, attempt to reorient him frequently. Place a calendar or clock in the patient's room to aid in orientation.

If the patient with encephalitis is disoriented, place a calendar or clock in the room.



Guillain-Barré syndrome

Guillain-Barré syndrome, or acute idiopathic polyneuritis, is also known as *infectious polyneuritis*. It's an acute, rapidly progressive, and potentially fatal form of polyneuritis that causes muscle weakness and mild distal sensory loss.

Equal opportunity syndrome

This syndrome can occur at any age but is most common in people between ages 30 and 50. It affects both sexes equally.

Recovery is spontaneous and complete in about 95% of patients; however, mild motor or reflex deficits may persist in the feet and legs. The prognosis is best when symptoms resolve sooner than 15 to 20 days after onset.

Three-phase syndrome

Guillain-Barré syndrome occurs in three phases:

1. The acute phase begins with the onset of the first definitive symptom and ends 1 to 3 weeks later. Further deterioration doesn't occur after the acute phase.
2. The plateau phase lasts several days to 2 weeks.
3. The recovery phase coincides with remyelination and regrowth of axonal processes. Recovery commonly takes 4 to 6 months but may take as long as 2 to 3 years in severe cases.

In a severe case, the patient with Guillain-Barré syndrome may take up to 3 years to recover.



Commonly complicated syndrome

Common complications include thrombophlebitis, pressure ulcers, muscle wasting, sepsis, joint contractures, respiratory tract infections, respiratory failure, and loss of bladder and bowel control.

What causes it

The precise cause of Guillain-Barré syndrome isn't known, but it may be a cell-mediated immune response to a virus. About 50% of patients with Guillain-Barré syndrome have a recent history of minor febrile illness, usually an upper respiratory tract infection or, less commonly, gastroenteritis. When infection precedes the onset of Guillain-Barré syndrome, signs of infection subside before neurologic features appear.

Possible precipitators

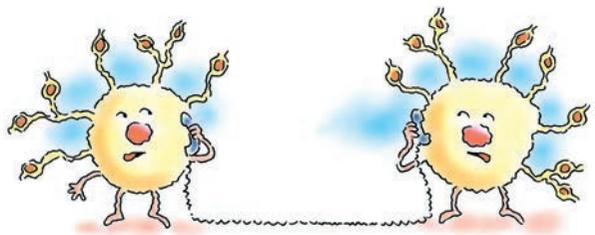
Other possible precipitating factors include:

- surgery
- rabies or swine influenza vaccination
- Hodgkin's or other malignant disease
- systemic lupus erythematosus.

How it happens

The major pathologic feature of Guillain-Barré syndrome is segmental demyelination of the peripheral nerves, which prevents the normal transmission of electrical impulses along the sensorimotor nerve roots.

My sensorimotor nerve roots are fried . . . nothing's getting through! Hello . . . ?



Double trouble

Guillain-Barré syndrome causes inflammation and degenerative changes in both posterior (sensory) and the anterior (motor) nerve roots. That's why

signs of sensory and motor losses occur simultaneously. Additionally, autonomic nerve transmission may be impaired.

What to look for

During your assessment, look for symptoms that are progressive and include:

- symmetrical muscle weakness (the major neurologic sign), appearing first in the legs (in the ascending type of the syndrome, which is the most common form) and then extending to the arms and facial nerves within 24 to 72 hours, due to impaired anterior nerve root transmission
- muscle weakness developing in the brain stem, in the cranial nerves, and progressing downward in the arms first (in the descending type of the syndrome) or in the arms and legs simultaneously, due to impaired anterior nerve root transmission
- normal muscle strength (in mild forms of the syndrome) or weakness affecting only the cranial nerves
- paresthesia, sometimes preceding muscle weakness but vanishing quickly, due to impairment of the dorsal nerve root transmission
- diplegia, possibly with ophthalmoplegia (ocular paralysis), from impaired motor nerve root transmission and involvement of cranial nerves III, IV, and VI
- dysphagia or dysarthria and, less commonly, weakness of the muscles supplied by CN XI (the spinal accessory nerve)
- hypotonia and areflexia from interruption of the reflex arc.

What tests tell you

These tests help diagnose Guillain-Barré syndrome:

- CSF analysis reveals protein levels that begin to increase several days after the onset of symptoms and peak in 4 to 6 weeks.
- CBC early in illness shows leukocytosis and immature forms of white blood cells (WBCs; immature neutrophils, called *bands*).
- Electromyography may show repeated firing of the same motor unit instead of widespread sectional stimulation.
- Nerve conduction velocities slow soon after paralysis develops.
- Serum immunoglobulin levels are elevated due to an inflammatory response.

How it's treated

- Treatments are primarily supportive and include endotracheal (ET) intubation or tracheotomy if respiratory muscle involvement causes difficulty in clearing secretions.
- A trial dose (7 days) of prednisone (Deltasone) is given to reduce inflammatory response if the disease is relentlessly progressive; if prednisone produces no noticeable improvement, the drug is discontinued.
- Plasmapheresis is useful during the initial phase but of no benefit if started 2 weeks after onset.
- Continuous ECG is used to monitor for possible arrhythmias due to autonomic dysfunction. Propranolol (Inderal) is used to reduce tachycardia and hypertension. Atropine is given for bradycardia. Volume replacement is used in treating patients with severe hypotension.

Stand by to provide supportive treatments, such as ET intubation or tracheotomy.



What to do

When caring for a patient with Guillain-Barré syndrome:

- Watch for ascending sensory loss, which precedes motor loss.
- Monitor vital signs and LOC.
- Assess and treat patients with respiratory dysfunction.
- Auscultate breath sounds, turn and position the patient, and encourage coughing and deep breathing. Begin respiratory support at the first sign of respiratory failure, which may include ET intubation and mechanical ventilation.
- Provide meticulous skin care to prevent skin breakdown.
- Perform passive ROM exercises within the patient's pain limits.
- To prevent aspiration, test the gag reflex and elevate the head of the bed before the patient eats. If the gag reflex is absent, give NG tube feedings until the gag reflex returns.
- As the patient regains strength and can tolerate a vertical position, be alert for hypotension. Change the patient's position slowly.
- Apply antiembolism stockings and a sequential compression device to the legs.
- If the patient has facial paralysis, provide eye and mouth care every 4 hours.
- Watch for urine retention. Use an indwelling urinary catheter if necessary.
- To prevent constipation, provide a high-fiber diet and offer prune juice. Administer a suppository or bisacodyl (Fleet enema), as ordered.
- Refer the patient to physical therapy.

Head injury

Head injury is any traumatic insult to the brain that causes physical, intellectual, emotional, social, or vocational changes. Children ages 6 months to 2 years, adults ages 15 to 24, and elderly adults are most at risk for head injury.

It's no surprise that children are particularly at-risk for head injury.

To put it bluntly

Head injury is generally categorized as closed trauma or open trauma. Closed (or blunt) trauma is more common. It typically occurs when the head strikes a hard surface or a rapidly moving object strikes the head. The dura mater is intact, and no brain tissue is exposed to the external environment.

In open trauma, as the name suggests, an opening in the scalp, skull, meninges, or brain tissue (the dura mater) exposes the cranial contents to the environment. The risk of infection is high.



Complications are possible

Possible complications include:

- increased ICP
- infection (in open trauma)
- respiratory depression and failure
- brain herniation.

On the decline

Mortality from head injury has declined as a result of:

- advances in preventive measures, such as air bags, seat belts, and helmets
- quicker emergency response and transport times
- improved treatment measures.

Think safety.
Always wear your helmet on the field.

What causes it

Head injury commonly results from:

- motor vehicle collisions (the number one cause)
- falls
- sports-related accidents
- assaults and other crimes.

