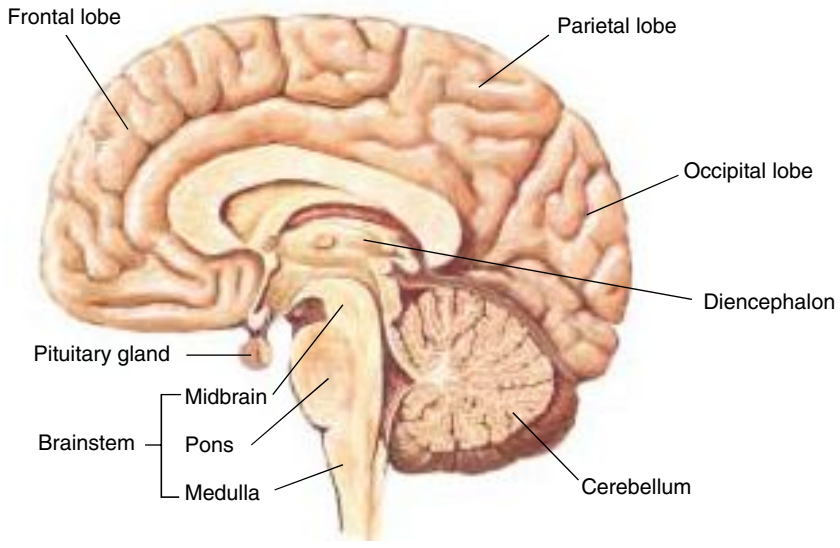
CHAPTER

16 The Nervous System

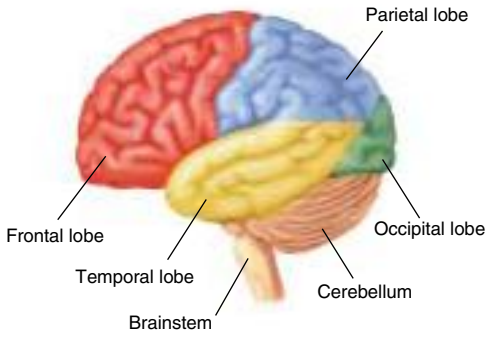
ANATOMY AND PHYSIOLOGY

This section deals briefly with structures, functions, and concepts that relate directly to the neurologic examination. After a short description of the brain, spinal cord, cranial and peripheral nerves, and reflexes, it summarizes important motor and sensory pathways. Common or concerning symptoms, health promotion and counseling, and a preview of the pertinent write-up then follow. Next comes *Techniques of Examination* for the nervous system, including mental status, the cranial nerves, the motor and sensory systems, and reflexes.

As you review this material, note that the *central nervous system* consists of the brain and the spinal cord. The *peripheral nervous system* consists of the 12 pairs of cranial nerves and the spinal and peripheral nerves. Most of the peripheral nerves contain both motor and sensory fibers.



RIGHT HALF OF THE BRAIN, MEDIAL VIEW



LEFT LATERAL VIEW OF THE BRAIN

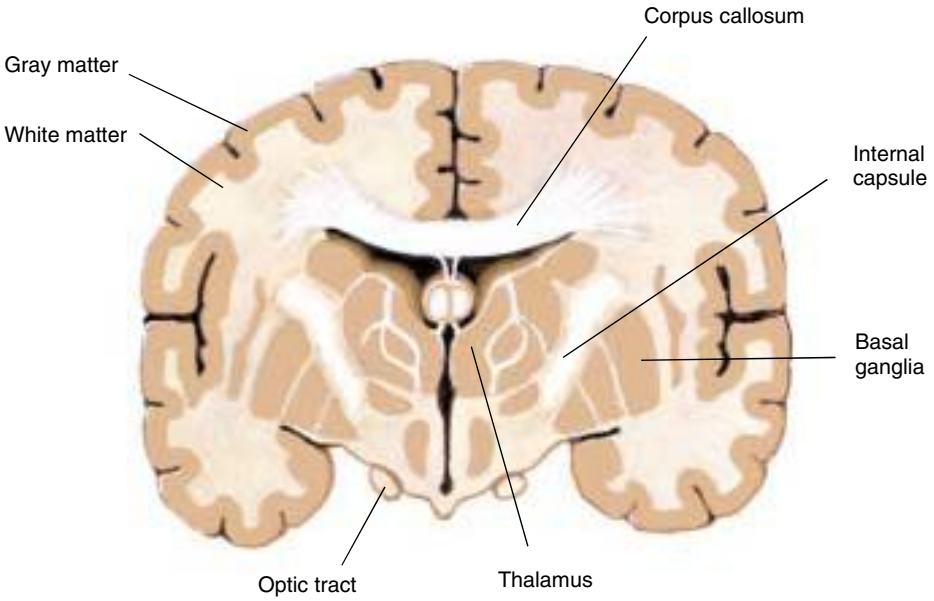
Central Nervous System

THE BRAIN

The brain has four regions: the cerebrum, the diencephalon, the brainstem, and the cerebellum. The cerebral hemispheres contain the greatest mass of brain tissue. Each hemisphere is subdivided into frontal, parietal, temporal, and occipital lobes.

The brain is a vast network of interconnecting *neurons* (nerve cells). These consist of cell bodies and their *axons*—single long fibers that conduct impulses to other parts of the nervous system.

Brain tissue may be gray or white. *Gray matter* consists of aggregations of neuronal cell bodies. It rims the surfaces of the cerebral hemispheres, forming the cerebral cortex. *White matter* consists of neuronal axons that are coated with myelin. The myelin sheaths, which create the white color, allow nerve impulses to travel more rapidly.



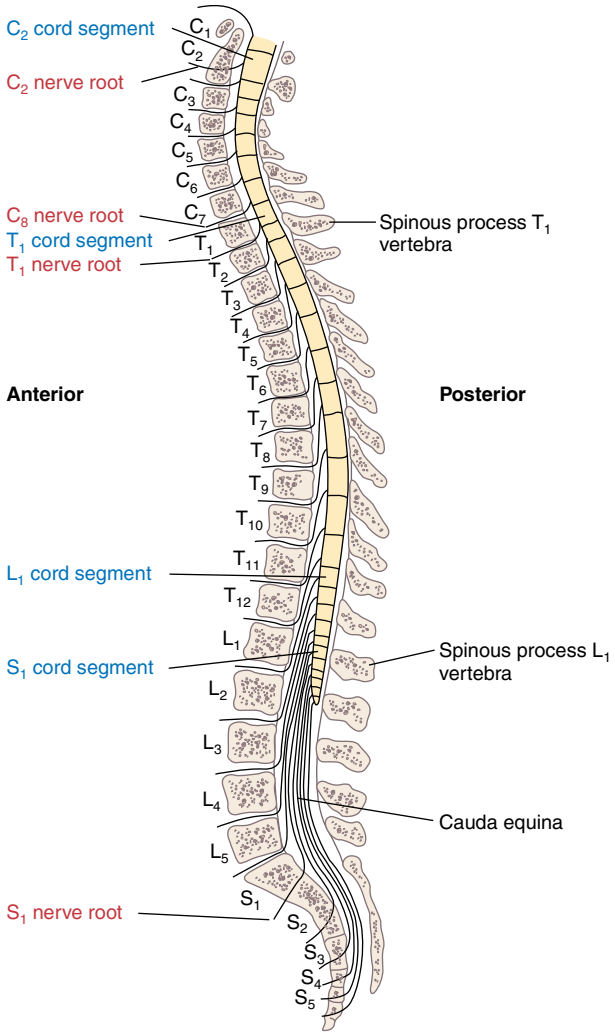
CORONAL SECTION OF THE BRAIN

Deep in the brain lie additional clusters of gray matter. These include the *basal ganglia,* which affect movement, and the thalamus and the hypothalamus (structures in the diencephalon). The *thalamus* processes sensory impulses and relays them to the cerebral cortex. The *hypothalamus* maintains homeostasis and regulates temperature, heart rate, and blood pressure. The hypothalamus affects the endocrine system and governs emotional behaviors such as anger and sexual drive. Hormones secreted in the hypothalamus act directly on the pituitary gland.

In contrast, note the *internal capsule,* a white matter structure where myelinated fibers converge from all parts of the cerebral cortex and descend into the brainstem. The *brainstem,* which connects the upper part of the brain with the spinal cord, has three sections: the midbrain, the pons, and the medulla.

Consciousness depends on the interaction between intact cerebral hemispheres and an important structure in the diencephalon and upper brainstem, the *reticular activating (arousal) system*.

The *cerebellum*, which lies at the base of the brain, coordinates all movement and helps maintain the body upright in space.



THE SPINAL CORD, LATERAL VIEW

THE SPINAL CORD

The *spinal cord* is a cylindrical mass of nerve tissue encased within the bony vertebral column, extending from the medulla to the first or second lumbar vertebra. It contains important motor and sensory nerve pathways that exit and enter the cord via anterior and posterior nerve roots and spinal and peripheral nerves. The spinal cord also mediates reflex activity of the deep tendon (or spinal nerve) reflexes. Motor and sensory tracts and the deep tendon reflexes are further discussed on pp. 541–546).

The spinal cord is divided into five segments: cervical (C1–8), thoracic (T1–12), lumbar (L1–5), sacral (S1–5), and coccygeal.

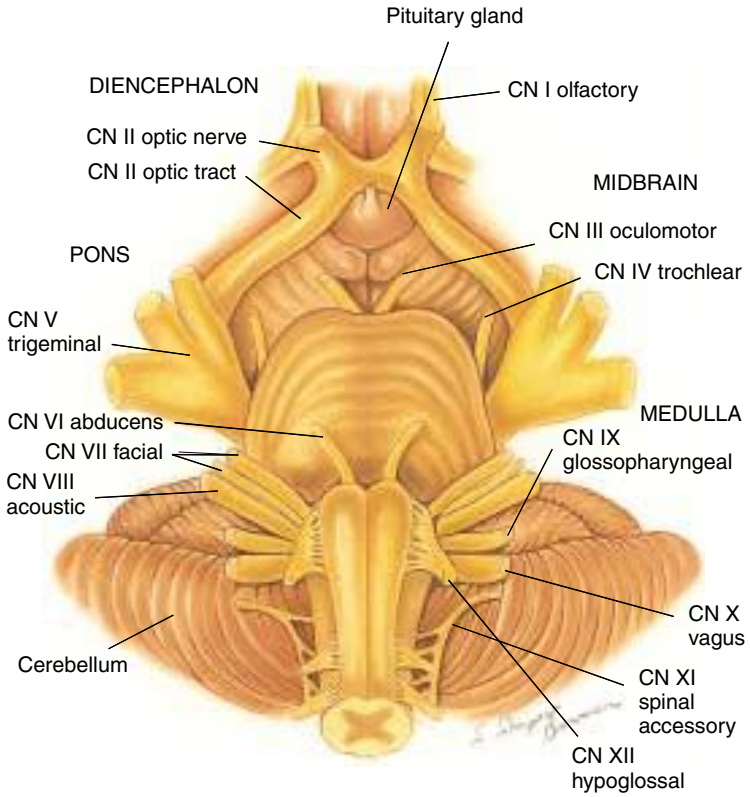
Note that the spinal cord is not as long as the vertebral canal. The level of the nerve roots exiting the cord differs from the adjacent vertebral level. The lumbar and sacral roots travel the longest intraspinal distance. These roots fan out like a horse's tail at L1–2, giving rise to the term *cauda equina*. (To avoid injury to the cord, most lumbar punctures are performed at the L3–4 vertebral interspace.)

Peripheral Nervous System

THE CRANIAL NERVES

Twelve pairs of special nerves called *cranial nerves* emerge from within the skull or *cranium*. Cranial Nerves II through XII arise from the diencephalon and the brainstem, as illustrated below. (Cranial Nerves I and II are actually fiber tracts emerging from the brain.) Some cranial nerves are limited to general motor or sensory functions, whereas others are specialized, producing smell, vision, or hearing (I, II, VIII).

Functions of the cranial nerves (CN) most relevant to physical examination are summarized on the next page.

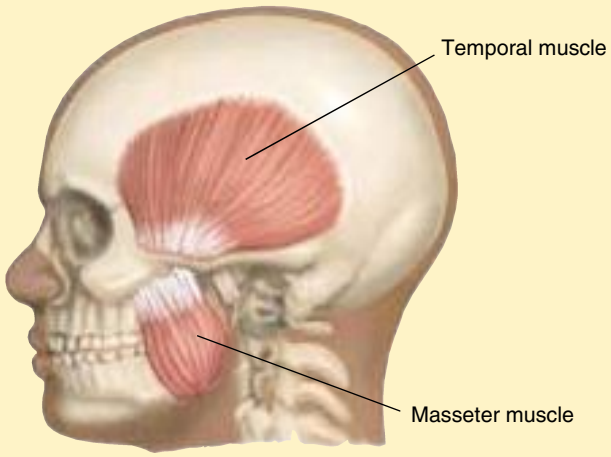


INFERIOR SURFACE OF THE BRAIN

THE PERIPHERAL NERVES

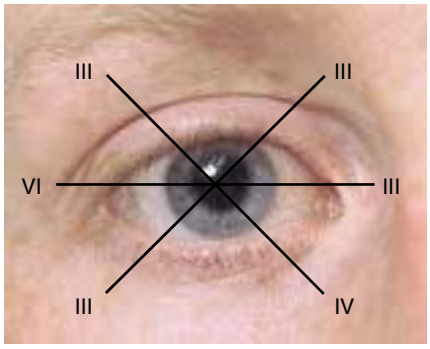
In addition to cranial nerves, the peripheral nervous system also includes spinal and peripheral nerves that carry impulses to and from the cord. Thirty-one pairs of nerves attach to the spinal cord: 8 cervical, 12 thoracic, 5 lumbar,

No.	Cranial Nerve	Function
I	**Olfactory**	Sense of smell
II	**Optic**	Vision
III	**Oculomotor**	Pupillary constriction, opening the eye, and most extraocular movements
IV	**Trochlear**	Downward, inward movement of the eye
VI	**Abducens**	Lateral deviation of the eye
V	**Trigeminal**	*Motor*—temporal and masseter muscles (jaw clenching), also lateral movement of the jaw *Sensory*—facial. The nerve has three divisions: (1) ophthalmic, (2) maxillary, and (3) mandibular.

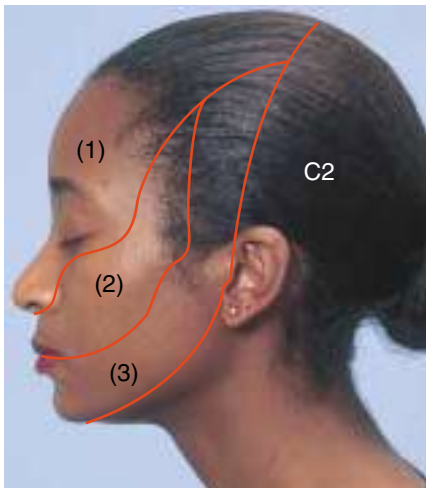


CN V—MOTOR

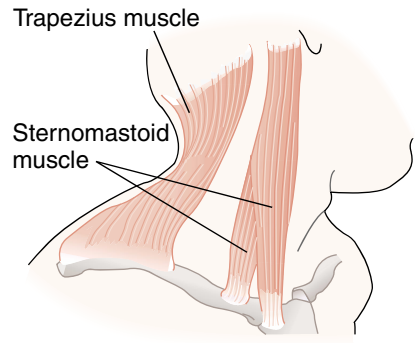
VII	**Facial**	*Motor*—facial movements, including those of facial expression, closing the eye, and closing the mouth *Sensory*—taste for salty, sweet, sour, and bitter substances on the anterior two thirds of the tongue
VIII	**Acoustic**	Hearing (cochlear division) and balance (vestibular division)
IX	**Glossopharyngeal**	*Motor*—pharynx *Sensory*—posterior portions of the eardrum and ear canal, the pharynx, and the posterior tongue, including taste (salty, sweet, sour, bitter)
X	**Vagus**	*Motor*—palate, pharynx, and larynx *Sensory*—pharynx and larynx
XI	**Spinal accessory**	*Motor*—the sternomastoid and upper portion of the trapezius
XII	**Hypoglossal**	*Motor*—tongue



RIGHT EYE (CN III, IV, VI)



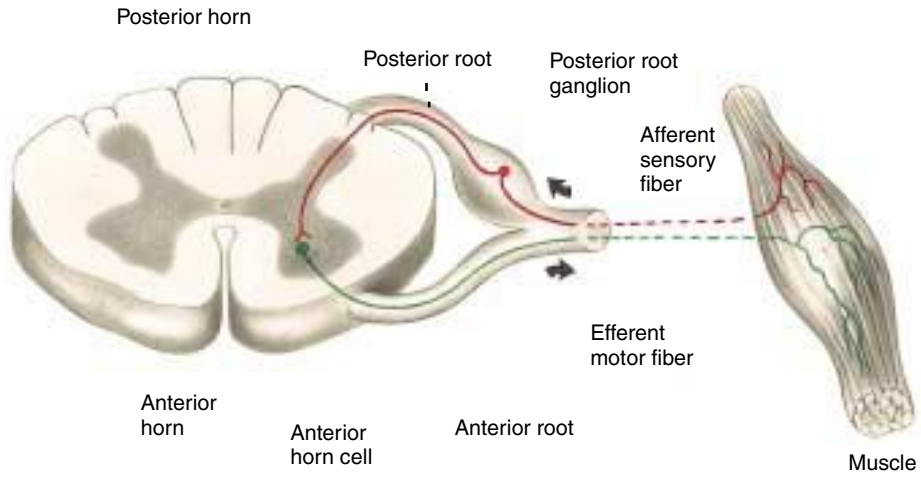
CN V—SENSORY



CN XI—MOTOR

5 sacral, and 1 coccygeal. Each nerve has an anterior (ventral) root containing motor fibers, and a posterior (dorsal) root containing sensory fibers. The anterior and posterior roots merge to form a short (<5 mm) *spinal nerve.* Spinal nerve fibers commingle with similar fibers from other levels to form *peripheral nerves.* Most peripheral nerves contain both *sensory* (afferent) and *motor* (efferent) fibers.

Like the brain, the spinal cord contains both gray matter and white matter. Nuclei of gray matter, which are aggregations of nerve cell bodies, are surrounded by white tracts of nerve fibers connecting the brain to the peripheral nervous system. Note the butterfly appearance of the gray matter nuclei, with anterior and posterior horns.



THE SPINAL CORD, CROSS SECTION

Spinal Reflexes: The Deep Tendon Response

The deep tendon or muscle stretch reflexes are relayed over structures of both the central and peripheral nervous systems. Recall that a *reflex* is an involuntary stereotypical response that may involve as few as two neurons, one afferent (sensory) and one efferent (motor), across a single synapse. The deep tendon reflexes in the arms and legs are such monosynaptic reflexes. They illustrate the simplest unit of sensory and motor function. (Other reflexes are polysynaptic, involving interneurons interposed between sensory and motor neurons.)

To elicit a deep tendon reflex, briskly tap the tendon of a partially stretched muscle. For the reflex to fire, all components of the reflex arc must be intact: sensory nerve fibers, spinal cord synapse, motor nerve fibers, neuromuscular junction, and muscle fibers. Tapping the tendon activates special sensory fibers in the partially stretched muscle, triggering a sensory impulse that travels to the spinal cord via a peripheral nerve. The stimulated sensory fiber

synapses directly with the anterior horn cell innervating the same muscle. When the impulse crosses the neuromuscular junction, the muscle suddenly contracts, completing the reflex arc.

Because each deep tendon reflex involves specific spinal segments, together with their sensory and motor fibers, an abnormal reflex can help you to locate a pathologic lesion. You should know the segmental levels of the deep tendon reflexes. You can remember them easily by their numerical sequence in ascending order from ankle to triceps: S1—L2, 3, 4,—C5, 6, 7.

Ankle reflex	Sacral 1 primarily
Knee reflex	Lumbar 2, 3, 4
Supinator (brachioradialis) reflex	Cervical 5, 6
Biceps reflex	Cervical 5, 6
Triceps reflex	Cervical 6, 7

Reflexes may be initiated by stimulating skin as well as muscle. Stroking the skin of the abdomen, for example, produces a localized muscular twitch. These superficial (cutaneous) reflexes and their corresponding spinal segments include:

Abdominal reflexes—upper	Thoracic 8, 9, 10
—lower	Thoracic 10, 11, 12
Plantar responses	Lumbar 5, Sacral 1

Motor Pathways

Motor pathways contain upper motor neurons, synapses in the brainstem or spinal cord, and lower motor neurons. Nerve cell bodies or *upper motor neurons* lie in the motor strip of the cerebral cortex and in several brainstem nuclei; their axons synapse with motor nuclei in the brainstem (for cranial nerves) and in the spinal cord (for peripheral nerves). *Lower motor neurons* have cell bodies in the spinal cord, termed anterior horn cells; their axons transmit impulses through the anterior roots and spinal nerves into peripheral nerves, terminating at the neuromuscular junction.

Three kinds of motor pathways impinge on the anterior horn cells: the corticospinal tract, the basal ganglia system, and the cerebellar system. There are additional pathways originating in the brainstem that mediate flexor and extensor tone in limb movement and posture; most notable in coma (see Table 16-16, p. 622).

All of these higher motor pathways affect movement only through the lower motor neurons—sometimes called the "final common pathway." Any movement, whether initiated voluntarily in the cortex, "automatically" in the

THE PRINCIPAL MOTOR PATHWAYS

- The ***corticospinal (pyramidal) tract***. The corticospinal tracts mediate voluntary movement and integrate skilled, complicated, or delicate movements by stimulating selected muscular actions and inhibiting others. They also carry impulses that inhibit *muscle tone,* the slight tension maintained by normal muscle even when it is relaxed. The corticospinal tracts originate in the motor cortex of the brain. Motor fibers travel down into the lower medulla, where they form an anatomical structure resembling a pyramid. There most of these fibers cross to the opposite or *contralateral* side of the medulla, continue downward, and synapse with anterior horn cells or with intermediate neurons. Tracts synapsing in the brainstem with motor nuclei of the cranial nerves are termed *corticobulbar.*
- The ***basal ganglia system***. This exceedingly complex system includes motor pathways between the cerebral cortex, basal ganglia, brainstem, and spinal cord. It helps to maintain muscle tone and to control body movements, especially gross automatic movements such as walking.
- The ***cerebellar system***. The cerebellum receives both sensory and motor input and coordinates motor activity, maintains equilibrium, and helps to control posture.

basal ganglia, or reflexly in the sensory receptors, must ultimately be translated into action via the anterior horn cells. A lesion in any of these areas will affect movement or reflex activity.

When the corticospinal tract is damaged or destroyed, its functions are reduced or lost below the level of injury. *When upper motor neurons are damaged above the crossover of its tracts in the medulla, motor impairment develops on the opposite or contralateral side. In damage below the crossover, motor impairment occurs on the same or ipsilateral side of the body.* The affected limb becomes weak or paralyzed, and skilled, complicated, or delicate movements are performed especially poorly when compared to gross movements. Muscle tone is increased and deep tendon reflexes are exaggerated.

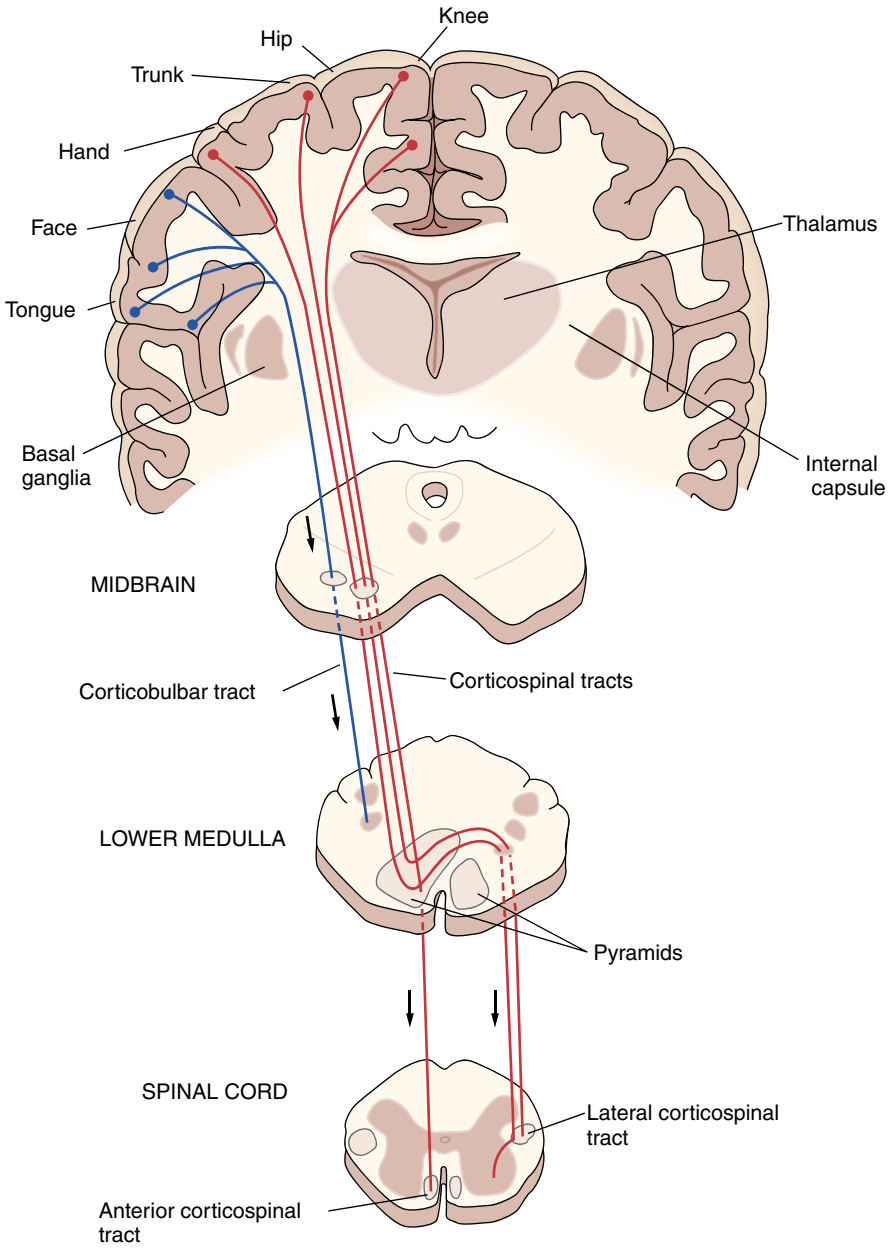
Damage to the lower motor neurons causes ipsilateral weakness and paralysis, but in this case muscle tone and reflexes are decreased or absent.

Disease of the basal ganglia system or cerebellar system does not cause paralysis, but can be disabling. Damage to the basal ganglia system produces changes in muscle tone (most often an increase), disturbances in posture and gait, a slowness or lack of spontaneous and automatic movements termed *bradykinesia*, and a variety of involuntary movements. Cerebellar damage impairs coordination, gait, and equilibrium, and decreases muscle tone.

Sensory Pathways

Sensory impulses not only participate in reflex activity, as previously described, but also give rise to conscious sensation, calibrate body position in

space, and help regulate internal autonomic functions like blood pressure, heart rate, and respiration.



