

Neurologic History and Examination

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Overview

The goal of the history and examination is to guide the diagnosis, workup, and treatment strategy and to provide a simple and clear record, so any subsequent changes can be easily determined.

- Avoid forcing the history and examination results into categories (e.g. cerebellar tremor) – just describe what you hear and observe precisely and quantitatively.
- Customize the history and examination for each patient.
- Do not substitute imaging studies and other tests for bedside clinical localization – for many neurologic diseases, such as headache syndromes, myasthenia, myopathies, Parkinson disease, and dystonias, imaging studies are not typically useful.
- Establish the neuroanatomic localization first, then consider the differential diagnosis.
- Try to formulate a specific hypothesis about localization and etiology as you obtain the history and then devise a strategy for critically testing this by follow-up questions and during the examination.
- Be definitive and precise about examination findings – avoid “equivocal,” “+/-,” “possible.”

Approach to history taking for the neurologic patient

- Ask open-ended questions and try to avoid listing choices.
- Beware of asking questions that merely confirm your preconceptions as patients may tend to tell you what they think you expect to hear.
- Focus on onset, recovery, timing, and pace of events.
- Characterize the nature and distribution of symptoms (e.g. is pain sharp, dull, aching, shooting, burning, tingling?), distribution, and comparators (e.g. like a toothache, hitting finger with hammer, etc.).
- Determine how witnesses describe the symptoms, especially for disorders of cognition or consciousness.
- Inquire about other medical conditions, review medical and neurologic history and systems, prescription drug use, drugs of abuse, HIV status, and family history, especially for similar conditions.

Approach to the neurologic examination

- The examination is tailored to the complaint, history, and initial findings.
- Not every bedside test is performed for each patient. *Always* tell the patient what you will be doing and what to expect throughout the examination.
- When useful, demonstrate what you want the patient to do.

Screening neurologic examination

- Mental status:
 - Assess language and cognition during the patient interview.
- Cranial nerves:
 - Confrontation visual fields. Visual acuity on Snellen card, eye movements in all six directions, funduscopy, pupillary reflex while focusing at distance, facial pinprick sensation, facial movement (close eyes tightly, smile), hearing to finger rub, whispered numbers, vocal clarity, movement of palate and uvula, shoulder shrug and neck turning, tongue protrusion.
- Motor:
 - Inspect bulk, tone, assess posture and movements.
 - Assess strength of shoulder shrug, elbow flexion and extension, wrist flexion and extension, grip, pronator drift with eyes closed, foot dorsiflexion and plantarflexion.
- Sensory:
 - Pinprick sensation in all four extremities.
 - Joint position sense, vibratory sensation in toes.
 - Detection of double simultaneous touch stimuli.
- Coordination:
 - Assess finger-nose-finger, coordination and speed of fine finger and rapid alternating movements.
 - Assess gait, tandem gait, Romberg test.
- Reflexes:
 - Biceps, triceps, brachioradialis, patellar, ankle, and plantar reflexes.

Mental status

- Carry out a formal mental status examination if there is any suggestion of abnormality during the history taking.
- Describe the deficits observed as simply as possible and give examples in your records.
- Memory and cognition cannot be fully evaluated in patients with aphasia or impaired alertness.
- See Chapter 6 for discussion of the examination of cognitive function, and Chapter 7 for other cognitive deficits and standardized screening tests – such as the mini mental status examination (MMSE) and Montreal Cognitive Assessment (MoCA).
- The Glasgow Coma Scale (see Chapter 41) is a screen developed for rapid assessment of head trauma and should not substitute for a full examination.

Principal components of the mental status examination

- Orientation and alertness:
 - Level of alertness.
 - Orientation to time, place, person.
 - Ability to spell “world” backwards.
- Memory and calculations:
 - Ability to retain three words at 5 minutes.
 - Ability to recall recent verifiable events or remote history.
 - Subtract 7 from 100 and continue subtracting.
- Language: Dysphasia or aphasia refer to acquired impairments of expressive or receptive language function. Dysphasia is impaired language; aphasia is a more complete disruption of

language production or understanding. In testing language, avoid providing non-verbal clues by pointing or facial expressions.