How it happens

The patient's brain is shielded by the cranial vault (composed of skin, bone, meninges, and CSF), which intercepts the force of a physical blow. Below a certain level of force, the cranial vault prevents energy from affecting the brain.



The degree of traumatic head injury is usually proportional to the amount of force reaching the cranial tissues. In addition, unless it's ruled out, you may presume that neck injuries are present in patients with traumatic head injuries.

Case closed

Closed trauma is typically a sudden acceleration–deceleration or coup–contrecoup injury. In coup–contrecoup injury, the head hits a more stationary object, injuring cranial tissues near the point of impact (coup); the remaining force then pushes the brain against the opposite side of the skull, causing a second impact and injury (contrecoup).

Contusions and lacerations may also occur during contrecoup as the brain's soft tissues slide over the rough bone of the cranial cavity. The cerebrum may endure rotational shear, damaging the upper mid-brain and areas of the frontal, temporal, and occipital lobes.

What to look for

Types of head trauma include:

- concussion
- contusion
- epidural hematoma
- subdural hematoma
- intracerebral hematoma
- skull fractures.

Each type is associated with specific signs and symptoms. (See *Types of head injury*, pages 116 to 119.) Signs and symptoms of head trauma in elderly patients may not be readily apparent. (See *Hidden hematoma*.)

What tests tell you

These tests help diagnose head injury:

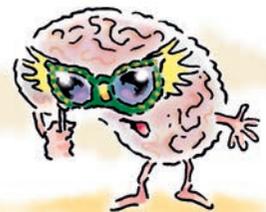
- Skull X-rays show the location of the fracture, unless the cranial vault is fractured. (A CT scan will show a fracture of the cranial vault.)
- CTA shows the location of vascular disruption due to internal pressure or injuries that result from cerebral contusion or skull fracture.
- CT scan shows intracranial hemorrhage from ruptured blood vessels; ischemic or necrotic tissue; cerebral edema; a shift in brain tissue; and subdural, epidural, and intracerebral hematomas.
- MRI may show intracranial hemorrhage, edema, or neuronal damage not seen on CT such as diffuse axonal injury.



Hidden hematoma

An older person with cerebral atrophy can tolerate a larger subdural hematoma for a longer time than a younger person can before the hematoma causes neurologic changes. That's why a hematoma in an older patient can become rather large before you see any symptoms, even in an acute condition.

Remember, signs and symptoms of head trauma in elderly patients may not be obvious at first. They often take longer to develop than in a younger person.



How it's treated

Treatment may be surgical or supportive.

It's surgical

Surgical treatment includes:

- evacuation of a hematoma
- elevation of a depressed skull fracture
- decompressive craniotomy.

The goal of surgery is reduce pressure on the brain, debride damaged tissue, and restore the cranial vault.

It's supportive

Provide supportive treatment, which includes:

- close observation to detect changes in neurologic status suggesting further damage or expanding hematoma
- cleaning and debridement of any wounds associated with skull fractures
- diuretics such as mannitol to reduce cerebral edema
- analgesics such as acetaminophen to relieve complaints of headache
- anticonvulsants such as phenytoin (Dilantin) or fosphenytoin (Cerebyx) to prevent seizures
- respiratory support, including mechanical ventilation and ET intubation for patients with respiratory failure from brain stem involvement
- only contaminated open fractures require antibiotics.

What to do

- Initially monitor vital signs continuously and check for additional injuries.
- Continue to check vital signs and neurologic status, including LOC and pupil size, every 15 minutes.
- Maintain a patent airway. Monitor oxygen saturation levels through pulse oximetry and ABG analysis as ordered.
- Assess hemodynamic parameters to aid in evaluating CPP.
- Administer medications as ordered. If necessary, use continuous infusions of such agents as midazolam (Versed), fentanyl (Sublimaze), morphine, or propofol (Diprivan) to reduce metabolic demand and the risk for increased ICP.
- Observe the patient closely for signs of hypoxia or increased ICP, such as headache, dizziness, irritability, anxiety, and changes in behavior such as agitation.

Check skull X-rays for the location of a fracture.



Surgical intervention may be needed to reduce a patient's risk for infection and further brain damage due to fractures.



(Text continues on page 120.)

Types of head injury

Here's a summary of the signs and symptoms and diagnostic test findings for different types of head injury.

Type	Description
Concussion (closed head injury)	<ul style="list-style-type: none"> • Characterized as a blow to the head hard enough to make the brain hit the skull but not hard enough to cause a cerebral contusion; causes temporary neural dysfunction • Recovery is usually complete within 24 to 48 hours. • Repeated injuries have a cumulative effect on the brain.
Epidural hematoma (bleeding above the dura mater)	<ul style="list-style-type: none"> • It's most common in 20- to 40-year-olds. • Most result from arterial bleeding. • Blood commonly accumulates between skull and dura mater. Injury to middle meningeal artery in parietotemporal area is most common and is typically accompanied by linear skull fractures in temporal region over middle meningeal artery. • It less commonly arises from dural venous sinuses.
Contusion	<ul style="list-style-type: none"> • Acceleration–deceleration or coup–contrecoup injuries disrupt normal nerve functions in bruised area. • Injury is directly beneath the site of impact when the brain rebounds against the skull from the force of a blow (e.g., a beating with a blunt instrument), when the force of the blow drives the brain against the opposite side of the skull, or when the head is hurled forward and stopped abruptly (as in an automobile crash when a driver's head strikes the windshield). • Brain continues moving and slaps against the skull (acceleration), then rebounds (deceleration). Brain may strike bony prominences inside the skull (especially the sphenoidal ridges), causing intracranial hemorrhage or hematoma that may result in tentorial herniation.

Signs and symptoms

- May occur without loss of consciousness. A short-term loss of consciousness is secondary to disruption of reticular activating system, possibly due to abrupt pressure changes in the areas responsible for consciousness, changes in polarity of the neurons, ischemia, or structural distortion of neurons.
 - Anterograde and retrograde amnesia (patient can't recall events immediately after the injury or events that led up to the traumatic incident) correlating with severity of injury; all related to disruption of reticular activating system
 - Irritability or lethargy
 - Behavior out of character
 - Complaints of dizziness, nausea, or severe headache
-
- Severe scalp wounds from direct injury
 - Brief period of unconsciousness after injury reflecting the concussive effects of head trauma, followed by a lucid interval varying from 10 to 15 minutes to hours or, rarely, days
 - Labored respiration and loss of consciousness secondary to increased pressure from bruising
 - Hemiparesis related to interrupted blood flow to the site of injury
 - Decorticate or decerebrate posturing from cortical damage or hemispheric dysfunction r/t increased ICP
 - Unequal pupillary response from brain stem involvement—third cranial nerve
-
- Drowsiness, confusion, disorientation, agitation, or violence from increased ICP associated with trauma
 - Severe headache
 - Edema forms around the contusion increasing the ICP, which can lead to decreased LOC, worsening of neurologic deficit—depending on the location of the contusion—may include.
 - Respirations, initially deep and labored, becoming shallow and irregular as brain stem is impacted
 - Contralateral motor deficits reflecting compression of corticospinal tracts that pass through the brain stem
 - Ipsilateral (same-side) pupillary dilation due to compression of third cranial nerve
 - Seizures possibly from high ICP
 - Continued bleeding leading to progressive neurologic degeneration (evidenced by bilateral pupillary dilation, bilateral decerebrate response, increased systemic blood pressure, decreased pulse, and profound coma with irregular respiratory patterns)

Diagnostic test findings

- CT scan or MRI reveals no sign of fracture, bleeding, or other nervous system lesion.
-
- CT scan shows changes in tissue density, possible displacement of the surrounding structures, and evidence of ischemic tissue, hematomas, and fractures.
 - EEG recordings directly over the area of contusion reveal progressive abnormalities by appearance of high-amplitude theta and delta waves.
-
- CT scan or MRI identifies abnormal masses or structural shifts within the cranium.

