

Emergency Neurology

Principles and Practice

Edited by

SID M. SHAH

*Michigan State University–
College of Human Medicine
Ingham Regional Medical Center
Lansing, Michigan*

KEVIN M. KELLY

*MCP Hahnemann University School of Medicine
Pittsburgh, Pennsylvania*

Foreword by JOHN G. WIEGENSTEIN



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Contents

<i>Preface</i>	<i>page xi</i>
<i>Foreword</i>	xiii
<i>Acknowledgments</i>	xv
<i>List of Contributors</i>	xvii

Part I. Neurological Examination and Neurodiagnostic Testing

1. The Neurological Examination	3
THOMAS F. SCOTT	
2. Neuroradiology	14
ANDREW L. GOLDBERG	
THOMAS J. FIX	
3. Electroencephalography	42
IVO DRURY	
AHMAD BEYDOUN	
4. Lumbar Puncture	47
JAMES P. VALERIANO	
DOUGLAS J. GELB	
5. Electromyography	57
JAMES W. ALBERS	
JOHN J. WALD	
6. Electronystagmography	71
NEIL T. SHEPARD	
7. Evoked Potentials	83
NAVIN K. VARMA	

Part II. Common Neurological Presentations

8. Altered Level of Consciousness	93
J. STEPHEN HUFF	
WILLIAM J. BRADY	
9. Headache	105
DOUGLAS GELB	
ASHOK HARWANI	
WILLIAM D. FALES	

CONTENTS

10.	Weakness	122
	GEORGE A. SMALL LYNN BROWN	
11.	Dizziness	132
	KEVIN M. KELLY STEVEN A. TELIAN	
12.	Seizures	154
	KEVIN M. KELLY JAMES P. VALERIANO JERALD A. SOLOT	
13.	Gait Disturbance	173
	JON BRILLMAN KATHLEEN COWLING	
Part III. Specific Neurological Conditions		
14.	Central Nervous System Infections in Adults	183
	PAUL BLACKBURN OLIVER W. HAYES GREG FERMANN	
15.	Viral Encephalitis	202
	EARL J. REISDORFF	
16.	Cerebrovascular Disease	214
	MICHAEL R. FRANKEL MARC CHIMOWITZ	
17.	Movement Disorders	232
	SID M. SHAH ROGER L. ALBIN	
18.	Neuromuscular Disorders	253
	JAMES W. ALBERS JOHN J. WALD	
19.	Musculoskeletal and Neurogenic Pain	273
	ROBERT G. KANIECKI L. R. SEARLS	
20.	Neuro-ophthalmology	288
	ERIC EGGENBERGER TIM HODGE	
21.	Multiple Sclerosis	303
	THOMAS F. SCOTT DANIELLE RAY	
22.	Dementia	312
	JUDITH L. HEIDEBRINK NORMAN L. FOSTER	

23.	Brain Tumors and Other Neuro-oncological Emergencies	326
	HERBERT B. NEWTON	
24.	Neuropsychiatry	342
	CRAIG A. TAYLOR	
	BARBARA C. GOOD	
25.	Neuroanesthesiology	350
	KEVIN J. GINGRICH	
26.	Increased Intracranial Pressure	356
	AMY BLASEN	
	RON JAKUBIAK	
27.	Idiopathic Intracranial Hypertension	368
	ERIC EGGENBERGER	
	SID M. SHAH	
	MARSHA D. RAPPLEY	
28.	Normal Pressure Hydrocephalus	378
	HENRY R. LANDSGAARD	
	OLIVER W. HAYES	
29.	Sleep Disorders	381
	A. SINAN BARAN	
Part IV. Neurological Trauma and Environmental Emergencies		
30.	Traumatic Brain Injury	391
	PATRICIA LANTER	
	BRIAN ZINK	
31.	Spinal Cord Injuries	405
	CHARLES H. BILL II	
32.	Peripheral Nerve Injuries	429
	ARIO KEYARASH	
	MARK BARATZ	
33.	Neurological Complications of Environmental Emergencies	437
	DAVID ROSSI	
	DAVID OVERTON	
Part V. Pediatric Neurological Emergencies		
34.	Hydrocephalus and Shunts in Children	455
	STEPHEN GUERTIN	
35.	Pediatric Infections of the Central Nervous System	471
	JANE L. TURNER	
36.	Pediatric Cerebrovascular Disorders	478
	IMAD JARJOUR	
37.	Pediatric Seizures	492
	RAE R. HANSON	

