

SECTION I. NEUROLOGICAL EXAMINATION AND NEURODIAGNOSTIC TESTING

1 Neurological Examination

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INTRODUCTION

The neurological history and examination provide information to help localize lesions of the nervous system. The neurological examination is incorporated in the context of the patient's overall health history and general physical examination. Evidence of systemic disease is considered in the interpretation of the neurological findings.

Goals of an emergency neurological examination:

1. Is there a neurological condition?
2. Where is (are) the lesion(s) located?
3. What are the possible causes?
4. Can the patient be discharged safely from the emergency department or is hospitalization required?

Neurological History

A detailed neurological history allows one to focus on important components of the neurological examination, thus saving time and resources. The more specific and detailed a history, the greater is the likelihood of making a definite diagnosis in the emergency department. About 75% of neurological diagnoses are made from the history alone. An account from family members and bystanders can be an important source of information. A detailed description of the event is more important than the patient or a bystander volunteering a diagnosis such as "I had a seizure." The history obtained from the patient can be considered a part of the mental status examination.

Important Historical Elements of a Focused Neurological Examination

1. Onset of symptoms: time and mode (Look beyond the symptoms, to the context in which they occur.)
2. Temporal relationships of symptoms

Table 1.1. The Brief Mental Status Examination				
Item	Score		=	Total
	Number of errors	× Weight		
What year is it now?	0 or 1	× 4	=	
What month is it?	0 or 1	× 3	=	
Present memory phrase: "Repeat this phrase after me and remember it: <i>John Brown, 42 Market Street, New York.</i> "				
About what time is it? (Answer correct if within one hour)	0 or 1	× 3	=	
Count backwards from 20 to 1.	0, 1, or 2	× 2	=	
Say the months in reverse.	0, 1, or 2	× 2	=	
Repeat memory phrase (Each underlined portion is worth 1 point.)	0, 1, 2, 3, 4, or 5	× 2	=	
Final score is the sum of the total			=	
Possible score range, from 0 to 28. 0–8 normal; 9–19 mildly impaired; 20–28 severely impaired. Source: Kaufman DM, Zun L. A quantifiable, brief mental status examination for emergency patients. <i>J Emerg Med.</i> 1995;13:449–56.				

- 3. Progression of symptoms
- 4. Associated symptoms (neurological and nonneurological)
- 5. Exacerbating and alleviating factors
- 6. Symptoms that indicate involvement of a particular region of central nervous system
- 7. History of similar event
- 8. History of medication use: illicit drug use, exposure to toxins, head trauma

Neurological Examination

- Mental status
- Cranial nerve function
- Motor function
- Deep tendon reflexes, cutaneous reflexes, and miscellaneous signs
- Sensory modalities
- Pathological reflexes

Mental Status

- See Table 1.1.
- Six elements of mental status evaluation, modified from Zun and Howes (1988), are:
- 1. Appearance, behavior, and attitude
 - Is dress appropriate?
 - Is motor behavior at rest appropriate?
 - Is the speech pattern normal?
 - 2. Disorders of thought
 - Are the thoughts logical and realistic?
 - Are false beliefs or delusions present?
 - Are suicidal or homicidal thoughts present?

Table 1.2. Glasgow Coma Scale	
<i>Eye opening</i>	
Opens eyes spontaneously	4
Opens eyes to verbal command	3
Opens eyes to pain	2
Does not open eyes	1
<i>Verbal response</i>	
Alert and oriented	5
Converses but disoriented	4
Speaking but nonsensical	3
Moans or makes unintelligible sounds	2
No response	1
<i>Motor response</i>	
Follows commands	6
Localizes pain	5
Movement or withdrawal from pain	4
Abnormal flexion (decorticate)	3
Abnormal extension (decerebrate)	2
No response	1
Total	3–15

- 3. Disorders of perception
 - Are hallucinations present?
- 4. Mood and affect
 - What is the prevailing mood?
 - Is the emotional content appropriate for the setting?
- 5. Insight and judgment
 - Does the patient understand the circumstances surrounding the visit?
- 6. Sensorium and intelligence
 - Is the level of consciousness normal?
 - Is cognition or intellectual functioning impaired?

