Starting in medical school neurologic examination has remained intimidating for many physicians. The examination has been perfected over many decades thanks to our French and German founding fathers. It has led to eponyms (e.g. Babinski sign, Hoffmann reflex) different techniques of detecting subtle signs of weakness (e.g. hand rolling, foot tapping) and even a wide collection of reflex hammers that can be proudly displayed in the office. How is one to summarize a skill or tool that can be directly used in daily practice? As with many medical specialties, the history is dependent on specific knowledge of neurologic disorders. And it has become a cliché to think that neurologic examination is dull and time consuming involving meticulous assessment of reflex asymmetries, sensory deficits and mental function. Some may argue whether a MRI would not suffice. Diagnostic tests have grown tremendously over the last decade and may have left the impression that neurologists, despite their acumen, may be inferior to them. Nothing seems further from the truth.

The level of this chapter will be for the medical student but also recently qualified physician in their first years of specialty training. Using the basic tenets of a comprehensive history, examination of the major components of brain, spinal cord, and neuromuscular unit function, a tentative diagnosis should be possible in approximately half of the cases. The principle of neurologic examination is localization of the lesion followed by a differential diagnosis of the cause of the lesion. This is a sacrosanct principle and cannot be ignored despite rapid development in technology. This chapter, however, has to ignore failure of some patients to describe a particular symptom, information forthcoming after repeated history and recognition of malingering. This chapter should have a practical value for the vast majority of physicians seeing patients in the clinic.

The History

- General Issues

Sometimes one is not off to a good start. Patients may present their medical history illustrating a plethora of physician encounters, interpretation of diagnostic tests in layman terms, and multiple diagnoses often contradicting each other. As expected, many of the requests for a neurologic examination pertains to the infinite symptoms of dizziness, weakness, fatigue, facial numbness, confusion, and longstanding headache. The physician task is using a series of leading questions to obtain the overriding presenting symptom, onset and time, and its progression. Neurologic diseases may have different evolution over time. The onset can be acute which may be defined in seconds to rapidly progressing in hours, fluctuating typically over weeks, a relenting rapidly progressing course and a fluctuating, remitting and relapsing course such that is seen in multiple sclerosis and myasthenia gravis. In any presenting symptom, associated symptoms are of the utmost importance and many of them may involve systemic features such as fever, nausea and vomiting, or weight loss. The neurologic history remains time consuming but with quick
pointers, one should be able to categorize a reliable account of the presentation. Family history is important in many neurologic disorders. A long list of genetic disorders has been described with a predominant neurologic presentation but many more of the common neurologic disorders run in families. These include hereditary peripheral neuropathy, multiple sclerosis, but also epilepsy, migraine, and cerebrovascular disease particularly intracranial aneurysms. The technique of history taking should remain respectful but may include prodding questions, constant verification of the answers, repetitive summaries to the patient and verification with family members.

Some leading questions are

1. Where in the body did it start?
2. When was the last time you were without any symptoms?
3. Why did it occur and are triggers known?
4. What did you do to relief the symptom?
5. How did it progress, evolve, came to a halt or improve?
6. Who was helping you out when it happened and what was done?

- **Specific Complaints**

There are many, but only a few are common.

- **Headache**

The cardinal features of headache are quality, severity, localization, relieving and precipitating factors, associated phenomena such as nausea or scotoma, as well as recurrence over time. The distinction between an acute and chronic persistent headache is usually easily apparent. Acute headache is often severe in intensity but acute should be further defined as split second (such as in a ruptured aneurysm), rapidly progressing (such as in migraine), or jabbing and jolting (such as in cluster headache and trigeminal neuralgia). The severity of pain is difficult to judge but it is important to know whether the pain is throbbing, lancinating, electric, stabbing, radiating, involving the entire head or unilateral, or more circumspect spots such as in a psychogenic headache. Factors that may aggravate the pain should be identified such as posture, straining, sneezing, coughing, movement, prior alcohol and even consumption of ice cream. Headache that changes with position is important. Many headaches associated with an increased intracranial pressure do worse with lying down and are severe at awakening in the morning and headaches associated with CSF hypotension are worse with standing up and immediately relieved with lying down. Headaches relieved with knee-chest position may be due to third ventricle obstructive tumors. The severity of the headache is difficult to measure. For example, a severe neck strain from overzealous gardening may look like an acute subarachnoid hemorrhage. Associated symptoms are important. Nausea and vomiting with acute new onset headache is commonly due to a structural lesion. Migraine may have specific features that may further classify the type of migraine such as aura (flickering lights, geometric distortions and even visual hallucinations), photophobia and sonophobia, and inability to accept any kitchen smells. Ptosis, nasal stuffing, redness of the conjunctiva may suggest a
cluster headache. Electric lancinating pain in the cheek, ear, or jaw mimicking a molar abscess may suggest a trigeminal neuralgia. Touching the face with make up, a cold breeze, chewing or toothbrushing may trigger a brief lancinating electric stab as if a finger is put in a socket. It is sometimes a spot diagnosis. A patient who requires a dark room likely has migraine or chronic tension headache. A tendency to move, rock back and forth, is characteristic of a patient in the middle of a cluster headache, and a patient with trigeminal neuralgia may assume a typical posture in which the palm of the hand is held close to the cheek supported by the other arm but without touching it. It is important to find factors that may precipitate or aggravate headache and factors that may provide relief. Certain drugs may cause immediate relief and can be used as a diagnostic test. This includes several liters of nasal oxygen for cluster headache, any of the tryptans for classic migraine, and NSAIDs for paroxysmal hemicrania. Nonetheless it is important to mention that a rapid response to over-the-counter pain medications and even narcotics do not exclude the possibility of a more severe disorder such as brain tumor.