CONTENTS

38.	Hypotonic Infant	503
	MARSHA D. RAPPLEY	
	SID M. SHAH	
	Part VI. Pregnancy-Related Neurological Emergencies	
39.	Pregnancy-Related Neurological Emergencies	515
	MARY HUGHES	
	Part VII. Neurotoxicology and Brain Resuscitation	
40.	Neurotoxicology	533
	MARY BETH MILLER	
41.	Neurotoxicology of Alcohol and Substances of Abuse	546
	SCOTT R. ZITTEL	
	MARY ELLEN WERMUTH	
	BRENT FURBEE	
	MARY BETH MILLER	
42.	Neurotoxicology of Envenomations	559
	JANET G. H. ENG	
	ROBERT J. PRODINGER	
	DAVID J. CASTLE	
43.	Brain Resuscitation	570
	W. LEE WARREN	
	JAMES E. WILBERGER, JR.	
	<i>Index</i>	577

1 The Neurological Examination

THOMAS F. SCOTT

SUMMARY The neurological history and examination provide physicians with information to localize lesions of the nervous system. Neuroanatomical localization allows formulation of a focused differential diagnosis, diagnostic plan, and treatment plan. Although aspects of the neurological examination differ based on the clinical situation, the standard elements of the neurological examination remain the same. A review of the neurological examination as it applies to emergency department evaluation is provided.

Introduction

An adequate neurological examination can differ significantly among various clinical situations. An emergency physician caring for a patient with a gastrointestinal problem may limit the neurological examination to observance of speech and motor movements during the interview and the general medical examination, and document an abbreviated neurological examination. A patient who has symptoms that suggest a neurological or a musculoskeletal problem requires a detailed neurological examination. The purpose of this chapter is to review the elements of a focused neurological examination for use in a busy emergency department.

The neurological examination is primarily a bedside tool that allows clinicians to localize lesions in the nervous system. Typically, a combination of findings on the examination allows this localization. If an examination suggests multiple lesions in the nervous system, the implications of each lesion are considered individually and in combinations. Evidence of *systemic disease* (involving more than one organ system) is considered in the interpretation of the neurological findings. Thus the neurolog-

ical examination is incorporated in the context of the patient's overall health history and general physical examination.^{1,2}

A standard neurological examination begins with a brief assessment of the patient's mental status, followed by testing of cranial nerve function, motor function, deep tendon reflexes, sensory modalities, and pathological reflexes (i.e., snout, grasp, and Babinski reflex) generally presented in that order in documentation.³ Time constraints in the emergency department frequently prevent a comprehensive neurological examination. However, an abbreviated and focused neurological examination is adequate for most patients presenting with a focal problem such as a typical migrainous headache, unilateral limb pain, or back pain. A focused neurological examination is directed at a specific clinical condition and includes site-specific components of neurological evaluation. Patients who are being admitted to the hospital for nonneurological conditions require a documented "abbreviated" evaluation that includes a brief mental status examination, testing of cranial nerves II to VII, motor function, and sensory function.

The goal of an emergency department neurological examination is the ability to answer confidently

the following questions: Is there a neurological condition? Where is (are) the lesion(s) located? What are the possible causes? Can the patient be discharged safely from the emergency department or is hospitalization required?

Emergency Department Evaluation

Neurological History

A detailed neurological history allows the emergency physician to focus on important components of the neurological examination, thus saving time and resources. A specific and detailed history enhances the likelihood of making a definite diagnosis in the emergency department. About 75% of neurological diagnoses are made while obtaining the patient’s history. One of the key elements in obtaining a neurological history in the emergency department is the *directed history*, which allows development of a relevant differential diagnosis. An account from family members and bystanders can be an important source of information in the emergency department. The history obtained from the patient can be considered a part of the mental status examination, providing information about the patient’s affect, speech, memory, logical thought, and any psychiatric symptoms. Important historical elements to elicit are the time and mode of onset, temporal relationship of symptoms, progression of symptoms, associated symptoms, and exacerbating and alleviating factors. Symptoms that indicate involvement of a particular region of the nervous system are explored. A history of a similar event in the past is of singular historical importance. A history of medication use, illicit drug use, exposure to toxins, and head trauma is important.