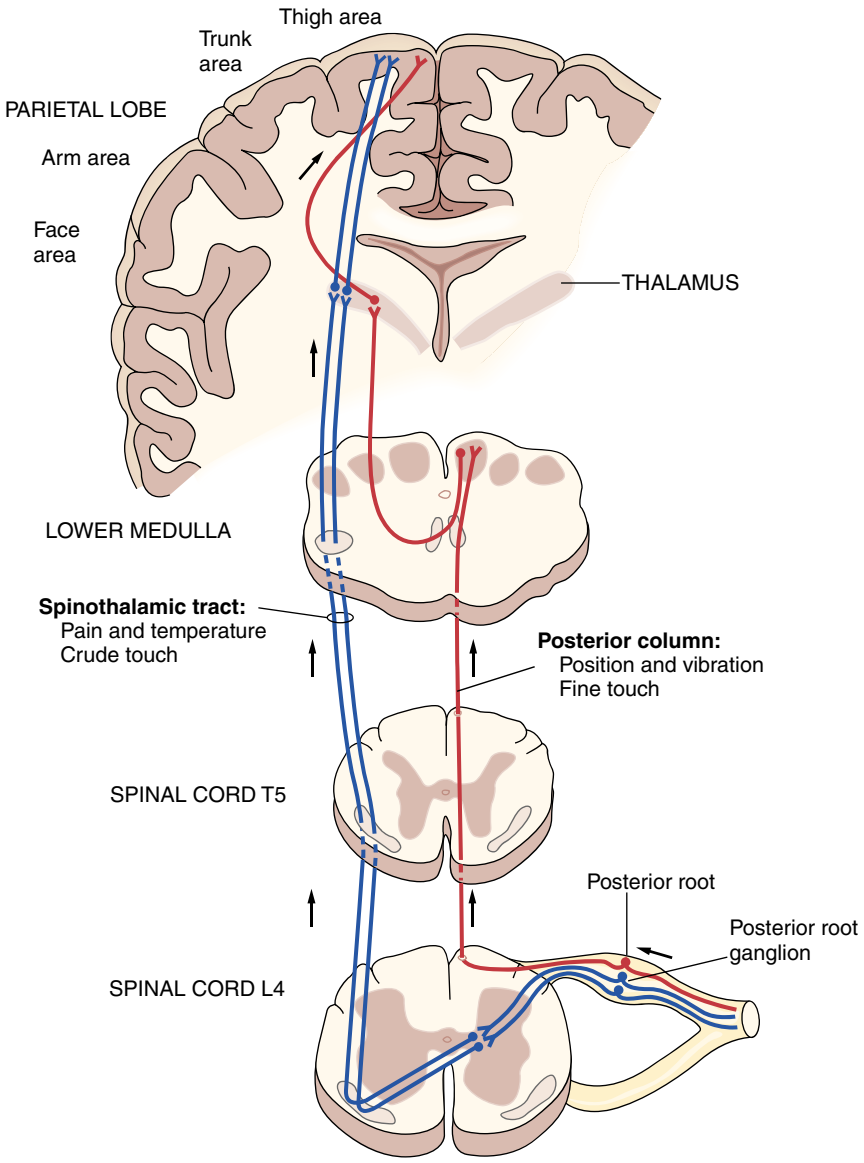
MOTOR PATHWAYS: CORTICOSPINAL AND CORTICOBULBAR TRACTS

A complex system of sensory receptors relays impulses from skin, mucous membranes, muscles, tendons, and viscera. Sensory fibers registering sensations such as pain, temperature, position, and touch, pass through the peripheral nerves and posterior roots and enter the spinal cord. Once inside the cord, sensory impulses reach the sensory cortex of the brain

via one of the two pathways: the spinothalamic tracts or the posterior columns.

Within one or two spinal segments from their entry into the cord, fibers conducting the sensations of *pain* and *temperature* pass into the posterior horn of the spinal cord and synapse with secondary sensory neurons. Fibers conducting *crude touch*—a sensation perceived as light touch but without accurate localization—also pass into the posterior horn and synapse with secondary neurons. The secondary neurons then cross to the opposite side and pass upward in the *spinothalamic tract* into the thalamus.

Fibers conducting the sensations of *position* and *vibration* pass directly into the *posterior columns* of the cord and travel upward to the medulla, together with fibers transmitting *fine touch*—touch that is accurately localized and finely discriminating. These fibers synapse in the medulla with secondary sensory neurons. Fibers projecting from secondary neurons cross to the opposite side at the medullary level and continue on to the thalamus.



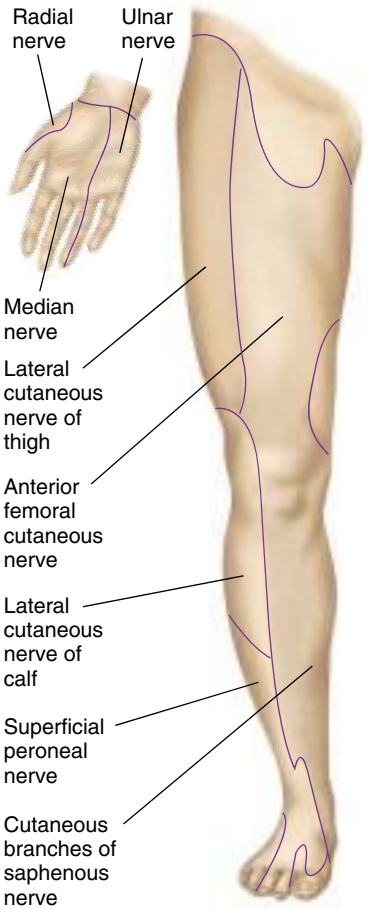
SENSORY PATHWAYS: SPINOTHALAMIC TRACT AND POSTERIOR COLUMNS

At the *thalamic level*, the general quality of sensation is perceived (e.g., pain, cold, pleasant and unpleasant), but fine distinctions are not made. For full perception, a third group of sensory neurons sends impulses from the thalamus to the *sensory cortex* of the brain. Here stimuli are localized and discriminations made among them.

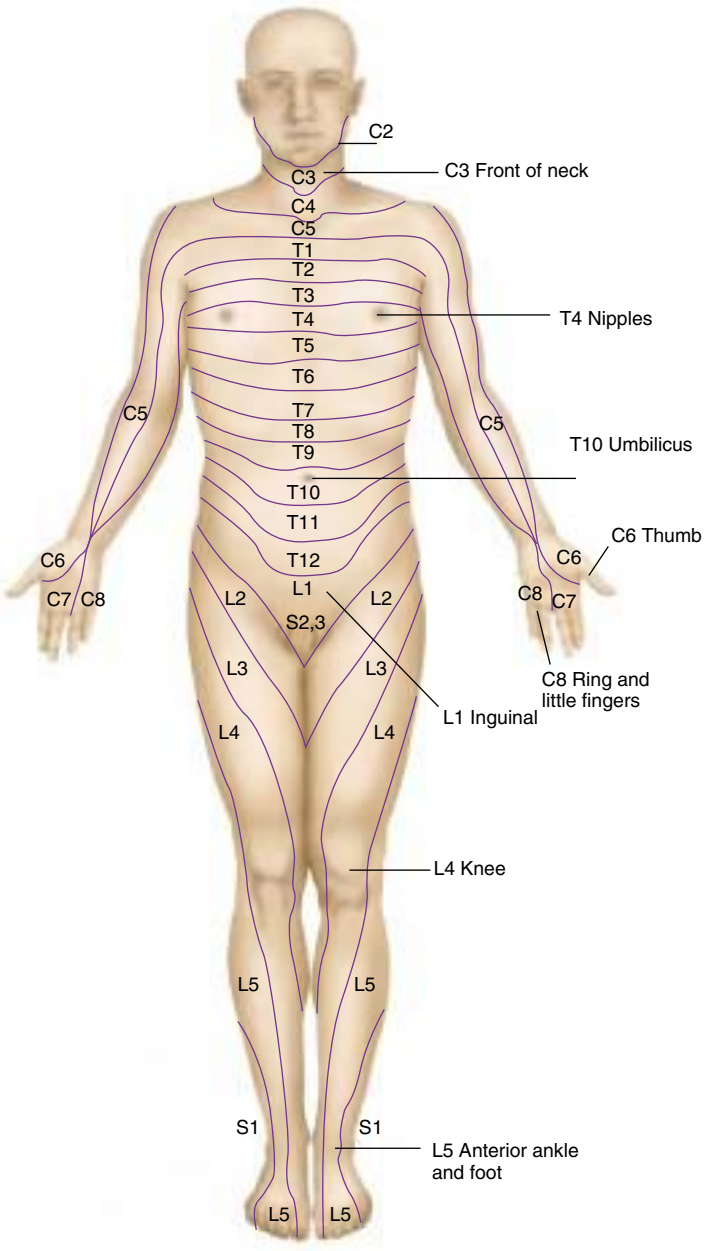
Lesions at different points in the sensory pathways produce different kinds of sensory loss. Patterns of sensory loss, together with their associated motor findings, help you to identify where the causative lesions might be. A lesion in the sensory cortex may not impair the perception of pain, touch, and position, for example, but does impair finer discrimination. A person so affected cannot appreciate the size, shape, or texture of an object by feeling it and therefore cannot identify it. Loss of position and vibration sense with

preservation of other sensations points to disease of the posterior columns, while loss of all sensations from the waist down, together with paralysis and hyperactive reflexes in the legs, indicates transection of the spinal cord (see Table 16-5, p. 603). Crude and light touch are often preserved despite partial damage to the cord because impulses originating on one side of the body travel up both sides of the cord.

A knowledge of *dermatomes* also aids in localizing neurologic lesions. *A dermatome is the band of skin innervated by the sensory root of a single spinal nerve.* Dermatome patterns are mapped in the next two figures. Their lev-



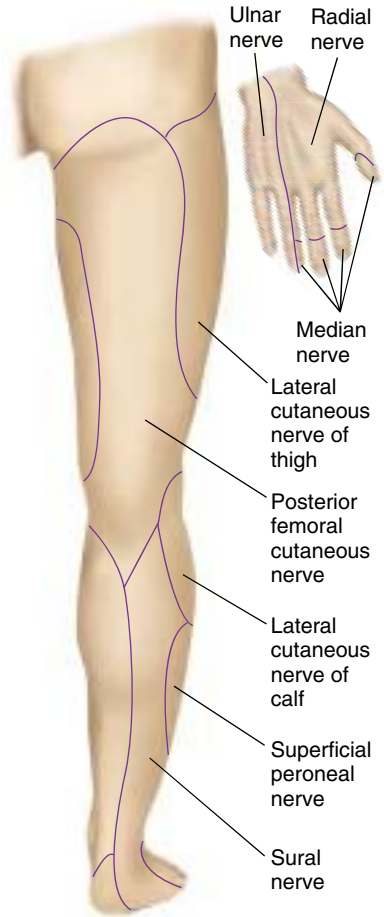
AREAS INNERVATED BY PERIPHERAL NERVES



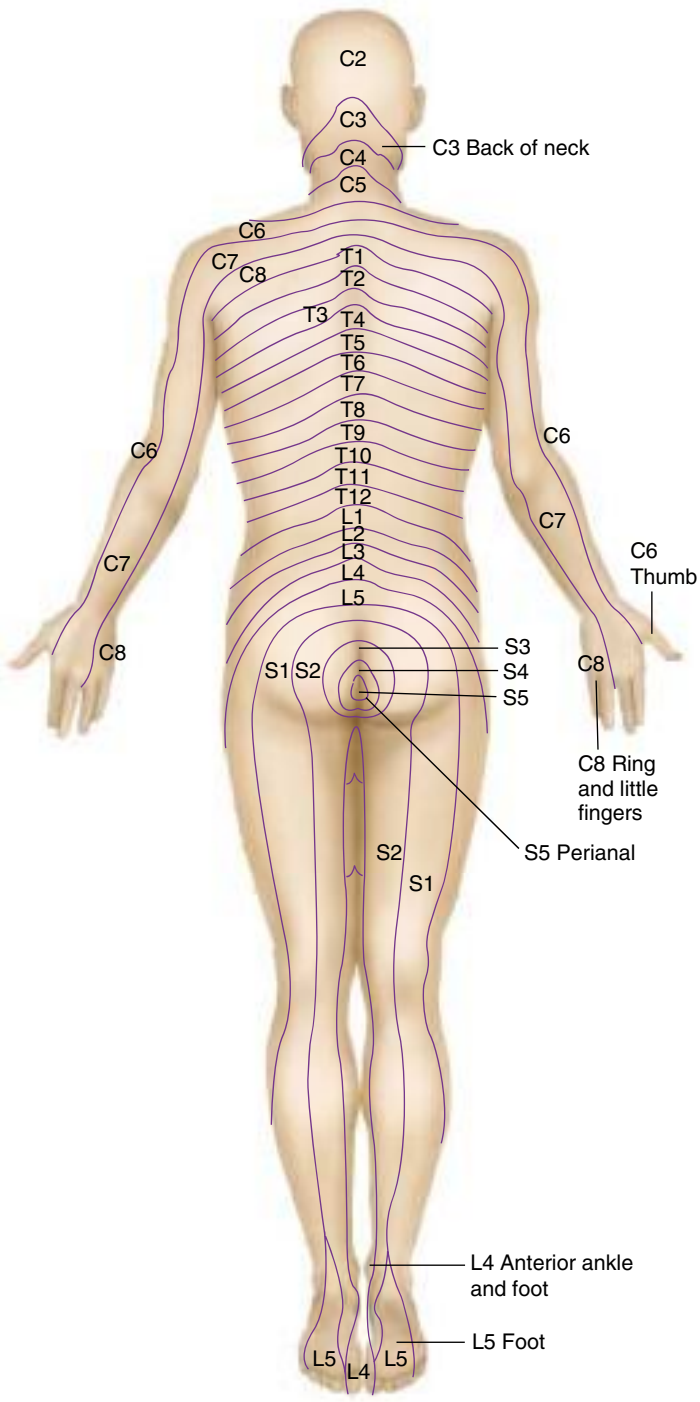
DERMATOMES INNERVATED BY POSTERIOR ROOTS

els are considerably more variable than the diagrams suggest, and dermatomes overlap each other. The sensory nerves from each side of the body overlap slightly across the midline. The distribution of a few key peripheral nerves is shown in the inserts on the left.

Do not try to memorize all the dermatomes. It is useful, however, to remember the locations of some, such as those shaded in yellow on the right side of the diagrams.



AREAS INNERVATED BY PERIPHERAL NERVES



DERMATOMES INNERVATED BY POSTERIOR ROOTS

Changes With Aging

Aging may affect all aspects of the nervous system, from mental status to motor and sensory function and reflexes. Age-related losses may take their toll on the mental function of an elderly person. These include the deaths of loved ones and friends, retirement from valued employment, diminution in income, decreased physical capacities including impairments in vision and hearing, and perhaps decreased stimulation or growing isolation. In addition, biologic changes affect the aging brain. Brain volume and the number of cortical brain cells decrease, and both microanatomic and biochemical changes have been identified. Nevertheless, most men and women adapt well to getting older. They maintain their self-esteem, they alter their activities in ways that are appropriate to their changing capacities and circumstances, and eventually they ready themselves for death.

In assessing the nervous system of an older person, it is sometimes difficult to distinguish the changes of normal aging from those of age-related or other diseases. Some findings that you would consider abnormal in younger people, however, occur often enough in the elderly that you may attribute them to aging alone. Alterations in hearing, vision, extraocular movements, and pupillary size, shape, and reactivity have been described in Chapter 5 (see pp. 134–136).

Changes in the motor system are common. Older persons move and react with less speed and agility than younger ones, and skeletal muscles decrease in bulk. The hands of an aged person often look thin and bony because their small muscles have atrophied. Look for such muscular wasting in the backs of the hands, where atrophy of the dorsal interosseous muscles may leave concavities or grooves. As illustrated on page 572, this change is often most evident between the thumb and the hand (1st and 2nd metacarpals) but may also be seen between the other metacarpals. Atrophy of small muscles may also flatten the thenar and hypothenar eminences of the palms. Muscle strength, though diminished, is relatively well maintained. Arm and leg muscles may also show atrophy. This sometimes exaggerates the apparent size of adjacent joints.

Occasionally, an older person develops a benign essential tremor in the head, jaw, lips, or hands that may be confused with parkinsonism (p. 608). Unlike parkinsonian tremors, however, benign tremors are slightly faster and disappear at rest, and there is no associated muscle rigidity.

Vibration sense is frequently decreased or lost in the feet and ankles (but not in the fingers or over the shins). Less commonly, position sense may diminish or disappear.

Aging may also alter some of the reflexes. The gag reflex may be diminished or absent. Ankle reflexes may be symmetrically decreased or absent, even when reinforced. Less commonly, knee reflexes are similarly affected. Abdominal reflexes may diminish or disappear and, partly because of musculoskeletal changes in the feet, the plantar responses become less obvious and more difficult to interpret.

If changes such as those described are accompanied by other neurologic abnormalities, or if atrophy and reflex changes are asymmetric, you should search for an explanation other than age alone.

Most elderly people do well on a mental status examination, but functional impairments may become evident, especially at advanced ages. Many older people complain about their memories. "Benign forgetfulness" is the usual explanation and may occur at any age. This term refers to a difficulty in recalling the names of people or objects or certain details of specific events. Naming this common phenomenon, when appropriate, may help to reassure a person who is worried that it signifies Alzheimer's disease. In addition to this circumscribed forgetfulness, elderly people retrieve and process data more slowly, and they take more time to learn new material. Their motor responses may slow, and their ability to perform complex tasks may become impaired.

The clinician must often try to distinguish these age-related changes from the manifestations of specific mental disorders, some of which are more prevalent with aging, such as depression and dementia. Sorting out these ailments from medical complaints may be difficult, particularly since both mood disturbances and cognitive changes may impair the patient's ability to recognize or report symptoms. Older patients are also more susceptible to delirium, a temporary confusional state that may be the first clue to infection or problems with medications. The clinician must learn to recognize these conditions promptly and to protect the patient from harm. (Information on detection of these conditions can be found on pp. 549–551, the Health History, on pp. 551–553, Health Promotion and Counseling, and in Table 16-1, Disorders of Mood, p. 599, and Table 16-5, Delirium and Dementia, p. 603.)

THE HEALTH HISTORY

Common or Concerning Symptoms

- Changes in mood, attention, or speech
- Changes in orientation, memory, insight, or judgment
- Delirium or dementia
- Headache
- Dizziness or vertigo
- Generalized, proximal, or distal weakness
- Numbness, abnormal or loss of sensations
- Loss of consciousness, syncope, or near-syncope
- Seizures
- Tremors or involuntary movements

Much of the information about the patient's *mental status* becomes evident during the interview. As you talk to the patient and listen to the patient's story, you should assess *level of consciousness,* general *appearance and mood,* and *ability to pay attention, remember, understand,* and *speak*. By placing the patient's vocabulary and general fund of information in the context of the patient's cultural and educational background, you can often make a rough estimate of intelligence. Likewise, the patient's responses to illness and life circumstances often tell you about his or her degree of *insight and judgment.* If the patient has unusual thoughts, preoccupations, beliefs, or perceptions, you should explore them as they arise during the interview. If you suspect a problem in *orientation and memory,* you can ask, "Let's see, your last clinic appointment was when . . . ?" . . . "And the date today?" The more you can integrate your exploration of mental status into a sensitive patient history, the less it will seem like an interrogation.

See Table 16-1, Disorders of Mood, p. 599, and Table 16-2, Disorders of Speech, p. 600.

See Table 16-3, Anxiety Disorders, p. 601, and Table 16-4, Psychotic Disorders, p. 602.

For some patients, you will need to supplement your interview with questions in specific areas. You may determine the need to go further and pursue a formal mental status examination. The components of the mental status examination are described in the section on Techniques of Examination, pp. 556–557.

All patients with documented or suspected brain lesions, psychiatric symptoms, or reports of vague or changed behavioral symptoms by family members need further systematic assessment. Patients may have subtle behavioral changes, difficulty taking medications properly, problems attending to household chores or paying bills, or loss of interest in their usual activities. Other patients may behave strangely after surgery or during an acute illness. Each problem should be identified as expeditiously as possible. Mental function influences the ability to hold a job and is often important in evaluating disability.

Possible signs of depression or dementia

See Table 16-5, Delirium and Dementia, p. 603.

Two of the most common symptoms in neurologic disorders are *headache* and *dizziness.* Turn to p. 136 and p. 139 to review the health history pertinent to these symptoms.

For *headache,* be sure to ask about location, severity, how long it lasts, and any associated symptoms, such as visual changes, weakness, or loss of sensation. Ask if the headache is affected by coughing, sneezing, or sudden movements of the head.

See Table 5-1, Headaches, pp. 170–173.

Subarachnoid hemorrhage may evoke "the worst headache of my life." Dull headache affected by such maneuvers, especially on awakening and recurring in the same location, is seen with mass lesions such as a brain tumor.

The complaint of *dizziness* can have many meanings. You will need to elicit exactly what the patient has experienced. Is the patient light-headed or feeling faint? Or is there *vertigo,* a perception that the room is spinning or rotating?

Light-headedness in palpitations, near syncope from vasovagal stimulation, low blood pressure, febrile illness, and others. Vertigo in middle-ear conditions, brainstem tumor. See Table 5-2, p. 174.

Especially in older patients, are any medications contributing to the dizziness? Are there any associated symptoms such as double vision, or *diplopia,* difficulty forming words, or *dysarthria,* or difficulty with gait or balance, or *ataxia*?

Diplopia, dysarthria, ataxia in posterior circulation transient ischemic attack (TIA) or stroke

What about any associated *weakness,* either generalized or in the face or a part of the body? Weakness is another common symptom and requires careful attention to detail. Probe for exactly what it means to the patient. Explore whether there is any *paralysis,* or inability to move a part or side of the body. Did the weakness start slowly or suddenly? Has it progressed? How? What areas of the body are involved? Does the weakness affect one or both sides? What movements are affected?

Weakness or paralysis in transient ischemic attack or stroke

Focal weakness may arise from ischemic, vascular, or mass lesions in the central nervous system; also from peripheral nervous system disorders, neuromuscular disorders, or the muscles themselves.

For weakness without light-headedness, try to distinguish between *proximal* and *distal weakness.* For proximal weakness, ask about combing hair, trying to reach something on a high shelf, or difficulty getting up out of a chair or taking a high step up. Does the weakness increase with repeated effort and improve after rest? Are there associated sensory or other symptoms? For distal weakness in the arms, inquire about hand movements such as opening a jar or can, or using hand tools such as scissors, pliers, or a screwdriver. For distal weakness in the legs, ask about frequent tripping.

Bilateral proximal weakness in myopathy. Bilateral, predominantly distal weakness in polyneuropathy. Weakness made worse with repeated effort and improved with rest suggests myasthenia gravis.

Find out if the patient has had any *loss of sensation*. Ask if there has been any *numbness,* but clarify its meaning and location. Has there been loss of sensation, difficulty moving a limb, or altered sensations such as tingling or pins and needles? There may be peculiar sensations without an obvious stimulus, called *paresthesias.* These occur commonly when an arm or leg "goes to sleep" following compression of a nerve, and may be described as tingling, prickling, or feelings of warmth, coldness, or pressure. *Dysesthesias* are distorted sensations in response to a stimulus and may last longer than the stimulus itself. For example, a person may perceive a light touch or pinprick as a burning or tingling sensation that is irritating or unpleasant. *Pain* may arise from neurologic causes but is usually reported with symptoms of other body systems, such as the head and neck or the musculoskeletal system.

Loss of sensation, paresthesias, and dysesthesias in central lesions in the brain and spinal cord, as well as disorders of peripheral sensory roots and nerves; paresthesias in the hands and around the mouth in hyperventilation.

See Table 5-1, Headaches, pp. 170–173, Table 15-2, Pains in the Neck, p. 523, and Table 15-1, Low Back Pain, p. 522.

"Have you ever fainted or passed out?" leads the discussion to any *loss of consciousness.* It is important to begin by exploring what the patient means by loss of consciousness. Did the patient black out completely, or could voices be heard throughout the episode, indicating some consciousness? Be sure to use descriptive terms carefully and precisely. *Syncope* is the sudden but temporary loss of consciousness that occurs with decreased blood flow to the brain, commonly described as *fainting.* Symptoms of feeling faint, lightheaded, or weak, but without actual loss of consciousness, are called *near syncope* or *presyncope.*

See Table 16-6, Syncope and Similar Disorders, pp. 604–605.

Get as complete and unbiased a description of the event as you can. What brought on the episode? Were there any warning symptoms? Was the patient standing, sitting, or lying down when the episode began? How long did it last? Could voices be heard while passing out and coming to? How rapidly did the patient recover? In retrospect, were onset and offset slow or fast?

Young people with emotional stress and warning symptoms of flushing, warmth, or nausea may have *vasodepressor (or vasovagal) syncope* of slow onset, slow offset. *Cardiac syncope* from arrhythmias, more common in older patients, often with sudden onset, sudden offset.

Also ask if anyone observed the episode. If so, what did the patient look like before losing consciousness, during the episode, and afterward? Was there any seizure-like movement of the arms or legs? Any incontinence of the bladder or bowel? Any drowsiness or impaired memory after the episode ended?

Tonic-clonic motor activity, bladder or bowel incontinence, and *postictal state* suggest a generalized *seizure.* Unlike syncope, injury such as tongue biting or bruising of limbs may occur.

A *seizure* is a paroxysmal disorder caused by sudden excessive electrical discharge in the cerebral cortex or its underlying structures. Seizures can be of several types. Depending on the type, there may or may not be loss of consciousness. With some types of seizures, there may be abnormal feelings, thought processes, and sensations, including smells, as well as abnormal movements. Asking "Have you ever had any seizures or 'spells'?" . . . "Any fits or convulsions?" can open the discussion. As with syncope, aim for a full and complete description, including precipitating circumstances, warnings, and behavior and feelings both during the attack and afterward. Ask about age at onset, frequency, any change in frequency or symptom pattern, and use of medications. Is there any history of prior head injury or other conditions that may be causally related?

See Table 16-7, Seizure Disorders, pp. 606–607.

Tremors and other *involuntary movements* occur with or without additional neurologic manifestations. Ask about any trembling, shakiness, or body movements that the patient seems unable to control.

See Table 16-8, Involuntary Movements, pp. 608–609.

Distinct from these symptoms is an almost indescribable *restlessness of the legs* that typically develops at rest and is accompanied by an urge to move about. Walking gives relief.

The common but often overlooked restless legs syndrome, usually benign

HEALTH PROMOTION AND COUNSELING

Important Topics for Health Promotion and Counseling

- Screening for depression and suicidality
- Screening for dementia
- Prevention of TIAs or stroke

Up to a third of all primary care visits involve mental health—depressed mood, anxiety, somatic concerns, and more serious disorders of mood and mental function. The burden of suffering imposed by these disorders is great. For the general population, focus health promotion and counseling on depression, suicidality, and dementia, three important conditions often overlooked. You should also screen for use of drugs or alcohol (see pp. 43–45).

The lifetime prevalence of major depression meeting formal diagnostic criteria is 5% to 10% in men and 10% to 20% in women. Primary-care providers fail to diagnose major depression in up to 50% of affected patients, often missing early clues such as low self-esteem, anhedonia (failure to find pleasure in daily activities), sleep disorders, and difficulty in concentrating or making decisions. Routine general screening has not been shown to improve outcomes; rather, target diagnosis and treatment to patients who are symptomatic. Watch carefully for depressive symptoms, especially in patients who are young, female, single, divorced, separated, seriously or chronically ill, or bereaved. Patients with a prior history of depression or positive family history are also at risk. Failure to diagnose depression can have consequences that are fatal—suicide rates in patients with major depression are eight times higher than in the general population.*

Clinicians must be adept at eliciting suicidal ideation or intent (see Chap 2, p. 47). Suicide rates are highest among men over age 65, but have been increasing in teenagers and young adults. Risk factors include any history of psychiatric illness (especially if linked to a hospital admission), substance abuse, personality disorder, prior suicide attempt, or family history of suicide. Clinicians should ask about domestic firearms and screen for alcohol dependence: guns are present in the home of more than half of all suicide victims, and alcohol intoxication is associated with nearly 25% of suicide deaths. Any evidence of suicidal ideation must be further assessed. Has a weapon been obtained? Is there a plan or a note? Such patients should be promptly referred for mental health and psychiatric care and for treatment of any related problems of alcohol or drug abuse.