- Dizziness and Vertigo

Neurologists are sometimes disillusioned when their clinic day only involves patients with dizziness and vertigo. As an isolated symptom it is rarely due to a neurologic cause. Dizziness is often described by the patient as wooziness, giddiness or faintness. In elucidating these umbrella terms it is important to determine whether the dizziness is true vertigo, in which the patient experiences a true rotational effect, or whether it consists of a light-headedness or presyncopal sensation. It is important to inquire about vertigo with other signs of brain stem dysfunction such as diplopia, dysarthria, dysphagia, hypesthesia, and acute ataxia. On the other hand the presence of ringing in the ear, hearing loss and violent vomiting may suggest a peripheral (labyrinth) rather than a central (brainstem) cause. vertigo with position change is commonly due to a peripheral cause as well. Intermittent "dizzy spells" are commonly hyperventilation. Failure to perform a hyperventilation test (have the patient breathe in and out for one to two minutes trying to produce patients own recognizable symptoms) in these patients must be considered a mistake. In these dizzy patients who do not have the classic tingling fingers and tight lips during the attack a series of expensive MRI and ENT tests have been performed but they are only waiting to be recognized by an astute physician. Similarly it should be noted that some patients describe dizziness to point out a gait disorder and we have seen patients with early Parkinson’s disease and instability undergo several ENT evaluations until the tendency to pro- or retropulse or cogwheel rigidity is detected.

- Sensory Symptoms

Numbness is vague term used by patients and may indicate weakness, pain or itching. True tingling, in which the patient describes a constant pins and needles sensation should be differentiated from a tight band, tight shoe, walking on air or rough surface sensation which indicates an abnormality in the posterior columns. Typically numbness has been present for quite some time in extremities. Extremity numbness expanding in a clockwise fashion, although sounding functional, may indicate a cervical spinal cord lesion. Lack of temperature sense needs to be addressed. Often patients are unable to distinguish hot and
cold while taking a shower. Failure to recognize objects in a purse or in a pocket may also indicate significant loss of proprioception and is commonly found in a cervical spine lesion such as cervical spondylosis or syringomyelia. Some of these patients may demonstrate spontaneous finger movements in an attempt to orient them in space (pseudoathetosis).

- **Cognitive Decline**

Typically memory decline is a gradual process over years, but an alleged acute worsening may sometimes prompt early evaluation. Sometimes it is a child from out of state that visits after a while, only to find out that personal hygiene has tumbled downhill. In some patients family members are surprised that the patient does not know the date, the name of the president or any recent encounter when specifically asked. Memory decline often involves consistent difficulty with finding words, names, way to one’s own house, inability to describe routes to the clinic, but also more subtle problems such as inability to maintain a coherent conversation or failure to complete complex dinners.

A history of nocturnal confusion with wandering through the house and opening of drawers without a purpose may be obtained in a patient with advanced Alzheimer’s disease or any of the other dementias. Transient focal signs including dysarthria, aphasia, or hemiparesis may point to a multivascular dementia. A recent head trauma, often a car accident in the months before memory decline may indicate a subdural hematoma, and as expected it is not volunteered by the patient. Complete loss of memory is known as transient global amnesia and essentially the patient has no recollection of this episode. During this period the patient is constantly asking for where he is, whom he is with, and what he has been doing to be in this place. Complete loss of memory with a defined period of time, (or example, memory loss of three months) is typically psychogenic. Loss of moral behavior and critical judgment may indicate an advanced stage of dementia but depending on the underlying personality. Loss of a patient’s own identity is a very late stage of dementia often emerging when patients become bed - bound. Its early presentation should indicate pseudodementia. It is important to inquire about depressive symptoms such as anhedonia, weight loss, and suicidal thoughts to exclude the possibility of a treatable depression but patients with Alzheimers, particularly high strung individuals, may become depressed when defects are becoming noticed.

- **Speech Disorders**

Speech disorders can be grossly distinguished between a dysarthria in which there is a major disturbance of articulation and aphasia in which the patient’s speech is distorted and words or letters become substituted. A dysarthria is not only slurring of words but also output is hesitant, explosive, and staccato. A fluent aphasia can be so severe (“word salad”) that no content is discernible. The patient also then has difficulty with repetition and naming. A nonfluent aphasia is present when the speech is fragmentary, telegram style, with many substitutions and neologisms. Failure to speak (muteness) is uncommon but may occur in extreme advanced forms of Parkinson’s disease and due to bifrontal cerebral infarcts. Muteness with retained ability to recognize objects by word recognition and complete retention of writing is rarely structural and commonly psychogenic.
• Weakness

Weakness may involve one or two extremities. Unilateral weakness has more significance than a generalized sensation of weakness. In many patients, weakness is progressive; fluctuating weakness should point to the possibility of myasthenia gravis, particularly when it occurs after fatiguing the muscle. Again the time frame of weakness is important. Rapid onset weakness within days may indicate a Guillain-Barré syndrome, inflammatory myopathy, or a vasculitis. Unexplained weakness without any sensory symptoms in one or two limbs should point to the possibility of ALS and often the patient is not aware of associated fasciculations or muscle atrophy and will only indicate weight loss.

• Visual abnormalities

Visual abnormalities may involve blindness in one or both eyes, double vision, or blurring of vision. Blurring of vision in itself may indicate an intrinsic ophthalmological cause or inability of the patient to express diplopia. Diplopia is binocular, meaning that covering one eye will lead to its disappearance. In addition, degree of diplopia increases as the gaze proceeds in the direction of the action of the paralyzed muscle. Monocular blindness can be due to transient ischemic attack in the retina. This disorder presents with the gradual onset (minutes) of a full gray or black field and sometimes with a small peephole indicating macular sparing. In other patients an altitudinal hemianopia is seen in which the defining line between visual loss and normal vision is horizontal. An hemianopia is often homonymous, typically the left eye deals with vision to the left and the right eye to the right and examination will often delineate the visual fields defect. It is important to additionally inquire about eye pain. Optic neuritis or painful ophthalmoplegia due to cavernous sinus syndrome or migraine may all present with blindness.