The Glasgow Coma Scale is often used as a method of briefly quantitating neurological dysfunction (see Table 1.2).

More specific testing of higher cortical functions is often added to the mental status examination in patients with evidence of focal lesions. Delineation of aphasias can involve detailed testing but is usually limited to gross observation of speech output, conduction (ability to repeat), and comprehension. Other tests include naming objects, distinguishing between right and left, and testing for visual and sensory neglect (especially important in parietal and thalamic lesions).

Cranial Nerve Function

Evaluating the Cranial Nerves

- I. Olfactory

Evaluate: Smell

Anatomic Location: Olfactory bulb and tract

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Tests: Odor recognition
Significant Findings: Lack of odor perception in one or both sides

Lesions of the olfactory groove (typically meningioma) can have associated psychiatric symptoms related to frontal lobe injury.

► **II. Optic**

Evaluate: Vision
Anatomic Location: Optic nerve, chiasma, and tracts
Tests: Visual acuity
Significant Findings: Reduced vision
Tests: Pupillary light reflex
Significant Findings: Afferent pupillary defect (Marcus-Gunn pupil +/–)
Tests: Visual field testing

Testing visual acuity is important when primarily ocular lesion(s) are suspected. Papilledema: increased intracranial pressure due to tumor, hydrocephalus, or other causes. Hollenhorst plaque: a bright-appearing cholesterol or atheromatous embolus visualized by funduscopy examination of the retinal vessels, implies an embolic process. Optic nerve lesions: monocular visual disturbance. See comment 1.

► **III. Oculomotor**

Evaluate: Eye movement, pupil contraction and accommodation, eyelid elevation
Anatomic Location: Midbrain
Tests: Extraocular eye movements (EOM)
Significant Findings: Impairment of one or more eye movements or disconjugate gaze
Tests: Pupillary light reflex
Significant Findings: Pupillary dilatation, ptosis

Cranial nerves III, IV, VI – Check for EOM. Dysfunction of these nerves can be localized by noting the direction of gaze, which causes or worsens a diplopia, and any loss of upgaze, downgaze, or horizontal movements in either eye. Diplopia that worsens on lateral gaze suggests an ipsilateral palsy of cranial nerve VI or lateral rectus weakness. See comment 2.

► **IV. Trochlear**

Evaluate: Eye movement
Anatomic Location: Midbrain
Tests: Extraocular eye movements
Significant Findings: Impairment of one or more eye movements or disconjugate gaze

► **V. Trigeminal**

Evaluate: Facial sensation, mastication
Anatomic Location: Pons

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Tests: Sensation above eye, between eye and mouth, below the mouth to angle of jaw
Significant Findings: Reduced sensation in one or divisions of cranial nerve V
Tests: Corneal reflex
Significant Findings: Impaired
Tests: Palpation of masseter muscles
Significant Findings: Reduced strength in masseter or pterygoid muscles

*If an abnormality is found in only one or two divisions of cranial nerve V (V₁–V₃), the findings imply a lesion distal to the gasserian ganglion.
See comment 3.*

► VI. Abducens

Evaluate: Ocular movement
Anatomic Location: Pons
Tests: Extraocular eye movements
Significant Findings: Reduced eye abduction

► VII. Facial

Evaluate: Facial expression secretions, taste, visceral and cutaneous sensibility
Anatomic Location: Pons
Tests: Facial expression
Significant Findings: Weakness of upper or lower face or eye closure
Tests: Corneal reflex
Significant Findings: Impaired
Tests: Taste on anterior $\frac{2}{3}$ tongue
Significant Findings: Impaired

Seventh cranial nerve lesions can be either central or peripheral. In central lesions, located proximal to the seventh nerve nucleus and contralateral to the resulting facial droop, the upper face (periorbital area and forehead) will be relatively spared. The palpebral fissure may be slightly larger ipsilateral to the facial droop. In peripheral lesions, weakness is ipsilateral to the lesion of the seventh cranial nerve nucleus or the nerve itself. Other brainstem signs are seen typically when a lesion involves the nerve nucleus; the term Bell’s palsy commonly refers to lesions of the nerve distal to the nucleus. Eye closure may be lost in severe cases of peripheral seventh nerve lesions. Hyperacusis is due to loss of the seventh nerve’s dampening influence on the stapes.