- Oral expression: evaluated for tempo, errors.
- Understanding spoken words: avoiding visual cues such as gestures, test spoken commands (close your eyes, lift your left hand).
- Written expression: open-ended and to dictation.
- Reading: commands such as open your mouth, point to your right ear, and read aloud.
- Naming objects: fingers, coins.
- Repeating phrases.
- Apraxia: This is an acquired inability to perform a task despite having the motor ability and comprehension needed.
 - Ask the patient to draw a clock with the current time (constructional apraxia).
 - Ask the patient to show how they would use a comb, scissors, or toothbrush.
- Agnosia: This is an impairment of perception of sensory stimuli in the presence of intact primary sensory or visual modalities.
 - Is the patient unable to recognize faces of famous people (prosopagnosia)?
 - Does the patient not recognize their body parts, especially on the left (asomatognosia)?
 - Is the patient unaware of limitations such as paresis caused by their illness (anosognosia)?
 - When testing visual fields, see if finger movement is detected in each quadrant when presented simultaneously bilaterally. Test light touch similarly on the backs of both hands. Does the patient detect visual or sensory stimuli on both sides, but neglects one if they are presented simultaneously (visual or sensory hemi-neglect)?
 - Visual neglect can also be detected by asking a patient to draw a line across the middle of a horizontal line or by placing short lines at different angles all across a page and asking the patient to cross each line.
 - Does the patient have difficulty recognizing coins or a safety pin (astereognosis) or numbers traced on the palm (agraphesthesia)?

Elements of examination of the comatose patient (see also Chapter 4)

- Coma scale rating is not a substitute for performing and recording a careful examination.
- Begin with airway, breathing pattern, circulation, and vital sign assessment.
- Examine for bruises and lacerations, jaundice, cyanosis, needle marks.
- Assess consciousness:
 - Does the patient open his eyes to voice and look at examiner?
 - If unarousable to gentle stimuli, what response is seen to painful stimuli? Be humane and avoid bruising and skin damage. Try repeated pinprick before stronger stimuli such as controlled pressure using the stem of a reflex hammer on the nailbed.
- Test pupillary symmetry and reactivity, funduscopic examination, corneal reflex. After excluding neck injury, test oculoccephalic reflex (doll's eyes): rotate head to left then right and observe full eye movement to other side. Cold caloric testing is performed by ice water instillation in the ears with the neck at 30 degrees (initially 1 mL after visualizing an intact eardrum, then up to 50 mL). The expected response in coma (with preserved brainstem function) is full conjugate deviation to the side of irrigation. Test facial movement elicited by pinprick or supraorbital pressure and gag reflex.
- In patients not following commands, movements and sensory level are tested by response to painful stimuli. Are the responses purposeful (moves away from pain in non-stereotyped manner or reaches toward stimulus with other hand) or stereotyped (triple flexion, decorticate, decerebrate responses)?
- Evaluate reflexes and extensor-plantar responses.