• Miscellaneous historical facts

Similar as in other branches of medicine, the occupation of the patient needs to be noted. Several neurologic disorders can be caused by poisons and heavy metal exposure. Lead, arsenic, insecticides, nitric oxide may all cause peripheral neuropathy. Drug-induced neurologic disorders are rare but many prescription drugs may have neurotoxic side effects. (An important reference is Neurotoxic Side Effects of Prescription Drugs.) A history of a recent infectious diseases, insect bite, tropic travel, and previous hospitalizations should be included as well as a survey of the marital history, alcohol and drug use (alcohol causes subdural hematomas and ecstasy or cocaine causes intracranial hemorrhages) when assessing a patient’s personality. Family history should be scrutinized for possible hereditary neurologic disorders (e.g. - Charcot-Marie-Tooth polyneuropathy, Huntington disease).
THE NEUROLOGICAL EXAMINATION

Neurologic examination follows a standardized pattern. Experience may tailor the full examination and result in focusing more on the most pertinent signs and symptoms. In addition often certain abnormalities should be reexamined over and over again to assure the abnormality.

- **Consciousness and Evaluation of Cognition**

Level of consciousness is measured with the Glasgow *Coma* Scale. This simple scoring system does not indicate the cause of decreased level of consciousness or *coma* but only indicates the depth of *coma* using three simple components. The spontaneous verbal eye and motor response is assessed followed by response to voice and pain. The pain stimulus is standardized using compression of the supraorbital nerve, nailbed, or temporomandibular joint. These noxious stimuli can produce standardized responses and these are outlined in the following table.

<table>
<thead>
<tr>
<th>Table 1 - The Glasgow Coma Score (GCS)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
</tr>
<tr>
<td>Spontaneous (4)</td>
</tr>
<tr>
<td>To speech (3)</td>
</tr>
<tr>
<td>To pain (2)</td>
</tr>
<tr>
<td>Remain closed (1)</td>
</tr>
<tr>
<td><strong>Best verbal response</strong></td>
</tr>
<tr>
<td>Oriented (5)</td>
</tr>
<tr>
<td>Confused (4)</td>
</tr>
<tr>
<td>Inappropriate words (3)</td>
</tr>
<tr>
<td>Incomprehensible words (2)</td>
</tr>
<tr>
<td>Mute (1)</td>
</tr>
<tr>
<td><strong>Best motor response</strong></td>
</tr>
<tr>
<td>Obeying commands (6)</td>
</tr>
<tr>
<td>Localizing pain (5)</td>
</tr>
<tr>
<td>Quick withdrawal to pain (4)</td>
</tr>
<tr>
<td>Flexion (coordinated movement to the chest; decorticate) to pain (3)</td>
</tr>
<tr>
<td>Extension (endo rotation and stiffening; decerebrate) to pain (2)</td>
</tr>
<tr>
<td>None (1)</td>
</tr>
</tbody>
</table>
Paradoxically the most severe form of coma, persistent vegetative state, the patient has the eyes open and will appear to look about but is unable to track any visual object (awake but not aware). Apart from determining the depth of coma it is important to evaluate hourly fluctuations. Fluctuation in level of consciousness may be caused sedative drugs, sleep deprivation but also by a disorder called nonconvulsive status epilepticus in which fluctuating level of consciousness is associated with eye lid jerking, staring as well as fumbling with hands and picking at clothes and bed linen. Overt jerking of extremities is not seen despite continuous electrographic spike and wave activity.

Cognitive function is tested using a series of batteries. Cognitive decline, as alluded to earlier, starts insidiously. In the very old (more than 85) a dividing line between dementia or some decline in memory function remains often difficult to draw. Several memory scales have been developed which test not only memory but also orientation, general knowledge, calculation, abstract thinking and so forth. Table 2 shows the individual components that are evaluated with a comprehensive bedside mental status examination.

**Table 2 - Mental Status Examination (MSE)**

1. **Orientation**: Patient needs to state her full name, address, building, city, and state and current date.

2. **Attention**: is tested by giving a patient a series of numbers. The patient needs to repeat seven digits forward, usually using a full phone number.

3. **Learning**: the patient is asked to repeat and memorize four words that are unconnected to each other, such as, apple, shoe, crying, and Mr. Murphy.

4. **Calculation**: is tested by subtracting 7 from 100 including other arithmetic problems. For example, asking to multiply 5 x 13 or add 11 and 29.

5. **Abstraction**: is tested by having the patient interpret similarities such as orange, banana, horse, dog, table, bookcase, and to ask the patient to explain common proverbs such as people who live in glass houses shouldn’t throw stones or don’t cry over spilled milk.

6. **Judgment**: can also be investigated by asking the patient what to do if he would see a person collapse while crossing the street.

7. **Construction**: is tested by having a patient draw a cube and a clock showing the hands directed at a certain time.

Failure to perform any of those tests, but usually a combination, may indicate a cognitive decline and would justify more extensive psychometric testing. Other investigations of higher cortical function are important. Apraxia is due to a disturbance of skilled movement or due to a disconnection of the speech area in the area of cortex that integrates motor tasks. Patients are unable to perform tasks such as a salute, form interlocking fingers, comb hair, stick out tongue, or pucker as if to kiss.
• **Speech and language**

Spontaneous speech, naming, repetition and reading is assessed. It requires experience and often a speech pathologist is needed to carefully categorize the abnormality. The major abnormalities are shown in Table 3.

<table>
<thead>
<tr>
<th>Types</th>
<th>Fluency</th>
<th>Repetition</th>
<th>Comprehension</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Frontoparietal lobe</td>
</tr>
<tr>
<td>Broca</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>Posterior part of inferior frontal gyrus</td>
</tr>
<tr>
<td>Transcortical</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>Watershed infarct</td>
</tr>
<tr>
<td>Wernicke</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>Superior temporal gyrus</td>
</tr>
<tr>
<td>Conduction</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>Arcuate fasciculus</td>
</tr>
<tr>
<td>Anomic</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Angular gyrus, temporal lobe</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type</th>
<th>Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hoarse, nasal</td>
<td>Myopathy, Myasthenia gravis</td>
</tr>
<tr>
<td>Tremor, irregular; variation in loudness</td>
<td>Cerebellum</td>
</tr>
<tr>
<td>Strained hyper nasal mono pitch</td>
<td>Corticobulbar tracts / bilateral</td>
</tr>
<tr>
<td>Weak phonation, poor fluency, breathy</td>
<td>Extrapyramidal</td>
</tr>
</tbody>
</table>

• **Cranial Nerve Examination**

The examination of 12 cranial nerves is simple in its execution but complex in interpretation.