► VIII. Acoustic

Evaluate: Hearing, equilibrium
Anatomic Location: Pons
Tests: Auditory and vestibular
Significant Findings: Reduced hearing

The eighth cranial nerve consists of an auditory component and a vestibular component. Deafness rarely results from cortical lesions, which more often cause difficulty with

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sound localization. Common bedside testing involves comparison for gross symmetry with a high-pitched tuning fork (512 or 256 Hz) or by finger rubbing near the ear, and the Weber and Rinne tests (for air conduction compared to bone conduction of sound). Lesions of the vestibular nuclei and the vestibular portion of the eighth cranial nerve can produce vertigo, nausea, vomiting, and nystagmus.

► IX. Glossopharyngeal

Evaluate: Taste, glandular secretions, swallowing, visceral sensibility (pharynx, tongue, tonsils)

Anatomic Location: Medulla

Tests: Gag reflex

Significant Findings: Reduced gag

Tests: Speech (phonation)

Significant Findings: Dysarthria

Tests: Swallowing

Significant Findings: Dysarthria

Lesions of cranial nerve IX may be undetected clinically.

► X. Vagus

Evaluate: Involuntary muscle and gland control (pharynx, larynx, trachea, bronchi, lungs, digestive system, heart), swallowing, phonation, visceral and cutaneous sensibility, taste

Anatomic Location: Medulla

Tests: Phonation

Significant Findings: Hoarseness

Tests: Coughing

Significant Findings: Impaired

Hoarseness and dysphagia can be seen with unilateral or bilateral injury to cranial nerve X.

► XI. Accessory

Evaluate: Movement of head and shoulders

Anatomic Location: Cervical

Tests: Resisted head turning

Significant Findings: Weakness of trapezius and sternocleidomastoid muscle

The loss of strength is often greater with nuclear or peripheral lesions as opposed to a supranuclear injury of cranial nerve XI.

► XII. Hypoglossal

Evaluate: Tongue movement

Anatomic Location: Medulla

Tests: Tongue protrusion

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On protrusion, a unilateral weak tongue deviates toward the side of weakness in lesions of the nucleus and peripheral nerve injury, but away from supranuclear lesions. Nuclear and peripheral lesions are associated with atrophy when chronic.

Comment 1. Visual field defects include (a) homonymous hemianopsia, a large hemispheric lesion or lesion of the lateral geniculate ganglion, (b) bitemporal hemianopsia, a lesion of the pituitary area compressing the optic chiasm, (c) central scotoma, a lesion of the optic nerve that typically occurs with optic neuritis, (d) superior quadrantanopsia, a contralateral temporal lobe lesion.

The pupillary light reflex includes the swinging flashlight test that may reveal a consensual response (contralateral pupillary constriction with stimulation) despite a relatively poor direct response ipsilaterally (afferent pupillary defect, also known as a Marcus-Gunn pupil) due to an optic nerve lesion.

Bilateral pinpoint pupils in a comatose patient with apneustic or agonal respirations imply a pontine lesion or a narcotics overdose. Loss of the oculocephalic reflex, or “doll’s eyes,” is rarely seen in drug overdose and implies brainstem injury (normally, eye movements are opposite to rotary movements of the head performed by the examiner). A unilateral dilated pupil in a comatose patient implies brainstem herniation, usually related to contralateral hemispheric mass effect. Bilateral dilated and fixed pupils and loss of all brainstem reflexes and respiratory drive occur in brain death. Paralytic agents can produce a similar clinical presentation, but typically pupils are not affected.