(continued)

Types of head injury *(continued)*

Type	Description
Subdural hematoma	<ul style="list-style-type: none">• Meningeal hemorrhage results from accumulation of blood in subdural space (between dura mater and arachnoid mater).• It may be acute, subacute, and chronic: unilateral or bilateral.• It's usually associated with torn connecting veins in cerebral cortex; rarely from arteries.• Large acute hematomas are a surgical emergency.• Subacute hematomas have better prognosis because they occur over a longer period of time.
Intracerebral hematoma	<ul style="list-style-type: none">• Traumatic or spontaneous disruption of cerebral vessels in brain parenchyma cause neurologic deficits, depending on site and amount of bleeding.• Shear forces from brain movement frequently cause vessel laceration and hemorrhage into the parenchyma.• Frontal and temporal lobes are common sites. Trauma is associated with few intracerebral hematomas; most are caused by hypertension.
Skull fracture	<ul style="list-style-type: none">• There are four types: linear, comminuted, depressed, and basilar.• Fractures of anterior and middle fossae are associated with severe head trauma and are more common than those of posterior fossa.• A blow to the head causes one or more of the types. It may not be problematic unless the brain is exposed or bone fragments are driven into neural tissue.

Signs and symptoms

- Similar to epidural hematoma but significantly slower in onset because bleeding is typically of venous origin

- Headache
- May have loss of consciousness—depends on size of hematoma
- Possible motor deficits and decorticate or decerebrate responses from compression of corticospinal tracts and brain stem

- May not produce symptoms, depending on underlying brain trauma
- Discontinuity and displacement of bone structure with severe fracture
- Motor sensory and cranial nerve dysfunction with associated facial fractures
- May cause periorbital ecchymosis (raccoon eyes), anosmia (loss of smell due to first cranial nerve involvement), and pupil abnormalities (second and third cranial nerve involvement) in those with anterior fossa basilar skull fractures
- CSF rhinorrhea (leakage through nose), CSF otorrhea (leakage from the ear), hemotympanum (blood accumulation at the tympanic membrane), ecchymosis over the mastoid bone (Battle's sign), and facial paralysis (seventh cranial nerve injury) in those with middle fossa basilar skull fractures
- Signs of medullary dysfunction such as cardiovascular and respiratory failure in those with posterior fossa basilar skull fractures

Diagnostic test findings

- CT scan, confirming a hematoma
- CT scan or MRI reveals evidence of masses and tissue shifting.
- CT scan or MRI /MRA Identify the hemorrhage

- CT scan and MRI reveal swelling and intracranial hemorrhage from ruptured blood vessels.
- Skull X-ray may reveal a fracture.

- Monitor elderly patients especially closely because they may have brain atrophy and, therefore, more space for cerebral edema. This means ICP may increase without showing signs.
- If an ICP monitoring system is inserted, continuously monitor ICP waveforms and pressure.
- Carefully observe the patient for CSF leakage. Check the bed sheets for a blood-tinged spot surrounded by a lighter ring (halo sign). If the patient has CSF leakage or is unconscious, elevate the head of the bed 30 degrees.
- Position the patient so that secretions drain properly. If you detect CSF leakage from the nose, place a gauze pad under the nostrils. Don't suction through the nose but use the mouth. If CSF leaks from the ear, position the patient so his ear drains naturally.
- Monitor intake and output frequently to maintain fluid balance.
- Institute seizure precautions as necessary. Use safety precautions to minimize the risk of injury.
- Cluster nursing activities to provide rest periods, thus reducing metabolic demands and reducing the risk of sustained increases in ICP.
- Prepare the patient for craniotomy as indicated.
- After the patient is stabilized, clean and dress superficial scalp wounds using strict sterile technique. Monitor wounds for signs and symptoms of infection.
- Explain all procedures and treatments to the patient and his family.

Meningitis

In meningitis, the brain and the spinal cord meninges become inflamed, usually because of bacterial infection. Such inflammation may involve all three meningeal membranes—the dura mater, arachnoid mater, and pia mater.

Promptness improves prognosis

If meningitis is recognized early and the infecting organism responds to treatment, the prognosis is good. Complications are rare and may include increased ICP, hydrocephalus, cerebral infarction, cranial nerve deficits causing optic neuritis and deafness, brain abscess, seizures, or coma.

Time matters.
Prompt recognition
and treatment
improves the
meningitis patient's
prognosis.



What causes it

Meningitis is usually a complication of bacteremia, especially from pneumonia, empyema, osteomyelitis, or endocarditis. Aseptic meningitis may result from a virus or other organism. Sometimes no causative organism can be found.

Uh-oh, other infections

Other infections associated with meningitis include:

- sinusitis
- otitis media
- encephalitis
- myelitis
- brain abscess, usually caused by *Neisseria meningitidis*, *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Escherichia coli*.

Any opening

Meningitis may follow trauma or invasive procedures, including skull fracture, penetrating head wound, lumbar puncture, and ventricular shunting.

How it happens

Meningitis commonly begins as inflammation of the pia–arachnoid tissue. It may progress to congestion of adjacent tissues and destroy some nerve cells.

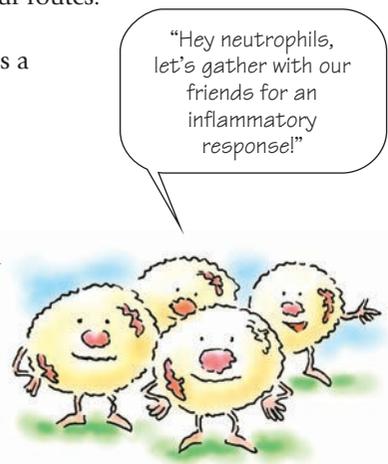
It enters here . . .

The causative organism typically enters the CNS by one of four routes:

1. the blood (most common)
2. a direct opening between the CSF and the environment as a result of trauma
3. along the cranial and peripheral nerves
4. through the mouth or nose.

. . . and triggers a response . . .

The invading organism triggers an inflammatory response in the meninges. To ward off the invasion, neutrophils gather in the area and produce an exudate in the subarachnoid space, causing the CSF to thicken. The thickened CSF flows less readily around the brain and spinal cord. This can block the arachnoid villi, further obstructing CSF flow and causing hydrocephalus.



... and yet more responses

The exudate also:

- exacerbates the inflammatory response, increasing the pressure in the brain
- can extend to the cranial and peripheral nerves, triggering additional inflammation
- irritates the meninges, disrupting their cell membranes, and causing edema.

Truth or consequences

The consequences of meningitis are:

- elevated ICP
- engorged blood vessels
- disrupted cerebral blood supply
- possible thrombosis or rupture
- cerebral infarction if ICP isn't reduced
- possible encephalitis (a secondary infection of the brain tissue).

In aseptic meningitis, lymphocytes infiltrate the pia–arachnoid layers but usually not as severely as in bacterial meningitis; no exudate is formed. Thus, this type of meningitis is self-limiting.

What to look for

Look for the signs of meningitis, which typically include:

- fever, chills, and malaise resulting from infection and inflammation
- headache, vomiting and, rarely, papilledema (inflammation and edema of the optic nerve) from increased ICP.

Bacterial meningitis (meningococcal) has a characteristic petechial rash.

Signs of irritation

Signs of meningeal irritation include:

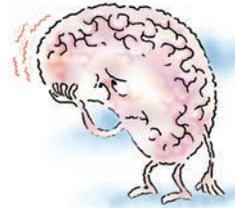
- nuchal rigidity
- positive Brudzinski's and Kernig's signs
- exaggerated and symmetrical deep tendon reflexes
- opisthotonos (a spasm more common in infants and children in which the back and extremities arch backward so that the body rests on the head and heels).