• **Cranial Nerve I (Olfactory Nerve)**

Smell and taste is often impaired due to other systemic illnesses including banalities such as the flu. It is uncommonly tested during a routine neurologic examination but smell cards have been devised. Standard odors include peppermint, cloves, musk, and floral powders, as well as coffee and lemon extracts. The distinction between odors has more importance than its precise recognition. Anosmia can be excluded if the patient appreciates at least one odorized powder. The abnormalities of the olfactory nerve are typically caused by severe traumatic brain injury or a meningioma arising from the olfactory groove.
• Cranial Nerve II (Optic Nerve)

The optic nerve is examined using several tests starting with visual acuity. Each eye is tested separately using Snellen test card. The letters and the line designated 20 should be read at 20 feet recording 20/20 vision. When a refractory error is considered, the patient needs to view these letters through a pinhole using a piece of paper and creating a hole of approximately 1 mm. Marked deterioration of vision is recorded using several standard landmarks. For example a vision of 1/60 is present when a patient is able to see finger counting at 1-m distance, 1/200 when moving of the hand is observed. 1/¥ when only light perception is present and zero when completely blind. These abnormalities are typically seen in patients with a lesion of the optic nerve, often due to optic neuritis or anterior ischemic optic neuropathy. The visual fields are tested with a confrontation method in which the patient faces the examiner, covers one eye with his hand, and fixes his gaze on the examiner’s nose. The examiner’s wiggling finger is then brought in along all four quadrants and mentioned by the patient when it comes into view. Visual field defects are named hemianopsia when there is loss of vision in one half field of one eye. Loss vision in corresponding halves of both visual fields is called homonymous hemianopsia. Localization of a homonymous hemianopsia is typically in the occipital cortex. However, macular (central) sparing may occur due to significant collateral branches from the middle cerebral artery. Lesions in the temporal lobe produce a "pie in the sky" homonymous defect. A lesion in the parietal lobe produces a lower quadrant defect. Examination is followed by fundoscopy in which the optic disk is assessed. Dilatation of the pupil is not needed for most purposes but when in doubt a more complete examination with assessment of the macula should follow. Disk swelling is apparent with loss of the normal venous pulse first followed by loss of sharp temporal or nasal margins. Papilledema in advanced forms assumes the configuration of a champagne cork and peripheral hemorrhages are seen. Papilledema indicates increased intracranial pressure from a mass or due to cerebral venous obstruction. It may also seen in a central venous occlusion and may at times be difficult to distinguish from congenital lesions such as a drusen optic disk or anomalous elevation.
Figure 1. Keep both yours and the person's eyes Have the patient focus on a distant object Look at right fundus with your right eye Ophthalmoscope should be close to your eyes. Your head and the scope should move together Set the lens opening at +8 to +10 diopters. With the ophthalmoscope 12-15 inches from the patient's eye, check for the red reflex and for opacities in lens or aqueous. While adjusting the diopter setting, approach the patient more closely and systematically inspect the disc, noting the color, shape, margins and cup-to-disc ratio. Inspect the vessels, noting obstruction, caliber and arterial/venous ratio. Note the presence of arterial/venous nicking and arterial light reflex. Check the background by inspecting for pigmentation, hemorrhages and hard or soft exudates. Next, try to identify the macula. Have the patient look at light Normal: Disc margins are sharp color: yellowish orange to creamy pink shape: round or oval Cup to disc ratio: less than half Vessels AV ratio AV crossing: no indentation No arterial light reflex Fundus background No exudates or hemorrhages color : red to purplish Macula macula is located 2.5 disc distance temporal to disc no vessels are noted around Macula it may be slightly pigmented.

Figure 2. Position yourself in front of the patient. Test the patient's visual acuity, each eye separately, covering one at a time. Snellen's chart is used by Ophthalmologists. Visual acuity is recorded as a fraction. The numerator indicates the distance (in feet) from the chart which the subject can read the line. The denominator indicates the distance at which a normal eye can read the line. Normal vision is 20/20. A pocket screener is used at the bedside. Hold the pocket screener at a distance of 12-14 inches. At this distance the letters are equivalent to those on Snellen's chart. In children the techniques used are "E" card: If the child cannot read letters or numbers Fixation and following: In infants. Have the child follow a toy. Pinhole test: To differentiate refraction errors from organic disease. If vision improves with pinhole it is refractory error.
Figure 3. By confrontation Position yourself in front of the patient. The nose normally cuts off the medial field of vision. Hence, compare the patient's right eye to your left eye and vice versa. Instruct the patient to look straight at you and not to move their eyes. Compare your field of vision with the subject's. Bring your finger from the right field of vision until it is recognized. Test one quadrant at a time. Wiggle your fingers to see whether the patient can recognize the movement. Some like to have the patient count fingers, i.e., 1, 2 or 5. Test all four quadrants in a similar fashion. When abnormality is detected, would require automated methods of testing in the lab Normal: Assuming the examiner has normal field of vision, patient should have the same extent of field of vision.

- Cranial Nerves III, IV, and V (Oculomotor, Trochlear, and Abducens)

The pupil size and reflexes are tested typically in a darkened room. Pupils are normally equal in size, although a 1-mm difference may be physiologic. Bright light will produce constriction except in a blind eye. The differential diagnosis of myosis or mydriasis is shown in Table 4.
### Table 4 – Myosis, Mydriasis, and Horner Syndrome Differentials

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td><strong>Myosis</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Narcotic drugs</td>
</tr>
<tr>
<td></td>
<td>Acute metabolic encephalopathy</td>
</tr>
<tr>
<td></td>
<td>Acute pontine lesion</td>
</tr>
<tr>
<td></td>
<td>Nonketotic hyperglycemia</td>
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<tr>
<td><strong>Mydriasis</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Delirium, anticholinergic agents, magnesium excess</td>
</tr>
<tr>
<td></td>
<td>Norepinephrine</td>
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<tr>
<td><strong>Horner syndrome</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Carotid dissection</td>
</tr>
<tr>
<td></td>
<td>Brachial plexopathy</td>
</tr>
<tr>
<td></td>
<td>Lateral medulla oblongata lesion (Wallenberg syndrome)</td>
</tr>
</tbody>
</table>

When anisocoria is noticed on should determine change in dim or bright light. As a general rule, increase in difference in bright light indicates an abnormality in the sphincter (iris damage, atropine), decrease in bright light indicates iris dilator weakness (Horner syndrome, Adie syndrome, uveitis).