Cranial nerves

- I. Olfactory
 - Test each nostril with a fruit slice or cup of juice at bedside, or better with identical vials of peppermint or orange extract.
 - Ammonia detection, a noxious stimulus, does not discern deficits of olfaction.
- II. Optic
 - Test pupillary light responses in reduced lighting. Pupils constrict bilaterally with unilateral stimulus. Paradoxical enlargement of the pupil when moving the light from one pupil to the other indicates a relative afferent defect (swinging flashlight test).
 - Test pupillary accommodation to fixation on a finger moving slowly towards the bridge of the nose.
 - Sitting opposite the patient, test visual fields using fingers or, more accurately, using red and white hatpins (obtainable at a craft store). Unilateral reduction of red intensity (red desaturation) is sensitive for detecting optic nerve dysfunction such as optic neuritis.
 - Test acuity in each eye with correction using Snellen card. Refractive errors can be partly compensated using a pinhole for testing.
 - Examine optic disk, vessels, and eyegrounds using an ophthalmoscope.
- III, IV, VI. Ocular motor (oculomotor, trochlear, abducens)
 - Have the patient follow your finger to all positions as well as look from one hand to the other held at opposite extremes. Note diplopia and inquire about double vision; note nystagmus, smoothness of pursuit. Note lid retraction or ptosis.
- V. Trigeminal
 - Corneal reflex: have the patient look up and away. Moving slowly to avoid eliciting eye closure to a visual threat, touch the cornea edge with twisted cotton wool. Both eyes fail to close with a sensory V deficit; unilateral failure to close occurs with motor (facial) weakness.
 - Compare light touch and pinprick on forehead (V1), upper cheeks (V2), and lower lip (V3).
 - Jaw jerk: when you place your index finger firmly above the chin and tap sharply downward on your finger with the reflex hammer you will feel and see the jaw close slightly.
 - Ask the patient to clasp their teeth (while applying opposing pressure outside the mouth) and to open their jaw against resistance, ask the patient to move the jaw from side to side.
- VII. Facial
 - Observe for asymmetry of palpebral fissures and nasolabial folds. Look for weakness of forehead wrinkling on upward gaze. Have patients close their eyes tightly and squeeze their lips together tightly while testing resistance to opening. Lower motor neuron facial weakness involves entire hemi-face, whereas central upper motor neuron facial weakness usually spares the forehead.
- VIII. Acoustic (vestibulocochlear)
 - Test detection of finger rub and comprehension to whispering numbers in each ear.
 - Rinne test for non-neural conduction defect: compare 516 Hz tuning fork base on mastoid process (bone conduction) to tones held outside ear (air conduction). Normal: air louder than bone. Bone conduction is increased in a conduction defect.
 - Weber test: hold fork at center top of forehead. Louder in bad ear with conduction defect, in good ear with sensorineural defect.
 - Vestibular tests: evaluate eye movements for nystagmus and gait as described below. Special tests for dizziness include the head thrust test, Fukuda stepping test and Dix–Hallpike test.
 - Head thrust test identifies unilateral hypofunction of the peripheral vestibular system. The examiner sits in front of the patient and asks them to look at the examiner's nose while the examiner abruptly rotates the head through a small arc (10–20 degrees) left and right.

Normally, the gaze is held relatively stable. If the vestibular ocular reflex is abnormal, a corrective saccade is seen on the side of reduced vestibular function back toward the examiner's nose. This corrective saccade supports peripheral vestibular hypofunction on the side toward which the head rotation occurred.

- Stepping test: the patient steps in place for 1 minute with their eyes closed. The normal response is to continue facing in the same direction. A patient with an acute vestibular deficit slowly rotates toward the side of the deficit.
- Dix–Hallpike test: reposition the patient from a sitting position to reclining with their head hanging and chin 45° to the left; get them to hold the position for at least a minute while inquiring about symptoms and observing for nystagmus. Repeat with chin to the right. In benign paroxysmal positional vertigo, vertigo and rotatory nystagmus begin after a latency of about 5 to 20 seconds, usually improving within 1 minute and decreasing with repeat of the process.
- IX, X: Glossopharyngeal and vagus
 - Evaluate symmetry of palate elevation while the patient says "ah" in a deep voice and while eliciting the gag reflex with stimulation on each side.
- XI: Spinal accessory
 - Get the patient to shrug their shoulder against resistance – the contracting trapezius can be seen and palpated. Rotate the patient's head to each side against resistance – the contracting sternocleidomastoid muscle can be seen and palpated.
- XII: Hypoglossal
 - Evaluate the symmetry of tongue protrusion by having the patient push their tongue against the inside of their cheek on each side against the examiner's hand held outside the cheek.