Further features

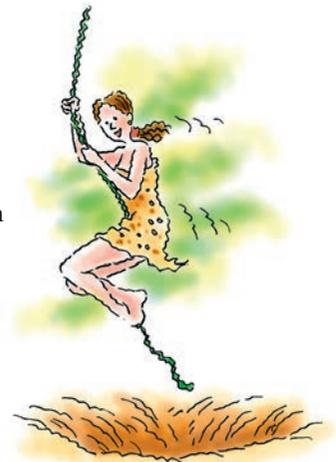
Other features of meningitis may include:

- sinus arrhythmias due to irritation of autonomic nerves
- irritability due to increasing ICP
- photophobia, diplopia, and other visual problems due to cranial nerve irritation

Thickened CSF flows slowly around the brain and spinal cord, sometimes causing hydrocephalus.



Fever is one of the classic signs of meningitis. Others include chills, malaise, headache, and, in some cases, papilledema.



- delirium, deep stupor, and coma due to increased ICP and cerebral edema.

What tests tell you

- Lumbar puncture shows elevated CSF pressure (from obstructed CSF outflow at the arachnoid villi), cloudy or milky-white CSF, high protein levels, positive Gram stain and culture (unless a virus is responsible), presence of enterovirus by Xpert EV test, and decreased glucose concentration.
- Positive Brudzinksi's and Kernig's signs indicate meningeal irritation.
- Cultures of blood, urine, and nose and throat secretions reveal the offending organism.
- Chest X-ray may reveal pneumonitis or lung abscess, tubercular lesions, or granulomas secondary to a fungal infection.
- Sinus and skull X-rays may identify paranasal sinusitis as the underlying infectious process or a skull fracture as the mechanism for entrance of microorganisms.
- WBC count reveals leukocytosis.

Various specimens will be collected and tests ordered to confirm the diagnosis and identify a causative organism.



How it's treated

Treatment includes administration of:

- antibiotic therapy, usually for 2 weeks
- digoxin (Lanoxin) to control arrhythmia
- mannitol to decrease cerebral edema
- anticonvulsant to prevent seizures
- sedative to reduce restlessness
- acetaminophen to relieve headache and fever.

Supportive measures

Supportive measures include bed rest; fever reduction, which may include tepid baths or cooling the patient with a hyperthermia-hypothermia blanket; and isolation, if necessary.

What to do

Take the following steps when caring for a patient with meningitis:

- Assess neurologic function often.
- Watch for deterioration, especially temperature increase, deteriorating LOC, onset of seizures, and altered respirations.
- Monitor fluid balance. Maintain adequate fluid intake to avoid dehydration but avoid fluid overload because of the danger of cerebral edema.
- Position the patient to prevent joint stiffness and neck pain. Assist with ROM exercises.
- Maintain adequate nutrition and elimination.
- Maintain a quiet environment.

- Follow strict sterile technique when treating patients with head wounds or skull fractures.
- Provide emotional support.
- Administer medications as ordered.

Seizure disorder

Seizure disorder, or epilepsy, is a condition of the brain characterized by recurrent seizures (paroxysmal events associated with abnormal electrical discharges of neurons in the brain).

Primary and secondary

Primary seizure disorder or epilepsy is idiopathic without apparent structural changes in the brain.

Secondary epilepsy, characterized by structural changes or metabolic alterations of the neuronal membranes, causes increased automaticity.

Who's affected . . .

Epilepsy affects 1% to 2% of the population; approximately 2 million people live with epilepsy. The incidence is highest in childhood and old age. The prognosis is good if the patient adheres strictly to the prescribed treatment.

. . . and how

Complications of epilepsy may include hypoxia or anoxia due to airway occlusion, traumatic injury, brain damage, and depression and anxiety.

What causes it

In about one-half of seizure disorder cases, the cause is unknown. Some possible causes are:

- birth trauma (such as inadequate oxygen supply to the brain, blood incompatibility, or hemorrhage)
- perinatal infection
- anoxia
- infectious diseases (meningitis, encephalitis, or brain abscess)
- head injury or trauma.

How it happens

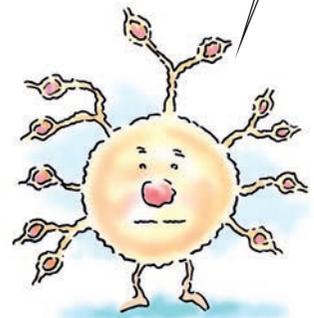
Some neurons in the brain may depolarize easily or be hyperexcitable, firing more readily than normal when stimulated. On stimulation, the electrical current spreads to surrounding cells, which fire in turn. The impulse thus cascades to:

- one side of the brain (a partial seizure)
- both sides of the brain (a generalized seizure)
- cortical, subcortical, and brain stem areas.

Remember:
Meningitis is usually due to infection, so use sterile technique for patients with a head or skull injury.



Don't be fooled . . . without the right treatment, I'm prone to recurrent seizures and possible hypoxia, traumatic injury, and even brain damage.



Increase O₂, or else

The brain's metabolic demand for oxygen increases dramatically during a seizure. If this demand isn't met, hypoxia and brain damage result.

Firing of inhibitory neurons causes the excited neurons to slow their firing and eventually stop. Without this inhibitory action, the result is status epilepticus (seizures occurring one right after another). Without treatment, resulting anoxia is fatal.

What to look for

The hallmarks of seizure disorders are recurring seizures, which can be classified as partial or generalized. Some patients are affected by more than one type. (See *Types of seizures*, page 126.)

What tests tell you

The results of primary diagnostic tests for seizure disorders may include the following:

- CT scan to show density readings of the brain may indicate abnormalities in internal structures.
- MRI may indicate abnormalities in internal brain structures.
- EEG is used to confirm the diagnosis of epilepsy by documenting changes in the brain's electrical conduction.
- Long-term or continuous EEG monitoring can be used to confirm seizure spikes.
- PET scan can help locate the focus of the seizures.

How it's treated

Generally, treatment consists of drug therapy specific to the type of seizures. The goal is to reduce seizures using a combination of the fewest drugs.

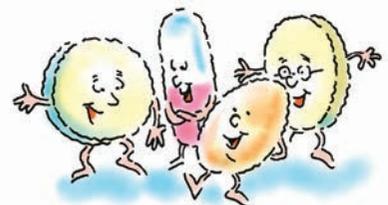
For tonic-clonic seizures

The most commonly prescribed drugs for generalized tonic-clonic seizures (alternating episodes of muscle spasm and relaxation) include phenytoin, carbamazepine (Tegretol), phenobarbital, and primidone (Mysoline).

For absence seizures

Drugs commonly prescribed for absence seizures (brief changes in LOC) include valproic acid (Depakene), clonazepam (Klonopin), and ethosuximide (Zarontin).

"The fewer the better. The goal is to reduce seizures using the fewest of us as possible!"



Types of seizures

Use the guidelines below to understand different seizure types. Keep in mind that some patients may be affected by more than one type.

Partial seizures

Partial seizure activity arising from a localized area in the brain may spread to the entire brain, causing a generalized seizure. There are several types and subtypes of partial seizures:

- simple partial seizures, which include jacksonian and sensory seizures
- complex partial seizures
- secondarily generalized partial seizures (partial onset leading to generalized tonic-clonic seizure).

Jacksonian seizures

A jacksonian seizure begins as a localized motor seizure, characterized by a spread of abnormal activity to adjacent areas of the brain.

The patient experiences stiffening or jerking in one extremity, with a tingling sensation in the same area. The patient seldom loses consciousness, but the seizure may progress to a generalized tonic-clonic seizure.

Sensory seizure

Symptoms of a sensory seizure include hallucinations, flashing lights, tingling sensations, vertigo, *déjà vu*, and smelling a foul odor.