Comment 2. Cranial nerve III and sympathetic fibers are responsible for eye opening; consequently, ptosis, without or with a Horner’s syndrome (ptosis, miosis, anhidrosis), is recorded as part of the extraocular muscle examination (although the pupil abnormalities associated with these syndromes can be recorded as part of the visual examination). A classic finding of abnormal ocular motility is referred to as an internuclear ophthalmoplegia (INO). See Chapter 18, “Neuro-Ophthalmological Emergencies,” for definition of INO. Abnormal ipsilateral adduction with visual tracking of the eye is seen with lacunar infarcts of the medial longitudinal fasciculus (MLF) or with multiple sclerosis plaques in the MLF.

Comment 3. Distinct splitting of sensory function at the midline face can imply a functional disorder. Vibration is not tested for cranial nerve V function, but splitting of vibratory sensation across the forehead or skull is further evidence of a functional component in a clinical presentation.

Motor Function**Tone and Power**

Muscle tone is evaluated by passively moving joints through a range of motion at varying velocities. Rigidity occurs in extrapyramidal disorders such as Parkinson’s disease. Tremor plus rigidity yields “cogwheel” rigidity. Muscle tone can be increased in both pyramidal and extrapyramidal disturbances. Acute central nervous system (CNS) lesions involving the pyramidal tracts often produce hypotonia. This finding evolves over days, producing hyperreflexia and hypertonicity, referred to as spasticity. Hypertonicity can occur acutely in brainstem lesions (decorticate or decerebrate posturing). Hypotonicity may be present chronically in neuromuscular disease.

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Muscle group power is graded on a scale of 0 to 5:

Grade 0	No muscle contraction
Grade 1	Muscle contraction without joint movement
Grade 2	Partial movement with gravity eliminated
Grade 3	Movement against gravity
Grade 4	Movement against some resistance
Grade 5	Normal strength

The two most commonly performed tests for detection of mild weakness:

Pronator drift	Patient with arms extended and supinated tends to pronate and lower the whole arm with flexion at the elbow
Gait observation	Includes heel and toe walking

Coordination

Equilibrium refers to the coordination and balance of the whole body. The presence of ataxia and the result of tests for Romberg sign and tandem gait are sensitive markers of dysequilibrium.

Truncal ataxia is tested by observing sitting, balance when standing, and gait (classically “wide-based” in cases of mild to moderate ataxia).

Limb ataxia (appendicular ataxia) can be present in a single extremity (usually an arm) but is more often seen in an ipsilateral arm and leg pattern, with the patient exhibiting a tendency to fall to that side. Limb ataxia is demonstrated by testing finger-to-nose and heel-to-shin movements. Limb ataxia causes intention tremor (see below) and dysidiadochokinesia (impairment of rapid alternating movements), classic indicators of lesions of the cerebellar system.

When limb ataxia is combined with weakness, the term *ataxic hemiparesis* is used. Ataxic hemiparesis is a classic finding for an internal capsule or pontine lacunar stroke when presenting as a pure motor stroke syndrome. Limb ataxia in the absence of weakness suggests a lesion of the cerebellar hemispheres and their projections, whereas truncal ataxia in isolation suggests a lesion of midline cerebellar structures and their projections.

Abnormal Movements

See Chapter 13, Movement Disorders.

Deep Tendon Reflexes, Cutaneous Reflexes, and Miscellaneous Signs

When deep tendon reflexes (DTR) are increased or hyperactive, reflex “spread” occurs in other local muscles, resulting in an increased intensity of muscle contraction.

Deep Tendon Reflexes

Deep tendon reflexes are generally graded on a 0 to 4 basis.