Neurological Examination

Mental Status Examination. The mental status examination is performed and documented as the first part of the neurological examination because its findings have bearing on the reliability of the remainder of the examination. For example, patients with an abnormal affect may be more likely to show signs of functional (somatoform) illness, and demented or encephalopathic patients may not be able to cooperate fully during the examination. Due to time constraints of the emergency department, the reporting of mental

TABLE 1.1. 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

status is often abbreviated, sometimes being reduced to a single phrase such as “patient alert and a good historian.” Such limited documentation can make changes in mental status difficult to assess during the course of a hospitalization. Ideally, the record adequately reflects the patient’s baseline mental functioning prior to hospitalization.

Level of alertness is noted first, as either alert, confused, drowsy, stuporous (tending to drift into sleep during the examination, or arousable for only brief periods), or comatose (with or without spontaneous or purposeful movements). An assigned number on a coma scale is no substitute for a precise description of mental status. However, the Glasgow Coma Scale is often used as a method of briefly quantitating neurological dysfunction (see Table 1.1).⁴ Coma with certain combinations of ocular findings and breathing patterns can indicate specific neuroanatomical substrates for the coma.⁵ Bilateral pinpoint pupils in a comatose patient with apneustic or agonal respirations implies a pontine lesion with high morbidity and mortality, but these findings can also occur as a result of 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.

In alert patients, a comment regarding affect is made when noting the patient's behavior and insight during the interview and examination. Orientation is checked in four "spheres": person (self and others), place, time, and purpose. The remainder of the mental status examination varies among clinicians, but several of the following tests are performed at the bedside routinely: naming presidents, "serial sevens," registration of three objects and recalling them at five minutes, repeating digit span forward and backward, interpreting proverbs and similarities, complex figure drawing, spelling "world" forward and backward, and naming five cities. A standardized mini mental status examination such as the Folstein MMSE is used occasionally. Determination of probable mild to severe dementia is often made with only a brief neurological examination using these tests.

More specific bedside 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. Bedside testing also includes object naming, awareness of right/left, and testing for visual and sensory neglect (especially important in parietal and thalamic lesions).

Cranial Nerve Examination. An abnormality on cranial nerve examination may relate to lesion(s) in the cortex, deep gray matter (for example, thalamus), brainstem (including nuclei), or along the course of a cranial nerve through soft and bony tissues. Certain patterns of cranial nerve dysfunction allow localization of lesions to these areas, and many such patterns are considered to be "classic" findings related to specific neuroanatomical substrates. It is often necessary to combine a cranial

nerve finding with other neurological deficits in order to localize a lesion precisely (see Fig. 1.1).⁶

- *Cranial Nerve I.* Although the first cranial nerve is often omitted as part of the routine examination, a deficit of smell is often an important clue to a diagnosis. Lesions of the olfactory groove (typically, a meningioma) can have associated psychiatric symptoms related to frontal lobe injury, and loss of sense of smell due to compression of the olfactory nerves. Loss of sense of smell is also common after head trauma and is due to shearing of the branches of the olfactory nerves as they pass through the cribriform plate. Coffee grounds are often used to test sense of smell. Noxious smells such as isopropyl alcohol should be avoided because they stimulate cranial nerve V.
- *Cranial Nerve II.* Visual disturbances are listed as part of the cranial nerve examination regardless of the location of the lesion. A visual field disturbance related to hemispheric injury (e.g., a homonymous hemianopsia) is noted. Lesions limited to the optic nerve produce monocular visual disturbance. Typically, visual acuity is tested when the patient's complaints are primarily ocular. In patients with near blindness, the distance at which the patient can count fingers is sometimes noted. A funduscopic examination is done routinely and reported as negative when the retina, retinal vessels, and optic discs are free of lesions. Papilledema (swelling of the optic disc) is a classic finding of increased intracranial pressure due to tumor, hydrocephalus, or other causes. Visual field neglect, frequently associated with contralateral parietal lesions, is usually noted as part of the mental status examination or under the topic of "higher cortical functions." When intact, several optic nerve functions are commonly summarized by the abbreviation PERRLA (pupils equal, round, and reactive to light and accommodation). The swinging flashlight test 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.