Complex partial seizure

Signs and symptoms of a complex partial seizure are variable but usually include purposeless behavior, including a glassy stare, picking at clothes, aimless wandering, lip-smacking or chewing motions, and unintelligible speech.

An aura may occur first, and seizures may last a few seconds to

20 minutes. Afterward, mental confusion may last for several minutes and may be mistaken for alcohol or drug intoxication or psychosis. The patient has no memory of his actions during the seizure.

Secondarily generalized partial seizure

A secondarily generalized partial seizure can be simple or complex and can progress to a generalized seizure. An aura may occur first, with loss of concentration immediately or 1 to 2 minutes later.

Generalized seizure

Generalized seizures cause a generalized electrical abnormality in the brain. Types include absence, myoclonic, clonic, tonic, generalized tonic-clonic, and atonic.

Absence seizure

Absence seizure, also known as *petit mal seizure*, is most common in children. It usually begins with a brief change in the LOC, signaled by blinking or rolling of the eyes, a blank stare, and slight mouth movements. The patient retains his posture and continues pre-seizure activity without difficulty.

Such seizures last 1 to 10 seconds, and impairment is so brief that the patient may be unaware of it. If not properly treated, seizures can recur up to 100 times a day and progress to a generalized tonic-clonic seizure.

Myoclonic seizure

Myoclonic seizure is marked by brief, involuntary muscle jerks of the body

or extremities and typically occurs in early morning.

Clonic seizure

Clonic seizure is characterized by bilateral rhythmic movements.

Tonic seizure

Tonic seizure is characterized by a sudden stiffening of muscle tone, usually of the arms but may also include the legs.

Generalized tonic-clonic seizure

Typically, a generalized tonic-clonic seizure begins with a loud cry, caused by air rushing from the lungs and through the vocal cords. The patient falls to the ground, losing consciousness. The body stiffens (tonic phase) and then alternates between episodes of muscle spasm and relaxation (clonic phase). Tongue biting, incontinence, labored breathing, apnea, and cyanosis may also occur.

The seizure stops in 2 to 5 minutes, when abnormal electrical conduction of the neurons is completed. Afterward, the patient regains consciousness but is somewhat confused. He may have difficulty talking and may have drowsiness, fatigue, headache, muscle soreness, and arm or leg weakness. He may fall into a deep sleep afterward.

Atonic seizure

Atonic seizure is characterized by a general loss of postural tone and temporary loss of consciousness. It occurs in children and is sometimes called a *drop attack* because the child falls.



Take charge!

Understanding status epilepticus

Status epilepticus is a continuous seizure state that must be interrupted by emergency measures. It can occur during all types of seizures. For example, generalized tonic-clonic status epilepticus is a continuous generalized tonic-clonic seizure without an intervening return of consciousness.

Always an emergency

Status epilepticus is accompanied by respiratory distress. It can result from withdrawal of antiepileptic medications, hypoxic or metabolic encephalopathy, acute head trauma, or septicemia secondary to encephalitis or meningitis.

Act fast

Emergency treatment usually consists of diazepam, phenytoin, or phenobarbital; I.V. dextrose 50% when seizures are secondary to hypoglycemia; and I.V. thiamine in patients with chronic alcoholism or those who are undergoing withdrawal.

Surgery and emergencies

Emergency treatment for patients with status epilepticus usually includes:

- administration of diazepam (Valium), lorazepam (Ativan), fosphenytoin (Cerebyx), or phenobarbital
- 50% dextrose I.V. (when seizures are secondary to hypoglycemia)
- thiamine I.V. (in chronic alcoholism or withdrawal). (See *Understanding status epilepticus*.)

A nonpharmacologic approach for managing seizures is vagus nerve stimulation. The vagus nerve stimulation device acts on the brain the way a pacemaker acts on the heart. It sends electrical signals to the brain to inhibit seizure activity.

Because the device is implanted in the chest and neck, adverse effects include voice changes, throat discomfort, and shortness of breath, all of which usually occur when the device is turned on.

What to do

- Monitor a patient taking anticonvulsants constantly for signs of toxicity, such as nystagmus, ataxia, lethargy, dizziness, drowsiness, slurred speech, irritability, nausea, and vomiting.
- When administering fosphenytoin I.V., use a large vein, administer according to guidelines (not more than 150 mg/minute), and monitor vital signs often.

Did you know that the vagus nerve stimulation device is like a pacemaker for the brain? It sends electrical signals to me to inhibit seizure activity.



- Encourage the patient and his family to express their feelings about the patient's condition.
- Stress the need for compliance with the prescribed drug schedule.
- Emphasize the importance of having blood levels of anticonvulsants checked at regular intervals.

Tonic-clonic interventions

Generalized tonic-clonic seizures may necessitate the following interventions:

- Avoid restraining the patient during a seizure.
- Help the patient to a lying position, loosen any tight clothing, and place something flat and soft, such as a pillow, under his head.
- Clear the area of hard objects.
- Don't force anything into the patient's mouth if his teeth are clenched.
- Turn the patient's head or turn him on his side to provide an open airway.
- After the seizure, reassure the patient that he's all right, orient him to time and place, and tell him that he had a seizure.

The key during a generalized tonic-clonic seizure is to take necessary precautions to prevent injury and maintain a patent airway.



Stroke

Stroke, also known as a *cerebrovascular accident* or *brain attack*, is a sudden impairment of cerebral circulation in one or more blood vessels. A stroke interrupts or diminishes oxygen supply and commonly causes serious damage or necrosis in the brain tissues.

The sooner, the better

The sooner circulation returns to normal after a stroke, the better your patient's chances are for a complete recovery. However, about one-half of the patients who survive a stroke remain permanently disabled and experience a recurrence within weeks, months, or years. It's the leading cause of admission to long-term care.

Numbers and odds

Stroke is the third most common cause of death in the United States and the most common cause of neurologic disability. It affects more than 700,000 people each year and is fatal in about one-half of these cases.

What causes it

Stroke typically results from one of three causes:

1. thrombosis of the cerebral arteries supplying the brain or of the intracranial vessels occluding blood flow

2. embolism from a thrombus outside the brain, such as in the heart, aorta, or common carotid artery
3. hemorrhage from an intracranial artery or vein, such as from hypertension, ruptured aneurysm, AVM, trauma, hemorrhagic disorder, or septic embolism.

Risk factor facts

Risk factors that predispose patients to stroke include:

- hypertension
- family history of stroke
- history of TIA (See *TIA and elderly patients*.)
- cardiac disease, including arrhythmias, coronary artery disease, acute myocardial infarction, dilated cardiomyopathy, and valvular disease
- diabetes
- familial hyperlipidemia
- cigarette smoking
- increased alcohol intake
- obesity, sedentary lifestyle (See *Obesity and stroke*.)
- use of hormonal contraceptives.

How it happens

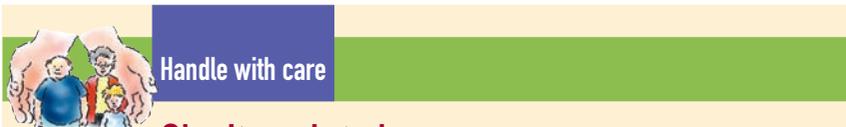
Regardless of the cause, the underlying event leading to stroke is oxygen and nutrient deprivation. Here's what happens:

- Normally, if the arteries become blocked, autoregulatory mechanisms maintain cerebral circulation until collateral circulation develops to deliver blood to the affected area.
- If the compensatory mechanisms become overworked or cerebral blood flow remains impaired for more than a few minutes, oxygen deprivation leads to infarction of brain tissue.



TIA and elderly patients

During your assessment, ask an elderly patient about recent falls—especially frequently occurring falls. This is important because an older patient is less likely to forget about or minimize frequent falls than he is to report other signs of a TIA.