Grade 0	Reflex is not elicited
Grade 1	Hypoactive reflex or one that is present only with reinforcing maneuvers
Grade 2	Normal reflex
Grade 3	Reflexes that appear to be hyperactive but may not necessarily be pathological
Grade 4	Clonic reflexes that may or may not be pathological

Cutaneous Reflexes

Abdominal reflex	Abdominal wall muscle contraction
Cremasteric reflex (stimulation of the skin over the scrotal area)	Testicular elevation
Anal wink reflex	Anal contraction with stimulation

Sensory Modalities

Objective findings of a sensory examination are difficult to interpret, particularly in a concise emergency department (ED) examination, since the response of both the patient and the examiner can be subjective. A quick and effective ED examination consists of checking for recognition of numbers written on the palms of hand with eyes closed. A major sensory deficit is unlikely if the patient is able to recognize the numbers and letters. A cooperative patient can outline the area of sensory deficit by marking or with pinpricks. A “sensory level” can be outlined by the patient by having the patient run his or her own finger up the body until there is a sensory change.

The sensory examination is documented as a response to five modalities: light touch, pinprick, vibration, joint position, and temperature.

Light touch and pinprick sensation assesses the integrity of the peripheral nervous system and spinal cord sensory tracts. It can also be used to assess the presence of a cortical lesion (e.g., “extinction” in parietal lobe lesions occurs when bilateral stimuli are presented and the sensory stimulus is neglected contralateral to the lesion).

Perception of temperature or pain requires integrity of unmyelinated peripheral nerves (which originate as bipolar neurons in the dorsal root ganglia), the spinothalamic tracts of the spinal cord and brainstem, the ventral posterolateral and ventral posteromedial thalami, and thalamic projections to the parietal lobes.

Sensation of light touch is transmitted similarly, but it is also likely transmitted through the posterior columns. Sensory loss to light touch and pinprick can

occur in the distribution of a single nerve, nerve root, plexus pattern, hemi cord pattern, transverse cord pattern, or crossed brainstem pattern (see later), or it can occur somatotopically, corresponding to lesions above the brainstem (e.g., contralateral face-arm-leg). A lesion is localized to the brainstem when sensory loss occurs on one side of the face and contralateral body. A “stocking-glove” pattern is usually seen with polyneuropathy, often due to diabetes. Perception of vibratory and position sense requires integrity of myelinated nerve fibers (originating as bipolar neurons in the dorsal root ganglion), the posterior columns, the medial lemniscus, ventral posterolateral nucleus of the thalamus, and cortex. Lesions of the posterior columns are demonstrated by loss of vibratory and position sense disproportionate to the loss of other modalities (e.g., B₁₂ deficiency). Vibratory sensation is best tested with a 128-Hz tuning fork, and position sense is tested by small excursions of the distal digits.

Pathological Reflexes

Pathological reflexes can be associated with the following signs:

- Frontal release signs consist of glabellar, snout, suck, root, grasp, and palmomental reflexes. These signs usually indicate bilateral frontal lobe disease.
- Hoffmann’s sign indicates hyperreflexia in the upper extremities, elicited by brisk tapping of the distal digits in the hand and observing for flexion of the thumb.
- Babinski sign occurs when plantar stimulation of the foot with a blunt object produces extension of the great toe and fanning of the other toes. This reflex is synonymous with an extensor plantar response and is a sign of upper motor neuron dysfunction. Other methods of eliciting an “upgoing toe” involve stimulation of the lateral foot (Chaddock’s sign) or pinprick over the dorsum of the foot (Bing’s sign).

Anatomical Basis of Neurological Examination

A simple method to remember the anatomic basis of neurological examination is to focus on five “levels” of the CNS, which are the brain, the brainstem, the spinal cord, the peripheral nerves, and the muscles.

Brain (hemispheres)	(a) Alteration of thought processes or consciousness, language problems (dysphasia/aphasia), neglect. (b) Seizures and involuntary movements. (c) When motor and sensory deficits are present, they occur on the same side.
Brainstem	(a) Cranial nerve(s) deficits in association with motor and sensory deficits (b) Diplopia, vertigo, dysarthria, dysphagia, disequilibrium
Spinal cord	(a) Well-demarcated “level” – sensory or motor. Normal function above and below the level. (b) Sensory dissociation – decreased pain on one side and decreased vibration and position on the other side;