Findings of the neurological examination that are listed under cranial nerve II include the following: a Hollenhorst plaque, a bright-appearing

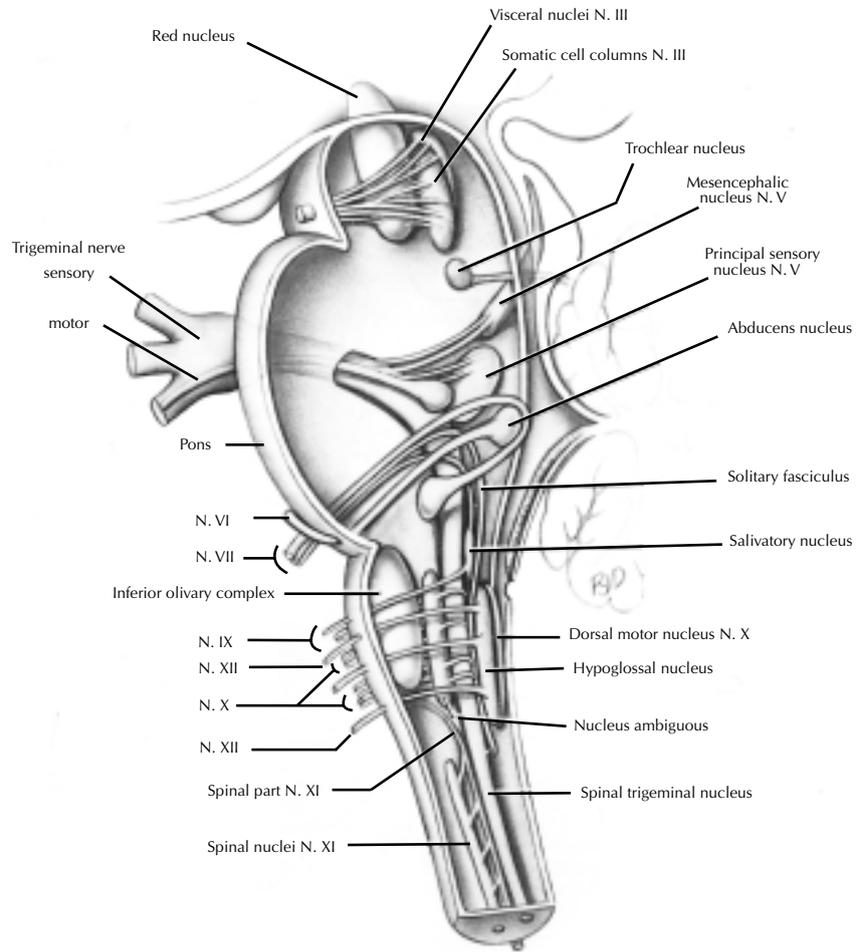


FIGURE 1.1. Sagittal view of the brainstem showing selected anatomical features to demonstrate cranial nerve nuclei, cranial nerves, and their anatomical relationships. An abnormality of cranial nerve function may localize a lesion to the brainstem when associated with other specific neurological deficits.

cholesterol or atheromatous embolus visualized by funduscopic examination of the retinal vessels, implying an embolic process. Visual field defects include: homonymous hemianopsia, a large hemispheric lesion or lesion of the lateral geniculate ganglion; bitemporal hemianopsia, a lesion of the pituitary area compressing the optic chiasm; central scotoma, a lesion of the optic nerve that typically occurs with optic neuritis, superior quadrantanopsia, a contralateral temporal lobe lesion.

- *Cranial Nerves III, IV, VI.* Cranial nerves that control eye movements are frequently described by the abbreviation EOMI (extraocular muscles intact). 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

(while the patient tracks a hand-held object or finger) suggests an ipsilateral palsy of cranial nerve VI or lateral rectus weakness. 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). 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.

- *Cranial Nerve V.* Facial sensation is tested with light touch, pinprick, and temperature. A cooled tuning fork or ice bag can be used to test tempera-

ture. If an abnormality is found in only one or two divisions of V_1 – V_3 , the findings imply a lesion distal to the gasserian ganglion. Distinct splitting of sensory function at the midline face is unusual and 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 patient's clinical presentation.