Obesity and stroke

The degree of obesity—defined by BMI, waist circumference, or waist-to-hip ratio—has been found to be a significant risk factor for ischemic stroke incidence, regardless of gender or race. It's important to encourage your obese patients to lose weight, eat a healthy diet, and exercise as potential measures to reduce the incidence of ischemic stroke.

- The brain cells cease to function because they can't engage in anaerobic metabolism or store glucose or glycogen for later use.

Ischemic stroke

Here's what happens when a thrombotic or embolic stroke causes ischemia:

- Some of the neurons served by the occluded vessel die from lack of oxygen and nutrients.
- The result is cerebral infarction, in which tissue injury triggers an inflammatory response that in turn increases ICP.
- Injury to the surrounding cells disrupts metabolism and leads to changes in ionic transport, localized acidosis, and free radical formation.
- Calcium, sodium, and water accumulate in the injured cells, and excitatory neurotransmitters are released.
- Consequent continued cellular injury and swelling set up a vicious cycle of further damage.

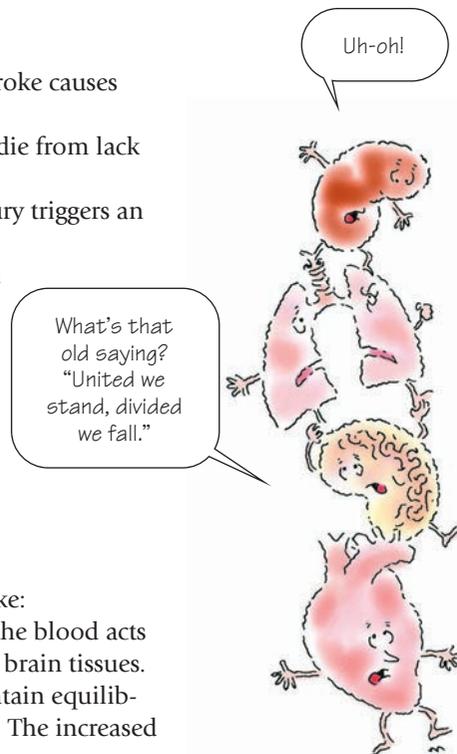
Hemorrhagic stroke

Here's what happens when a hemorrhage causes a stroke:

- Impaired cerebral perfusion causes infarction, and the blood acts as a space-occupying mass, exerting pressure on the brain tissues.
- The brain's regulatory mechanisms attempt to maintain equilibrium by increasing blood pressure to maintain CPP. The increased ICP forces CSF out, thus restoring equilibrium.
- If the hemorrhage is small, the patient may have minimal neurologic deficits. If the bleeding is heavy, ICP increases rapidly and perfusion stops. Even if the pressure returns to normal, many brain cells die.
- Initially, the ruptured cerebral blood vessels may constrict to limit the blood loss. This vasospasm further compromises blood flow, leading to more ischemia and cellular damage.
- If a clot forms in the vessel, decreased blood flow also promotes ischemia. If the blood enters the subarachnoid space, meningeal irritation occurs.
- Blood cells that pass through the vessel wall into the surrounding tissue may break down and block the arachnoid villi, causing hydrocephalus.

What to look for

Clinical features of stroke vary, depending on the artery affected (and, consequently, the portion of the brain it supplies), the severity



of the damage, and the extent of collateral circulation that develops to help the brain compensate for decreased blood supply. (See *Stroke signs and symptoms*, page 132.)

Left is right and right is left

A stroke in the left hemisphere produces symptoms on the right side of the body; in the right hemisphere, symptoms on the left side.

Common signs and symptoms of stroke include sudden onset of:

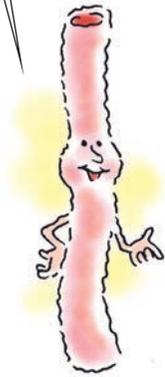
- hemiparesis on the affected side (may be more severe in the face and arm than in the leg)
- unilateral sensory defect (such as numbness, or tingling) generally on the same side as the hemiparesis
- slurred or indistinct speech or the inability to understand speech
- blurred or indistinct vision, double vision, or vision loss in one eye (usually described as a curtain coming down or gray-out of vision)
- mental status changes or loss of consciousness (particularly if associated with one of the above symptoms)
- very severe headache (with hemorrhagic stroke).

What tests tell you

Here are some test findings that can help diagnose a stroke:

- CT scan discloses structural abnormalities, edema, hemorrhage, and lesions, such as nonhemorrhagic infarction and aneurysms. Results are used to differentiate a stroke from other disorders, such as a tumor or hematoma. Patients with TIA generally have a normal CT scan. CT scan shows evidence of hemorrhagic stroke immediately and of ischemic (thrombotic or embolic) stroke within 72 hours after onset of symptoms. CT scan should be obtained within 25 minutes after the patient arrives in the ED, and results should be available within 45 minutes of arrival to determine whether hemorrhage is present. If hemorrhagic stroke is present, thrombolytic therapy is contraindicated.
- MRI is used to identify areas of ischemia and infarction and cerebral swelling. MRA/CTA can be used to evaluate the cerebral vessels.
- Cerebral angiography shows details of disruption or displacement of the cerebral circulation by occlusion or hemorrhage and can be used to treat occlusion or vasospasm.
- Carotid duplex scan is a high-frequency ultrasound that shows blood flow through the carotid arteries and reveals stenosis due to atherosclerotic plaque and blood clots.
- Transcranial Doppler studies are used to evaluate the velocity of blood flow through major intracranial vessels, which can indicate vessel diameter.

"The features of stroke vary, depending on the artery affected."



A CT scan should be taken within 25 minutes after the patient arrives in the emergency department. This will determine within minutes whether hemorrhage is present.



Stroke signs and symptoms

With stroke, functional loss reflects damage to the area of the brain that's normally perfused by the occluded or ruptured artery. Although one patient may experience only mild hand weakness, another may develop unilateral paralysis.

Hypoxia and ischemia may produce edema that affects distal parts of the brain, causing further neurologic deficits. Here are the signs and symptoms that accompany stroke at different sites.

Site	Signs and symptoms	Site	Signs and symptoms
Middle cerebral artery	<ul style="list-style-type: none"> • Aphasia • Dysphasia • Dyslexia (reading problems) • Dysgraphia (inability to write) • Visual field cuts • Hemiparesis on the affected side, which is more severe in the face and arm than in the leg 	Anterior cerebral artery	<ul style="list-style-type: none"> • Confusion • Weakness • Numbness on the affected side (especially in the arm) • Paralysis of the contralateral foot and leg • Incontinence • Poor coordination • Impaired motor and sensory functions • Personality changes, such as flat affect and distractibility
Internal carotid artery	<ul style="list-style-type: none"> • Headaches • Weakness • Paralysis • Numbness • Sensory changes • Visual disturbances such as blurring on the affected side • Altered LOC • Bruits over the carotid artery • Aphasia • Dysphagia • Ptosis 	Vertebral or basilar artery	<ul style="list-style-type: none"> • Mouth and lip numbness • Dizziness • Weakness on the affected side • Visual deficits, such as color blindness, lack of depth perception, and diplopia • Poor coordination • Dysphagia • Slurred speech • Amnesia • Ataxia
		Posterior cerebral artery	<ul style="list-style-type: none"> • Visual field cuts • Sensory impairment • Dyslexia • Coma • Blindness from ischemia in the occipital area

- Brain scan shows ischemic areas but may not be conclusive for up to 2 weeks after stroke.
- Single photon emission CT scanning and PET scan show areas of altered metabolism surrounding lesions that aren't revealed by other diagnostic tests.
- No laboratory tests confirm the diagnosis of stroke, but some tests aid diagnosis and some are used to establish a baseline for thrombolytic treatment. A blood glucose test shows whether the patient's symptoms are related to hypoglycemia. Hb level and HCT may be elevated in severe occlusion. Baselines obtained before thrombolytic therapy begins include CBC, platelet count, PTT, PT, fibrinogen level, and chemistry panel.