Dementia, a "global impairment of cognitive function that interferes with normal activities,"† affects 16% of Americans over 65. Prominent features include short- and long-term memory deficits and impaired judgment. Thought processes are impoverished and speech may be hesitant due to difficulty in finding words. Loss of orientation to place may make navigating by foot or car problematic or even dangerous. Most dementias represent Alzheimer's disease (~50%–85%) or vascular multi-infarct dementia (~10%–20%). Be watchful for Alzheimer's disease in individuals with a pos-

*U.S. Preventive Services Task Force: Ch. 49: "Screening for Depression." In *Guide to Clinical Preventive Services.* Baltimore, Williams and Wilkins, pp. 541–546, 1996.
†U.S. Preventive Services Task Force: Ch. 48: "Screening for Dementia." In *Guide to Clinical Preventive Services.* Baltimore, Williams and Wilkins, pp. 531–541, 1996.

itive family history, since their risk is three times higher than in the general population.

Dementia often has a slow, insidious onset and may escape detection by both families and clinicians, especially in its initial stages. Currently there are no reliable screening tests to help you detect dementia early in its course. Clinicians should be alert to evidence of change in cognitive function or activities of daily living, and to family complaints about new or unusual patient behaviors. Use of the Mini-Mental State Examination is helpful for assessing cognitive impairment (although scores may be affected by level of education and cultural variables such as language). Once cognitive change is identified, be sure to address the possible role of medications, depression, or metabolic abnormalities. Couple cognitive and behavioral assessment with a careful neurologic examination during your evaluation of the patient. Be sure to look for other medical and psychiatric conditions that could be contributing to changes in behavior or level of daily activity. For demented patients and affected families, counseling about the potential for disruptive behavior, accidents and falls, and termination of driving privileges is warranted. Clinicians can foster discussion of legal matters such as power of attorney and advanced directives while the patient is still able to contribute to decision-making.

Finally, direct clinical attention to averting cerebrovascular accidents (CVAs). Strokes, or CVAs, are the third leading cause of death in the United States and contribute to extensive disability in the workforce and general population. The incidence of stroke increases with age and is 60% higher in African Americans compared to Caucasians. The clinician's first task in stroke prevention is to control hypertension. Hypertension accelerates atherosclerotic changes in the carotid, vertebral, and cerebral arteries and disturbs autoregulation of cerebral blood pressure. It is the leading risk factor for both ischemic and hemorrhagic stroke, which account for approximately 85% and approximately 10% of all CVAs, respectively. In addition, clinicians should counsel patients to modify conditions contributing to atherosclerosis: smoking, hyperlipidemia, and diabetes. Drug users should be warned of the link between stroke and cocaine.

Clinicians should be alert to symptoms of transient ischemic attacks (TIAs), generally defined as neurologic events that resolve within 24 hours. TIAs can be viewed as CVA warning signals, the anginal equivalent of the brain. In the first year after a TIA, risk of CVA is 6%–7%, and the CVA usually occurs in the same vascular distribution as the TIA. Common symptoms of TIAs include visual loss (especially transient monocular blindness from emboli), aphasia, dysarthria, and changes in facial movement or sensation. For TIAs affecting motor or sensory pathways, watch for clumsiness, weakness, paralysis, or tingling or paresthesias of the arm, leg, or hemibody.

Preview: Recording the Examination—The Nervous System

Note that initially you may use sentences to describe your findings; later you will use phrases. The style below contains phrases appropriate for most write-ups. Unfamiliar terms are explained in the next section, Techniques of Examination. Note that there are five components to the examination and write-up of the nervous system.

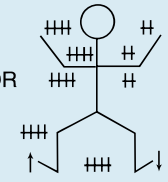
"*Mental Status:* Alert, relaxed, and cooperative. Thought process coherent. Oriented to person, place, and time. Detailed cognitive testing deferred. *Cranial Nerves:* I—not tested; II through XII intact. *Motor:* Good muscle bulk and tone. Strength 5/5 throughout. Cerebellar: Rapid alternating movements (RAMs), finger-to-nose (F→N), heel-to-shin (H→S) intact. Gait with normal base. Romberg—maintains balance with eyes closed. No pronator drift. *Sensory:* Pinprick, light touch, position, and vibration intact. *Reflexes:* 2+ and symmetric with plantar reflexes downgoing."

OR

"*Mental Status:* The patient is alert and tries to answer questions but has difficulty finding words. *Cranial Nerves:* I—not tested; II—visual acuity intact; visual fields full; III, IV, VI—extraocular movements intact; V motor—temporal and masseter strength intact, sensory corneal reflexes present; VII motor—prominent right facial droop and flattening of right nasolabial fold, left facial movements intact, sensory—taste not tested; VIII—hearing intact bilaterally to whispered voice; IX, X—gag intact; XI—strength of sternomastoid and trapezius muscles 5/5; XII—tongue midline. *Motor:* strength in right biceps, triceps, iliopsoas, gluteals, quadriceps, hamstring, and ankle flexor and extensor muscles 3/5 with good bulk but increased tone and spasticity; strength in comparable muscle groups on the left 5/5 with good bulk and tone. Gait—unable to test. Cerebellar—unable to test on right due to right arm and leg weakness; RAMs, F→N, H→S intact on left. Romberg—unable to test due to right leg weakness. Right pronator drift present. *Sensory:* decreased sensation to pinprick over right face, arm, and leg; intact on the left. Stereognosis and two-point discrimination not tested. *Reflexes* (can record in two ways):

	Biceps	Triceps	Brach	Knee	Ankle	PI
RT	4+	4+	4+	4+	4+	↑
LT	2+	2+	2+	2+	1+	↓

OR



Suggests left hemispheric CVA in distribution of the left middle cerebral artery, with right-sided hemiparesis

TECHNIQUES OF EXAMINATION

Important Areas of Examination

- Mental status: appearance and behavior, speech and language, mood, thoughts and perceptions, cognition
- Cranial Nerves I through XII
- Motor system: muscle bulk, tone, and strength; coordination, gait, and stance
- Sensory system: pain and temperature, position and vibration, light touch, discrimination
- Deep tendon, abdominal, and plantar reflexes

Three important questions govern the neurologic examination:

- Is the mental status intact?
- Are right-sided and left-sided findings symmetric?
- And, if the findings are asymmetric or otherwise abnormal, does the causative lesion lie in the central nervous system or the peripheral nervous system?

In this section, you will learn the techniques for a practical and reasonably comprehensive examination of the nervous system. It is important to master the techniques for a thorough examination. At first these techniques may seem difficult, but with practice, dedication, and supervision you will come to feel comfortable evaluating neurologic symptoms and disease. You should be active in your learning and ask your instructors or even neurologists to review your skills.

The detail of an appropriate neurologic examination varies widely. As you gain experience, you will find that in healthy persons your examination will come to be relatively brief. When you detect abnormal findings, your examination will become more comprehensive. Be aware that neurologists may use many other techniques in specific situations.

For efficiency, you should integrate certain portions of the neurologic assessment with other parts of your examination. Survey the patient's mental status and speech during the interview, for example, even though you may wish to do further testing during your neurologic evaluation. Assess some of the cranial nerves as you examine the head and neck, and inspect the arms and legs for neurologic abnormalities while you also observe the peripheral vascular and musculoskeletal systems. Chapter 3 provides an outline for this kind of integrated approach. Think about and describe your findings, however, in terms of the nervous system as a unit.

Organize your thinking into five categories: (1) mental status, speech and language, (2) cranial nerves, (3) the motor system, (4) the sensory system, and (5) reflexes. If your findings are abnormal, begin to group them into patterns of central or peripheral disorders.

Mental Status

Your assessment of mental status, such as the General Survey, begins with the first words of the interview. As you gather the health history, you will quickly discern the patient's level of *alertness* and *orientation, mood, attention,* and *memory.* As the history unfolds, you will learn about the patient's *insight* and *judgment,* as well as any *recurring or unusual thoughts or perceptions.* For some, you will need to supplement your interview with specific questions and a more formal evaluation of mental status. Just as symptoms, blood pressure, and valvular murmurs help you to distinguish, for example, health from disease in the cardiovascular system, specific components of mental function illuminate the workings of the mind. Although these components do not encompass all the aspects of human thought and feeling, they serve as useful and continually important clinical tools.

Components of the mental status examination include:

- Appearance and behavior
- Speech and language
- Mood
- Thoughts and perceptions
- Cognitive function, including memory, attention, information and vocabulary, calculations, and abstract thinking and constructional ability.

Many of these terms are familiar to you from social conversation. Take the time to learn their special meaning in the context of a formal mental status evaluation.

COMPONENTS OF THE MENTAL STATUS EXAMINATION

Level of consciousness	Alertness or state of awareness of the environment
Attention	The ability to focus or concentrate over time on one task or activity—an inattentive or distractible person with impaired consciousness has difficulty giving a history or responding to questions.
Memory	The process of registering or recording information, tested by asking for immediate repetition of material, followed by storage or retention of information. *Recent or short-term memory* covers minutes, hours, or days; *remote or long-term memory* refers to intervals of years.

Orientation	Awareness of personal identity, place, and time; requires both memory and attention
Perceptions	Sensory awareness of objects in the environment and their interrelationships (external stimuli); also refers to internal stimuli such as dreams or hallucinations
Thought processes	The logic, coherence, and relevance of the patient's thought as it leads to selected goals, or *how* people think
Thought content	*What* the patient thinks about, including level of insight and judgment
Insight	Awareness that symptoms or disturbed behaviors are normal or abnormal; for example, distinguishing between daydreams and hallucinations that seem real
Judgment	Process of comparing and evaluating alternatives when deciding on a course of action; reflects values that may or may not be based on reality and social conventions or norms
Affect	An observable, usually episodic, feeling tone expressed through voice, facial expression, and demeanor
Mood	A more sustained emotion that may color a person's view of the world (mood is to affect as climate is to weather)
Language	A complex symbolic system for expressing, receiving, and comprehending words; as with consciousness, attention, and memory, language is essential for assessing other mental functions
Higher cognitive functions	Assessed by vocabulary, fund of information, abstract thinking, calculations, construction of objects that have two or three dimensions

Distinguishing the interplay of body and mind in relation to these attributes is very important but not always easy. Mental disorders such as anxiety or depression may take the form of somatic complaints. Likewise, physical illness can cause mental and emotional responses and in older patients, can impair mental function without causing typical symptoms or signs such as fever or pain. Always look carefully for physical or pharmacologic causes as you try to understand the context and emotional meaning of changes in mental status. Some mental status evaluations are complicated by personality factors, psychodynamics, or the patient's personal experiences, areas that can be explored during the interview (but not covered in this chapter). By integrating and correlating all the relevant data, the clinician tries to understand the person as a whole.

As a student, you may feel reluctant to perform mental status examinations, wondering if they will upset patients, invade their privacy, or result in labeling their thoughts or behavior as pathologic. Such concerns are understandable and appropriate. An insensitive examination of mental status may alarm

a patient, and even a skillful examination may bring to conscious awareness an embarrassing or upsetting deficit that the patient was trying to ignore. You may wish to discuss some of these concerns with your instructor or other experienced clinicians. As with other realms of interviewing and assessment, your skills and confidence will improve with practice and rewards will follow. Remember that many patients will appreciate an understanding listener, and some will owe their health, their safety, or even their lives to your attention.

The format that follows should help to organize your observations, but it is not intended as a step-by-step guide. When a full examination is indicated, you should be flexible in your approach but thorough in what you cover. In some situations, however, sequence is important. If during your initial interview the patient's consciousness, attention, comprehension of words, or ability to speak seems impaired, assess this attribute promptly. Such a patient cannot give a reliable history and you will not be able to test most of the other mental functions.

APPEARANCE AND BEHAVIOR

Use here all the relevant observations made throughout the course of your history and examination. Include these areas:

Level of Consciousness. Is the patient awake and alert? Does the patient seem to understand your questions and respond appropriately and reasonably quickly, or is there a tendency to lose track of the topic and fall silent or even asleep?

See the table on Level of Consciousness (Arousal), p. 595.

If the patient does not respond to your questions, escalate the stimulus in steps:

- Speak to the patient by name and in a loud voice.

Lethargic patients are drowsy but open their eyes and look at you, respond to questions, and then fall asleep.

- Shake the patient gently, as if awakening a sleeper.

Obtunded patients open their eyes and look at you, but respond slowly and are somewhat confused.

If there is no response to these stimuli, promptly assess the patient for stupor or coma—severe reductions in the level of consciousness (see p. 595).

Posture and Motor Behavior. Does the patient lie in bed, or prefer to walk about? Note body posture and the patient's ability to relax. Observe the pace, range, and character of movements. Do they seem to be under voluntary control? Are certain parts immobile? Do posture and motor activity change with topics under discussion or with activities or people around the patient?

Tense posture, restlessness, and fidgetiness of anxiety; crying, pacing, and handwringing of agitated depression; hopeless, slumped posture and slowed movements of depression; singing, dancing, and expansive movements of a manic episode.

TECHNIQUES OF EXAMINATION	EXAMPLES OF ABNORMALITIES
Dress, Grooming, and Personal Hygiene. How is the patient dressed? Is clothing clean, pressed, and properly fastened? How does it compare with clothing worn by people of comparable age and social group? Note the patient's hair, nails, teeth, skin, and, if present, beard. How are they groomed? How do the person's grooming and hygiene compare with those of other people of comparable age, lifestyle, and socioeconomic group? Compare one side of the body with the other.	Grooming and personal hygiene may deteriorate in depression, schizophrenia, and dementia. Excessive fastidiousness may be seen in an obsessive–compulsive disorder. One-sided neglect may result from a lesion in the opposite parietal cortex, usually the nondominant side.
Facial Expression. Observe the face, both at rest and when the patient is interacting with others. Watch for variations in expression with topics under discussion. Are they appropriate? Or is the face relatively immobile throughout?	Expressions of anxiety, depression, apathy, anger, elation. Facial immobility of parkinsonism
Manner, Affect, and Relationship to Persons and Things. Using your observations of facial expressions, voice, and body movements, assess the patient's affect. Does it vary appropriately with topics under discussion, or is the affect labile, blunted, or flat? Does it seem inappropriate or extreme at certain points? If so, how? Note the patient's openness, approachability, and reactions to others and to the surroundings. Does the patient seem to hear or see things that you do not or seem to be conversing with someone who is not there?	Anger, hostility, suspiciousness, or evasiveness of paranoid patients. Elation and euphoria of the manic syndrome. Flat affect and remoteness of schizophrenia. Apathy (dulled affect with detachment and indifference) in dementia. Anxiety, depression

SPEECH AND LANGUAGE

Throughout the interview, note the characteristics of the patient's speech, including the following:

Quantity. Is the patient talkative or relatively silent? Are comments spontaneous or only responsive to direct questions?

Rate. Is speech fast or slow?

Slow speech of depression; accelerated rapid, loud speech in mania

Loudness. Is speech loud or soft?

Articulation of Words. Are the words spoken clearly and distinctly? Is there a nasal quality to the speech?

Dysarthria refers to defective articulation. *Aphasia* refers to a disorder of language. See Table 16-2, Disorders of Speech, p. 600.

Fluency. This involves the rate, flow, and melody of speech and the content and use of words. Be alert for abnormalities of spontaneous speech such as these:

- Hesitancies and gaps in the flow and rhythm of words
- Disturbed inflections, such as a monotone
- Circumlocutions, in which phrases or sentences are substituted for a word the person cannot think of, such as "what you write with" for "pen"

These abnormalities suggest aphasia. The patient may have so much difficulty in talking or in understanding others that you may not be able to obtain a history. You may also falsely suspect a psychotic disorder.

- Paraphasias, in which words are malformed ("I write with a den"), wrong ("I write with a bar"), or invented ("I write with a dar").

If the patient's speech lacks meaning or fluency, proceed with further testing as outlined in the following table.

Testing for Aphasia

Word Comprehension	Ask the patient to follow a one-stage command, such as "Point to your nose." Try a two-stage command: "Point to your mouth, then your knee."
Repetition	Ask the patient to repeat a phrase of one-syllable words (the most difficult repetition task): "No ifs, ands, or buts."
Naming	Ask the patient to name the parts of a watch.
Reading Comprehension	Ask the patient to read a paragraph aloud.
Writing	Ask the patient to write a sentence.

These tests help you to decide what kind of aphasia the patient may have. Remember that deficiencies in vision, hearing, intelligence, and education may also affect performance. Two common kinds of aphasia—Wernicke's and Broca's—are compared in Table 16-2, Disorders of Speech, p. 600.

A person who can write a correct sentence does not have aphasia.

MOOD

Assess mood during the interview by exploring the patient's own perceptions of it. Find out about the patient's usual mood level and how it has varied with life events. "How did you feel about that?", for example, or, more generally, "How are your spirits?" The reports of relatives and friends may be of great value.

Moods include sadness and deep melancholy; contentment, joy, euphoria, and elation; anger and rage; anxiety and worry; and detachment and indifference.

What has the patient's mood been like? How intense has it been? Has it been labile or fairly unchanging? How long has it lasted? Is it appropriate to the patient's circumstances? In case of depression, have there also been episodes of an elevated mood, suggesting a bipolar disorder?

For depressive and bipolar disorders, see Table 16-1, Disorders of Mood, p. 599.

If you suspect depression, assess its depth and any associated risk of suicide. A series of questions such as the following is useful, proceeding as far as the patient's positive answers warrant.

> Do you get pretty discouraged (or depressed or blue)?
> How low do you feel?
> What do you see for yourself in the future?
> Do you ever feel that life isn't worth living? Or that you would just as soon be dead?
> Have you ever thought of doing away with yourself?
> How did (do) you think you would do it?
> What would happen after you were dead?

Asking about suicidal thoughts does not implant the idea in the patient's mind, and it may be the only way to get the information. Although many

student clinicians feel uneasy about exploring this topic, most patients can discuss their thoughts and feelings about it freely with you, sometimes with considerable relief. By such discussion, you demonstrate your interest and concern for what may well be the patient's most serious and threatening problem. By avoiding the issue, you may miss the most important feature of the patient's illness.

THOUGHT AND PERCEPTIONS

Thought Processes. Assess the logic, relevance, organization, and coherence of the patient's thought processes as they are revealed in words and speech throughout the interview. Does speech progress in a logical manner toward a goal? Here you are using the patient's speech as a window into the patient's mind. Listen for patterns of speech that suggest disorders of thought processes, as outlined in the table below.

Variations and Abnormalities in Thought Processes

		EXAMPLES OF ABNORMALITIES
Circumstantiality	Speech characterized by indirection and delay in reaching the point because of unnecessary detail, although the components of the description have a meaningful connection. Many people without mental disorders speak circumstantially.	Observed in obsessional persons
Derailment (Loosening of Associations)	Speech in which a person shifts from one subject to others that are unrelated or only obliquely related without realizing that the subjects are not meaningfully connected. Ideas slip off the track between clauses, not within them.	Observed in schizophrenia, manic episodes, and other psychotic disorders
Flight of Ideas	An almost continuous flow of accelerated speech in which a person changes abruptly from topic to topic. Changes are usually based on understandable associations, plays on words, or distracting stimuli, but the ideas do not progress to sensible conversation.	Most frequently noted in manic episodes
Neologisms	Invented or distorted words, or words with new and highly idiosyncratic meanings	Observed in schizophrenia, other psychotic disorders, and aphasia
Incoherence	Speech that is largely incomprehensible because of illogic, lack of meaningful connections, abrupt changes in topic, or disordered grammar or word use. Shifts in meaning occur within clauses. Flight of ideas, when severe, may produce incoherence.	Observed in severely disturbed psychotic persons (usually schizophrenic)
Blocking	Sudden interruption of speech in midsentence or before completion of an idea. The person attributes this to losing the thought. Blocking occurs in normal people.	Blocking may be striking in schizophrenia.
Confabulation	Fabrication of facts or events in response to questions, to fill in the gaps in an impaired memory	Common with amnesia
Perseveration	Persistent repetition of words or ideas	Occurs in schizophrenia and other psychotic disorders
Echolalia	Repetition of the words and phrases of others	Occurs in manic episodes and schizophrenia
Clanging	Speech in which a person chooses a word on the basis of sound rather than meaning, as in rhyming and punning speech. For example, "Look at my eyes and nose, wise eyes and rosy nose. Two to one, the ayes have it!"	Occurs in schizophrenia and manic episodes

Thought Content. You should ascertain most of the information relevant to thought content during the interview. Follow appropriate leads as they occur rather than using stereotyped lists of specific questions. For example, "You mentioned a few minutes ago that a neighbor was responsible for your entire illness. Can you tell me more about that?" Or, in another situation, "What do you think about at times like these?"

You may need to make more specific inquiries. If so, couch them in tactful and accepting terms. "When people are upset like this, they sometimes can't keep certain thoughts out of their minds," or ". . . things seem unreal. Have you experienced anything like this?"

In these ways find out about any of the patterns shown in the following table.

Abnormalities of Thought Content

Compulsions	Repetitive behaviors or mental acts that a person feels driven to perform in order to produce or prevent some future state of affairs, although expectation of such an effect is unrealistic
Obsessions	Recurrent, uncontrollable thoughts, images, or impulses that a person considers unacceptable and alien
Phobias	Persistent, irrational fears, accompanied by a compelling desire to avoid the stimulus
Anxieties	Apprehensions, fears, tensions, or uneasiness that may be focused (phobia) or free floating (a general sense of ill-defined dread or impending doom)
Feelings of Unreality	A sense that things in the environment are strange, unreal, or remote
Feelings of Depersonalization	A sense that one's self is different, changed, or unreal, or has lost identity or become detached from one's mind or body
Delusions	False, fixed, personal beliefs that are not shared by other members of the person's culture or subculture. Examples include: ■ *Delusions of persecution* ■ *Grandiose delusions* ■ *Delusional jealousy* ■ *Delusions of reference,* in which a person believes that external events, objects, or people have a particular and unusual personal significance (e.g., that the radio or television might be commenting on or giving instructions to the person) ■ *Delusions of being controlled* by an outside force ■ *Somatic delusions* of having a disease, disorder, or physical defect ■ *Systematized delusions,* a single delusion with many elaborations or a cluster of related delusions around a single theme, all systematized into a complex network

Compulsions, obsessions, phobias, and anxieties are often associated with neurotic disorders. See Table 16-3, Anxiety Disorders (p. 601).

Delusions and feelings of unreality or depersonalization are more often associated with psychotic disorders. See Table 16-4, Psychotic Disorders (p. 602). Delusions may also occur in delirium, severe mood disorders, and dementia.

Perceptions. Inquire about false perceptions in a manner similar to that used for thought content. For example, "When you heard the voice speaking to you, what did it say? How did it make you feel?" Or, "After you've been drinking a lot, do you ever see things that aren't really there?" Or, "Sometimes after major surgery like this, people hear peculiar or frightening things. Have you experienced anything like that?" In these ways find out about the following abnormal perceptions.

Abnormalities of Perception		
Illusions	Misinterpretations of real external stimuli	Illusions may occur in grief reactions, delirium, acute and posttraumatic stress disorders, and schizophrenia.
Hallucinations	Subjective sensory perceptions in the absence of relevant external stimuli. The person may or may not recognize the experiences as false. Hallucinations may be auditory, visual, olfactory, gustatory, tactile, or somatic. (False perceptions associated with dreaming, falling asleep, and awakening are not classified as hallucinations.)	Hallucinations may occur in delirium, dementia (less commonly), posttraumatic stress disorder, schizophrenia, and alcoholism.

Insight and Judgment. These attributes are usually best assessed during the interview.

Insight. Some of your very first questions to the patient often yield important information about insight: "What brings you to the hospital?" "What seems to be the trouble?" "What do you think is wrong?" More specifically, note whether or not the patient is aware that a particular mood, thought, or perception is abnormal or part of an illness.

Patients with psychotic disorders often lack insight into their illness. Denial of impairment may accompany some neurologic disorders.

Judgment. You can usually assess judgment by noting the patient's responses to family situations, jobs, use of money, and interpersonal conflicts. "How do you plan to get the help you'll need after leaving the hospital?" "How are you going to manage if you lose your job?" "If your husband starts to abuse you again, what will you do?" "Who will attend to your financial affairs while you are in the nursing home?"

Judgment may be poor in delirium, dementia, mental retardation, and psychotic states. Judgment is affected also by anxiety, mood disorders, intelligence, education, socioeconomic options, and cultural values.

Note whether decisions and actions are based on reality or, for example, on impulse, wish fulfillment, or disordered thought content. What values seem to underlie the patient's decisions and behavior? Allowing for cultural variations, how do these compare with mature adult standards? Because judgment is part of the maturational response, it may be variable and unpredictable during adolescence.

COGNITIVE FUNCTIONS

Orientation. By skillful questioning you can often determine the patient's orientation in the context of the interview. For example, you can ask quite naturally for specific dates and times, the patient's address and

Disorientation occurs especially when memory or attention is impaired, as in delirium.

telephone number, the names of family members, or the route taken to the hospital. At times—when rechecking the status of a delirious patient, for example—simple, direct questions may be indicated.

"Can you tell me what time it is now . . . and what day is it?" In either of these ways, determine the patient's orientation for the following:

- *Time* (e.g., the time of day, day of the week, month, season, date and year, duration of hospitalization)
- *Place* (e.g., the patient's residence, the names of the hospital, city, and state)
- *Person* (e.g., the patient's own name, and the names of relatives and professional personnel)

Attention. These tests of attention are commonly used:

Digit Span. Explain that you would like to test the patient's ability to concentrate, perhaps adding that people tend to have trouble with that when they are in pain, or ill, or feverish. Recite a series of digits, starting with two at a time and speaking each number clearly at a rate of about one per second. Ask the patient to repeat the numbers back to you. If this repetition is accurate, try a series of three numbers, then four, and so on as long as the patient responds correctly. Jotting down the numbers as you say them helps to ensure your own accuracy. If the patient makes a mistake, try once more with another series of the same length. Stop after a second failure in a single series.

Causes of poor performance include delirium, dementia, mental retardation, and performance anxiety.

In choosing digits you may use street numbers, zip codes, telephone numbers, and other numerical sequences that are familiar to you, but avoid consecutive numbers, easily recognized dates, and sequences that possibly are familiar to the patient.

Now, starting again with a series of two, ask the patient to repeat the numbers to you backward.

Normally, a person should be able to repeat correctly at least five digits forward and four backward.

Serial 7s. Instruct the patient, "Starting from a hundred, subtract 7, and keep subtracting 7. . . ." Note the effort required and the speed and accuracy of the responses. (Writing down the answers helps you keep up with the arithmetic.) Normally, a person can complete serial 7s in 1½ minutes, with fewer than four errors. If the patient cannot do serial 7s, try 3s or counting backward.

Poor performance may be due to delirium, the late stage of dementia, mental retardation, loss of calculating ability, anxiety, or depression. Also consider the possibility of limited education.

Spelling Backward. This can substitute for serial 7s. Say a five-letter word, spell it, e.g., W-O-R-L-D, and ask the patient to spell it backward.

Remote Memory. Inquire about birthdays, anniversaries, social security number, names of schools attended, jobs held, or past historical events such as wars relevant to the patient's past.

Remote memory may be impaired in the late stage of dementia.

Recent Memory (e.g., the events of the day). Ask questions with answers that you can check against other sources so that you will know whether or not the patient is confabulating (making up facts to compensate for a defective memory). These might include the day's weather, today's appointment time, and medications or laboratory tests taken during the day. (Asking what the patient had for breakfast may be a waste of time unless you can check the accuracy of the answer.)