- *Cranial Nerve VII.* Seventh cranial nerve lesions are referred to as 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.
- *Cranial Nerve VIII.* 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 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.
- *Cranial Nerves IX and X.* The examiner records symmetry of palatal elevation and the gag reflex, functions subserved by these cranial nerves. Hoarseness and dysphagia can be seen with unilateral or bilateral injury to cranial nerve X (vagus); however, lesions of cranial nerve IX may be undetectable clinically.
- *Cranial Nerve XI.* Strength of the sternocleidomastoid and trapezius muscles is tested with re-

sistance to head turning and shoulder shrug, respectively. The loss of strength is often greater with nuclear or peripheral lesions as opposed to a supranuclear injury.

- *Cranial Nerve XII.* 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.

The Motor System. Tone and Power. Normal muscle tone refers to the slight tension present in muscles at rest. Muscle tone can be increased in both pyramidal and extrapyramidal disturbances. Muscle tone is evaluated at bedside by passive movements of joints through a range of motion at varying velocities. In slowly evolving extrapyramidal disorders such as Parkinson's disease, rigidity occurs.

Tremor plus rigidity yields "cogwheel" rigidity. 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. Spasticity is a velocity-dependent increase in tone, waxing and waning through the range of motion. Hypertonicity can occur acutely in brainstem lesions (decorticate or decerebrate posturing). Hypotonicity may be present chronically in neuromuscular disease.

After resistance to passive manipulation is tested manually, individual muscle group power is graded on a scale of 0–5 as follows: 0 equals no muscle contraction, 1 equals muscle contraction without joint movement, 2 equals partial movement with gravity eliminated, 3 equals movement against gravity, 4 equals resistance given, 5 equals normal strength. For detection of mild weakness, the examiner assesses for pronator drift (the patient with arms extended and supinated tends to pronate and lower the whole arm with flexion at the elbow) and observes the gait (including heel and toe walking).

The neurological history and examination usually can localize weakness to upper motor neuron dysfunction, lower motor neuron dysfunction, or the neuromuscular junction or muscle (see Table 1.2).⁷ Predominant proximal muscle weakness or atrophy, when symmetrical, usually suggests a myopathic

TABLE 1.2. Changes in Motor Function

	Loss of Power	Tone	Atrophy	Fasciculations	Ataxia
Spinomuscular lesion					
a. Anterior horn cell	Focal	Flaccid	Present	Present	Absent
b. Nerve root, plexus, peripheral nerve	Focal or segmental	Flaccid	Present	Occasionally present	Absent
c. Neuromuscular junction	Diffuse	Usually normal	Usually normal	Absent	Absent
d. Muscle	Diffuse	Flaccid	Present but later than a. and b.	Absent	Absent
Extrapyramidal lesion	None or mild	Rigid	Absent	Absent	Absent
Corticospinal tract lesion	Generalized Incomplete	Spastic	Absent	Absent	Absent
Cerebellar lesion	None; ataxia may simulate loss of power	Hypotonic (ataxia)	Absent	Absent	Absent
Psychogenic disorder	Bizarre No true loss of power May simulate any type	Normal or variable Often	Absent	Absent	Absent (may simulate ataxia)

Source: From Haerer AF. 5th ed. *DeJong's The Neurologic Examination*. Philadelphia, Pa: JB Lippincott Co; 1992. Courtesy of and with permission from publisher, Lippincott-Raven.

condition. Flabby or flaccid weak muscles, often atrophic, are seen in lower motor neuron disorders and are associated with a decrease in deep tendon reflexes (e.g., peripheral neuropathy, spinal muscle atrophy). Upper motor neuron disorders are distinguished by spasticity, increased tone, and increased deep tendon reflexes. In some neurological disorders (e.g., amyotrophic lateral sclerosis, vitamin B₁₂ deficiency) a mixture of upper motor neuron and lower motor neuron dysfunction evolves over time.

Coordination. Equilibrium refers to the coordination and balance of the whole body; when equilibrium is impaired, it is referred to as truncal ataxia. This is tested at bedside by observing sitting, balance

when standing, and gait (classically “wide-based” in cases of mild to moderate ataxia). The examination is refined with testing of tandem gait (placing one heel directly in front of the opposite toes), and by testing for the Romberg sign (swaying and truncal movements) when the eyes are closed after the feet are placed together with the arms at the sides.

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. When limb ataxia is combined with weakness, the term *ataxic hemiparesis* applies (classic for an internal capsule or pontine lacunar stroke when present-

TABLE 1.2. Changes in Motor Function (cont.)