After a definitive diagnosis has been made, treatment with thrombolytics begins—if the patient meets the criteria—within 60 minutes after arrival in the emergency department.

How it's treated

The goal is to begin treatment within 3 hours of symptom onset.

Drugs of choice

Thrombolytics (also called *fibrinolytics*) are the drugs of choice in treating a stroke patient. The patient must first meet certain criteria to be considered for this type of treatment. (See *Who's suited for thrombolytic therapy?*, page 134.)



Drugs of choice for management

Drug therapy for the management of stroke includes:

- thrombolytics for emergency treatment of ischemic stroke (See *Adult suspected stroke algorithm*, page 135.)
- aspirin or clopidogrel (Plavix) as an antiplatelet agent to prevent recurrent stroke
- benzodiazepines to treat patients with seizure activity
- anticonvulsants to treat seizures or to prevent them after the patient's condition has stabilized
- stool softeners to avoid straining, which increases ICP
- antihypertensives and antiarrhythmics to treat patients with risk factors for recurrent stroke
- analgesics to relieve the headaches that may follow a hemorrhagic stroke.

Antihypertensives and antiarrhythmics are given to patients with risk factors for recurrent stroke.

Medical management

Medical management of stroke commonly includes physical rehabilitation, dietary and drug regimens to reduce risk factors, surgery, and care measures to help the patient adapt to deficits, such as motor impairment and paralysis.



Who's suited for thrombolytic therapy?

Not every stroke patient is a candidate for thrombolytic therapy. Each is evaluated to see whether established criteria are met.

Criteria that must be present

Criteria that must be present for a patient to be considered for thrombolytic therapy include:

- acute ischemic stroke associated with significant neurologic deficit
- onset of symptoms less than 3 hours before treatment begins
- age 18 or older.

Criteria that must not be present

In addition to meeting the above criteria, the patient must not:

- have a history of head trauma or prior stroke in the past 3 months
- exhibit evidence of subarachnoid hemorrhage during pretreatment evaluation
- have a history of arterial puncture at a noncompressible site in the past 7 days
- have a history of previous intracranial hemorrhage
- have an elevated blood pressure (systolic blood pressure greater than 185 mm Hg or diastolic blood pressure less than 110 mm Hg) at the time of treatment
- have evidence of active bleeding on examination
- have a blood glucose concentration less than 50 mg/dl (2.7 mmol/L)
- have a CT scan that demonstrates multilobar infarction (hypodensity greater than 1/3 cerebral hemisphere)
- have a known bleeding diathesis, involving but not limited to:
 - platelet count less than 100,000/mm³
 - receipt of heparin within 48 hours before the onset of stroke and having an elevated activated PTT (greater than the upper limit of normal)
 - current use of oral anticoagulants such as warfarin, international normalized ratio greater than 1.7, or PT greater than 15 seconds.

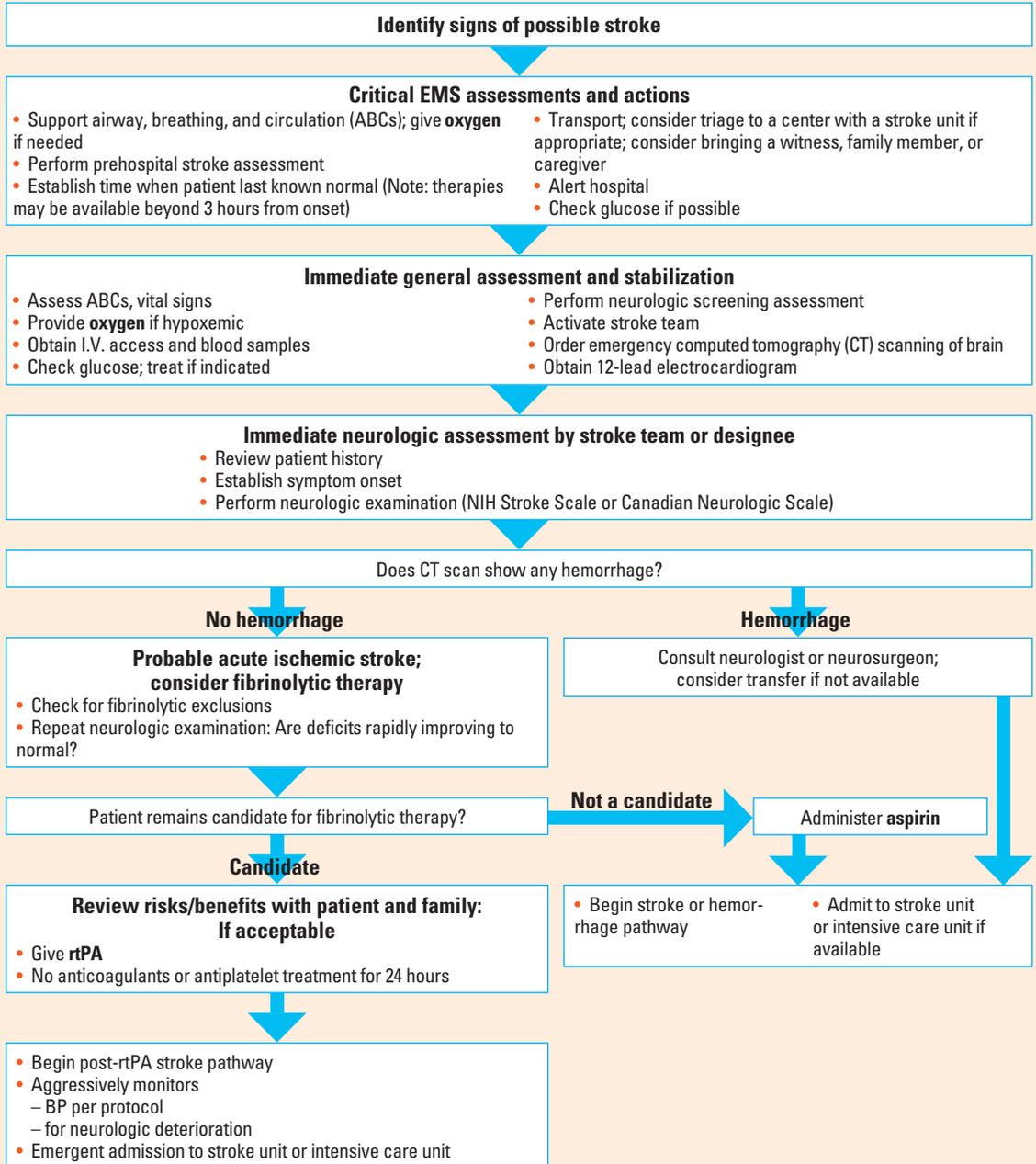
Criteria that must be evaluated individually

Recent experience suggests that under some circumstances and with careful evaluation, a patient may receive fibrinolytic therapy despite the presence of one or more of the following criteria. The doctor must weigh the risk to benefit for each patient.

The criteria include:

- evidence of only minor or rapidly improving stroke symptoms that clear spontaneously
- seizure at onset with postictal residual neurologic impairments
- major surgery or serious trauma within previous 14 days
- GI or urinary tract hemorrhage within the previous 21 days
- acute myocardial infarction within the previous 3 months.

Adult suspected stroke algorithm



Reproduced with permission from Hazinski, M. F., & Samson, R. (Eds.). (2010). *2010 Handbook of Emergency Cardiovascular Care For Healthcare Providers*. © 2010, American Heart Association.

Under the knife

Depending on the cause and extent of the stroke, the patient may undergo:

- a craniotomy to remove a hematoma
- a carotid endarterectomy to remove atherosclerotic plaques from the inner arterial wall
- an extracranial bypass to circumvent an artery that's blocked by occlusion or stenosis.