Recent memory is impaired in dementia and delirium. See Table 16-5, Delirium and Dementia, p. 603. *Amnestic disorders* impair memory or new learning ability significantly and reduce a person's social or occupational functioning, but they do not have the global features of delirium or dementia. Anxiety, depression, and mental retardation may also impair recent memory.

New Learning Ability. Give the patient three or four words such as "83 Water Street and blue," or "table, flower, green, and hamburger." Ask the patient to repeat them so that you know that the information has been heard and registered. This step, like digit span, tests registration and immediate recall. Then proceed to other parts of the examination. After about 3 to 5 minutes, ask the patient to repeat the words. Note the accuracy of the response, awareness of whether or not it is correct, and any tendency to confabulate. Normally, a person should be able to remember the words.

HIGHER COGNITIVE FUNCTIONS

Information and Vocabulary. Information and vocabulary, when observed clinically, provide a rough estimate of a person's intelligence. Assess them during the interview. Ask a student, for example, about favorite courses, or inquire about a person's work, hobbies, reading, favorite television programs, or current events. Explore such topics first with simple questions, then with more difficult ones. Note the person's grasp of information, the complexity of the ideas expressed, and the vocabulary used.

If considered in the context of cultural and educational background, information and vocabulary are fairly good indicators of intelligence. They are relatively unaffected by any but the most severe psychiatric disorders, and may be helpful for distinguishing mentally retarded adults (whose information and vocabulary are limited) from those with mild or moderate dementia (whose information and vocabulary are fairly well preserved).

More directly, you can ask about specific facts, such as these:

The name of the president, vice president, or governor
The names of the last four or five presidents
The names of five large cities in the country

Calculating Ability. Test the patient's ability to do arithmetical calculations, starting at the rote level with simple addition ("What is 4 + 3? . . . 8 + 7?") and multiplication ("What is 5 × 6? . . . 9 × 7?"). The task can be made more difficult by using two-digit numbers ("15 + 12" or "25 × 6") or longer, written examples.

Poor performance may be a useful sign of dementia or may accompany aphasia, but it must be assessed in terms of the patient's intelligence and education.

Alternatively, pose practical and functionally important questions, such as "If something costs 78 cents and you give the clerk one dollar, how much should you get back?"

Abstract Thinking. The capacity to think abstractly can be tested in two ways.

Concrete responses are often given by persons with mental retardation, delirium, or dementia, but may also be simply a function of limited education. Schizophrenics may respond concretely or with personal, bizarre interpretations.

Proverbs. Ask the patient what people mean when they use some of the following proverbs:

A stitch in time saves nine.
Don't count your chickens before they're hatched.
The proof of the pudding is in the eating.
A rolling stone gathers no moss.
The squeaking wheel gets the grease.

Note the relevance of the answers and their degree of concreteness or abstractness. For example, "You should sew a rip before it gets bigger" is concrete, while "Prompt attention to a problem prevents trouble" is abstract. Average patients should give abstract or semiabstract responses.

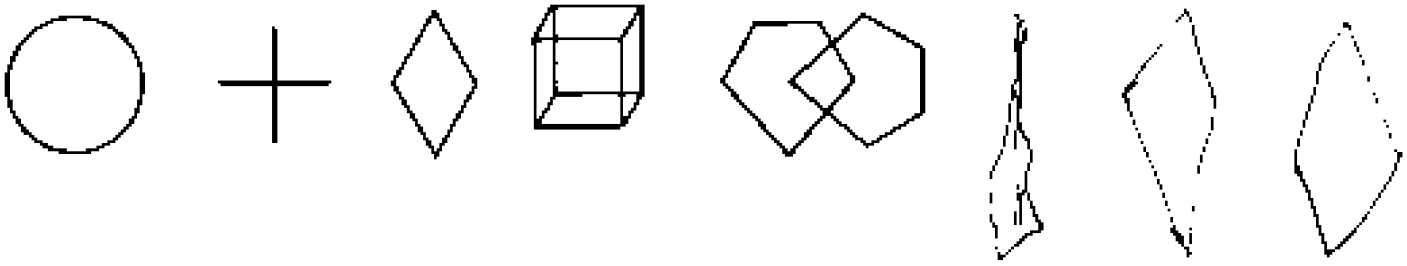
Similarities. Ask the patient to tell you how the following are alike:

An orange and an apple	A church and a theater
A cat and a mouse	A piano and a violin
A child and a dwarf	Wood and coal

Note the accuracy and relevance of the answers and their degree of concreteness or abstractness. For example, "A cat and a mouse are both animals" is abstract, "They both have tails" is concrete, and "A cat chases a mouse" is not relevant.

Constructional Ability. The task here is to copy figures of increasing complexity onto a piece of blank unlined paper. Show each figure one at a time and ask the patient to copy it as well as possible.

The three diamonds below are rated poor, fair, and good (but not excellent).



(Strub RL, Black FW: The Mental Status Examination in Neurology, 2nd ed. Philadelphia, FA Davis, 1985)

In another approach, ask the patient to draw a clock face complete with numbers and hands. The example below is rated excellent.

These three clocks are poor, fair, and good.

(Strub RL, Black FW: The Mental Status Examination in Neurology, 2nd ed. Philadelphia, FA Davis, 1985)

If vision and motor ability are intact, poor constructional ability suggests dementia or parietal lobe damage. Mental retardation may also impair performance.

The Cranial Nerves

Overview. The examination of the cranial nerves (often abbreviated as CN) can be summarized as follows:

I	Smell
II	Visual acuity, visual fields, and ocular fundi
II, III	Pupillary reactions
III, IV, VI	Extraocular movements
V	Corneal reflexes, facial sensation, and jaw movements
VII	Facial movements
VIII	Hearing
IX, X	Swallowing and rise of the palate, gag reflex
V, VII, X, XII	Voice and speech
XI	Shoulder and neck movements
XII	Tongue symmetry and position

Cranial Nerve I—Olfactory. Test the *sense of smell* by presenting the patient with familiar and nonirritating odors. First be sure that each nasal passage is open by compressing one side of the nose and asking the patient to sniff through the other. The patient should then close both eyes. Occlude one nostril and test smell in the other with such substances as cloves, coffee, soap, or vanilla. Ask if the patient smells anything and, if so, what. Test the other side. A person should normally perceive odor on each side, and can often identify it.

Loss of smell has many causes, including nasal disease, head trauma, smoking, aging, and the use of cocaine. It may be congenital.

Cranial Nerve II—Optic. Test *visual acuity* (see pp. 144–145).

Inspect the *optic fundi* with your ophthalmoscope, paying special attention to the optic discs (see pp. 151–155).

Optic atrophy, papilledema

Screen the visual fields by confrontation (see pp. 145–146). Occasionally—in a stroke patient, for example—screening indicates a visual field defect, such as a homonymous hemianopsia, that you cannot confirm by testing one eye at a time. This screening observation, nevertheless, is significant.

These findings suggest visual *extinction,* a subtle impairment detectable only when testing both eyes simultaneously. It suggests a lesion in the parietal cortex.

Cranial Nerves II and III—Optic and Oculomotor. Inspect the size and shape of the pupils, and compare one side with the other. Test the *pupillary reactions to light*; if these are abnormal, examine the *near response* also (see p. 149).

See Table 5-9, Pupillary Abnormalities (p. 181).

Cranial Nerves III, IV, and VI—Oculomotor, Trochlear, and Abducens. Test the *extraocular movements* in the six cardinal directions of gaze, and look for loss of conjugate movements in any of the six directions. Check convergence of the eyes. Identify any nystagmus, noting the direction of gaze in which it appears, the plane in which movements occur (horizontal, vertical, rotary, or mixed), and the direction of the quick and slow components (see pp. 149–151).

See Table 5-10, Deviations of the Eyes (p. 182).

See Table 16-9, Nystagmus (pp. 610–611).

Look for *ptosis* (drooping of the upper eyelids). A slight difference in the width of the palpebral fissures may be noted in about one third of all normal people.

Ptosis in 3rd nerve palsy, Horner's syndrome (ptosis, meiosis, anhidrosis), myasthenia gravis

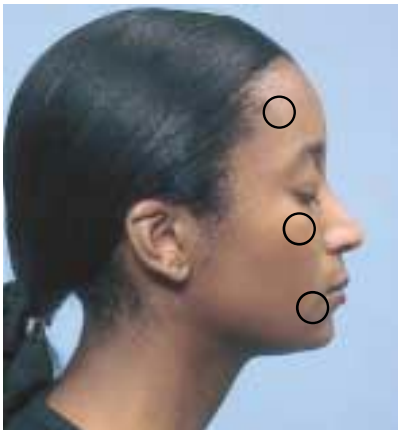
Cranial Nerve V—Trigeminal

Motor. While palpating the temporal and masseter muscles in turn, ask the patient to clench his or her teeth. Note the strength of muscle contraction.

Weak or absent contraction of the temporal and masseter muscles on one side suggests a lesion of CN V. Bilateral weakness may result from peripheral or central involvement. When the patient has no teeth, this test may be difficult to interpret.

PALPATING TEMPORAL MUSCLES

PALPATING MASSETER MUSCLES



Sensory. After explaining what you plan to do, test the forehead, cheeks, and jaw on each side for *pain sensation*. Suggested areas are indicated by the circles. The patient's eyes should be closed. Use a safety pin or other suitable sharp object,* occasionally substituting the blunt end for the point as a stimulus. Ask the patient to report whether it is "sharp" or "dull" and to compare sides.

Unilateral decrease in or loss of facial sensation suggests a lesion of CN V or of interconnecting higher sensory pathways. Such a sensory loss may also be associated with a conversion reaction.

*To avoid transmitting infection, use a new object with each patient. You can create a sharp wood splinter by breaking or twisting a cotton swab. The cotton end of the swab can also be used as a dull stimulus.

If you find an abnormality, confirm it by testing *temperature sensation*. Two test tubes, filled with hot and ice-cold water, are the traditional stimuli. A tuning fork may also be used. It usually feels cool. If you are near running water, the fork is easily made colder or warm. Dry it before use. Touch the skin and ask the patient to identify "hot" or "cold."

Then test for *light touch*, using a fine wisp of cotton. Ask the patient to respond whenever you touch the skin.

Test *the corneal reflex*. Ask the patient to look up and away from you. Approaching from the other side, out of the patient's line of vision, and avoiding the eyelashes, touch the cornea (not just the conjunctiva) lightly with a fine wisp of cotton. If the patient is apprehensive, however, first touching the conjunctiva may allay fear.

Look for blinking of the eyes, the normal reaction to this stimulus. (The sensory limb of this reflex is carried in CN V, the motor response in CN VII.) Use of contact lenses frequently diminishes or abolishes this reflex.

Absence of blinking suggests a lesion of CN V. A lesion of CN VII (the nerve to the muscles that close the eyes) may also impair this reflex.

Cranial Nerve VII—Facial. Inspect the face, both at rest and during conversation with the patient. Note any asymmetry (e.g., of the nasolabial folds), and observe any tics or other abnormal movements.

Flattening of the nasolabial fold and drooping of the lower eyelid suggest facial weakness.

A peripheral injury to CN VII, as in Bell's palsy, affects both the upper and the lower face; a central lesion affects mainly the lower face. See Table 16-10, Types of Facial Paralysis (pp. 612–613).

Ask the patient to:

1. Raise both eyebrows.

2. Frown.

3. Close both eyes tightly so that you cannot open them. Test muscular strength by trying to open them, as illustrated.

In unilateral facial paralysis, the mouth droops on the paralyzed side when the patient smiles or grimaces.

4. Show both upper and lower teeth.

5. Smile.

6. Puff out both cheeks.

Note any weakness or asymmetry.

Cranial Nerve VIII—Acoustic. Assess *hearing*. If hearing loss is present, (1) test for *lateralization,* and (2) compare *air and bone conduction* (see pp. 156–158).

See Table 5-19, Patterns of Hearing Loss (pp. 196–197).

Specific tests of *vestibular function* are seldom included in the usual neurologic examination. Consult textbooks of neurology or otolaryngology as the need arises.

Nystagmus may indicate vestibular dysfunction. See Table 16-9, Nystagmus (pp. 610–611).

Cranial Nerves IX and X—Glossopharyngeal and Vagus. Listen to the patient's *voice*. Is it hoarse or does it have a nasal quality?

Hoarseness in vocal cord paralysis; a nasal voice in paralysis of the palate

Is there difficulty in swallowing?

Pharyngeal or palatal weakness

Ask the patient to say "ah" or to yawn as you watch the *movements of the soft palate and the pharynx*. The soft palate normally rises symmetrically, the uvula remains in the midline, and each side of the posterior pharynx moves medially, like a curtain. The slightly curved uvula seen occasionally in a normal person should not be mistaken for a uvula deviated by a 10th nerve lesion.

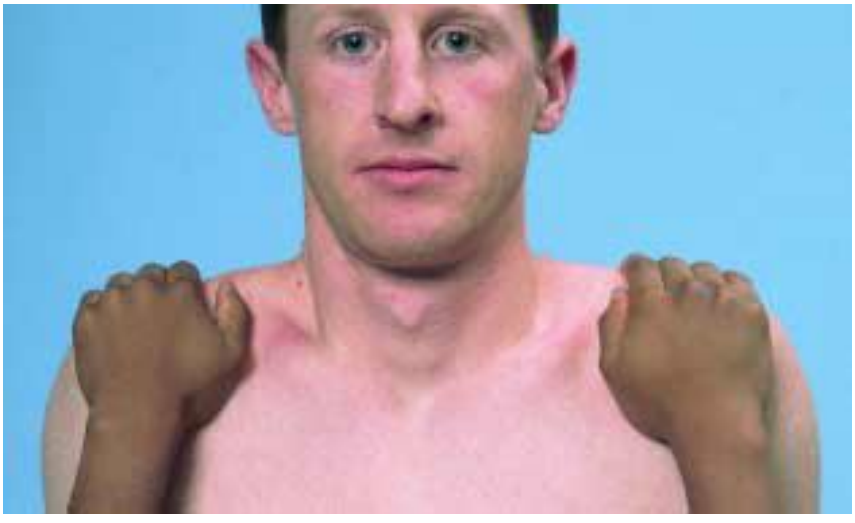
The palate fails to rise with a bilateral lesion of the vagus nerve. In unilateral paralysis, one side of the palate fails to rise and, together with the uvula, is pulled toward the normal side (see p. 162).

Warn the patient that you are going to test the *gag reflex*. Stimulate the back of the throat lightly on each side in turn and note the gag reflex. It may be symmetrically diminished or absent in some normal people.

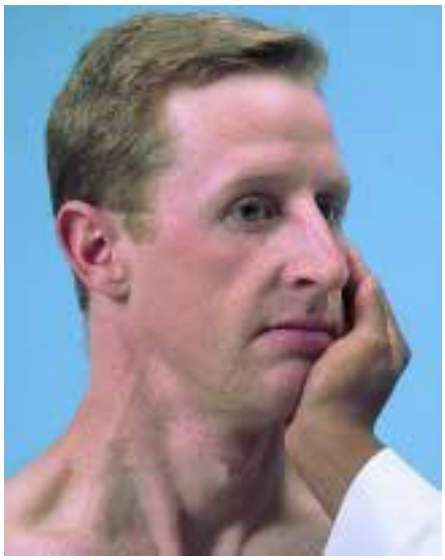
Unilateral absence of this reflex suggests a lesion of CN IX, perhaps CN X.

Cranial Nerve XI—Spinal Accessory. From behind, look for atrophy or fasciculations in the trapezius muscles, and compare one side with the

Weakness with atrophy and fasciculations indicates a peripheral nerve disorder. When the trapezius is paralyzed, the shoulder droops and the scapula is displaced downward and laterally.



other. Ask the patient to shrug both shoulders upward against your hands. Note the strength and contraction of the trapezii.



Ask the patient to turn his or her head to each side against your hand. Observe the contraction of the opposite sternomastoid and note the force of the movement against your hand.

A supine patient with bilateral weakness of the sternomastoids has difficulty raising the head off the pillow.

Cranial Nerve XII—Hypoglossal. Listen to the articulation of the patient's words. This depends on Cranial Nerves V, VII, and X as well as XII. Inspect the patient's tongue as it lies on the floor of the mouth. Look for any atrophy or *fasciculations* (fine, flickering, irregular movements in small groups of muscle fibers). Some coarser restless movements are often seen in a normal tongue. Then, with the patient's tongue protruded, look for asymmetry, atrophy, or deviation from the midline. Ask the patient to move the tongue from side to side, and note the symmetry of the movement. In ambiguous cases, ask the patient to push the tongue against the inside of each cheek in turn as you palpate externally for strength.

For poor articulation, or *dysarthria*, see Table 16-2, Disorders of Speech (p. 600). Atrophy and fasciculations in amyotrophic lateral sclerosis, polio

In a unilateral cortical lesion, the protruded tongue deviates transiently in a direction away from the side of the cortical lesion.

The Motor System

As you assess the motor system, focus on body position, involuntary movements, characteristics of the muscles (bulk, tone, and strength), and coordination. These components are described below in sequence. You may either use this sequence or check each component in the arms, legs, and trunk in turn. If you see an abnormality, identify the muscle(s) involved. Think about whether the abnormality is central or peripheral in origin, and begin to learn which nerves innervate the affected muscles.

Body Position. Observe the patient's body position during movement and at rest.

Abnormal positions alert you to neurologic deficits such as paralysis.

Involuntary Movements. Watch for involuntary movements such as tremors, tics, or fasciculations. Note their location, quality, rate, rhythm, and amplitude, and their relation to posture, activity, fatigue, emotion, and other factors.

See Table 16-8, Involuntary Movements (pp. 608–609).

Muscle Bulk. Compare the size and contours of muscles. Do the muscles look flat or concave, suggesting atrophy? If so, is the process unilateral or bilateral? Is it proximal or distal?

When looking for atrophy, pay particular attention to the hands, shoulders, and thighs. The thenar and hypothenar eminences should be full and convex, and the spaces between the metacarpals, where the dorsal interosseous muscles lie, should be full or only slightly depressed. Atrophy of hand muscles may occur with normal aging, however, as shown on the right below.

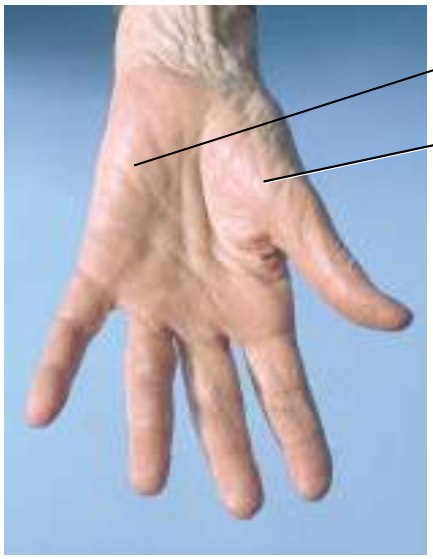
Muscular *atrophy* refers to a loss of muscle bulk (wasting). It results from diseases of the peripheral nervous system such as diabetic neuropathy, as well as diseases of the muscles themselves. *Hypertrophy* refers to an increase in bulk with proportionate strength, while increased bulk with diminished strength is called *pseudohypertrophy* (seen in the Duchenne form of muscular dystrophy).

Hand of a 44-year-old woman

Hand of an 84-year-old woman

Flattening of the thenar and hypothenar eminences and furrowing between the metacarpals suggest atrophy. Localized atrophy of the thenar and hypothenar eminences suggests damage to the median and ulnar nerves, respectively.

Hand of a 44-year-old woman



Hand of an 84-year-old woman

Other causes of muscular atrophy include motor neuron diseases, disuse of the muscles, rheumatoid arthritis, and protein-calorie malnutrition.

Be alert for fasciculations in atrophic muscles. If you see none, a tap on the muscle with a reflex hammer may stimulate them.

Fasciculations suggest lower motor neuron disease as a cause of atrophy.

Muscle Tone. When a normal muscle with an intact nerve supply is relaxed voluntarily, it maintains a slight residual tension known as muscle tone. This can be assessed best by feeling the muscle's resistance to passive stretch. Persuade the patient to relax. Take one hand with yours and, while supporting the elbow, flex and extend the patient's fingers, wrist, and elbow, and put the shoulder through a moderate range of motion. With practice, these actions can be combined into a single smooth movement. On each side, note muscle tone—the resistance offered to your movements. Tense patients may show increased resistance. You will learn the feel of normal resistance only with repeated practice.

Decreased resistance suggests disease of the peripheral nervous system, cerebellar disease, or the acute stages of spinal cord injury. See Table 16-11, Disorders of Muscle Tone (p. 614).

If you suspect decreased resistance, hold the forearm and shake the hand loosely back and forth. Normally the hand moves back and forth freely but is not completely floppy.

Marked floppiness indicates *hypotonic* or flaccid muscles.

If resistance is increased, determine whether it varies as you move the limb or whether it persists throughout the range of movement and in both directions, for example, during both flexion and extension. Feel for any jerkiness in the resistance.

Increased resistance that varies, commonly worse at the extremes of the range, is called *spasticity.* Resistance that persists throughout the range and in both directions is called *lead-pipe rigidity.*

To assess muscle tone in the legs, support the patient's thigh with one hand, grasp the foot with the other, and flex and extend the patient's knee and ankle on each side. Note the resistance to your movements.

Muscle Strength. Normal individuals vary widely in their strength, and your standard of normal, while admittedly rough, should allow for such variables as age, sex, and muscular training. A person's dominant side is usually slightly stronger than the other side. Keep this difference in mind when you compare sides.

Test muscle strength by asking the patient to move actively against your resistance or to resist your movement. Remember that a muscle is strongest when shortest, and weakest when longest.

Impaired strength is called weakness (*paresis*). Absence of strength is called paralysis (*plegia*). *Hemiparesis* refers to weakness of one half of the body; *hemiplegia* to paralysis of one half of the body. *Paraplegia* means paralysis of the legs; *quadriplegia,* paralysis of all four limbs.

If the muscles are too weak to overcome resistance, test them against gravity alone or with gravity eliminated. When the forearm rests in a pronated position, for example, dorsiflexion at the wrist can be tested against gravity alone. When the forearm is midway between pronation and supination, extension at the wrist can be tested with gravity eliminated. Finally, if the patient fails to move the body part, watch or feel for weak muscular contraction.

See Table 16-12, Disorders of the Central and Peripheral Nervous Systems (pp. 615–617).

Muscle strength is graded on a 0 to 5 scale:

0—No muscular contraction detected
1—A barely detectable flicker or trace of contraction
2—Active movement of the body part with gravity eliminated
3—Active movement against gravity
4—Active movement against gravity and some resistance
5—Active movement against full resistance without evident fatigue. This is normal muscle strength.

More experienced clinicians make further distinctions by using plus or minus signs toward the stronger end of this scale. Thus 4+ indicates good but not full strength, while 5− means a trace of weakness.

Methods for testing the major muscle groups are described below. The spinal root innervations and the muscles affected are shown in parentheses. To localize lesions in the spinal cord or the peripheral nervous system more precisely, additional testing may be necessary. For these specialized methods, refer to detailed texts of neurology.

Test flexion (C5, C6—biceps) *and extension* (C6, C7, C8—triceps) *at the elbow* by having the patient pull and push against your hand.

FLEXION

EXTENSION

EXTENSION AT WRIST

Test extension at the wrist (C6, C7, C8, radial nerve) by asking the patient to make a fist and resist your pulling it down.

Weakness of extension is seen in peripheral nerve disease (e.g., radial nerve damage) and in central nervous system disease producing hemiplegia (e.g., stroke or multiple sclerosis).

Test the grip (C7, C8, T1). Ask the patient to squeeze two of your fingers as hard as possible and not let them go. (To avoid getting hurt by hard squeezes, place your own middle finger on top of your index finger.) You should normally have difficulty removing your fingers from the patient's grip. Testing both grips simultaneously with arms extended or in the lap facilitates comparison.

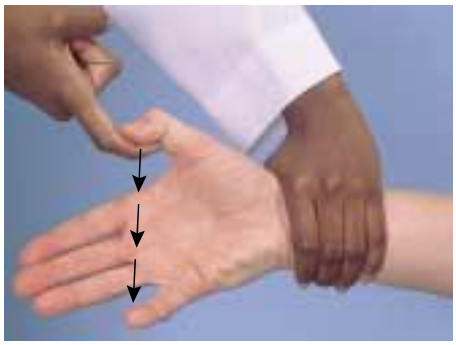
A weak grip may be due to either central or peripheral nervous system disease. It may also result from painful disorders of the hands.

Test finger abduction (C8, T1, ulnar nerve). Position the patient's hand with palm down and fingers spread. Instructing the patient not to let you move the fingers, try to force them together.

Weak finger abduction in ulnar nerve disorders

Test opposition of the thumb (C8, T1, median nerve). The patient should try to touch the tip of the little finger with the thumb, against your resistance.

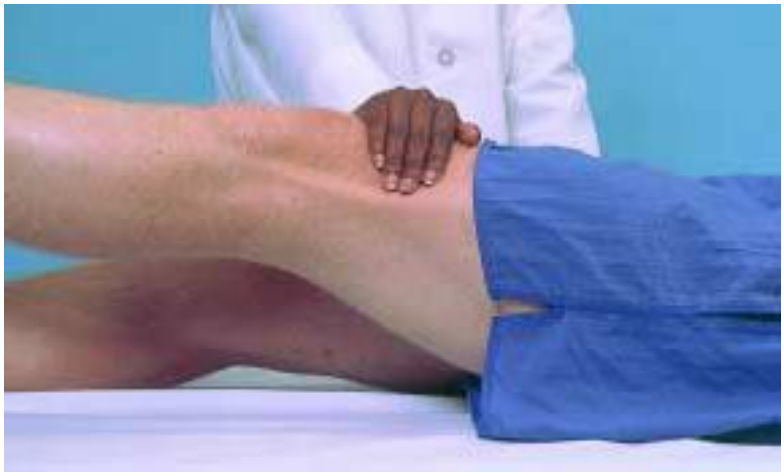
Weak opposition of the thumb in median nerve disorders such as carpal tunnel syndrome



Assessment of *muscle strength of the trunk* may already have been made in other segments of the examination. It includes:

- Flexion, extension, and lateral bending of the spine, and
- Thoracic expansion and diaphragmatic excursion during respiration

Test flexion at the hip (L2, L3, L4—iliopsoas) by placing your hand on the patient's thigh and asking the patient to raise the leg against your hand.



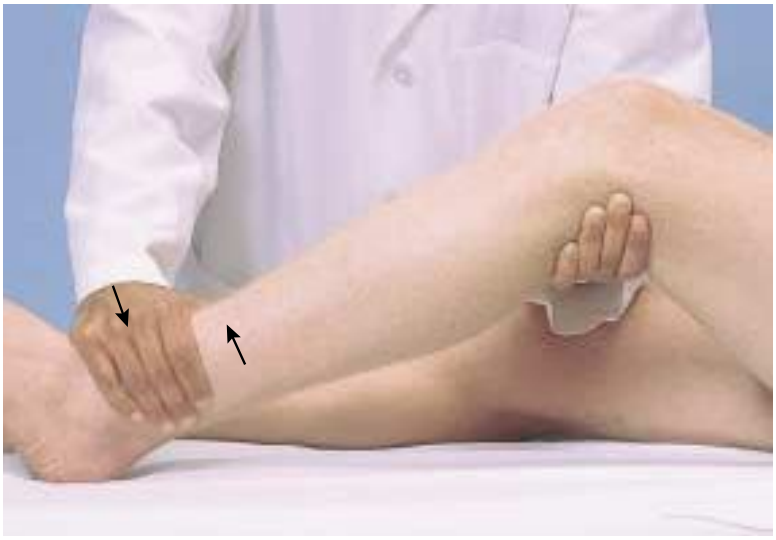
Test adduction at the hips (L2, L3, L4—adductors). Place your hands firmly on the bed between the patient's knees. Ask the patient to bring both legs together.