Reflexes	Abnormal Movements	Pathologic Associated Movements
Decreased or absent	None except for fasciculations	Absent
Decreased or absent	None except for rare fasciculations	Absent
Usually normal	None	Absent
Decreased	None	Absent
Muscle stretch reflexes normal or variable	Present	Absent
Superficial reflexes normal or slightly increased		
No corticospinal tract responses		
Muscle stretch reflexes hyperactive	None	Absent
Superficial reflexes diminished to absent		
Corticospinal tract responses		
Muscle stretch reflexes diminished or pendular	May be present (intention tremor and ataxia)	Absent
Superficial reflexes normal		
No corticospinal tract responses		
Muscle stretch reflexes normal or increased (range)	May be present	Absent
Superficial reflexes normal or increased		
No corticospinal tract responses		

ing as a pure motor stroke syndrome). Limb ataxia is demonstrated by testing finger-to-nose and heel-to-shin movements. Although limb ataxia can be seen in sensory disorders (pseudoathetosis), it is a classic indicator of lesions of the cerebellar system, producing intention tremor (see below) and dysdiadochokinesia (impairment of rapid alternating movements). 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. Essential or physiological tremor (commonly 8–11 Hz) can be a normal

finding. Essential tremor is assessed by having the patient forcibly extend the arms and digits; tremor is demonstrated distally. The term *benign essential tremor* denotes a pathological idiopathic high-frequency tremor that is often familial. Essential tremor can be accentuated or attenuated by drugs and disease states. Tremulousness generally refers to transient high-frequency tremor associated with acute illness or anxiety. In severe metabolic disturbances, tremor can coexist with other abnormal movements such as myoclonus (rapid and tense contractions of large muscle groups) and asterixis (intermittent lapses of tone-interrupting voluntary movements).

Intention tremor refers to to-and-fro motions that increase in amplitude as the patient's finger approaches a target. This tremor is usually demonstrated on finger-to-nose testing in patients with lesions of cerebellar hemispheres and their projections into the brainstem and ventral posterolateral thalami.

Parkinsonian tremor is typically at a lower frequency than essential tremor and is often described as a "pill-rolling" tremor. Unlike the "action" tremors described above, tremor in Parkinson's disease is present at rest (at least intermittently), is variably affected by changes in position, and is associated with stiffness (hence cogwheel rigidity) and bradykinesia.

Bradykinesia, Akinesia, and Dyskinesia:

Bradykinesia is a reduction of normal spontaneous or unconscious semipurposive movements such as blinking, shifting movements, and facial expressions. A classic symptom of Parkinson's disease, bradykinesia also occurs in other neurodegenerative syndromes, multi-infarct state, and depression.

Akinetic mutism is a condition of extreme lack of movement and interaction, verbal and nonverbal, that occurs in patients with brainstem lesions or bilateral hemispheric or deep gray matter lesions. It also occurs in patients with end-stage neurodegenerative disorders. Catatonic mutism refers to a similar condition – regarded as a manifestation of severe psychiatric disturbance, not structural lesions – and generally is associated with waxy flexibility (maintaining an unusual posture positioned passively) or rigidity.

Dyskinesia is a nonspecific term for complex irregular involuntary movements involving multiple muscle groups. Most patients with dyskinesia have medication-induced side effects of neuroleptics and antiparkinsonian drugs containing L-dopa. Tardive dyskinesia refers to the late-developing, neuroleptic-induced choreoathetoid movements primarily of the face, head, shoulders, and upper trunk.

Rapid dyskinetic movements are often referred to as *choreiform*. This term accurately describes many patients with tardive dyskinesia, Huntington's disease, and Sydenham chorea. Dystonic movements are slower and associated with increased tone or rigidity (for example, dystonia musculorum deformans) and can occur in patients with Parkinson's disease and as acute reactions to neuroleptics. Athetoid movements are intermittent in speed be-

tween chorea and dystonia, and have a writhing or more rhythmic quality.

Other miscellaneous abnormal movements include ballismus, tics, and akathisia:

- Ballismus refers to an abrupt flailing movement of an extremity that can occur following stroke involving the subthalamic nucleus.
- Tics refer to rapid movements that are stereotyped and repetitive. Tics are classic in Tourette's syndrome but can occur with mental retardation.
- Akathisia can be thought of as the opposite of bradykinesia. Normal spontaneous movements increase in the waking state. Patients with akathisia may be "fidgety" and often pace.