The ocular movements are investigated by having the patient turn the eyes in a horizontal and vertical plane tracking the physician finger. Vertical gaze tends to diminish with age. It is important to record saccades which are "stammering" eye movements often caused by drugs, also degenerative neurologic disorders such as Parkinson’s disease or progressive supranuclear palsy. In addition, convergence is examined. The examination may also be further examined using the optokinetic [nystagmus](#) in which the patients look in front of a drum containing a series of lines. Particularly patients with parietal lesions have an abnormal optokinetic [nystagmus](#). Diplopia is difficult to assess but certain rules can be applied. These are the following:

1. The distance between the true and the false image increases with direction of action of the paretic muscle. (In a sixth nerve palsy on the right the images are widest apart when looking to the right.)
2. Horizontal diplopia occurs with lesions of the medial or lateral recti muscles.
3. Vertical diplopia occurs with a superior or inferior recti or oblique muscles. The more peripherally seen image is always the false image.

[Nystagmus](#) is noted as well. Typically a [nystagmus](#) is a pendular movement in which the movements are of equal velocity. Eye jerk is divided into a fast and slow phase. First degree nystagmus to the right is revealed on a right lateral gaze and shows fast phase to the right. [Nystagmus](#) on forward gaze is second degree and on left lateral gaze is called third degree. [Nystagmus](#) in the vestibular nucleus is jerk type rotation. The differentiation between a...
central and peripheral nystagmus is difficult. Central vestibular nystagmus is often vertical, purely torsional and worse looking down and out. In addition vision does not suppress the centrally mediated nystagmus and vertigo is mild. Nystagmus from a central lesion often is part of a symptom complex with other brain stem signs. In some patients a congenital nystagmus is found and is recognized by irregular conjugate, horizontal and in up gaze, accentuated by fixation and anxiety and significantly diminished by convergence.

Figure 4. Inspect the eyes. Look for symmetry of eyelids. Note the alignment of the eyes at rest. Ductions: Movement of one eye at a time Versions: Both eye movement Have the patient follow an object into each of the nine cardinal fields of gaze. Note that both eyes move together into each field. Eye movements should be smooth and without jerking. Eyelids should be gently lifted up by the examiner's fingers when testing downward gaze. Jerky, oscillatory eye movements (nystagmus) may be abnormal, especially if sustained or asymmetrical. Hirschberg light reflex test: Use a penlight in middle of field of vision. Note
light reflection on both cornea. Let patient gaze in different directions, while noting the position of light reflection in cornea. If they are asymmetrical it indicated there is strabismus. Look up how to perform cover-uncover test to evaluate non paralytic strabismus. Normal: Full conjugate eye movements. No nystagmus in any direction

Figure 5. Have the patient look at a distant object. Look at size, shape and symmetry of pupils. Shine a light into each eye and observe constriction of pupil. Flash a light on one pupil and watch it contract briskly. Flash the light again and watch the opposite pupil constrict (consensual reflex). Repeat this procedure on the opposite eye. Normal: Pupils are subtle, mild anisocoria (unequal in size) by itself and not necessarily an abnormal finding. Pupil size is 3-5 mm in diameter. They react briskly to light. Both pupils constrict consensually.
Figure 6. Ask the patient to follow your finger as you bring it toward the bridge of his nose. Note the convergence of the eyes and pupillary constriction. Normal: Convergence should be sustainable to within 5-8 cm and both pupils constrict.

- Cranial Nerve V (Trigeminal Nerve)

The trigeminal nerve consists of motor and sensory fibers. The sensory dermatome involves the scalp close to the line of the ear to forehead, eye, cheek, and chin. It can be tested by light touch using a cotton Q-tip, pin, and temperature using hot and cold tubes. The corneal reflex is tested using a cotton ball gently striking the outer rim rather than centrally on the cornea causing a reflective blink. The patient should also indicate touch. In addition, the jaw jerk is elicited by tapping on the apex of the jaw. The response is only significant when it is exaggerated and may indicates a brain stem lesion.

Figure 7. Trigeminal nerve has motor and sensory components
• **Motor**

Have patient clench teeth and feel the Masseters and Temporal muscles and compare sides. Note the strength of contractions. Edentulous patients may not be able to clench "teeth".

• **Sensory**

With a light touch of cotton, check the patient's ability to detect light touch in all areas which are supplied by the three divisions of the fifth cranial nerve. Instruct the patient to close his eyes and respond by saying "yes" every time he feels the sensation of cotton touching his face.

• Compare corresponding contralateral segments of his face.
• Test pain sensation with a pin in each of the three divisions, comparing both sides.

Test corneal (blink) reflex with a wisp of cotton lightly touched to the edge of the cornea. There should be a consensual eyeblink normally.

---

![Figure 8. A, The corneal reflex, B, examination of the sensory part of the trigeminal nerve](image-url)

• **Cranial Nerve VII (Facial Nerve)**

This is tested having the patient elevate eyebrows, closing eyelids forcefully in which the eyelashes disappear, and producing a voluntary smile. When a paralysis of the facial nerve exists, pronounciation of sounds that require closure of the lip such as pot and boy is disturbed. In a peripheral seventh nerve palsy the platysma is also abnormal and can be examined after the patient draws the lower lip and the angle of the mouth downwards. Taste may be abnormal but only when the lesion is peripheral to its junction with the cordae tympany. It is examined using sugar, salt, and sometimes tartaric acid but the results are difficult to interpret. A common peripheral facial paralysis called Bell’s palsy can be recognized by involvement of all three branches, inability to blink and close the
eyelid, tearing, and a so-called Bell’s phenomenon in which with forceful closure of the eye the globe turns upward.