Symmetric weakness of the proximal muscles suggests a *myopathy* or muscle disorder; symmetric weakness of distal muscles suggests a *polyneuropathy,* or disorder of peripheral nerves.

Test abduction at the hips (L4, L5, S1—gluteus medius and minimus). Place your hands firmly on the bed outside the patient's knees. Ask the patient to spread both legs against your hands.

Test extension at the hips (S1—gluteus maximus). Have the patient push the posterior thigh down against your hand.

Test extension at the knee (L2, L3, L4—quadriceps). Support the knee in flexion and ask the patient to straighten the leg against your hand. The quadriceps is the strongest muscle in the body, so expect a forceful response.

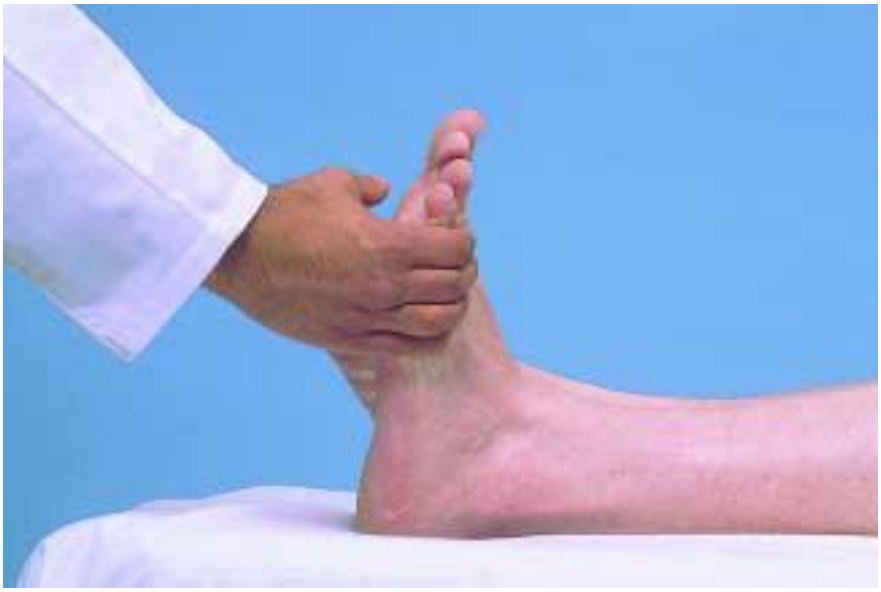


EXTENSION AT THE KNEE

Test flexion at the knee (L4, L5, S1, S2—hamstrings) as shown below. Place the patient's leg so that the knee is flexed with the foot resting on the bed. Tell the patient to keep the foot down as you try to straighten the leg.

FLEXION AT THE KNEE

Test dorsiflexion (mainly L4, L5) and *plantar flexion* (mainly S1) at the ankle by asking the patient to pull up and push down against your hand.



DORSIFLEXION

PLANTAR FLEXION

Coordination. Coordination of muscle movement requires that four areas of the nervous system function in an integrated way:

- The motor system, for muscle strength
- The cerebellar system (also part of the motor system), for rhythmic movement and steady posture

- The vestibular system, for balance and for coordinating eye, head, and body movements
- The sensory system, for position sense.

To assess coordination, observe the patient's performance in:

- Rapid alternating movements
- Point-to-point movements
- Gait and other related body movements
- Standing in specified ways.

Rapid Alternating Movements

ARMS. Show the patient how to strike one hand on the thigh, raise the hand, turn it over, and then strike the back of the hand down on the same place. Urge the patient to repeat these alternating movements as rapidly as possible.

Observe the speed, rhythm, and smoothness of the movements. Repeat with the other hand. The nondominant hand often performs somewhat less well.

In cerebellar disease, one movement cannot be followed quickly by its opposite and movements are slow, irregular, and clumsy. This abnormality is called *dysdiadochokinesis.* Upper motor neuron weakness and basal ganglia disease may also impair rapid alternating movements, but not in the same manner.

Show the patient how to tap the distal joint of the thumb with the tip of the index finger, again as rapidly as possible. Again, observe the speed, rhythm, and smoothness of the movements. The nondominant side often performs less well.

LEGS. Ask the patient to tap your hand as quickly as possible with the ball of each foot in turn. Note any slowness or awkwardness. The feet normally perform less well than the hands.

Dysdiadochokinesis in cerebellar disease

Point-To-Point Movements

ARMS. Ask the patient to touch your index finger and then his or her nose alternately several times. Move your finger about so that the patient has to alter directions and extend the arm fully to reach it. Observe the accuracy and smoothness of movements and watch for any tremor. Normally the patient's movements are smooth and accurate.

In cerebellar disease, movements are clumsy, unsteady, and inappropriately varying in their speed, force, and direction. The finger may initially overshoot its mark, but finally reaches it fairly well. Such movements are termed *dysmetria.* An intention tremor may appear toward the end of the movement (see p. 608).

Now hold your finger in one place so that the patient can touch it with one arm and finger outstretched. Ask the patient to raise the arm overhead and lower it again to touch your finger. After several repeats, ask the patient to close both eyes and try several more times. Repeat on the other side. Normally a person can touch the examiner's finger successfully with eyes open or closed. These maneuvers test position sense and the functions of both the labyrinth and the cerebellum.

Cerebellar disease causes incoordination that may get worse with eyes closed. Inaccuracy that appears with eyes closed suggests loss of position sense. Repetitive and consistent deviation to one side (referred to as *past pointing*), worse with the eyes closed, suggests cerebellar or vestibular disease.

LEGS. Ask the patient to place one heel on the opposite knee, and then run it down the shin to the big toe. Note the smoothness and accuracy of the movements. Repetition with the patient's eyes closed tests for position sense. Repeat on the other side.

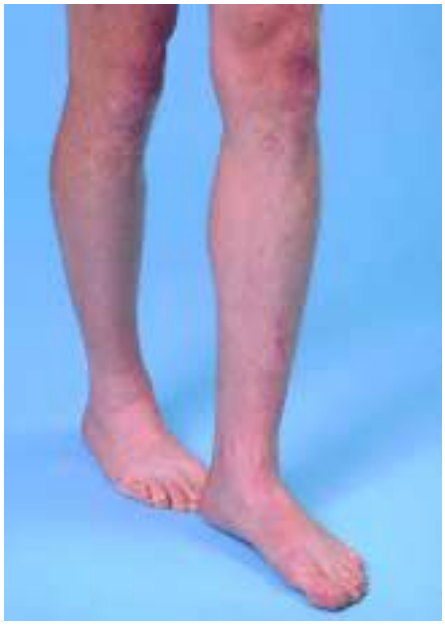
In cerebellar disease, the heel may overshoot the knee and then oscillate from side to side down the shin. When position sense is lost, the heel is lifted too high and the patient tries to look. With eyes closed, performance is poor.

Gait. Ask the patient to:

Abnormalities of gait increase risk of falls.

- *Walk across the room* or down the hall, then turn, and come back. Observe posture, balance, swinging of the arms, and movements of the legs. Normally balance is easy, the arms swing at the sides, and turns are accomplished smoothly.

A gait that lacks coordination, with reeling and instability, is called *ataxic.* Ataxia may be due to cerebellar disease, loss of position sense, or intoxication. See Table 16-13, Abnormalities of Gait and Posture (pp. 618–619).

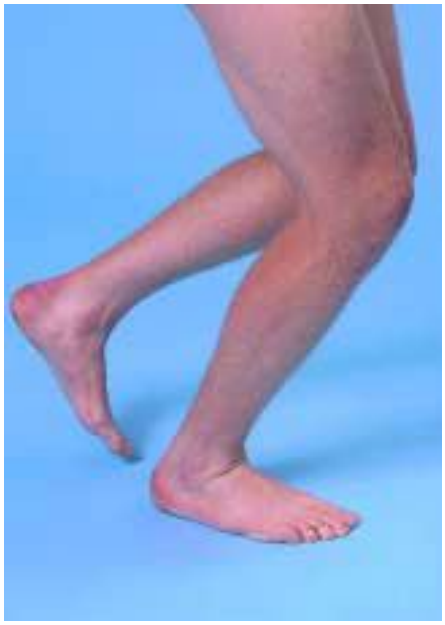


- *Walk heel-to-toe* in a straight line—a pattern called *tandem walking*.

Tandem walking may reveal an ataxia not previously obvious.

- *Walk on the toes,* then *on the heels*—sensitive tests respectively for plantar flexion and dorsiflexion of the ankles, as well as for balance.

Walking on toes and heels may reveal distal muscular weakness in the legs. Inability to heel-walk is a sensitive test for corticospinal tract weakness.



- *Hop in place* on each foot in turn (if the patient is not too ill). Hopping involves the proximal muscles of the legs as well as the distal ones and requires both good position sense and normal cerebellar function.

Difficulty with hopping may be due to weakness, lack of position sense, or cerebellar dysfunction.

- *Do a shallow knee bend,* first on one leg, then on the other. Support the patient's elbow if you think the patient is in danger of falling.

Difficulty here suggests proximal weakness (extensors of the hip), weakness of the quadriceps (the extensor of the knee), or both.

- *Rising from a sitting position* without arm support and *stepping up* on a sturdy stool are more suitable tests than hopping or knee bends when patients are old or less robust.

People with proximal muscle weakness involving the pelvic girdle and legs have difficulty with both of these activities.

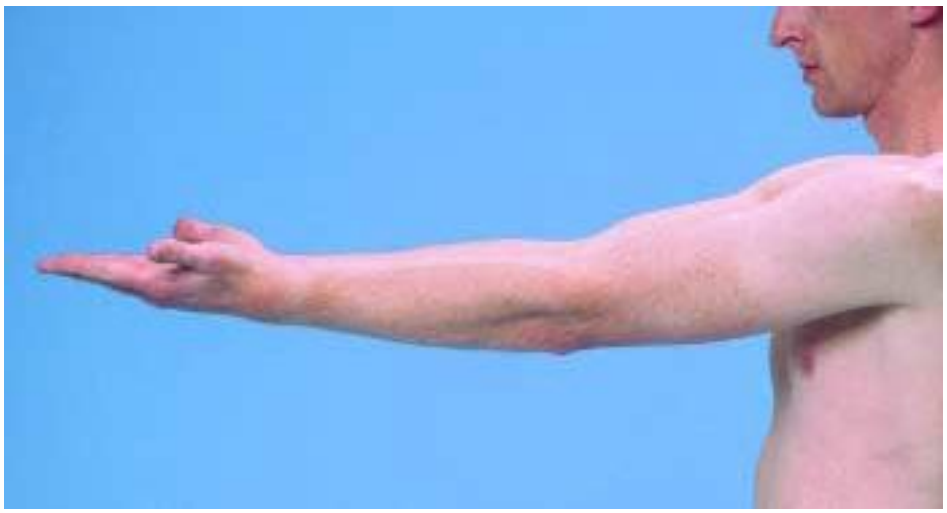
Stance. The following two tests can often be performed concurrently. They differ only in the patient's arm position and in what you are looking for. In each case, stand close enough to the patient to prevent a fall.

THE ROMBERG TEST. This is mainly a test of position sense. The patient should first stand with feet together and eyes open and then close both eyes for 20 to 30 seconds without support. Note the patient's ability to maintain an upright posture. Normally only minimal swaying occurs.

In ataxia due to loss of position sense, vision compensates for the sensory loss. The patient stands fairly well with eyes open but loses balance when they are closed, a *positive Romberg sign.* In cerebellar ataxia, the patient has difficulty standing with feet together whether the eyes are open or closed.

TEST FOR PRONATOR DRIFT. The patient should stand for 20 to 30 seconds with both arms straight forward, palms up, and with eyes closed. A person who cannot stand may be tested for a pronator drift in the sitting position. In either case, a normal person can hold this arm position well.

The pronation of one forearm suggests a contralateral lesion in the corticospinal tract; downward drift of the arm with flexion of fingers and elbow may also occur. These movements are called a *pronator drift,* shown below.



A sideward or upward drift, sometimes with searching, writhing movements of the hands, suggests loss of position sense.

Now, instructing the patient to keep the arms up and eyes shut, as shown above, *tap the arms briskly downward.* The arms normally return smoothly to the horizontal position. This response requires muscular strength, coordination, and a good sense of position.

A weak arm is easily displaced and often remains so. A patient lacking position sense may not recognize the displacement and, if told to correct it, does so poorly. In cerebellar incoordination, the arm returns to its original position but overshoots and bounces.

The Sensory System

To evaluate the sensory system, you will test several kinds of sensation:

- Pain and temperature (spinothalamic tracts)
- Position and vibration (posterior columns)
- Light touch (both of these pathways)
- Discriminative sensations, which depend on some of the above sensations but also involve the cortex

Familiarize yourself with each kind of test so that you can use it as indicated. When you detect abnormal findings, correlate them with motor and reflex activity. Is the underlying lesion central or peripheral?

See Table 16-12, Disorders of the Central and Peripheral Nervous Systems (pp. 615–617).

Patterns of Testing. Because sensory testing quickly fatigues many patients and then produces unreliable results, conduct the examination as efficiently as possible. Pay special attention to those areas (1) where there are symptoms such as numbness or pain, (2) where there are motor or reflex abnormalities that suggest a lesion of the spinal cord or peripheral nervous system, and (3) where there are trophic changes, such as absent or excessive sweating, atrophic skin, or cutaneous ulceration). Repeated testing at another time is often required to confirm abnormalities.

Meticulous sensory mapping helps to establish the level of a spinal cord lesion and to determine if a more peripheral lesion is in a nerve root, a major peripheral nerve, or one of its branches.

The following patterns of testing help you to identify sensory deficits accurately and efficiently.

- *Compare symmetric areas* on the two sides of the body, including the arms, legs, and trunk.

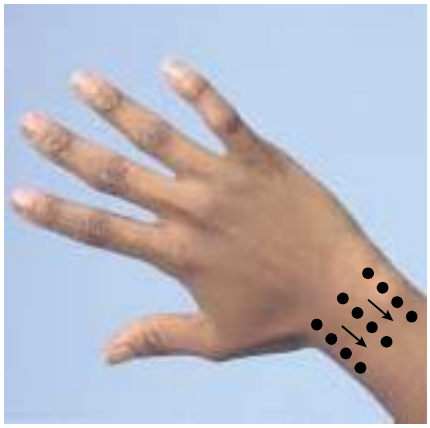
Hemisensory loss due to a lesion in the spinal cord or higher pathways

- When testing pain, temperature, and touch sensation, also *compare the distal with the proximal areas* of the extremities. Further, scatter the stimuli so as to sample most of the dermatomes and major peripheral nerves (see pp. 542–546). One suggested pattern includes both shoulders (C4), the inner and outer aspects of the forearms (C6 and T1), the thumbs and little fingers (C6 and C8), the fronts of both thighs (L2), the medial and lateral aspects of both calves (L4 and L5), the little toes (S1), and the medial aspect of each buttock (S3).

Symmetric distal sensory loss suggests a polyneuropathy, as described in the example on the next page. You may miss this finding unless you compare distal and proximal areas.

- When testing vibration and position sensation, first test the fingers and toes. If these are normal, you may safely assume that more proximal areas will also be normal.
- *Vary the pace of your testing.* This is important so that the patient does not merely respond to your repetitive rhythm.

- When you detect an area of sensory loss or hypersensitivity, *map out* its *boundaries* in detail. Stimulate first at a point of reduced sensation, and move by progressive steps until the patient detects the change. An example is shown at right.



Here all sensation in the hand is lost. Repetitive testing in a proximal direction reveals a gradual change to normal sensation at the wrist. This pattern fits neither a peripheral nerve nor a dermatome (see pp. 542–546). If bilateral, it suggests the "glove and stocking" sensory loss of a polyneuropathy, often seen in alcoholism and diabetes.

By identifying the distribution of sensory abnormalities and the kinds of sensations affected, you can infer where the causative lesion might be. Any motor deficit or reflex abnormality also helps in this localizing process.

Before each test below, show the patient what you plan to do and what responses you want. Unless otherwise specified, the patient's eyes should be closed during actual testing.

Pain. Use a sharp safety pin or other suitable tool. Occasionally, substitute the blunt end for the point. Ask the patient, "Is this sharp or dull?" or, when making comparisons, "Does this feel the same as this?" Apply the lightest pressure needed for the stimulus to feel sharp, and try not to draw blood.

Analgesia refers to absence of pain sensation, *hypalgesia* to decreased sensitivity to pain, and *hyperalgesia* to increased sensitivity.

To prevent transmitting a blood-borne infection, discard the pin or other device safely. Do not reuse it on another person.

Temperature. (This is often omitted if pain sensation is normal, but include it if there is any question.) Use two test tubes, filled with hot and cold water, or a tuning fork heated or cooled by water. Touch the skin and ask the patient to identify "hot" or "cold."

Light Touch. With a fine wisp of cotton, touch the skin lightly, avoiding pressure. Ask the patient to respond whenever a touch is felt, and to compare one area with another. Calloused skin is normally relatively insensitive and should be avoided.

Anesthesia is absence of touch sensation, *hypesthesia* is decreased sensitivity, and *hyperesthesia* is increased sensitivity.

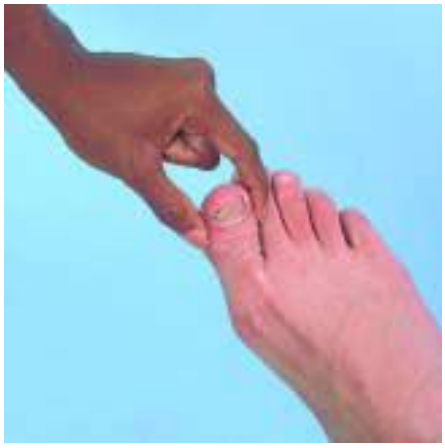
Vibration. Use a relatively low-pitched tuning fork of 128 Hz . Tap it on the heel of your hand and place it firmly over a distal interphalangeal joint of the patient's finger, then over the interphalangeal joint of the big toe. Ask what the patient feels. If you are uncertain whether it is pressure or vibration, ask the patient to tell you when the vibration stops, and then touch the fork to stop it. If

Vibration sense is often the first sensation to be lost in a peripheral neuropathy. Common causes include diabetes and alcoholism. Vibration sense is also lost in posterior column disease, as in tertiary syphilis or vitamin B_{12} deficiency.

Testing vibration sense in the trunk may be useful in estimating the level of a cord lesion.

vibration sense is impaired, proceed to more proximal bony prominences (e.g., wrist, elbow, medial malleolus, patella, anterior superior iliac spine, spinous processes, and clavicles).

Position. Grasp the patient's big toe, holding it by its sides between your thumb and index finger, and then pull it away from the other toes so as to avoid friction. (These precautions prevent extraneous tactile stimuli from revealing position changes that might not otherwise be detected.) Demonstrate "up" and "down" as you move the patient's toe clearly upward and downward. Then, with the patient's eyes closed, ask for a response of "up" or "down" when moving the toe in a small arc.



Loss of position sense, like loss of vibration sense, suggests either posterior column disease or a lesion of the peripheral nerve or root.

Repeat several times on each side, avoiding simple alternation of the stimuli. If position sense is impaired, move proximally to test it at the ankle joint. In a similar fashion, test position in the fingers, moving proximally if indicated to the metacarpophalangeal joints, wrist, and elbow.

Discriminative Sensations. Several additional techniques test the ability of the sensory cortex to correlate, analyze, and interpret sensations. Because discriminative sensations are dependent on touch and position sense, they are useful only when these sensations are either intact or only slightly impaired.

When touch and position sense are normal or only slightly impaired, a disproportionate decrease in or loss of discriminative sensations suggests disease of the sensory cortex. Stereognosis, number identification, and two-point discrimination are also impaired by posterior column disease.

Screen a patient with *stereognosis,* and proceed to other methods if indicated. The patient's eyes should be closed during all these tests.

- *Stereognosis.* Stereognosis refers to the ability to identify an object by feeling it. Place in the patient's hand a familiar object such as a coin, paper clip, key, pencil, or cotton ball, and ask the patient to tell you what it is. Normally a patient will manipulate it skillfully and identify it correctly. Asking the patient to distinguish "heads" from "tails" on a coin is a sensitive test of stereognosis.

Astereognosis refers to the inability to recognize objects placed in the hand.

- *Number identification (graphesthesia).* When motor impairment, arthritis, or other conditions pre-

The inability to recognize numbers, like astereognosis, suggests a lesion in the sensory cortex.

vent the patient from manipulating an object well enough to identify it, test the ability to identify numbers. With the blunt end of a pen or pencil, draw a large number in the patient's palm. A normal person can identify most such numbers.

- *Two-point discrimination.* Using the two ends of an opened paper clip, or the sides of two pins, touch a finger pad in two places simultaneously. Alternate the double stimulus irregularly with a one-point touch. Be careful not to cause pain.

  Find the minimal distance at which the patient can discriminate one from two points (normally less than 5 mm on the finger pads). This test may be used on other parts of the body, but normal distances vary widely from one body region to another.

  Lesions of the sensory cortex increase the distance between two recognizable points.

- *Point localization.* Briefly touch a point on the patient's skin. Then ask the patient to open both eyes and point to the place touched. Normally a person can do so accurately. This test, together with the test for extinction, is especially useful on the trunk and the legs.

  Lesions of the sensory cortex impair the ability to localize points accurately.

- *Extinction.* Simultaneously stimulate corresponding areas on both sides of the body. Ask where the patient feels your touch. Normally both stimuli are felt.

  With lesions of the sensory cortex, only one stimulus may be recognized. The stimulus on the side opposite the damaged cortex is extinguished.

Deep Tendon Reflexes

To elicit a *deep tendon reflex*, persuade the patient to relax, position the limbs properly and symmetrically, and strike the tendon briskly, using a rapid wrist movement. Your strike should be quick and direct, not glancing. You may use either the pointed or the flat end of the hammer. A properly weighted hammer is important. The pointed end is useful in striking small areas, such as your finger as it overlies the biceps tendon, while the flat end gives the patient less discomfort over the brachioradialis. Hold the reflex hammer between your thumb and index

finger so that it swings freely within the limits set by your palm and other fingers. Note the speed, force, and amplitude of the reflex response. Always compare one side with the other.

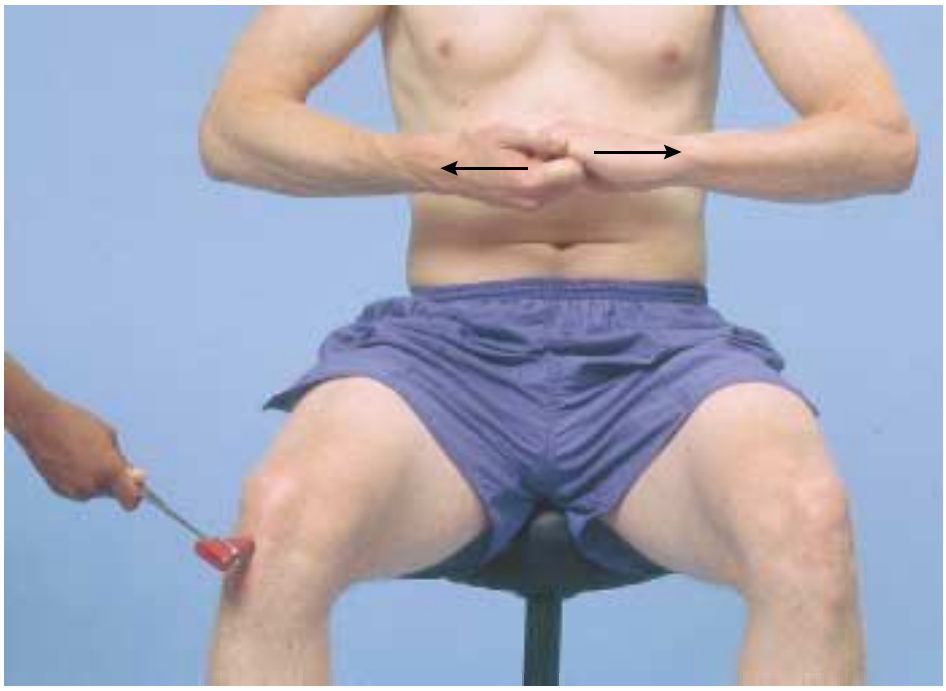
Reflexes are usually graded on a 0 to 4+ scale:

4+	Very brisk, hyperactive, with *clonus* (rhythmic oscillations between flexion and extension)
3+	Brisker than average; possibly but not necessarily indicative of disease
2+	Average; normal
1+	Somewhat diminished; low normal
0	No response

Hyperactive reflexes suggest central nervous system disease. Sustained clonus confirms it. Reflexes may be diminished or absent when sensation is lost, when the relevant spinal segments are damaged, or when the peripheral nerves are damaged. Diseases of muscles and neuromuscular junctions may also decrease reflexes.

Reflex response depends partly on the force of your stimulus. Use no more force than you need to provoke a definite response. Differences between sides are usually easier to assess than symmetric changes. Symmetrically diminished or even absent reflexes may be found in normal people.

If the patient's reflexes are symmetrically diminished or absent, use *reinforcement*, a technique involving isometric contraction of other muscles that may increase reflex activity. In testing arm reflexes, for example, ask the patient to clench his or her teeth or to squeeze one thigh with the opposite hand. If leg reflexes are diminished or absent, reinforce them by asking the patient to lock fingers and pull one hand against the other. Tell the patient to pull just before you strike the tendon.



REINFORCEMENT OF KNEE REFLEX

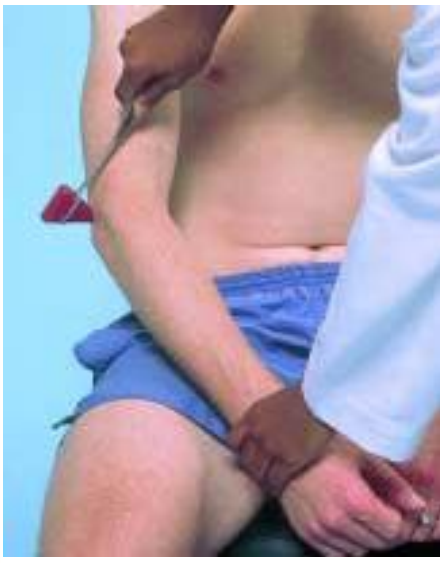
The Biceps Reflex (C5, C6). The patient's arm should be partially flexed at the elbow with palm down. Place your thumb or finger firmly on the biceps tendon. Strike with the reflex hammer so that the blow is aimed directly through your digit toward the biceps tendon.

PATIENT SITTING

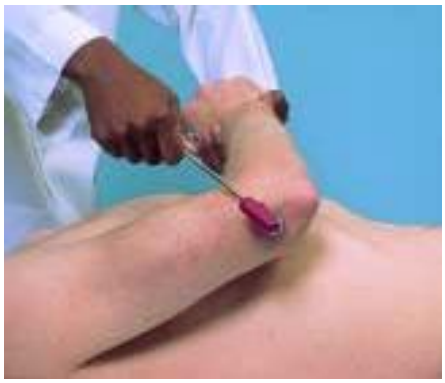
PATIENT LYING DOWN

Observe flexion at the elbow, and watch for and feel the contraction of the biceps muscle.

The Triceps Reflex (C6, C7). Flex the patient's arm at the elbow, with palm toward the body, and pull it slightly across the chest. Strike the triceps tendon above the elbow. Use a direct blow from directly behind it. Watch for contraction of the triceps muscle and extension at the elbow.