Deep Tendon Reflexes, Cutaneous Reflexes, and

Miscellaneous Signs. Deep tendon reflexes are elicited by percussion over tendon insertions that produces a rapid muscle stretch. These reflexes are mediated by reflex arcs originating in intramuscular organs that are sensitive to stretching; they transmit impulses to alpha motor neurons within the spinal cord, which produces a contraction of the percussed muscle. When deep tendon reflexes are increased or hyperactive, reflex "spread" occurs in other local muscles, resulting in an increased intensity of muscle contraction. Deep tendon reflexes are generally graded on a 0–4 basis: 0 indicates that a reflex is not elicited; 1 indicates a hypoactive reflex or one that is present only with reinforcing maneuvers; 2 indicates a normal reflex; 3 indicates reflexes that appear to be hyperactive but may not necessarily be pathological; 4 indicates clonic reflexes that may or may not be pathological.

Cutaneous reflexes consist of the abdominal reflex (abdominal wall muscle contraction), elicited by stimulation of the skin over the four quadrants of the abdomen, the cremasteric reflex (testicular elevation), elicited by stimulation of the skin over the scrotal area, and the anal wink reflex (anal contraction with stimulation).

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).

Psychogenic disorders can be associated with the following findings:

- Splitting the tuning fork test is administered to patients with psychogenic sensory disturbance. These patients typically have a sharply demarcated loss of sensation to the midface and may complain of a lack of vibratory sensation when tested on the affected side of the forehead, with intact vibratory sensation on the unaffected side.
- The hand-face drop test is used in patients with psychogenic coma who appear flaccid. When the patient's hand is held over the face and dropped, the patient in psychogenic coma typically avoids letting the hand hit the face with subtle movements to the side.
- Hoover's sign (of psychogenic neurological dysfunction): The examiner places one hand under each heel with the patient in the supine position. The patient is asked to raise one leg, and the examiner feels downward pressure of the opposite leg when voluntary effort is intact. Absence of pressure suggests lack of effort and possible contralateral functional weakness.
- Astasia – abasia is a lurching, unusual gait symptomatic of psychogenic ataxia.

Sensory Examination. Similar to the rest of the neurological examination, the sensory examination is an organized assessment of neuroanatomical structures and systems. It is usually performed last because findings related to cognition and higher cortical functions have bearing on its interpretation. Testing of light touch and pinprick sensation assesses the integrity of the peripheral nervous system and spinal cord sensory tracts, and 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).

The sensory examination is routinely documented as a response to five modalities: pinprick, light touch, vibration, position, and temperature. The tools used for these tests include a safety pin or other sharp object, a cotton swab, a 128-Hz tuning fork, an ice bag or metal object such as a tuning fork placed over a cooling vent or in ice water, and calipers to test two-point discrimination. 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 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, hemicord pattern, transverse cord pattern, or crossed brainstem pattern (see below), or somatotopically, corresponding to lesions above the brainstem (for example, 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 in patients 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.

Sample Examinations

The following two examinations are examples of neurological examinations as they might appear in a hospital chart in typed, dictated, or handwritten form. Handwritten examinations tend to include several commonly used abbreviations.

EXAMINATION:

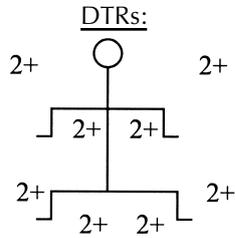
Mental status: 0 × 4, alert, appropriate, digits 6# ⇔ presidents✓, 3/3 5 min. memory, proverbs✓, serial 7s ✓

Cranial nerves: II - XII intact (PERRLA, EOMI, face-gag-palatal symmetrical, V₁-V₃ ✓) fundi ⊖

Motor: Strength 5/5, normal tone/bulk, ∅ drift, F→N✓, gait heel/toe/tandem✓, H→S

Sensory:

- Vibration✓
- Position✓
- Light touch✓
- Pinprick✓
- Temp✓



Imp: Right upper extremity paresthesias of uncertain etiology (normal exam)

FIGURE 1.2. A representative chart note of a normal neurological examination: 0 × 4 denotes orientation in four spheres, digits 6# ⇔ denotes digit span, PERRLA denotes pupils, equal, round, and reactive to light and accommodation, EOMI denotes extraocular muscles intact, V₁-V₃ denotes intact fifth nerve, ∅ drift denotes no pronator drift, F→N denotes finger-to-nose testing, H→S denotes heel-to-shin testing, stick figure denotes basic deep tendon reflexes. For example: 2+ over knee means that the knee is normal, whereas 1+ means trace reflexes and ∅ denotes loss of reflexes.