Motor examination

Overview

- Note tremor, abnormalities of posture, wasting, fasciculations, and tone (see below).
- A comprehensive motor examination is indicated for symptoms of weakness.
- Have the patient maintain arms outstretched in front with palms up and evaluate for drift or rotation suggestive of mild corticospinal deficit (pronator drift).
- Evaluate fine finger movements: demonstrate and ask the patient to play the piano in midair or to drum on a table rapidly using individual fingers. Rapid foot taps can also be evaluated.
- Firmly support the limb proximal to each joint to be tested.
- Evaluate pattern of weakness: hemiparesis, paraparesis, distal, proximal.
- Determine consistency of weakness and fatigability.
- Quantify strength of individual muscles according to the MRC scale (Box 1.1).

BOX 1.1 MRC SCALE FOR MUSCLE STRENGTH

- 0 No contraction
- 1 Trace contraction
- 2 Active movement with gravity
- 3 Active movement against gravity
- 4– Active movement against slight resistance
- 4 Active movement against moderate resistance
- 4+ Active movement against strong resistance
- 5 Full strength

8 Part 1: Introduction

- Tests for individual muscles are described in *Aids to the Examination of the Peripheral Nervous System* (see Reading list).

Abnormalities of tone

- Hypotonia: usually associated with muscle weakness and has diverse causes.
- Cogwheel rigidity: cogwheel-like catching with slow pronation-supination movements of forearm or extension and flexion of elbow by examiner. Characteristic of Parkinsonism.
- Paratonia (gegenhalten): irregular “gumby-like” resistance to limb movement. Varies with the resistance or effort put forth by the examiner. Associated with diffuse cortical disease.
- Spasticity: increased tone with sudden passive flexion of limb, such as extending the elbow or lifting the knee joint off the examining table. Stiffness depends on speed of passive movement. Associated with upper motor neuron deficits and clasp knife phenomenon, in which resistance suddenly decreases when the joint is passively moved.
- Myotonia: slow relaxation of muscle contraction. Percussion myotonia is elicited by tapping on the muscle.

CLINICAL PEARLS

- Cerebral upper motor neuron weakness preferentially affects the upper extremity: shoulder abduction > elbow extension = wrist and finger extension; lower extremity: hip flexion, knee flexion, and ankle dorsiflexion > extensors.
- In fine motor movements, corticospinal deficit shows slowing and reduced excursions. Cerebellar deficit shows variable amplitude and speed.
- Radial nerve palsy and a hand area stroke can both cause extensor weakness in the arm. The former can be distinguished by involvement of the brachioradialis, which can be felt to contract during elbow flexion against resistance with the thumb towards the ceiling.
- Non-organic motor weakness: variable; apparent strength on moving greater than when testing; normal tone and reflexes. Resistance tends to vary with the force used to test. Can be overcome with weak force, but shows more strength that is similarly overcome when testing with more force. Hoover sign may be present: place hand under opposite heel while the reclining patient lifts leg against resistance. A physiologic response is when the opposite leg pushes downwards when one leg lifted.

Sensory examination

Overview

- The most difficult part of the neurologic examination is to assess accurately and reproducibly due to physiologic differences in sensation, and individual patients tend either to exaggerate physiologically normal perceptual variation or underreport sensory deficits.
- Start from area of deficit, if present, and delineate transition to normal sensation.
- It is neither practical nor necessary to test the entire skin for every sensory modality.
- For a routine examination, test the face for pinprick and touch, and four extremities for pinprick, light touch, and joint position sense.
- Compare proximal to distal and right to left.
- Light touch: use a cotton wisp and avoid skin hairs.
- Pain sensation: test with new safety pin (disposed after use) using slow, light touches.
- If abnormal pinprick sensation is detected, test temperature sensation in that area. Temperature can be screened by comparing sides of a cool tuning fork warmed with your hand on one side, or with tubes filled with hot and cold water.