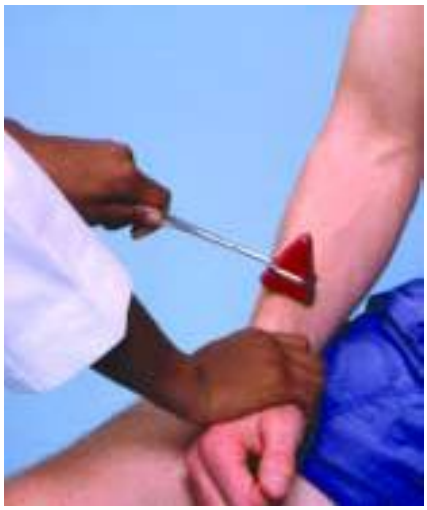
PATIENT SITTING



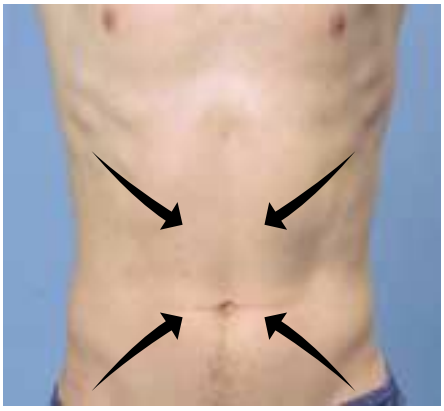
PATIENT LYING DOWN

If you have difficulty getting the patient to relax, try supporting the upper arm as illustrated on the right. Ask the patient to let the arm go limp, as if it were "hung up to dry." Then strike the triceps tendon.

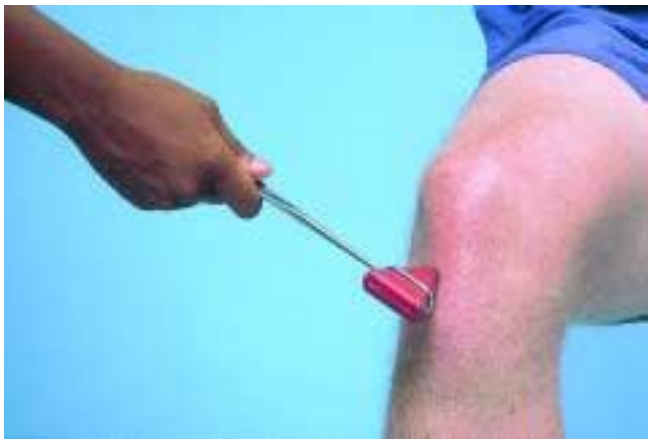
The Supinator or Brachioradialis Reflex (C5, C6). The patient's hand should rest on the abdomen or the lap, with the forearm partly pronated. Strike the radius about 1 to 2 inches above the wrist. Watch for flexion and supination of the forearm.



The Abdominal Reflexes. Test the abdominal reflexes by lightly but briskly stroking each side of the abdomen, above (T8, T9, T10) and below (T10, T11, T12) the umbilicus, in the directions illustrated. Use a key, the wooden end of a cotton-tipped applicator, or a tongue blade twisted and split longitudinally. Note the contraction of the abdominal muscles and deviation of the umbilicus toward the stimulus. Obesity may mask an abdominal reflex. In this situation, use your finger to retract the patient's umbilicus away from the side to be stimulated. Feel with your retracting finger for the muscular contraction.



Abdominal reflexes may be absent in both central and peripheral nervous system disorders.

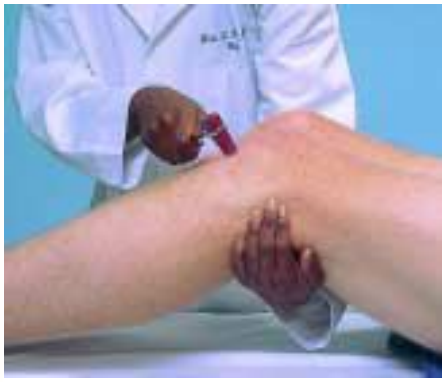


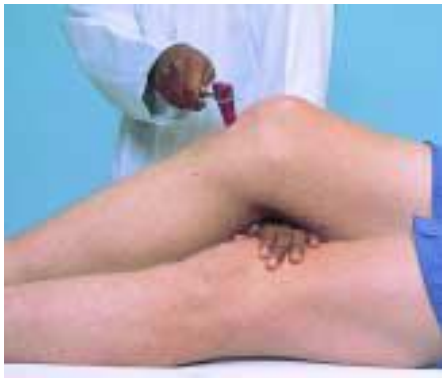
PATIENT SITTING

The Knee Reflex (L2, L3, L4). The patient may be either sitting or lying down as long as the knee is flexed. Briskly tap the patellar tendon just

below the patella. Note contraction of the quadriceps with extension at the knee. A hand on the patient's anterior thigh lets you feel this reflex.

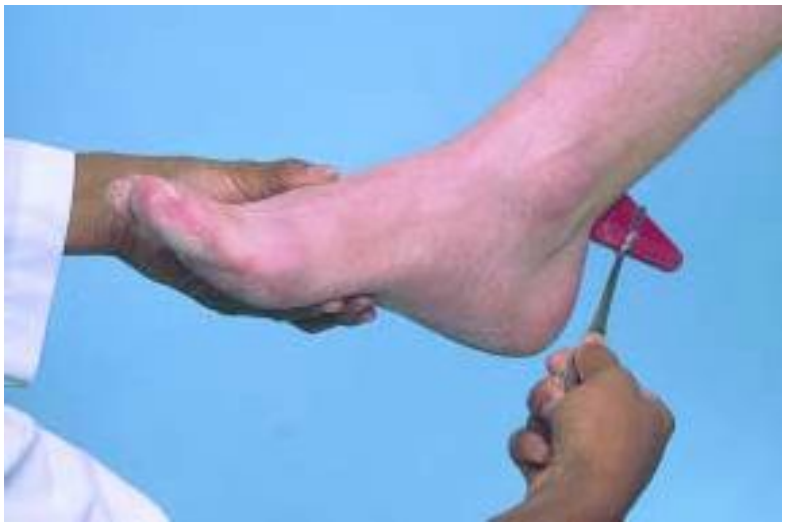
Two methods are useful when examining the supine patient. Supporting both knees at once, as shown below on the left, allows you to assess small differences between knee reflexes by repeatedly testing one reflex and then the other. Sometimes, however, supporting both legs is uncomfortable for both the examiner and the patient. You may wish to rest your supporting arm under the patient's leg, as shown below on the right. Some patients find it easier to relax with this method.





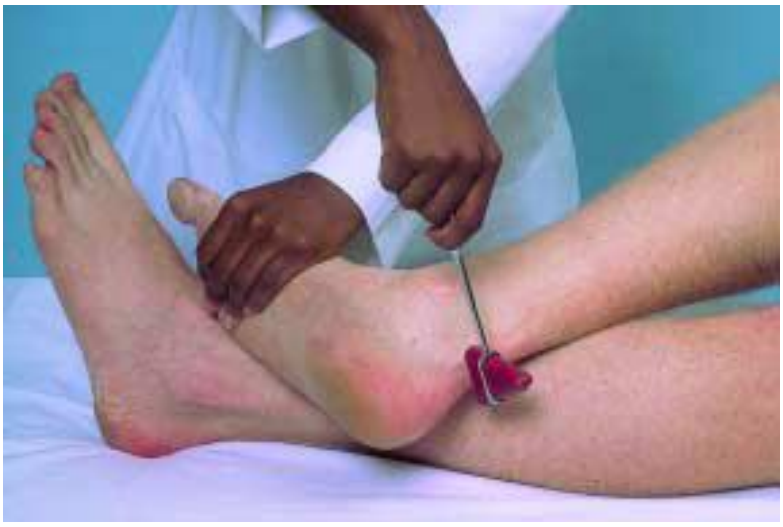
The Ankle Reflex (primarily S1). If the patient is sitting, dorsiflex the foot at the ankle. Persuade the patient to relax. Strike the Achilles tendon. Watch and feel for plantar flexion at the ankle. Note also the speed of relaxation after muscular contraction.

The slowed relaxation phase of reflexes in hypothyroidism is often easily seen and felt in the ankle reflex.



PATIENT SITTING

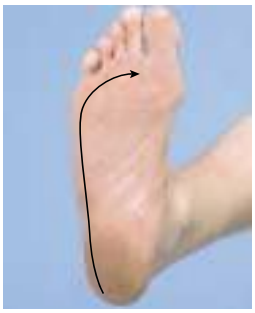
When the patient is lying down, flex one leg at both hip and knee and rotate it externally so that the lower leg rests across the opposite shin. Then dorsiflex the foot at the ankle and strike the Achilles tendon.

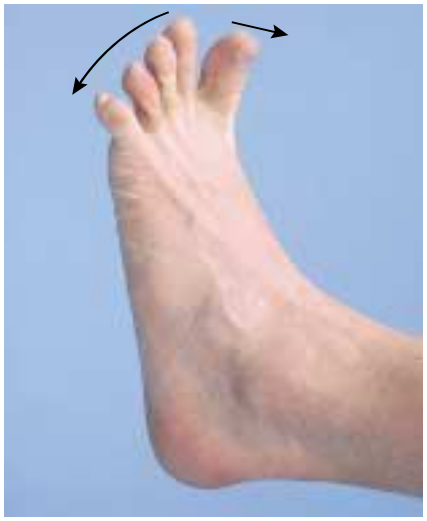


PATIENT LYING DOWN

The Plantar Response (L5, S1). With an object such as a key or the wooden end of an applicator stick, stroke the lateral aspect of the sole from the heel to the ball of the foot, curving medially across the ball. Use the lightest stimulus that will provoke a response, but be increasingly firm if necessary. Note movement of the toes, normally flexion.

Dorsiflexion of the big toe, often accompanied by fanning of the other toes, constitutes a Babinski response. It often indicates a central nervous system lesion in the corticospinal tract.





A Babinski response may also be seen in unconscious states due to drug or alcohol intoxication or in the postictal period following a seizure.

Some patients withdraw from this stimulus by flexing the hip and the knee. Hold the ankle, if necessary, to complete your observation. It is sometimes difficult to distinguish withdrawal from a Babinski response.

A marked Babinski response is occasionally accompanied by reflex flexion at hip and knee.

Clonus. If the reflexes seem hyperactive, test for *ankle clonus.* Support the knee in a partly flexed position. With your other hand, dorsiflex and plantar flex the foot a few times while encouraging the patient to relax, and then sharply dorsiflex the foot and maintain it in dorsiflexion. Look and feel for rhythmic oscillations between dorsiflexion and plantar flexion. In most normal people, the ankle does not react to this stimulus. A few clonic beats may be seen and felt, especially when the patient is tense or has exercised.

Sustained clonus indicates central nervous system disease. The ankle plantar flexes and dorsiflexes repetitively and rhythmically.

Clonus may also be elicited at other joints. A sharp downward displacement of the patella, for example, may elicit patellar clonus in the extended knee.

Special Techniques

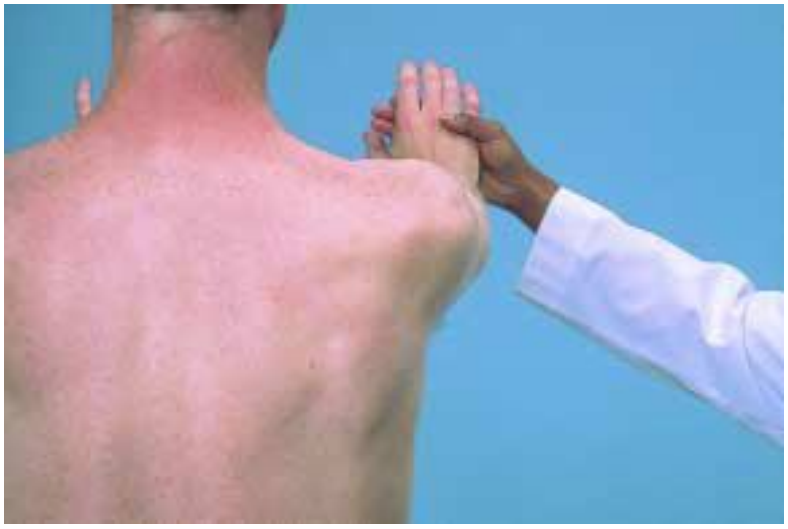
Mini-Mental State Examination (MMSE). This brief test is useful in screening for cognitive dysfunction or dementia and following their course over time. For more detailed information regarding the MMSE, contact the Publisher, Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549.

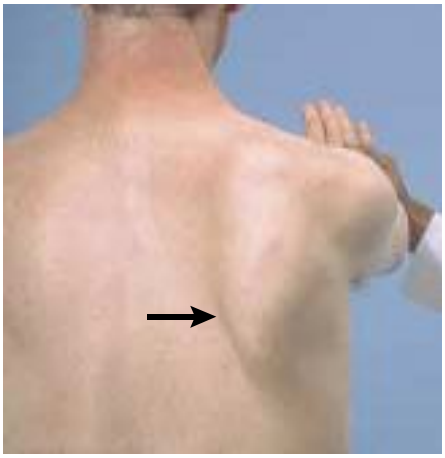
Asterixis. Asterixis helps identify a metabolic encephalopathy in patients whose mental functions are impaired. Ask the patient to "stop traffic" by extending both arms, with hands cocked up and fingers spread. Watch for 1 to 2 minutes, coaxing the patient as necessary to maintain this position.

Sudden, brief, nonrhythmic flexion of the hands and fingers indicates asterixis.

Winging of the Scapula. When the shoulder muscles seem weak or atrophic, look for winging. Ask the patient to extend both arms and push against your hand or against a wall. Observe the scapulae. Normally they lie close to the thorax.

In winging, shown below, the medial border of the scapula juts backward. It suggests weakness of the serratus anterior muscle, as in muscular dystrophy or injury to the long thoracic nerve.





In very thin but normal people, the scapulae may appear "winged" even when the musculature is intact.

Meningeal Signs. Testing for these signs is important if you suspect meningeal inflammation from infection or subarachnoid hemorrhage.

Neck Mobility. First make sure there is no injury to the cervical vertebrae or cervical cord. (In settings of trauma, this may require evaluation by x-ray.) Then, with the patient supine, place your hands behind the patient's head and flex the neck forward, until the chin touches the chest if possible. Normally the neck is supple and the patient can easily bend the head and neck forward.

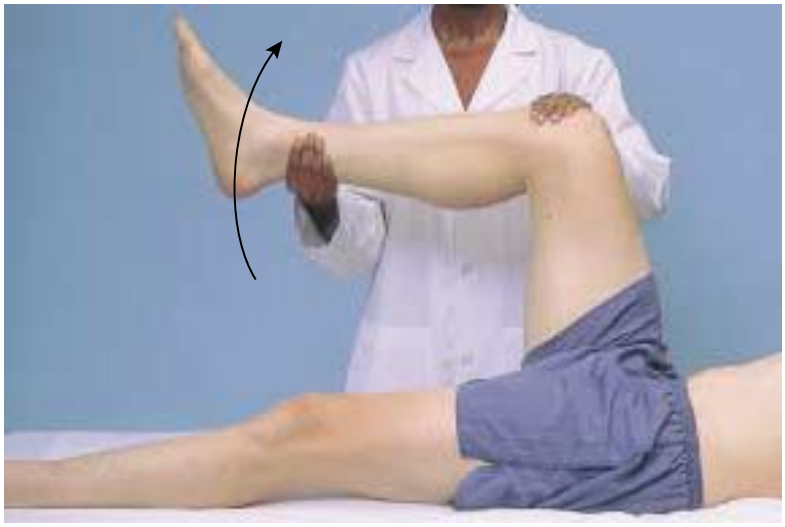
Pain in the neck and resistance to flexion can arise from meningeal inflammation, arthritis, or neck injury.

Brudzinski's Sign. As you flex the neck, watch the hips and knees in reaction to your maneuver. Normally they should remain relaxed and motionless.

Flexion of the hips and knees is a *positive Brudzinski's sign* and suggests meningeal inflammation.

Kernig's Sign. Flex the patient's leg at both the hip and the knee, and then straighten the knee. Discomfort behind the knee during full extension occurs in many normal people, but this maneuver should not produce pain.

Pain and increased resistance to extending the knee are a *positive Kernig's sign.* When bilateral, it suggests meningeal irritation.



Compression of a lumbosacral nerve root may also cause resistance, together with pain in the low back and the posterior thigh. Only one leg is usually involved.

Anal Reflex. Using a dull object, such as a cotton swab, stroke outward in the four quadrants from the anus. Watch for reflex contraction of the anal musculature.

Loss of the anal reflex suggests a lesion in the S2–3–4 reflex arc, as in a cauda equina lesion.

The Stuporous or Comatose Patient. Coma signals a potentially life-threatening event affecting the two hemispheres, the brainstem, or both. The usual sequence of history, physical examination, and laboratory evaluation does not apply. Instead, you must:

- First assess the ABCs (airway, breathing, and circulation)
- Establish the patient's level of consciousness
- Examine the patient neurologically. Look for focal or asymmetric findings, and determine whether impaired consciousness arises from a metabolic or a structural cause.

See Table 16-14, Metabolic and Structural Coma (p. 620).

Interview relatives, friends, or witnesses to establish the speed of onset and duration of unconsciousness, any warning symptoms, precipitating factors, or previous episodes, and the prior appearance and behavior of the patient. Any history of past medical and psychiatric illnesses is also useful.

As you proceed to the examination, remember two cardinal DON'Ts:

1. *Don't* dilate the pupils, the single most important clue to the underlying cause of coma (structural vs. metabolic), and
2. *Don't* flex the neck if there is any question of trauma to the head or neck. Immobilize the cervical spine and get an x-ray first to rule out fractures of the cervical vertebrae that could compress and damage the spinal cord.

Airway, Breathing, and Circulation. Quickly check the patient's color and pattern of breathing. Inspect the posterior pharynx and listen over

the trachea for stridor to make sure the airway is clear. If respirations are slowed or shallow, or if the airway is obstructed by secretions, consider intubating the patient as soon as possible while stabilizing the cervical spine.

Assess the remaining vital signs: pulse, blood pressure, and *rectal* temperature. If hypotension or hemorrhage is present, establish intravenous access and begin intravenous fluids. (Further emergency management and laboratory studies are beyond the scope of this text.)

Level of Consciousness. Level of consciousness primarily reflects the patient's capacity for arousal, or wakefulness. It is determined by the level of activity that the patient can be aroused to perform in response to escalating stimuli from the examiner.

Five clinical levels of consciousness are described in the table below, together with the techniques that may be used to elicit their characteristics. Increase your stimuli in a stepwise manner, depending on the patient's response.

When you examine patients with an altered level of consciousness, describe and record exactly what you see and hear. Summary terms such as lethargy, obtundation, stupor, or coma may have different meanings for other examiners.

Level of Consciousness (Arousal): Techniques and Patient Response

Level	Technique	Abnormal Response
Alertness	Speak to the patient in a normal tone of voice. An alert patient opens the eyes, looks at you, and responds fully and appropriately to stimuli (arousal intact).	
Lethargy	Speak to the patient in a loud voice. For example, call the patient's name or ask "How are you?"	A lethargic patient appears drowsy but opens the eyes and looks at you, responds to questions, and then falls asleep.
Obtundation	Shake the patient gently as if awakening a sleeper.	An obtunded patient opens the eyes and looks at you, but responds slowly and is somewhat confused. Alertness and interest in the environment are decreased.
Stupor	Apply a painful stimulus. For example, pinch a tendon, rub the sternum, or roll a pencil across a nail bed. (No stronger stimuli needed!)	A stuporous patient arouses from sleep only after painful stimuli. Verbal responses are slow or even absent. The patient lapses into an unresponsive state when the stimulus ceases. There is minimal awareness of self or the environment.
Coma	Apply repeated painful stimuli.	A comatose patient remains unarousable with eyes closed. There is no evident response to inner need or external stimuli.

Neurologic Evaluation

RESPIRATIONS. Observe the rate, rhythm, and pattern of respirations. Because neural structures that govern breathing in the cortex and brainstem overlap those that govern consciousness, abnormalities of respiration often occur in coma.

See Table 16-14, Metabolic and Structural Coma (p. 620), and Table 3-12, Abnormalities in Rate and Rhythm of Breathing (p. 93).

PUPILS. Observe the size and equality of the pupils and test their reaction to light. The presence or absence of the light reaction is one of the most important signs distinguishing structural from metabolic causes of coma. The light reaction often remains intact in metabolic coma.

See Table 16-15, Pupils in Comatose Patients (p. 621).

Structural lesions such as stroke may lead to asymmetrical pupils and loss of light reaction.

OCULAR MOVEMENT. Observe the position of the eyes and eyelids at rest. Check for horizontal deviation of the eyes to one side (*gaze preference*). When the oculomotor pathways are intact, the eyes look straight ahead.

In structural hemispheric lesions, the eyes "look at the lesion" in the affected hemisphere.

In irritative lesions due to epilepsy or early cerebral hemorrhage, the eyes "look away" from the affected hemisphere.

OCULOCEPHALIC REFLEX (DOLL'S EYE MOVEMENTS). This reflex helps to assess brainstem function in a comatose patient. Holding open the upper eyelids so that you can see the eyes, turn the head quickly, first to one side and then to the other. (Make sure the patient has no neck injury before performing this test.)

In a comatose patient with absence of doll's eye movements, shown below, the ability to move both eyes to one side is lost, suggesting a lesion of midbrain or pons.

In a comatose patient with an intact brainstem, as the head is turned the eyes move toward the opposite side (the doll's eye movements). In the adjacent photo, for example, the patient's head has been turned to the right; her eyes have moved to the left. Her eyes still seem to gaze at the camera. The doll's eye movements are intact.

OCULOVESTIBULAR REFLEX (WITH CALORIC STIMULATION). If the oculocephalic reflex is absent and you seek further assessment of brainstem function, test the oculovestibular reflex. Note that this test is almost never performed in an awake patient.

Make sure the eardrums are intact and the canals clear. You must elevate the patient's head to 30° to perform the test accurately. Place a kidney basin under the ear to catch any overflowing water. With a large syringe, inject ice water

through a small catheter that is lying in (but not plugging) the ear canal. Watch for deviation of the eyes in the horizontal plane. You may need to use up to 120 ml of ice water to elicit a response. In the comatose patient with an intact brainstem, the eyes drift *toward* the irrigated ear. Repeat on the opposite side, waiting 3 to 5 minutes if necessary for the first response to disappear.

No response to stimulation suggests brainstem injury.

POSTURE AND MUSCLE TONE. Observe the patient's posture. If there is no spontaneous movement, you may need to apply a painful stimulus (see p. 595). Classify the resulting pattern of movement as:

See Table 16-16, Abnormal Postures in the Comatose Patient (p. 622).

- *Normal–avoidant*—the patient pushes the stimulus away or withdraws.
- *Stereotypic*—the stimulus evokes abnormal postural responses of the trunk and extremities.

Two stereotypic responses predominate: *decorticate rigidity* and *decerebrate rigidity* (see Table 16-16, Abnormal Postures in the Comatose Patient, p. 622).

- *Flaccid paralysis or no response*

No response on one side suggests a corticospinal tract lesion.

Test muscle tone by grasping each forearm near the wrist and raising it to a vertical position. Note the position of the hand, usually only slightly flexed at the wrist.

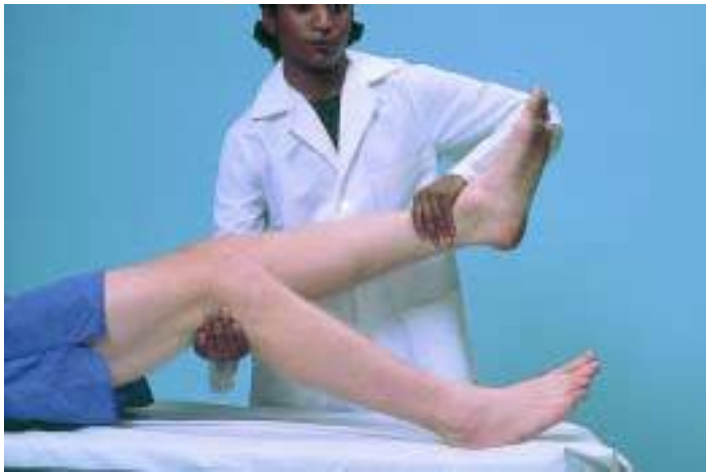
The hemiplegia of sudden cerebral accidents is usually flaccid at first. The limp hand drops to form a right angle with the wrist.

Then lower the arm to about 12 or 18 inches off the bed and drop it. Watch how it falls. A normal arm drops somewhat slowly.

A flaccid arm drops rapidly, like a flail.

Support the patient's flexed knees. Then extend one leg at a time at the knee and let it fall (see next page). Compare the speed with which each leg falls.

In acute hemiplegia, the flaccid leg falls more rapidly



Flex both legs so that the heels rest on the bed and then release them. The normal leg returns slowly to its original extended position.

In acute hemiplegia, the flaccid leg falls rapidly into extension, with external rotation at the hip.

Further Examination

As you complete the neurologic examination, check for facial asymmetry and asymmetries in motor, sensory, and reflex function. Test for meningeal signs if indicated.

Meningitis, subarachnoid hemorrhage.

As you proceed to the general physical examination, check for unusual odors.

Alcohol, liver failure, uremia

Look for abnormalities of the skin, including color, moisture, evidence of bleeding disorders, needle marks, and other lesions.

Jaundice, cyanosis, cherry red color of carbon monoxide poisoning

Examine the scalp and skull for signs of trauma.

Bruises, lacerations, swelling

Examine the fundi carefully.

Papilledema, hypertensive retinopathy

Check to make sure the corneal reflexes are intact. (Remember that use of contact lenses may abolish these reflexes.)

Reflex loss in coma and lesions affecting CN V or CN VII

Inspect the ears and nose, and examine the mouth and throat.

Blood or cerebrospinal fluid in the nose or the ears suggests a skull fracture; otitis media suggests a possible brain abscess.

Tongue injury suggests a seizure.

Be sure to evaluate the heart, lungs, and abdomen.

Tables 16-1 through 16-7 summarize the manifestations of selected disorders. They show how the data collected can be used diagnostically, and will help you to recognize and think about certain patterns of illness.

Tables 16-1, 16-3, and 16-4 are based, with permission, on the *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition, Text Revision (DSM IV-TR) Washington, D.C., American Psychiatric Association, 2000. For further details and criteria, the reader should consult this manual, its successor, or comprehensive textbooks of psychiatry.

TABLE 16-1 ■ Disorders of Mood

Mood disorders may be either depressive or bipolar. A bipolar disorder includes manic or hypomanic features as well as depressive ones. Four types of *episodes,* described below, are combined in different ways in diagnosis of *mood disorders.* A major depressive disorder includes only one or more major depressive episodes. A *bipolar I disorder* includes one or more manic or mixed episodes, usually accompanied by major depressive episodes. A *bipolar II disorder* includes one or more major depressive episodes accompanied by at least one hypomanic episode.

Dysthymic and *cyclothymic disorders* are chronic and less severe conditions that do not meet the criteria of the other disorders. *Mood disorders due to general medical conditions or substance abuse* are classified separately.

Major Depressive Episode

At least five of the symptoms listed below (including one of the first two) must be present during the same 2-week period. They must also represent a change from the person's previous state.

- Depressed mood (may be an irritable mood in children and adolescents) most of the day, nearly every day
- Markedly diminished interest or pleasure in almost all activities most of the day, nearly every day
- Significant weight gain or loss (not dieting) or increased or decreased appetite nearly every day
- Insomnia or hypersomnia nearly every day
- Psychomotor agitation or retardation nearly every day
- Fatigue or loss of energy nearly every day
- Feelings of worthlessness or inappropriate guilt nearly every day
- Inability to think or concentrate or indecisiveness nearly every day
- Recurrent thoughts of death or suicide, or a specific plan for or attempt at suicide

The symptoms cause significant distress or impair social, occupational, or other important functions. In severe cases, hallucinations and delusions may occur.