Call the "S" team

Your facility may have a stroke protocol and stroke team composed of specially trained nurses who respond to potential stroke patients. When a patient shows signs and symptoms of a stroke, first assess the patient using a stroke assessment tool such as the Cincinnati Stroke Scale. (See *Cincinnati prehospital stroke scale*.)

After your initial assessment, call the stroke team nurse, who will evaluate the patient; complete a neurologic assessment; report findings to the practitioner; and facilitate rapid and appropriate care of the patient, including emergency interventions, diagnostic tests, and transfer to the critical care unit.

Surgical intervention after stroke is called for in some cases.



Cincinnati prehospital stroke scale

The Cincinnati Prehospital Stroke Scale is a simplified scale for evaluating stroke patients that was derived from the National Institutes of Health Stroke Scale. It's used to evaluate facial palsy, arm weakness, and speech abnormalities. An abnormality in any one of the categories below is highly suggestive of stroke.

Facial droop (The patient shows teeth or smiles.)

- Normal—Both sides of the face move equally.
- Abnormal—One side of the face doesn't move as well as the other.

Arm drift (The patient closes eyes and extends both arms straight out for 10 seconds.)

- Normal—Both arms move the same or both arms don't move at all.
- Abnormal—One arm either doesn't move or one arm drifts downward as compared with the other.

Speech (The patient repeats, "The sky is blue in Cincinnati.")

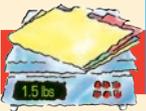
- Normal—The patient says the correct words with no slurring of words.
- Abnormal—The patient slurs words, says the wrong words, or can't speak.

From Kothari, R. U., Pancioli, A., Liu, T., Brott, T., & Broderick, J. (1999). Cincinnati Prehospital Stroke Scale: Reproducibility and validity. *Annals of Emergency Medicine*, 33, 373–378. Adapted with permission from the American College of Emergency Physicians.

What to do

- If the patient has an altered LOC, secure and maintain the patient's airway and anticipate the need for ET intubation and mechanical ventilation.
- Monitor oxygen saturation levels via pulse oximetry and ABG levels as ordered. Administer supplemental oxygen as ordered to maintain oxygen saturation greater than 90%.
- Place the patient on a cardiac monitor and monitor for cardiac arrhythmias. Monitor blood pressure carefully. The current guideline for patients with ischemic stroke that have not received tissue plasminogen activator (tPa) is for permissive hypertension—not treating unless the blood pressure is greater than 220 mm Hg systolic. For patients who have received tPa, blood pressure of greater than 180/105 mm Hg is treated to reduce the risk of posttreatment hemorrhage.
- Assess the patient's neurologic status frequently, at least every 15 to 30 minutes, initially, then hourly as indicated. Observe for signs of increased ICP.
- If cerebral edema is suspected, maintain ICP sufficient for adequate cerebral perfusion but low enough to avoid brain herniation. Elevate the head of the bed 25 to 30 degrees. (See *Positioning the head of the bed.*)

As you can see, nursing care of a stroke patient is pretty intensive. It demands careful monitoring and measures to prevent further progression of neurologic and other body system problems.



Weighing the evidence

Positioning the head of the bed

Positioning of the head of the bed must be individualized for each patient. The traditional positioning at 25 to 30 degrees is commonly used for potentially increased ICP. Stroke patients with increased ICP and chronic respiratory conditions may need head elevation for maximum oxygenation. The head of the bed should be elevated at least 30 degrees if the patient is at risk for aspiration or airway obstruction due to dysphagia.

Researchers haven't identified the optimal position of the head of the bed, but it seems that positioning depends on the individual patient's condition. Recent studies have suggested that positioning the head of the bed can facilitate an increase in cerebral blood flow and maximize oxygenation to cerebral tissue. A study using transcranial Doppler technology found that the head-flat position maximized blood flow to the brain. Further studies on head positioning of acute ischemic stroke patients need to be completed; if a patient has a lower risk for increased ICP and isn't at risk for aspiration, the head-down position has been shown to be beneficial.

Source: Summers, D., Leonard, A., Wentworth, D., Saver, J. L., Simpson, J., Spilker, J. A., . . . Mitchell, P. H. (2009). Comprehensive overview of nursing and interdisciplinary care of the acute ischemic stroke patient: A scientific statement from the American Heart Association. *Stroke, 40*(8), 2911–2944.

- Assess hemodynamic status frequently. Give fluids as ordered and monitor I.V. infusions to avoid overhydration, which may increase ICP.
- For a patient receiving thrombolytic therapy, assess the patient for signs and symptoms of bleeding every 15 to 30 minutes and institute bleeding precautions. Monitor results of coagulation studies.
- Monitor the patient for seizures and administer anticonvulsants as ordered. Institute safety precautions to prevent injury.
- If the patient had a TIA, administer antiplatelet agents.
- Turn the patient often and position him using careful body alignment. Apply antiembolism stockings or intermittent sequential compression devices.
- Take steps to prevent skin breakdown.
- Begin exercises as soon as possible. Perform passive ROM exercises for both the affected and unaffected sides. Teach and encourage the patient to use his unaffected side to exercise his affected side.
- Manage GI problems. Be alert for signs of straining at stool as it increases ICP. If the patient is receiving steroids, monitor for signs of GI irritation.
- Modify the patient's diet, as appropriate, such as by increasing fiber.
- Provide meticulous eye and mouth care.
- Maintain communication with the patient. If he's aphasic, set up a simple method of communicating.
- Provide psychological support.



Quick quiz

1. The most sensitive indicator of neurologic status change is:
 - A. LOC.
 - B. speech.
 - C. behavior.
 - D. cognitive function.

Answer: A. Change in LOC is the earliest and most sensitive indicator of neurologic status change.

2. Signs of an adverse reaction to contrast medium include all of the following except:
 - A. restlessness.
 - B. bradycardia.
 - C. urticaria.
 - D. facial flushing.

Answer: B. A sign of adverse reaction to the contrast medium is tachycardia.

3. The major neurologic symptom of Guillain-Barré syndrome is:
- A. headache.
 - B. nuchal rigidity.
 - C. muscle weakness.
 - D. altered LOC.

Answer: C. Muscle weakness usually appears in the legs first, then extends to the arms and face within 2 weeks or less.

4. Which type of seizure is characterized by brief, involuntary muscle movements?
- A. Jacksonian
 - B. Myoclonic
 - C. Generalized tonic-clonic
 - D. Akinetic

Answer: B. During myoclonic seizures, the patient has brief, involuntary muscle movements.

5. Which condition may have delayed symptoms in an older person?
- A. stroke
 - B. cerebral aneurysm
 - C. seizure disorder
 - D. subdural hematoma

Answer: D. An older person with cerebral atrophy can tolerate a larger subdural hematoma for a longer time than a younger person can before the hematoma causes neurologic changes.

6. In order for a patient experiencing an ischemic stroke to receive thrombolytic therapy, which criteria must be present?
- A. onset of symptoms less than 3 hours before treatment begins
 - B. evidence of only minor or rapidly improving stroke symptoms that clear spontaneously
 - C. seizure at onset with postictal residual neurologic impairments
 - D. evidence of active bleeding on examination

Answer: A. Criteria that must be present for a patient to be considered for thrombolytic therapy include acute ischemic stroke associated with significant neurologic deficit, onset of symptoms less than 3 hours before treatment begins, and age 18 or older.

Scoring

- ☆☆☆☆ If you answered all six questions correctly, you may already know this: You're a brainiac!
- ☆☆☆ If you answered six questions correctly, cheer up. You have all the brainpower you need to succeed.
- ☆☆ If you answered fewer than six questions correctly, don't become irritable. Review the chapter and then take the test again.

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