- Joint position sense: grasp sides of distal joint with one hand and sides of distal phalanx with the other. Move gently up and down, first assuring that there is no resistance to movement by the patient. Ask about the change in position in an unpredictable pattern – e.g. up, up, down, up, down. Determine size of movement reliably sensed. If absent, proceed to more proximal joint.
- Vibration sense: test over bony prominences with large 128 Hz tuning fork.
- Two-point discrimination can be quantified as the minimum distance the backs of two cotton swabs can be perceived.
- For findings with common cervical, lumbar, and sacral root syndromes, see Chapter 17.

CLINICAL PEARLS

- Sensory deficits are very suggestible. Confirm reliability by returning to area of deficit to retest.
- Test sacral sensation if urinary, bowel symptoms, bilateral leg weakness, or sensory loss to evaluate possible conus medullaris or cauda equina lesion.
- Non-organic sensory loss fails to follow anatomic distribution. Non-anatomic decreased facial sensation may stop at hairline and angle of jaw or on the trunk may proceed exactly to the midline. For hemisensory deficit try the crossed hand test: have the patient interweave their fingers with arms hyperpronated and rotate and fold in the arms so that pinkies of the clasped hands are held against the chest. With random testing of sensation of different fingers, a patient with non-organic hemisensory decrease will tend to confuse the fingers involved.
- Positive "functional" signs do not show that the patient does not have disease of the nervous system. Many patients with serious disease embellish their deficits or provide unreliable responses to examination.

Reflexes

Overview

- Tendon reflexes are a crucial and objective component of the examination.
- It is important to explain what you will do to get the patient to relax before hitting them with a reflex hammer. Gently move the joint to be tested to ascertain that the patient is relaxed.
- The tendon should be struck once in the correct spot with a short, free movement of the hammer.
- If the patient is tense, distract by asking to count backwards from 100.
- If reflexes are absent, try reinforcement: the patient links both hands with the fingers flexed and curved and just before the tendon is struck pulls the hands strongly in opposite directions. Clenching the opposite fist can be used to reinforce upper extremity reflexes. The timing of reinforcement is crucial as the effect is very brief.
- The standard reflexes are listed in Box 1.2.
- Grade reflexes from 0 to 4+, with 0 absent, 1+ trace, 2+ average, 3+ increased, and 4+ abnormally increased.
- Note the presence of clonus.
- Test for the presence or absence of the extensor-plantar reflex (Babinski sign) by slowly and firmly scraping the lateral edge of the sole with a tongue depressor or similar object. In a positive response the large toe moves upward.
- Other reflexes are listed in Table 1.1.

CLINICAL PEARLS

- Avoid confusing a pathologic withdrawal reflex (triple flexion) in a patient with upper motor paralysis of the lower extremities with voluntary withdrawal. The reflex can be identified by its stereotyped form and usually confirmed by stimulating with taps of a safety pin on the dorsum of the foot or top of the large toe. Unlike voluntary movement, which moves away from the painful stimulus, the flexion reflex will move the foot toward the pin stimulating the top of the foot.
- Symmetrically hyperactive or absent reflexes can be normal physiologic variants.

BOX 1.2 STANDARD DEEP TENDON REFLEXES

Biceps:	C5,6	Musculocutaneous nerve
Triceps:	C7,8	Radial nerve
Brachioradialis:	C5,6	Radial nerve
Patellar	L3,4	Femoral nerve
Achilles	S1	Sciatic nerve

Table 1.1 Reflex responses.