Mixed Episode

A mixed episode, which must last at least 1 week, meets the criteria for both manic and major depressive episodes.

Dysthymic Disorder

A depressed mood and symptoms for most of the day, for more days than not, over at least 2 years (1 year in children and adolescents). Freedom from symptoms lasts no more than 2 months at a time.

Manic Episode

A distinct period of abnormally and persistently elevated, expansive, or irritable mood must be present for at least a week (any duration if hospitalization is necessary). During this time, at least three of the symptoms listed below have been persistent and significant. (Four of the these symptoms are required if the mood is only irritable).

- Inflated self-esteem or grandiosity
- Decreased need for sleep (feels rested after sleeping 3 hours)
- More talkative than usual or pressure to keep talking
- Flight of ideas or racing thoughts
- Distractibility
- Increased goal-directed activity (either socially at work or school, or sexually) or psychomotor agitation
- Excessive involvement in pleasurable high-risk activities (buying sprees, foolish business ventures, sexual indiscretions)

The disturbance is severe enough to impair social or occupational functions or relationships. It may necessitate hospitalization for the protection of self or others. In severe cases, hallucinations and delusions may occur.

Hypomanic Episode

The mood and symptoms resemble those in a manic episode but are less impairing, do not require hospitalization, do not include hallucinations or delusions, and have a shorter minimum duration—4 days.

Cyclothymic Episode

Numerous periods of hypomanic and depressive symptoms that last for at least 2 years (1 year in children and adolescents). Freedom from symptoms lasts no more than 2 months at a time.

TABLE 16-2 ■ Disorders of Speech

Disorders of speech fall into three groups: those affecting (1) the voice, (2) the articulation of words, and (3) the production and comprehension of language.

Aphonia refers to a loss of voice that accompanies disease affecting the larynx or its nerve supply. *Dysphonia* refers to less severe impairment in the volume, quality, or pitch of the voice. For example, a person may be hoarse or only able to speak in a whisper. Causes include laryngitis, laryngeal tumors, and a unilateral vocal cord paralysis (Cranial Nerve X).

Dysarthria refers to a defect in the muscular control of the speech apparatus (lips, tongue, palate, or pharynx). Words may be nasal, slurred, or indistinct, but the central symbolic aspect of language remains intact. Causes include motor lesions of the central or peripheral nervous system, parkinsonism, and cerebellar disease.

Aphasia refers to a disorder in producing or understanding language. It is often caused by lesions in the dominant cerebral hemisphere (usually the left).

Compared below are two common types of aphasia: (1) Wernicke's, a fluent (receptive) aphasia, and (2) Broca's, a nonfluent (or expressive) aphasia. There are other less common kinds of aphasia, which may be distinguished from each other by differing responses on the specific tests listed. Neurologic consultation is usually indicated.

	Wernicke's Aphasia	Broca's Aphasia
Qualities of Spontaneous Speech	Fluent; often rapid, voluble, and effortless. Inflection and articulation are good, but sentences lack meaning and words are malformed (paraphasias) or invented (neologisms). Speech may be totally incomprehensible.	Nonfluent; slow, with few words and laborious effort. Inflection and articulation are impaired but words are meaningful, with nouns, transitive verbs, and important adjectives. Small grammatical words are often dropped.
Word Comprehension	Impaired	Fair to good
Repetition	Impaired	Impaired
Naming	Impaired	Impaired, though the patient recognizes objects
Reading Comprehension	Impaired	Fair to good
Writing	Impaired	Impaired
Location of Lesion	Posterior superior temporal lobe	Posterior inferior frontal lobe

While it is important to recognize aphasia early in your encounter with a patient, its full diagnostic meaning does not become clear until you integrate this information with your neurologic examination.

TABLE 16-3 ■ Anxiety Disorders

Anxiety disorders cause great distress and impair function, but those affected are not psychotic. The disorders are distinguished by the symptoms, the entities feared, or the stressors.

Panic Disorder	A panic disorder is defined by recurrent, unexpected panic attacks, at least one of which has been followed by a month or more of persistent concern about further attacks, worry over their implications or consequences, or a significant change in behavior in relation to the attacks. A *panic attack* is a discrete period of intense fear or discomfort that develops abruptly and peaks within 10 minutes. It involves at least four of the following symptoms: (1) palpitations, pounding heart, or accelerated heart rate, (2) sweating, (3) trembling or shaking, (4) shortness of breath or a sense of smothering, (5) a feeling of choking, (6) chest pain or discomfort, (7) nausea or abdominal distress, (8) feeling dizzy, unsteady, lightheaded, or faint, (9) feelings of unreality or depersonalization, (10) fear of losing control or going crazy, (11) fear of dying, (12) paresthesias (numbness or tingling), (13) chills or hot flushes. Panic disorder may occur with or without agoraphobia.
Agoraphobia	Agoraphobia is an anxiety about being in places or situations where escape may be difficult or embarrassing or help for sudden symptoms unavailable. Such situations are avoided, require a companion, or cause marked anxiety.
Specific Phobia	A specific phobia is a marked, persistent, and excessive or unreasonable fear that is cued by the presence or anticipation of a specific object or situation, such as dogs, injections, or flying. The person recognizes the fear as excessive or unreasonable, but exposure to the cue provokes immediate anxiety. Avoidance or fear impairs the person's normal routine, occupational or academic functioning, or social activities or relationships.
Social Phobia	A social phobia is a marked, persistent fear of one or more social or performance situations that involve exposure to unfamiliar people or to scrutiny by others. Those afflicted fear that they will act in embarrassing or humiliating ways, as by showing their anxiety. Exposure creates anxiety and possibly a panic attack, and the person avoids precipitating situations. He or she recognizes the fear as excessive or unreasonable. Normal routines, occupational or academic functioning, or social activities or relationships are impaired.
Obsessive–Compulsive Disorder	This disorder involves obsessions or compulsions that cause marked anxiety or distress. While they are recognized at some point as excessive or unreasonable, they are very time consuming and interfere with the person's normal routine, occupational functioning, or social activities or relationships.
Acute Stress Disorder	The person has been exposed to a traumatic event that involved actual or threatened death or serious injury to self or others and responded with intense fear, helplessness, or horror. During or immediately after this event, the person has at least three of these dissociative symptoms: (1) a subjective sense of numbing, detachment, or absence of emotional responsiveness; (2) a reduced awareness of surroundings, as in a daze; (3) feelings of unreality; (4) feelings of depersonalization; and (5) amnesia for an important part of the event. The event is persistently reexperienced, as in thoughts, images, dreams, illusions, and flashbacks, or distress from reminders of the event. The person is very anxious or shows increased arousal and tries to avoid stimuli that evoke memories of the event. The disturbance causes marked distress or impairs social, occupational, or other important functions. The symptoms occur within 4 weeks of the event and last from 2 days to 4 weeks.
Posttraumatic Stress Disorder	The event, the fearful response, and the persistent reexperiencing of the traumatic event resemble those in acute stress disorder. Hallucinations may occur. The person has increased arousal, tries to avoid stimuli related to the trauma, and has numbing of general responsiveness. The disturbance causes marked distress, impairs social, occupational, or other important functions, and lasts for more than a month.
Generalized Anxiety Disorder	This disorder lacks a specific traumatic event or focus for concern. Excessive anxiety and worry, which the person finds hard to control, are about a number of events or activities. At least three of the following symptoms are associated: (1) feeling restless, keyed up, or on edge, (2) being easily fatigued, (3) difficulty in concentrating or mind going blank, (4) irritability, (5) muscle tension, (6) difficulty in falling or staying asleep, or restless, unsatisfying sleep. The disturbance causes significant distress or impairs social, occupational, or other important functions.

TABLE 16-4 ■ Psychotic Disorders

Psychotic disorders involve grossly impaired reality testing. Specific diagnoses depend on the nature and duration of the symptoms and on a cause when it can be identified. Seven disorders are outlined below.

Schizophrenia	Schizophrenia impairs major functioning, as at work or school or in interpersonal relations or self care. For this diagnosis, performance of one or more of these functions must have decreased for a significant time to a level markedly below prior achievement. In addition, the person must manifest at least two of the following for a significant part of 1 month: (1) delusions, (2) hallucinations, (3) disorganized speech, (4) grossly disorganized or catatonic behavior,* and (5) negative symptoms such as a flat affect, alogia (lack of content in speech), or avolition (lack of interest, drive, and ability to set and pursue goals). Continuous signs of the disturbance must persist for at least 6 months. Subtypes of this disorder include paranoid, disorganized, and catatonic schizophrenia.
Schizophreniform Disorder	A schizophreniform disorder has symptoms similar to those of schizophrenia but they last less than 6 months, and the functional impairment seen in schizophrenia need not be present.
Schizoaffective Disorder	A schizoaffective disorder has features of both a major mood disturbance and schizophrenia. The mood disturbance (depressive, manic, or mixed) is present during most of the illness and must, for a time, be concurrent with symptoms of schizophrenia (listed above). During the same period of time, there must also be delusions or hallucinations for at least 2 weeks without prominent mood symptoms.
Delusional Disorder	A delusional disorder is characterized by nonbizarre delusions that involve situations in real life, such as having a disease or being deceived by a lover. The delusion has persisted for at least a month, but the person's functioning is not markedly impaired and behavior is not obviously odd or bizarre. The symptoms of schizophrenia except for tactile and olfactory hallucinations related to the delusion have not been present.
Brief Psychotic Disorder	In this disorder, at least one of the following psychotic symptoms must be present: delusions, hallucinations, disordered speech such as frequent derailment or incoherence, or grossly disorganized or catatonic behavior. The disturbance lasts at least 1 day but less than 1 month, and the person returns to his or her prior functional level.
Psychotic Disorder Due to a General Medical Condition	Prominent hallucinations or delusions may be experienced during a medical illness. For this diagnosis, they should not occur exclusively during the course of delirium. The medical condition should be documented and judged to be causally related to the symptoms.
Substance-Induced Psychotic Disorder	Prominent hallucinations or delusions may be induced by intoxication or withdrawal from a substance such as alcohol, cocaine, or opioids. For this diagnosis, these symptoms should not occur exclusively during the course of delirium. The substance should be judged to be causally related to the symptoms.

*Catatonic behaviors are psychomotor abnormalities that include stupor, mutism, negativistic resistance to instructions or attempts to move the person, rigid or bizarre postures, and excited, apparently purposeless activity.

TABLE 16-5 ■ Delirium and Dementia

Delirium and dementia are common and very important disorders that affect multiple aspects of mental status. Both have many possible causes. Some clinical features of these two conditions and their effects on mental status are compared below. A delirium may be superimposed on dementia.

	Delirium	Dementia
Clinical Features		
Onset	Acute	Insidious
Course	Fluctuating, with lucid intervals; worse at night	Slowly progressive
Duration	Hours to weeks	Months to years
Sleep/Wake Cycle	Always disrupted	Sleep fragmented
General Medical Illness or Drug Toxicity	Either or both present	Often absent, especially in Alzheimer's disease
Mental Status		
Level of Consciousness	Disturbed. Person less clearly aware of the environment and less able to focus, sustain, or shift attention	Usually normal until late in the course of the illness
Behavior	Activity often abnormally decreased (somnolence) or increased (agitation, hypervigilance)	Normal to slow; may become inappropriate
Speech	May be hesitant, slow or rapid, incoherent	Difficulty in finding words, aphasia
Mood	Fluctuating, labile, from fearful or irritable to normal or depressed	Often flat, depressed
Thought Processes	Disorganized, may be incoherent	Impoverished. Speech gives little information.
Thought Content	Delusions common, often transient	Delusions may occur.
Perceptions	Illusions, hallucinations, most often visual	Hallucinations may occur.
Judgment	Impaired, often to a varying degree	Increasingly impaired over the course of the illness
Orientation	Usually disoriented, especially for time. A known place may seem unfamiliar.	Fairly well maintained, but becomes impaired in the later stages of illness
Attention	Fluctuates. Person easily distracted, unable to concentrate on selected tasks	Usually unaffected until late in the illness
Memory	Immediate and recent memory impaired	Recent memory and new learning especially impaired
Examples of Cause	Delirium tremens (due to withdrawal from alcohol)	*Reversible:* Vitamin B_{12} deficiency, thyroid disorders
	Uremia	
	Acute hepatic failure	*Irreversible:* Alzheimer's disease, vascular dementia (from multiple infarcts), dementia due to head trauma
	Acute cerebral vasculitis	
	Atropine poisoning	

TABLE 16-6 ■ Syncope and Similar Disorders

Problem	Mechanism	Precipitating Factors
Vasodepressor Syncope *(the common faint)*	Sudden peripheral vasodilatation, especially in the skeletal muscles, without a compensatory rise in cardiac output. Blood pressure falls.	A strong emotion such as fear or pain
Postural *(orthostatic)* **Hypotension**	■ *Inadequate vasoconstrictor reflexes* in both arterioles and veins, with resultant venous pooling, decreased cardiac output, and low blood pressure	■ Standing up
	■ *Hypovolemia*, a diminished blood volume insufficient to maintain cardiac output and blood pressure, especially in the upright position	■ Standing up after hemorrhage or dehydration
Cough Syncope	Several possible mechanisms associated with increased intrathoracic pressure	Severe paroxysm of coughing
Micturition Syncope	Unclear	Emptying the bladder after getting out of bed to void
Cardiovascular Disorders		
Arrhythmias	Decreased cardiac output secondary to rhythms that are too fast (usually more than 180) or too slow (less than 35–40)	A sudden change in rhythm
Aortic Stenosis and Hypertrophic Cardiomyopathy	Vascular resistance falls with exercise, but cardiac output cannot rise.	Exercise
Myocardial Infarction	Sudden arrhythmia or decreased cardiac output	Variable
Massive Pulmonary Embolism	Sudden hypoxia or decreased cardiac output	Variable
Disorders Resembling Syncope		
Hypocapnia (decreased carbon dioxide) Due to Hyperventilation	Constriction of cerebral blood vessels secondary to hypocapnia that is induced by hyperventilation	Possibly a stressful situation
Hypoglycemia	Insufficient glucose to maintain cerebral metabolism; secretion of epinephrine contributes to symptoms.	Variable, including fasting
*Hysterical Fainting Due to a Conversion Reaction**	The symbolic expression of an unacceptable idea through body language	Stressful situation

* Important diagnostic observations in hysterical fainting include normal skin color and normal vital signs, sometimes bizarre and purposive movements, and occurrence in the presence of other people.

Predisposing Factors	Prodromal Manifestations	Postural Associations	Recovery
Fatigue, hunger, a hot, humid environment	Restlessness, weakness, pallor, nausea, salivation, sweating, yawning	Usually occurs when standing, possibly when sitting	Prompt return of consciousness when lying down, but pallor, weakness, nausea, and slight confusion may persist for a time.
■ Peripheral neuropathies and disorders affecting the autonomic nervous system; drugs such as antihypertensives and vasodilators; prolonged bed rest	■ Often none	■ Occurs soon after the person stands up	■ Prompt return to normal when lying down
■ Bleeding from the GI tract or trauma, potent diuretics, vomiting, diarrhea, polyuria	■ Lightheadedness and palpitations (tachycardia) on standing up	■ Usually occurs soon after the person stands up	■ Improvement on lying down
Chronic bronchitis in a muscular man	Often none except for cough	May occur in any position	Prompt return to normal
Nocturia, usually in elderly or adult men	Often none	Standing to void	Prompt return to normal
Organic heart disease and old age decrease the tolerance to abnormal rhythms.	Often none	May occur in any position	Prompt return to normal unless brain damage has resulted
Cardiac disorders	Often none. Onset is sudden.	Occurs with or after exercise	Usually a prompt return to normal
Coronary artery disease	Often none	May occur in any position	Variable
Deep venous thrombosis	Often none	May occur in any position	Variable
A predisposition to anxiety attacks and hyperventilation	Dyspnea, palpitations, chest discomfort, numbness and tingling of the hands and around the mouth lasting for several minutes. Consciousness is often maintained.	May occur in any position	Slow improvement as hyperventilation ceases
Insulin therapy and a variety of metabolic disorders	Sweating, tremor, palpitations, hunger; headache, confusion, abnormal behavior, coma. True syncope is uncommon.	May occur in any position	Variable, depending on severity and treatment
Hysterical personality traits	Variable	A slump to the floor, often from a standing position without injury	Variable, may be prolonged, often with fluctuating responsiveness

TABLE 16-7 ■ Seizure Disorders

Partial seizures are those that start with focal manifestations. They are further divided into *simple partial seizures,* which do not impair consciousness, and *complex partial seizures,* which do. Each of these two types may remain localized or progress into a third type, *partial seizures that become generalized*. Partial seizures of all kinds usually indicate a structural lesion in the cerebral cortex, such as a scar, tumor, or infarction. The quality of such seizures helps the clinician to localize the causative lesion in the brain.

Problem	Clinical Manifestations	Postictal *(Postseizure)* State
Partial Seizures		
Simple Partial Seizures		
■ With motor symptoms Jacksonian	Tonic and then clonic movements that start unilaterally in the hand, foot, or face and spread to other body parts on the same side	Normal consciousness
Other motor	Turning of the head and eyes to one side, or tonic and clonic movements of an arm or leg without the Jacksonian spread	Normal consciousness
■ With sensory symptoms	Numbness, tingling; simple visual, auditory, or olfactory hallucinations such as flashing lights, buzzing, or odors	Normal consciousness
■ With autonomic symptoms	A "funny feeling" in the epigastrium, nausea, pallor, flushing, lightheadedness	Normal consciousness
■ With psychiatric symptoms	Anxiety or fear; feelings of familiarity (déjà vu) or unreality; dreamy states; fear or rage; flashback experiences; more complex hallucinations	Normal consciousness
Complex Partial Seizures May start with simple partial seizures or with impaired consciousness. Automatisms may develop.	The seizure may or may not start with the autonomic or psychic symptoms that are outlined above. Consciousness is impaired and the person appears confused. Automatisms include automatic motor behaviors such as chewing, smacking the lips, walking about, and unbuttoning clothes; also more complicated and skilled behaviors such as driving a car.	The patient may remember initial autonomic or psychic symptoms (which are then termed an *aura*), but is amnesic for the rest of the seizure. Temporary confusion and headache may occur.
Partial Seizures That Become Generalized	Partial seizures that become generalized resemble tonic–clonic seizures (see next page). Unfortunately, the patient may not recall the focal onset and observers may overlook it.	As in a tonic–clonic seizure, described on the next page. *Two attributes indicate a partial seizure that has become generalized: (1) the recollection of an aura, and (2) a unilateral neurologic deficit during the postictal period.*

TABLE 16-7 ■ Seizure Disorders *(Continued)*

Generalized seizures, in contrast to partial ones, begin with either bilateral bodily movements or impairment of consciousness, or both. They suggest a widespread, bilateral cortical disturbance that may be either hereditary or acquired. When generalized seizures of the tonic–clonic (grand mal) variety start in childhood or young adulthood, they are often hereditary. When tonic–clonic seizures begin after the age of 30, suspect either a partial seizure that has become generalized or a general seizure caused by a toxic or metabolic problem. Toxic and metabolic causes include withdrawal from alcohol or other sedative drugs, uremia, hypoglycemia, hyperglycemia, hyponatremia and water intoxication, and bacterial meningitis.

Problem	Clinical Manifestations	Postictal *(Postseizure)* State
Generalized Seizures		
*Tonic–Clonic Convulsion (grand mal)**	The person loses consciousness suddenly, sometimes with a cry, and the body stiffens into tonic extensor rigidity. Breathing stops and the person becomes cyanotic. A clonic phase of rhythmic muscular contraction follows. Breathing resumes and is often noisy, with excessive salivation. Injury, tongue biting, and urinary incontinence may occur.	Confusion, drowsiness, fatigue, headache, muscular aching, and sometimes the temporary persistence of bilateral neurologic deficits such as hyperactive reflexes and Babinski responses. The person has amnesia for the seizure and recalls no aura.
Absence	A sudden brief lapse of consciousness, with momentary blinking, staring, or movements of the lips and hands but no falling. Two subtypes are recognized. *Petit mal absences* last less than 10 sec and stop abruptly. *Atypical absences* may last more than 10 sec.	No aura recalled. In petit mal absences, a prompt return to normal; in atypical absences, some postictal confusion
Atonic Seizure, or Drop Attack	Sudden loss of consciousness with falling but no movements. Injury may occur.	Either a prompt return to normal or a brief period of confusion
Myoclonus	Sudden, brief, rapid jerks, involving the trunk or limbs. Associated with a variety of disorders	Variable
Pseudoseizures		
May mimic seizures but are due to a conversion reaction (a psychological disorder).	The movements may have personally symbolic significance and often do not follow a neuroanatomic pattern. Injury is uncommon.	Variable

* *Febrile convulsions* that resemble brief tonic–clonic seizures may occur in infants and young children. They are usually benign but occasionally may be the first manifestation of a seizure disorder.

TABLE 16-8 ■ Involuntary Movements

Tremors

Tremors are relatively rhythmic oscillatory movements, which may be roughly subdivided into three groups: resting (or static) tremors, intention tremors, and postural tremors.

Resting (Static) Tremors

These tremors are most prominent at rest, and may decrease or disappear with voluntary movement. Illustrated is the common, relatively slow, fine, pill-rolling tremor of parkinsonism, about 5 per second.

Postural Tremors

These tremors appear when the affected part is actively maintaining a posture. Examples include the fine, rapid tremor of hyperthyroidism, the tremors of anxiety and fatigue, and benign essential (and sometimes familial) tremor. Tremor may worsen somewhat with intention.

Intention Tremors

Intention tremors, absent at rest, appear with activity and often get worse as the target is neared. Causes include disorders of cerebellar pathways, as in multiple sclerosis.

Oral–Facial Dyskinesias

Oral–facial dyskinesias are rhythmic, repetitive, bizarre movements that chiefly involve the face, mouth, jaw, and tongue: grimacing, pursing of the lips, protrusions of the tongue, opening and closing of the mouth, and deviations of the jaw. The limbs and trunk are involved less often. These movements may be a late complication of psychotropic drugs such as phenothiazines, and have then been termed *tardive* (late) dyskinesias. They also occur in long-standing psychoses, in some elderly individuals, and in some edentulous persons.

Tics

Tics are brief, repetitive, stereotyped, coordinated movements occurring at irregular intervals. Examples include repetitive winking, grimacing, and shoulder shrugging. Causes include Tourette's syndrome and drugs such as phenothiazines and amphetamines.

Chorea

Choreiform movements are brief, rapid, jerky, irregular, and unpredictable. They occur at rest or interrupt normal coordinated movements. Unlike tics, they seldom repeat themselves. The face, head, lower arms, and hands are often involved. Causes include Sydenham's chorea (with rheumatic fever) and Huntington's disease.

Athetosis

Athetoid movements are slower and more twisting and writhing than choreiform movements, and have a larger amplitude. They most commonly involve the face and the distal extremities. Athetosis is often associated with spasticity. Causes include cerebral palsy.

Dystonia

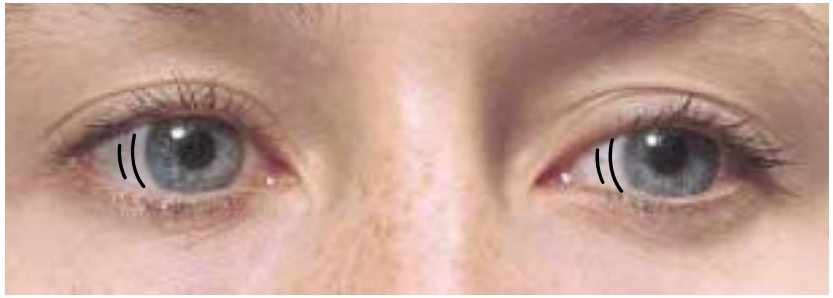
Dystonic movements are somewhat similar to athetoid movements, but often involve larger portions of the body, including the trunk. Grotesque, twisted postures may result. Causes include drugs such as phenothiazines, primary torsion dystonia and, as illustrated, spasmodic torticollis.

TABLE 16-9 ■ Nystagmus

Nystagmus is a rhythmic oscillation of the eyes, analogous to a tremor in other parts of the body. Its causes are multiple, including impairment of vision in early life, disorders of the labyrinth and the cerebellar system, and drug toxicity. Nystagmus occurs normally when a person watches a rapidly moving object (e.g., a passing train). Observe the three characteristics of nystagmus listed below and on the following page. Then refer to textbooks of neurology for differential diagnosis.

Direction of the Quick and Slow Components

Example: Nystagmus to the Left—A Slow Drift to the Right, Then a Quick Jerk to the Left in Each Eye



Nystagmus usually has both fast and slow movements, but is defined by its fast phase. For example, if the eyes jerk quickly to the patient's left and drift back slowly to the right, the patient is said to have nystagmus to the left.

Occasionally, nystagmus consists only of coarse oscillations without quick and slow components. It is then said to be *pendular*.

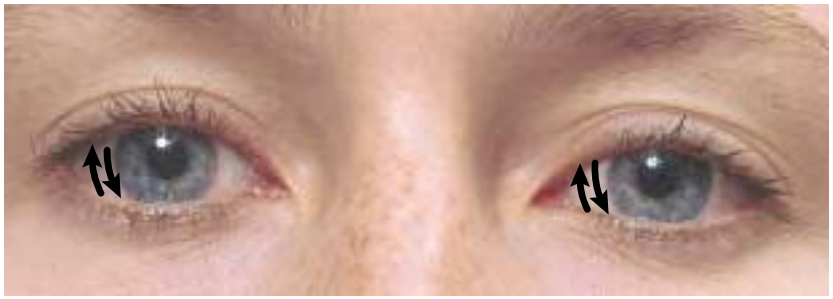
Plane of the Movements

Horizontal Nystagmus

The movements of nystagmus may occur in one or more planes (i.e., horizontal, vertical, or rotary). It is the plane of the movements, not the direction of the gaze, that defines this variable.

Vertical Nystagmus

Rotary Nystagmus

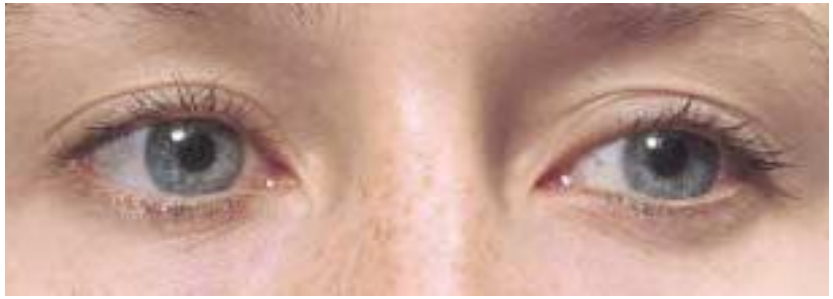


Direction of Gaze in Which Nystagmus Appears

Example: Nystagmus on Right Lateral Gaze

Nystagmus Present (Right Lateral Gaze)

Nystagmus Not Present (Left Lateral Gaze)



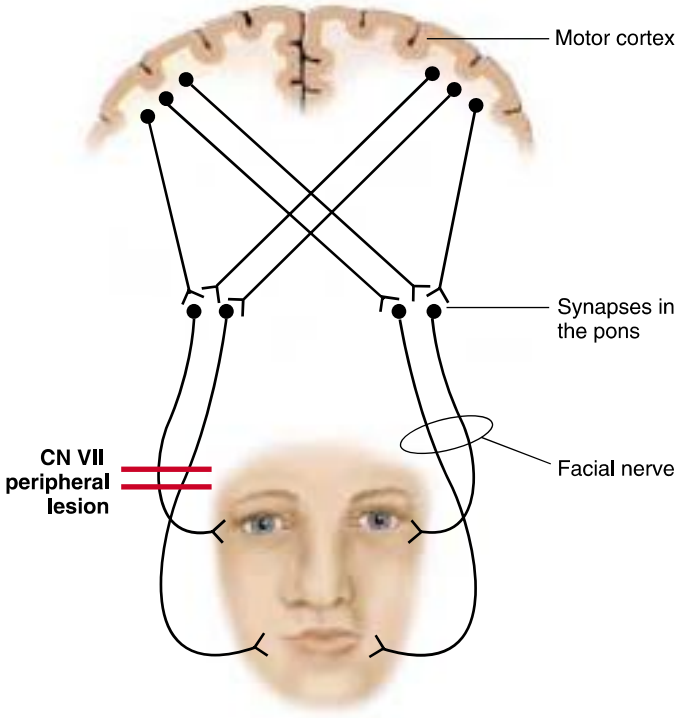
Although nystagmus may be present in all directions of gaze, it may appear or become accentuated only on deviation of the eyes (e.g., to the side or upward). On extreme lateral gaze, the normal person may show a few beats resembling nystagmus. Avoid making assessments in such extreme positions, and observe for nystagmus only within the field of full binocular vision.