Figure 9. Inspect the face. Look for asymmetry at rest, during conversation and when testing various muscles. Ask the patient to wrinkle his forehead or raise his eyebrows, enabling you to test the upper face (frontalis). Next, have the patient tightly close his eyes. Test the strength of the orbicularis oculi by gently trying to pry open the patient's upper eyelid. Instruct him to puff out both cheeks. Check tension by tapping his cheeks with your fingers. Have the patient smile broadly and show his teeth, testing the lower face. Normal: No facial asymmetry. Wrinkling of the forehead and smiling are equal and symmetrical.
• Cranial Nerve VIII (Acoustic Nerve)

Hearing is tested with a whisper voice. The examiner stands in front of the patient and whispers words (e.g.-66, Boston) while covering patient eyes with one hand and blocking the ear that is not tested with the other hand. Several tuning fork tests are available. The Weber test is a test in which tuning fork is placed in the middle of the skull in which hearing normally should be observed in both ears. Lateralization occurs on the same side in the middle ear involvement, on the opposite side when the cochlear nerve is involved. The Rinne test is performed after placing the vibrating tuning fork against the mastoid and when it can no longer be heard it is held in front of the ear. Positive result is when the tuning fork is heard longer by air than bone conduction. An abnormal test is a sign of middle ear defect or a blocking of the external auditory canal. Vestibular function can be examined with laboratory and caloric testing but also using the Barany test. The patient is seated on examining table and will be reclined backwards with the head hanging over the edge of the table. After a brief interval vertigo will set in and at the same time a brief rotary nystagmus appears. The patient is asked to look downwards. The test is sensitive for a benign positional nystagmus. BPPD is due to dysfunction of the vestibular organ. It is common and often misdiagnosed as vertebral - basilar insufficiency.

Figure 10. With eyes closed, the patient should be instructed to acknowledge hearing the gentle rubbing of the examiner's fingers approximately 3-4 inches away from his right and left ear. A watch, which the examiner can hear at a specific distance from his ear, is placed next to the patient's ear. Ask him to note when the watch sound disappears. Note that the examiner has to have normal hearing to do this exam (in at least one ear). Normal: In a quiet room, the patient should be able to hear the physician's fingers rubbed lightly together 3-4 inches from his ear. With aging Progressive bilateral Presbycusis (old hearing): Sensory neural loss Difficulty appreciating consonants
• Cranial Nerve IX (Glossopharyngeal Nerve)

This nerve is tested by putting a tongue depressor in the back of the throat which will produce a gag reflex. Midline elevation of the soft palate occurs. Its significance is dubious because many normal individuals have no gag with stimulation.

• Cranial Nerve X (Vagus Nerve)

The patient is asked to say "ah" and the soft palate will rise symmetrically. When there is weakness on one side, deviation will be to the intact side. Swallowing should not be impaired with unilateral involvement of the vagus but hoarseness occurs with involvement of the vocal cord on the affected side.

![Figure 11. Have patient say "ah" and observe movement of palate with flash light. Stimulate posterior pharynx (gag reflex) with cotton tipped probe one side at a time. Have the patient cough. Observe patients voice. Normal: No hoarseness is noted in voice and the cough is effective. Palate moves symmetrically and the uvula stays in mid line. Pharynx moves forwards with retching on stimulation of posterior pharynx.](image)

• Cranial Nerve XI (Accessory Nerve)

Accessory nerve is examined by having the patient turn the head forcefully against examiner hand and shrugging both shoulders against resistance. Paralysis of the trapezius muscle or sternocleidomastoid muscle can be observed and often is due to a peripheral nerve damage associated with a lymph biopsy lateral in the neck.
Figure 12. Inspect Trapezius and Sternocleidomastoid muscles. Note muscle size (bulk). Look for asymmetry, atrophy and fasciculation. Determine muscle power by gently trying to overpower contraction of each group of muscles. Have patient shrug shoulder against resistance and evaluate strength of Trapezius muscle. Have patient turn head to one side against resistance and evaluate strength and observe contracting sternomastoid muscle.

- Cranial Nerve XII (Hypoglossal Nerve)

The patient is asked to protrude the tongue and then also the tongue is investigated carefully for atrophy and fasciculations. Tongue fasciculations are strong indicators of ALS in the appropriate setting. It may appear like a bag of worms. The patient then is asked to rapidly move the tongue from left to right and the strength is tested by pushing the tongue against a tongue blade or against the cheek.
Figure 13. Pay attention to articulation. Inspect Tongue. Note muscle size (bulk). Look for asymmetry, atrophy and fasciculation. Determine muscle power by Having the patient to protrude the tongue and side to side, up and down. Note the resistance offered by the tongue to your finger pressing on the tongue through each cheek.

- Cranial nerve palsy examples

<table>
<thead>
<tr>
<th>CN III Palsy (a)</th>
<th>CN III Palsy (b)</th>
<th>Right CN VI Palsy Gaze left</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN VII (Bell's) Palsy (a)</td>
<td>CN VII (Bell's) Palsy (b)</td>
<td></td>
</tr>
</tbody>
</table>
### Examination of Muscle Function

Muscle examination includes inspection for atrophy, fasciculations, and tone. Tone can be rigid or decreased. Typically proximal and distal muscles are tested and are graded using the Medical Research Counsel (MRC) scale.
Muscle atrophy is seen in many diseases of the peripheral nerve but also in advanced myopathies. Generally muscle weakness in myopathies involves muscles in a proximal distribution and peripheral nerve in a distal distribution (hand and foot muscles).

Muscle weakness may involve a nerve root or single nerve. It is summarized in Table 8. Fasciculations are fine twitches in parts of muscle and typically do occur at areas of the limb that the examiner is not looking at ("the shooting star phenomenon"). Muscle tone is assessed after passive movement of the muscle and often muscle tone becomes clear to resistance. Hypotonia is apparent when a limb is shaken by the examiner documenting significant flailing. Loose and toneless muscles not only can be seen in peripheral nerve abnormalities also in the setting of acute cerebellar lesions. Spasticity is diagnosed with increasing resistance to passive movement followed by a sudden release of resistance, typically called a clasp knife reaction.