Name	Response	Significance
Babinski sign	Stimulate lateral sole → large toe moves upward	Corticospinal (upper motor neuron) dysfunction
Triple flexion	Stimulate lateral sole or foot → flexion of hip, knee, dorsiflexion of foot	Corticospinal (upper motor neuron) dysfunction
Hoffmann reflex	Flick distal phalanx of middle finger → flexion of thumb and fingers	Suggests hyperactive reflexes, but may be normal
Glabella reflex	Repeated tapping with finger on forehead over eyes → normal: eyes blink a few times then stop; abnormal: eyes continue to blink	Frontal release sign suggesting diffuse bilateral frontal lobe or cortical dysfunction as in dementia; also seen in Parkinsonism
Grasp reflex	Stroking the palm → involuntary grasp	Frontal release sign, similar to above
Palmomental reflex	Stroking the palm → wrinkling of chin mentalis muscle	Frontal release sign, similar to above
Decerebrate response	In coma, sternal pressure → rigid extension of neck and all four extremities	Severe dysfunction of brainstem superior colliculi and vestibular nuclei
Decorticate response	In coma, sternal pressure → flexion of arms and extension of legs	Brainstem dysfunction at a level higher than that causing decerebrate response involving both hemispheres, thalamus, or internal capsule

Gait and coordination (Box 1.3)**Overview**

- Examine posture (station) and walking. Pay attention to the width of the base and the symmetry of movements. Test heel-toe walking (tandem walk).

BOX 1.3 TYPES OF ABNORMAL GAIT

Hemiparetic: leg circumducts, decreased arm swing

Spastic: scissoring, stiff-legged

Ataxic: wide-based, staggering gait

Parkinsonian: slow, stooped posture, small steps

Magnetic: small steps, may appear as if the feet are glued to the ground

- Romberg sign: have the patient stand steadily feet together with their eyes open, then test if balance is maintained with the eyes closed.
- Finger-to-nose and finger-nose-finger tests: with their hands outstretched, have the patient touch their nose with each index finger, then move it back and forth from the examiner's finger to the patient's nose. Perform this with the patient's eyes open and closed. Evaluate for accuracy, smoothness, and tremor.
- Past pointing test: get the patient to extend their arm with their index finger touching the examiner's finger. The patient then raises their arm over their head with their eyes closed and brings the arm back to touch the examiner's finger.
- Heel-shin test of coordination: have the patient slowly rub the heel of one leg from the ankle to the knee of their other leg on the shin.
- Rapid alternating movements: this involves alternate tapping of the palm and back of hand on a flat surface. Examine and listen for speed and regularity.
- Note involuntary movements at rest and with movement (see further on).

Involuntary movements

- Tremor: constant, steady oscillation
 - Parkinsonian tremor: pill rolling, most prominent at rest, decreases with purposeful movement.
 - Essential tremor: head and voice often involved. Worsens with precise movement. Decreases at rest.
- Chorea: sudden, rapid, purposeless movements. Causes include Huntington disease, Sydenham chorea (post-rheumatic fever), polycythemia vera.
- Athetosis: slow writhing movements of arms and legs. Causes include cerebral palsy, Wilson disease, neurodegeneration with brain iron accumulation (NBIA), ataxia telangiectasia.
- Dystonia: sustained involuntary muscle contractions causing unnatural postures. Includes writer's cramp, blepharospasm, and generalized dystonia. Many genetic forms have been identified.
- Ballismus: wild, uncontrolled flinging movements with any attempt at movement. Caused by damage in the vicinity of the subthalamic red nucleus.
- Myoclonus: sudden, brief shock-like jerks of a group of muscles.

CLINICAL PEARLS

- Decrease in arm swing on one side is a sensitive sign for hemiparesis.
- Slow or magnetic gait associated with urinary incontinence may be normal pressure hydrocephalus.
- Parkinsonism may show retropulsion – difficulty in regaining center of gravity when gently pulled backwards while standing.

Reading list

Key reading sources for this chapter can be found online at www.mountsinaiaexpertguides.com

**Additional material for this chapter can be found online at:
www.mountsinaiaexpertguides.com**

This includes a reading list.