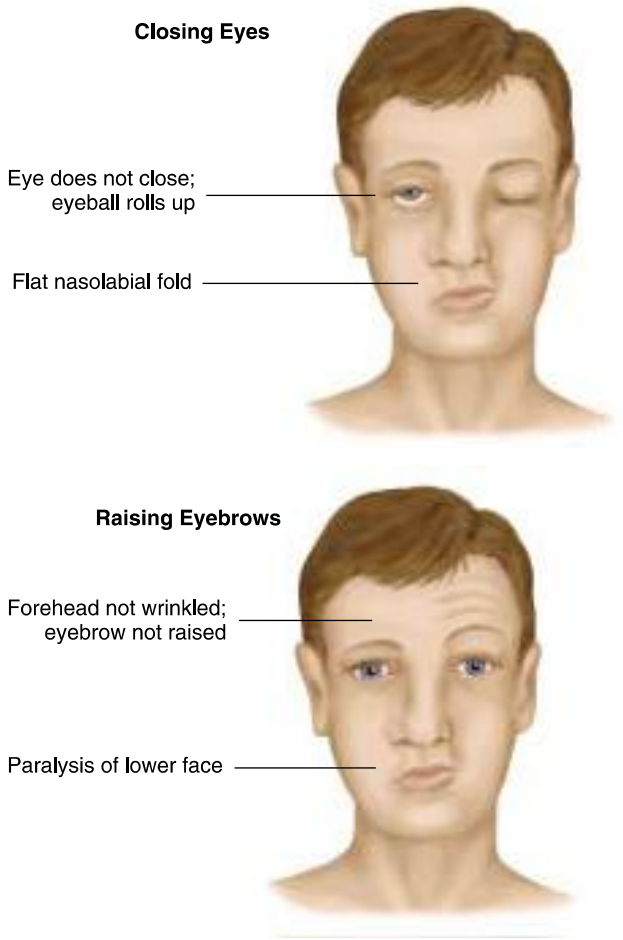
TABLE 16-10 ■ Types of Facial Paralysis

Facial weakness or paralysis may result either (1) from a peripheral lesion of CN VII, the facial nerve, anywhere from its origin in the pons to its periphery in the face, or (2) from a central lesion involving the upper motor neurons between the cortex and the pons. A peripheral lesion of CN VII, exemplified here by a Bell's palsy, is compared with a central lesion, exemplified by a left hemispheric cerebrovascular accident. Note their different effects on the upper part of the face, by which they can be distinguished.

CN VII—Peripheral Lesion

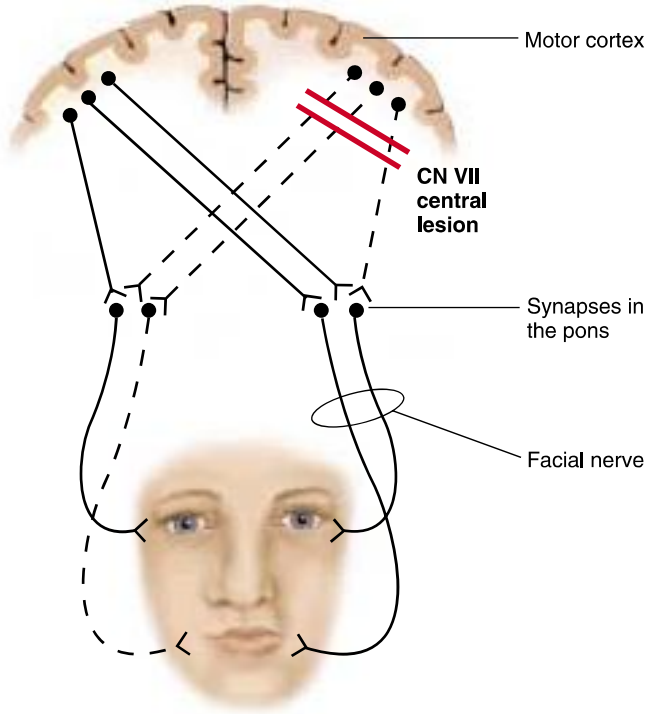
Peripheral nerve damage to CN VII paralyzes the entire right side of the face, including the forehead.





CN VII—Central Lesion

The lower part of the face normally is controlled by upper motor neurons located on only one side of the cortex—the opposite side. *Left-sided damage to these pathways, as in a stroke, paralyzes the right lower face.* The upper face, however, is controlled by pathways from both sides of the cortex. Even though the upper motor neurons on the left are destroyed, others on the right remain and the right upper face continues to function fairly well.



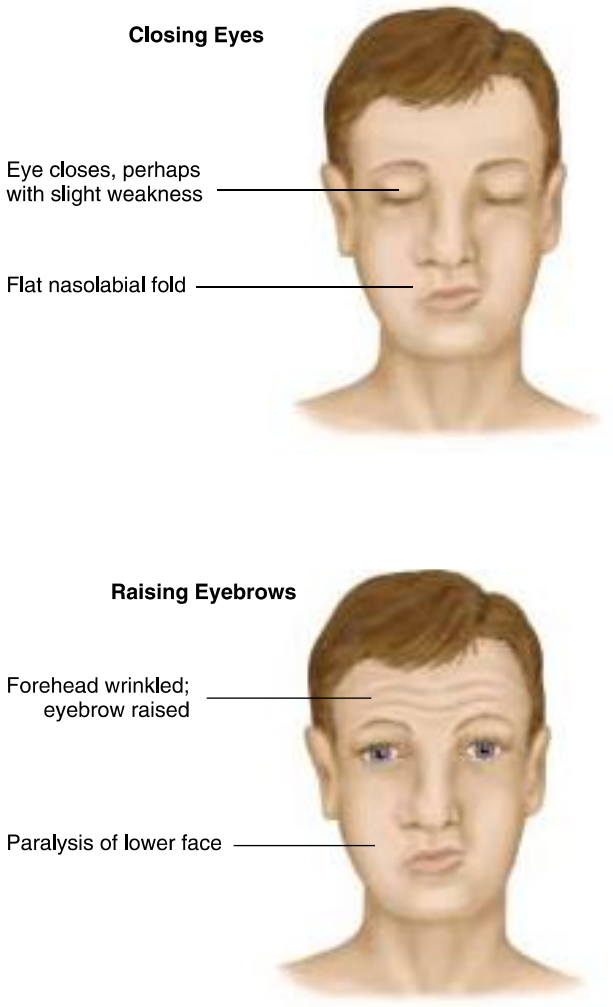


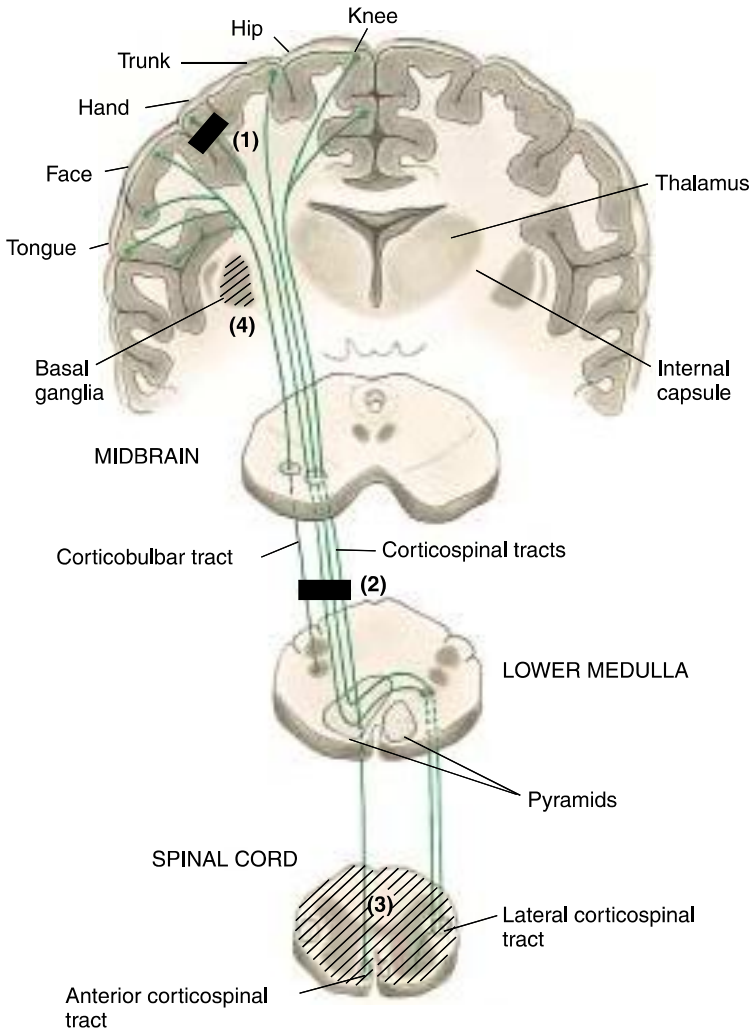
TABLE 16-10 ■ Types of Facial Paralysis

TABLE 16-11 ■ Disorders of Muscle Tone

	Spasticity	Rigidity	Flaccidity	Paratonia
Location of Lesion	Upper motor neuron of the corticospinal tract at any point from the cortex to the spinal cord	Basal ganglia system	Lower motor neuron at any point from the anterior horn cell to the peripheral nerves	Both hemispheres, usually in the frontal lobes
Description	Increased muscle tone (*hypertonia*) that is rate-dependent. Tone is greater when passive movement is rapid, and less when passive movement is slow. Tone is also greater at the extremes of the movement arc. During rapid passive movement, initial hypertonia may give way suddenly as the limb relaxes. This spastic "catch" and relaxation is known as "clasp-knife" resistance.	Increased resistance that persists throughout the movement arc, independent of rate of movement, is called *lead-pipe rigidity*. With flexion and extension of the wrist or forearm, a superimposed rachetlike jerkiness is called *cogwheel rigidity*.	Loss of muscle tone (*hypotonia*), causing the limb to be loose or floppy. The affected limbs may be hyperextensible or even flail-like.	Sudden changes in tone with passive range of motion. Sudden loss of tone that increases the ease of motion is called *mitgehen* (moving with). Sudden increase in tone making motion more difficult is called *gegenhalten* (holding against).
Common Cause	Stroke, especially late or chronic stage	Parkinsonism	Guillain–Barré syndrome; also initial phase of spinal cord injury (spinal shock) or stroke	Dementia

TABLE 16-12 ■ Disorders of the Central and Peripheral Nervous Systems

Central Nervous System Disorders



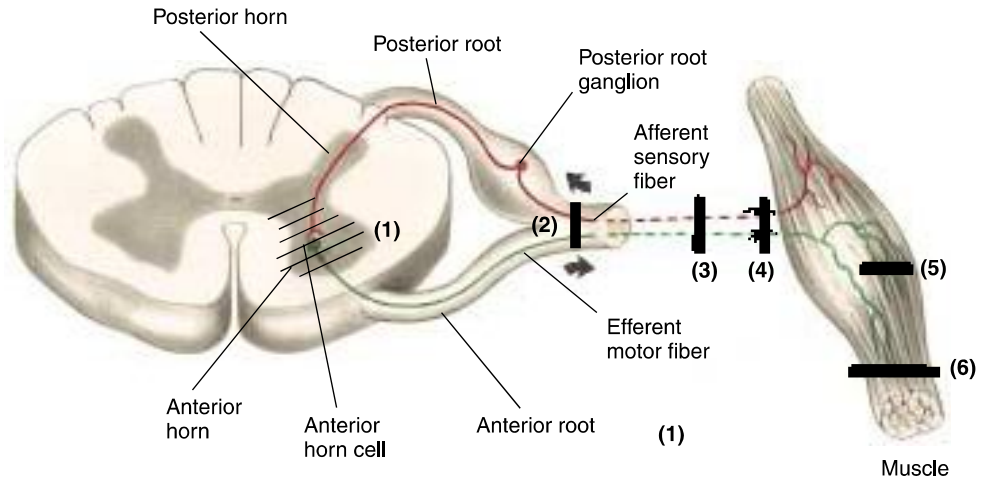
(table continues next page)

TABLE 16-12 ■ Disorders of the Central and Peripheral Nervous Systems *(Continued)*

Central Nervous System Disorders

Location of Lesion	Typical Findings: *Motor*	Typical Findings: *Sensory*	Typical Findings: *Deep Tendon Reflexes*	Examples of Cause
Cerebral Cortex (1)	Chronic contralateral upper motor neuron weakness and spasticity. Flexion is stronger than extension in the arm, plantar flexion is stronger than dorsiflexion in the foot, and the leg is externally rotated at the hip.	Contralateral sensory loss on the limbs and trunk on the same side as the motor deficits	↑	Cortical stroke
Brainstem (2)	Weakness and spasticity as above, plus cranial nerve deficits such as diplopia (from weakness of the extraocular muscles) and dysarthria	Variable. No typical sensory findings	↑	Brainstem stroke, acoustic neuroma
Spinal Cord (3)	Weakness and spasticity, as above, but often affecting both sides (when cord damage is bilateral), causing paraplegia or quadriplegia depending on the level of injury	Dermatomal sensory deficit on the trunk bilaterally at the level of the lesion, and sensory loss from tract damage below the level of the lesion	↑	Trauma, causing cord compression
Subcortical Gray Matter: Basal Ganglia (4)	Slowness of movement (bradykinesia), rigidity, and tremor	Sensation not affected	Normal or ↓	Parkinsonism
Cerebellar (not illustrated)	Hypotonia, ataxia, and other abnormal movements, including nystagmus, dysdiadochokinesis, and dysmetria	Sensation not affected	Normal or ↓	Cerebellar stroke, brain tumor

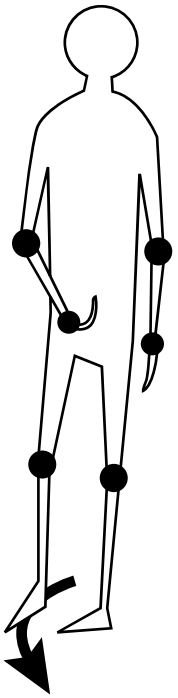
Peripheral Nervous System Disorders

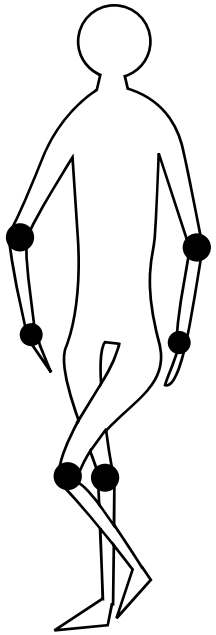


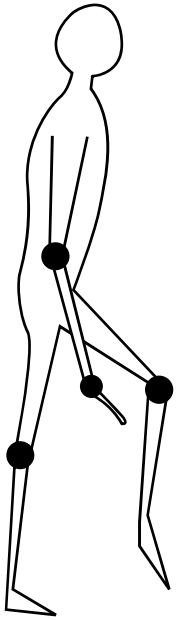
Typical Findings

Location of Lesion	*Motor*	*Sensory*	*Deep Tendon Reflexes*	Examples of Cause
Anterior Horn Cell (1)	Weakness and atrophy in a segmental or focal pattern; fasciculations	Sensation intact	↓	Polio, amyotrophic lateral sclerosis
Spinal Roots and Nerves (2)	Weakness and atrophy in a root-innervated pattern; sometimes with fasciculations	Corresponding dermatomal sensory deficits	↓	Herniated cervical or lumbar disc
Peripheral Nerve—Mononeuropathy (3)	Weakness and atrophy in a peripheral nerve distribution; sometimes with fasciculations	Sensory loss in the pattern of that nerve	↓	Trauma
Peripheral Nerve—Polyneuropathy (4)	Weakness, and atrophy more distal than proximal; sometimes with fasciculations	Sensory deficits, commonly in stocking–glove distribution	↓	Peripheral polyneuropathy of alcoholism, diabetes
Neuromuscular Junction (5)	Fatigability more than weakness	Sensation intact	Normal	Myasthenia gravis
Muscle (6)	Weakness usually more proximal than distal; fasciculations rare	Sensation intact	Normal or ↓	Muscular dystrophy

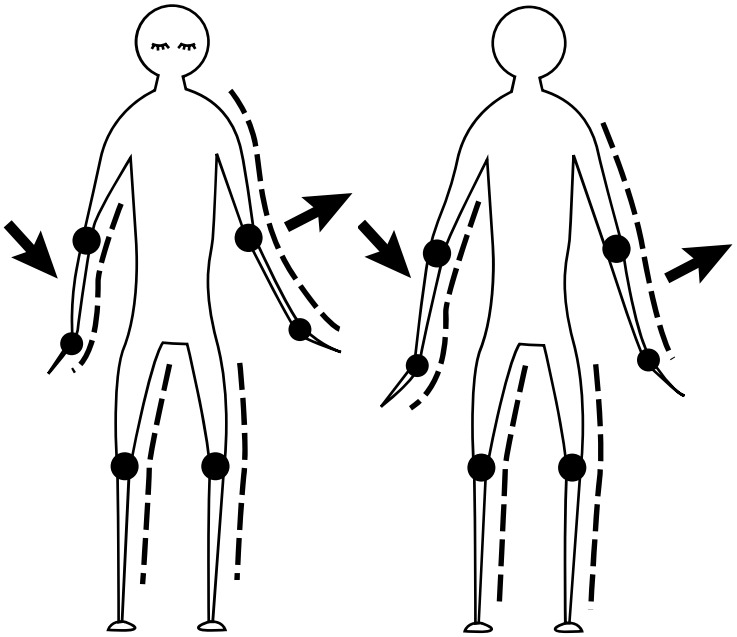
TABLE 16-13 ■ Abnormalities of Gait and Posture

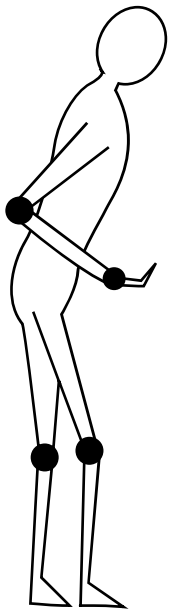


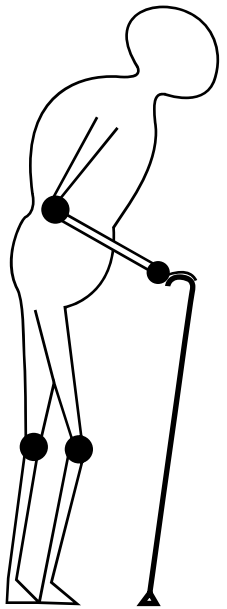




Underlying Defect	Spastic Hemiparesis	Scissors Gait	Steppage Gait
	Associated with lesion in corticospinal tract, as with stroke	Associated with bilateral spastic paresis of the legs	Associated with foot drop, usually secondary to lower motor neuron disease
Description	One arm is held immobile and close to the side, with elbow, wrist, and interphalangeal joints flexed. The leg is extended, with plantar flexion of the foot. On walking, the patient either drags the foot, often scraping the toe, or circles it stiffly outward and forward (*circumduction*).	The gait is stiff. Each leg is advanced slowly, and the thighs tend to cross forward on each other at each step. The steps are short. The patient appears to be walking through water.	These patients either drag their feet or lift them high, with knees flexed, and bring them down with a slap onto the floor, thus appearing to be walking up stairs. They are unable to walk on their heels. The steppage gait may involve one or both sides.







Underlying Defect	Sensory Ataxia	Cerebellar Ataxia	Parkinsonian Gait	Gait of Older Age
	Associated with loss of position sense in the legs, as from polyneuropathy or posterior column damage	Associated with disease of the cerebellum or associated tracts	Associated with the basal ganglia defects of Parkinson's disease	The aging process
Description	The gait is unsteady and wide based (with feet wide apart). These patients throw their feet forward and outward and bring them down, first on the heels and then on the toes, with a double tapping sound. They watch the ground for guidance while walking. With eyes closed, they cannot stand steadily with feet together (a positive Romberg sign) and the staggering gait worsens.	The gait is staggering, unsteady, and wide based, with exaggerated difficulty on the turns. These patients cannot stand steadily with feet together, whether their eyes are open or closed.	The posture is stooped, with head and neck forward and hips and knees slightly flexed. Arms are flexed at elbows and wrists. The patient is slow in getting started. Steps are short and often shuffling. Arm swings are decreased and the patient turns around stiffly—"all in one piece."	Speed, balance, and agility decrease with aging. Steps become short, uncertain, and even shuffling. The legs may be flexed at hips and knees. A cane may bolster lost confidence.

TABLE 16-14 ■ Metabolic and Structural Coma

Although there are many causes of coma, most can be classified as either structural or metabolic. Findings vary widely in individual patients; the features listed are general guidelines rather than strict diagnostic criteria. Remember that psychiatric disorders may mimic coma.

	Toxic–Metabolic	Structural
Pathophysiology	Arousal centers poisoned or critical substrates depleted	Lesion destroys or compresses brainstem arousal areas, either directly or secondary to more distant expanding mass lesions.
Clinical Features		
■ Respiratory pattern	If regular, may be normal or hyperventilation. If irregular, usually Cheyne–Stokes	Irregular, especially Cheyne–Stokes or ataxic breathing
■ Pupillary size and reaction	Equal, reactive to light. If *pinpoint* from opiates or cholinergics, you may need a magnifying glass to see the reaction. May be unreactive if *fixed and dilated* from anticholinergics or hypothermia	Unequal or unreactive to light (fixed) *Midposition, fixed*—suggests midbrain compression *Dilated, fixed*—suggests compression of CN III from herniation
■ Level of consciousness	Changes after pupils change	Changes before pupils change
Examples of Cause	Uremia, hyperglycemia	Epidural, subdural, or intracerebral hemorrhage
	Alcohol, drugs, liver failure	Cerebral infarct or embolus
	Hypothyroidism, hypoglycemia	Tumor, abscess
	Anoxia, ischemia	Brainstem infarct, tumor, or hemorrhage
	Meningitis, encephalitis	Cerebellar infarct, hemorrhage, tumor, or abscess
	Hyperthermia, hypothermia	

TABLE 16-15 ■ Pupils in Comatose Patients

Pupillary size, equality, and light reactions help in assessing the cause of coma and in determining the region of the brain that is impaired. Remember that unrelated pupillary abnormalities, including miotic drops for glaucoma or mydriatic drops for a better view of the ocular fundi, may have preceded the coma.

Small or Pinpoint Pupils

Bilaterally small pupils (1–2.5 mm) suggest (1) damage to the sympathetic pathways in the hypothalamus, or (2) metabolic encephalopathy (a diffuse failure of cerebral function that has many causes, including drugs). Light reactions are usually normal.

Pinpoint pupils (< 1 mm) suggest (1) a hemorrhage in the pons, or (2) the effects of morphine, heroin, or other narcotics. The light reactions may be seen with a magnifying glass.

Midposition Fixed Pupils

Pupils that are in the *midposition* or *slightly dilated* (4–6 mm) and are *fixed to light* suggest structural damage in the midbrain.

Large Pupils

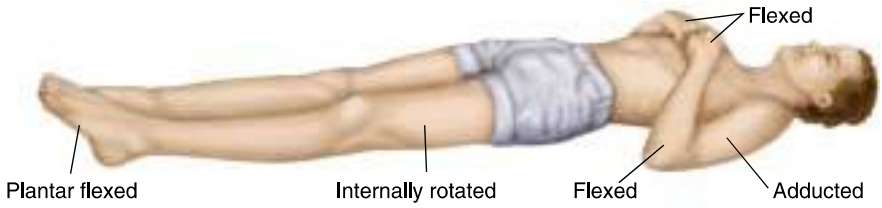
Bilaterally fixed and dilated pupils may be due to severe anoxia and its sympathomimetic effects, as seen after cardiac arrest. They may also result from atropinelike agents, phenothiazines, or tricyclic antidepressants.

Bilaterally large reactive pupils may be due to cocaine, amphetamine, LSD, or other sympathetic nervous system agonists.

One Large Pupil

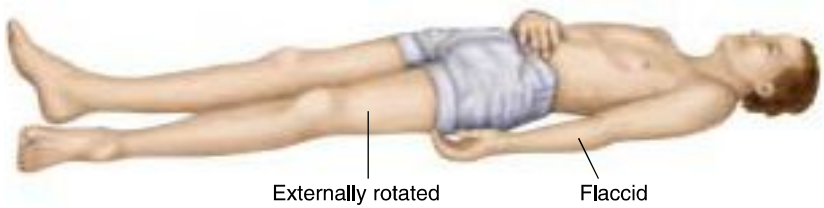
A pupil that is *fixed and dilated* warns of herniation of the temporal lobe, causing compression of the oculomotor nerve and midbrain.

TABLE 16-16 ■ Abnormal Postures in Comatose Patients



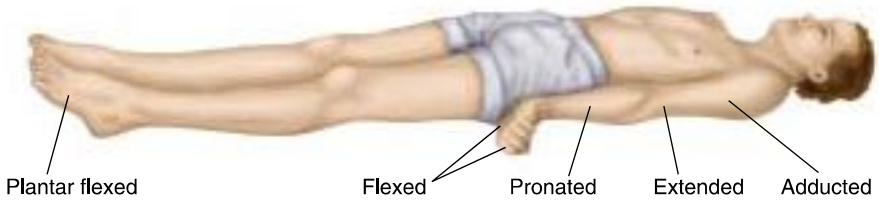
Decorticate Rigidity (Abnormal Flexor Response)

In decorticate rigidity, the upper arms are held tight to the sides with elbows, wrists, and fingers flexed. The legs are extended and internally rotated. The feet are plantar flexed. This posture implies a destructive lesion of the corticospinal tracts within or very near the cerebral hemispheres. When unilateral, this is the posture of chronic spastic hemiplegia.



Hemiplegia (Early)

Sudden unilateral brain damage involving the corticospinal tract may produce a hemiplegia (one-sided paralysis), which early in its course is flaccid. Spasticity will develop later. The paralyzed arm and leg are slack. They fall loosely and without tone when raised and dropped to the bed. Spontaneous movements or responses to noxious stimuli are limited to the opposite side. The leg may lie externally rotated. One side of the lower face may be paralyzed, and that cheek puffs out on expiration. Both eyes may be turned away from the paralyzed side.



Decerebrate Rigidity (Abnormal Extensor Response)

In decerebrate rigidity, the jaws are clenched and the neck is extended. The arms are adducted and stiffly extended at the elbows, with forearms pronated, wrists and fingers flexed. The legs are stiffly extended at the knees, with the feet plantar flexed. This posture may occur spontaneously or only in response to external stimuli such as light, noise, or pain. It is caused by a lesion in the diencephalon, midbrain, or pons, although severe metabolic disorders such as hypoxia or hypoglycemia may also produce it.