### Table 5 - MRC Scale

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Normal power</td>
</tr>
<tr>
<td>4</td>
<td>Reduced power, but still contracting muscle against resistance</td>
</tr>
<tr>
<td>3</td>
<td>Movement against gravity but not resistance</td>
</tr>
<tr>
<td>2</td>
<td>Movement with gravity eliminated</td>
</tr>
<tr>
<td>1</td>
<td>Flicker of movement only</td>
</tr>
<tr>
<td>0</td>
<td>No movement</td>
</tr>
</tbody>
</table>

### Table 6 - Nerve Roots and Peripheral Nerves Supplying Arm / Leg Muscles

<table>
<thead>
<tr>
<th>Nerve Roots</th>
<th>Muscles Supplied</th>
</tr>
</thead>
<tbody>
<tr>
<td>C₄</td>
<td>Levator scapular</td>
</tr>
<tr>
<td>C₅ - T₁</td>
<td>Pectoralis major</td>
</tr>
<tr>
<td>C₅ - C₆</td>
<td>Deltoid (axillary nerve)</td>
</tr>
<tr>
<td></td>
<td>Biceps (musculocutaneous nerve)</td>
</tr>
<tr>
<td></td>
<td>Brachioradialis (radial nerve)</td>
</tr>
<tr>
<td></td>
<td>Supinator (radial nerve)</td>
</tr>
<tr>
<td>C₆ - C₇</td>
<td>Pronator teres (median nerve)</td>
</tr>
<tr>
<td>C₆ - C₇ - C₈</td>
<td>Triceps (radial nerve)</td>
</tr>
<tr>
<td></td>
<td>Extensor carpi ulnaris (radial nerve)</td>
</tr>
<tr>
<td></td>
<td>Flexor carpi ulnaris (median and ulnar nerve)</td>
</tr>
<tr>
<td>Nerve Level</td>
<td>Muscles</td>
</tr>
<tr>
<td>------------</td>
<td>---------</td>
</tr>
<tr>
<td>C7-8</td>
<td>Digit extensors (radial nerve)</td>
</tr>
<tr>
<td>C7-8 - T1</td>
<td>Digit flexors (median and ulnar nerves)</td>
</tr>
<tr>
<td>C8 - T1</td>
<td>Thenar (median nerve)</td>
</tr>
<tr>
<td></td>
<td>Hypothenar (ulnar nerve)</td>
</tr>
<tr>
<td></td>
<td>Intersossei (ulnar nerve)</td>
</tr>
<tr>
<td>L2-3/4</td>
<td>Iliopsoas (femoral nerve)</td>
</tr>
<tr>
<td></td>
<td>Adductor thigh (obturator nerve)</td>
</tr>
<tr>
<td>L4-5 - S1</td>
<td>Hamstrings (sciatic nerve)</td>
</tr>
<tr>
<td></td>
<td>Toe extensors (peroneus nerve)</td>
</tr>
<tr>
<td>L2-3/4</td>
<td>Quadriceps (femoral nerve)</td>
</tr>
<tr>
<td>L4-5</td>
<td>Anterior tibial (peroneal nerve)</td>
</tr>
<tr>
<td>L5 - S1</td>
<td>Extensor hallucis longus (peroneal nerve)</td>
</tr>
<tr>
<td></td>
<td>Peronei (peroneal nerve)</td>
</tr>
<tr>
<td></td>
<td>Posterior tibial (tibial nerve)</td>
</tr>
<tr>
<td></td>
<td>Toe flexors (tibial nerve)</td>
</tr>
<tr>
<td>L5 - S1:2</td>
<td>Gluteus maximus (inferior gluteal nerve)</td>
</tr>
<tr>
<td></td>
<td>Gastrocnemius / Soleus (tibial nerve)</td>
</tr>
</tbody>
</table>

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Figure 14. Inspect the muscles of the shoulder, arm, forearm and hand. Note muscle size (bulk). Look for asymmetry, atrophy and fasciculation. Look for tremor and other abnormal movement at rest and with arms outstretched. Determine muscle power by gently trying to overpower contraction of each group of muscles. Shoulder: Abduction (Deltoid), Adduction (), Shrug (Trapezius) Elbow: flexion (Biceps) and extension (Triceps), Wrist: Flexion () and extension(). Hand: Grip, opposition of thumb and index finger, opposition of thumb and little finger and finger abduction and adduction. Determine limb tone (resistance to passive stretch). With the patient relaxed, gently move the limb at the shoulder, elbow and wrist joints and note whether tone is normal, increased or decreased Normal: Muscles are symmetrical in size with no involuntary movements. In some, muscles may be slightly larger on the dominant side. Muscle power obviously varies. You should not be able to overpower with reasonable resistance. You have to learn to appreciate the normal tone from practice.
Figure 15. Inspect the muscles of the hip, knee and ankle. Note muscle size (bulk). Look for asymmetry, atrophy and fasciculation. Look for abnormal movement. Determine muscle power by gently trying to overpower contraction of each group of muscles. Hip: Flexion (Iliopsoas), Extension (Gluteus maximus), Abduction, Adduction. Knee: Flexion (Hamstrings), Extension (Quadriceps) Ankle: Dorsiflexion (Tibialis anterior), Plantar flexion (Gastronemius). Determine limb tone (resistance to passive stretch). With the patient relaxed, gently move the limb at the hip, knee and ankle and note whether tone is normal, increased or decreased. Flex the hip and knee. Support the knee, dorsiflex the ankle sharply and hold the foot in this position checking for clonus.
Examination of motor power

Motor examination

Deltoid C5 Axillary N.
Biceps C6 Musculocutaneous N.

Triceps C7 Radial N.
Brachioradialis C6 Radial N.
Extensor Carpi Ulnaris C7 Radial (Posterior Interossious)

Extensor Digitorum C7 Radial (Posterior Interossious)
First Dorsal Interossious T1 Ulnar Nerve
Abductor Pollicis Brevis T1 Median N.