Sample Examination #1

A normal neurological examination (see Fig. 1.2):

Mental status: Patient is alert and oriented times four. Affect appropriate. Names presidents back to Carter easily, registers 3 out of 3 complex objects and recalls them at 5 minutes. Proverbs and serial 7's intact. Digit span 6 or 7 numbers forward and backward. On cranial nerve examination, fundi are benign. Pupils equal, round, and reactive to light and accommodation. Extraocular muscles intact. Face, gag, and palate elevation symmetrical. Facial sensation intact in all divisions of cranial nerve V. On motor examination, strength is 5/5 with normal tone and bulk. No pronator drift. Finger-to-nose testing and fine finger movements normal. Gait testing including heel, toe, and tandem walk intact. Heel-to-knee-to-shin intact. Deep tendon reflexes

are 2+ throughout and symmetrical with downgoing toes. No pathological reflexes. Sensory examination is intact to vibration, position, light touch, pinprick, and temperature.

Sample Examination #2

The following neurological examination could be recorded in a patient with an acute right middle cerebral artery distribution infarct and moderate idiopathic Parkinson's disease:

Mental status: Patient is slightly drowsy. Patient is oriented to place, year, season but not month, or day of the week, or time of day. Patient does not know how long he has been in the hospital or the reason for hospitalization. Patient refuses to consider the possibility that he might have left-sided weakness due to a stroke (anosognosia or denial). Patient seems to neglect the left visual field. Patient can name only one recent president. Patient knows his address and phone number but has trouble naming his four children. He is unable to register three complex objects. He is unable to perform simple calculations. On cranial nerve examination, the patient has a left central seventh nerve palsy and some mild tongue deviation on tongue protrusion. Sensory examination of V₁ through V₃ is not reliable. Patient does not cross the midline with conjugate gaze. Gag intact. Patient is noted to have a snout and glabellar tap reflex. On motor examination, the patient has increased tone and cogwheel rigidity on the right, and his left upper extremity is flaccid. The left lower extremity is remarkable for trace proximal movements to command, and proximal and distal withdrawal movements to deep pain. Bilateral Babinskis are present, and deep tendon reflexes are symmetrical and 2+. All sensory modalities are decreased on the left versus neglect on the left. Patient distinguishes different sensory modalities on the right but reliability is questionable.

PEARLS AND PITFALLS

- Examination findings for patients with neurological disease can be mistaken for hysteria.
- Abnormal findings on neurological examination can result from one or more disease processes. Findings are considered in isolation and in combination.

- An age-associated loss of upgaze and vibratory sensation is normal in elderly patients.
- A detailed neurological examination evaluating the entire neuroaxis is not practical in the emergency department setting; therefore, the examination is tailored to the patient's specific complaints to localize lesions.
- An abnormal gait can be the only sign of serious neurological disease.

REFERENCES

1. Adams RD, Victor M. 3rd ed. *Principles of Neurology*. New York, NY: McGraw-Hill Book Co.; 1985.
2. Rowland LP. 9th ed. *Merritt's Textbook of Neurology*. Baltimore, Md: Williams & Wilkins; 1995.
3. Mancall E. 2nd ed. *Alpers and Mancall's Essentials of the Neurologic Examination*. Philadelphia, Pa: FA Davis Co; 1981.
4. Henry GL. Coma and altered states of consciousness. In: Tintinalli JE, Ruiz E, Krome RL, eds. *Emergency Medicine: A Comprehensive Study Guide*. 4th ed. New York, NY: McGraw-Hill Book Co; 1996:227.
5. Plum F, Posner JB. 3rd ed. *The Diagnosis of Stupor and Coma*. Philadelphia, Pa: FA Davis Co; 1982.
6. Carpenter MB. 3rd ed. *Core Text of Neuroanatomy*. Baltimore, Md: Williams & Wilkins; 1985.
7. Haerer AF. 5th ed. *DeJong's The Neurologic Examination*. Philadelphia, Pa: JB Lippincott Co; 1992.