Psoas L1,2
Hamstring S1 Sciatic
Tibialis Anterior L4,5 Deep Peroneal N.
Reflexes

Tendon reflexes localize to various segments in the spinal cord. The biceps reflex (C5-C6), triceps (C6-C7), knee (L2, L3, and L4), and ankle (L5-S1). Deep tendon reflexes are also classified using a simple grading system (Table 7).

<table>
<thead>
<tr>
<th>Table 7 - Classification of Deep Tendon Reflexes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 = Absent</td>
</tr>
<tr>
<td>+/- = Present with enforcement</td>
</tr>
<tr>
<td>+ = Just present</td>
</tr>
<tr>
<td>2+ = Normal reflex</td>
</tr>
<tr>
<td>3+ = Brisk reflex, additional beat but still within normal limits</td>
</tr>
<tr>
<td>4+ = Pathological brisk reflex and clonus</td>
</tr>
</tbody>
</table>

Reflexes in patients may be depressed without any pathological meaning and in many patients voluntary contracting a muscle in other limb will facilitate the reflex (Jendrassik maneuver). Important reflexes are a normal plantar reflex (toes curling down), Babinski sign (unfortunately often called reflex, response or worse "the Babinskis") typically when a piece of metal or wood is applied to lateral surface of the foot or moved in a hockey stick curve from the heel to the front. It results in flexion of the great toe, spreading of the toes in
a same response as flexing the knee and contraction of the tensor fascia lata (so called triple response). Other reflexes that need to be examined are abdominal reflexes, stroking the surface of the abdomen in four segments. Contraction is seen, but in elderly obese, and patients with lax abdominal muscles, reflexes are most of the time absent. When the two lowest abdominal responses are absent, a localized spinal cord lesion is at the T10 level. Many other reflexes have been described. They include snout reflex by stimulating the lips, grasp reflex with persistent flexion of the fingers after insertion of two fingers in the palm, palmomental reflex with pressure on the palm causing a contraction of the ipsilateral mentalis muscle, all potentially indicating cortical inhibition. The Hoffmann-Trommer reflex is obtained by snapping the terminal phalanx of the middle finger causing the flexion response of all fingers. The abnormality has been falsely considered "the Babinski of the arm" but only asymmetries are of importance. It is often difficult to elicit.

Figure 16. Patient should be relaxed and positioned symmetrically, preferably lying supine. Biceps reflex: (C5-C6) With the arm gently flexed at the elbow, tap the biceps tendon with a reflex hammer. It may help to locate this tendon with your thumb, and strike your own thumb with the hammer. There should be a reflex contraction of the biceps brachii muscle (elbow flexion). Triceps reflex: (C7-C8) With the elbow in flexion, tap the triceps tendon, just proximal to the elbow, with a reflex hammer. The arm could also be abducted at the shoulder for this maneuver. There should be a reflex contraction of the triceps muscle (elbow extension). Brachiradialis reflex: (C5-C6) Knee reflex: (L2-L4) Slightly lift up the leg under the knee, and tap the patellar tendon with a reflex hammer. There should be a reflex contraction of the quadriceps muscle (knee extension). (If performed in a sitting position, have the legs dangle over the edge of the chair or table). Ankle reflex: (S1) Slightly externally rotate at the hip, and gently dorsiflex the foot, tapping the Achilles tendon with a reflex hammer. There should be a reflex contraction of the gastrocnemius muscle (plantar flexion). When the reflexes are absent try eliciting it after re-enforcing (Jendrassik maneuver0, by asking the patient to interlock and pull flexed fingers. Deep tendon reflexes should be graded on a scale of 0-4 as follows: 0 = absent despite reinforcement 1 = present
only with reinforcement 2 = normal 3 = increased but normal 4 = markedly hyperactive, with clonus

Figure 17. With the patient supine, support the weight of the foot at the ankle. With a pointed object, stroke the lateral aspect of the sole of the foot, from the heel up and across the ball of the foot. Normal: Note plantar flexion of the toes.

Sensory Examination

Sensory testing involves assessment of light touch, pinprick, vibration sense, and joint position sense, and in occasional situations temperature assessment. Light touch involves wisp of cotton ball. The skin is touched, not moved, along it. Pinprick is tested with a sterile pin. Vibration using a tuning fork has similar meaning as joint position sense. Typically movement of the toe up or down is assessed or the patient imitates the same movement in the other limb. When a sensory level is noted by the patient the margins of abnormality needs to be carefully localized. Important pointers are shoulders (C4), nipples (TH4) and navel (TH10). Significant loss of proprioception will cause pseudoathetosis in which the fingers constantly try to orient themselves in space. A 2-point discrimination is also assessing the posterior column and normally stimulus separated by 2 mm should be distinguished.
Figure 18. Test light touch with a wisp of cotton. Test pain sense with a blunted, disposable safety pin or splintered cotton tip applicator. For light touch and pain: Have patient close eyes and report each test stimulus. Test over sides of each foot, leg, thigh, hand, forearm and arm. Compare the right and left and distal with proximal. Test the trunk where indicated. Test position sense by moving the toe or finger up and down, held by its sides, and have the patient report its position with eyes closed. Vibration sense is tested with a vibrating tuning fork placed over bony prominences of the feet (ankles) and hands (knuckles). Ask the patient to report when the vibration sense is lost. Sensation is tested by comparing the right and left sides in cases of suspected root or nerve lesions, sensation in a dermatomal or peripheral nerve distribution is carefully tested. If a spinal cord lesion is suspected, check for sensory loss over the trunk and sacral areas. Normal: Light touch, pinprick, vibration and position sense are intact throughout.

Cerebellar Function

Cerebellar function is tested with a finger-to-nose test or finger-to-finger test typically using additional turning in the wrists to further test coordination. The most commonly neglected investigation in bed bound patients is a sitting position in which patient may fall to one side with a midline vermis lesion. Dysmetria is noted when the patient cannot smoothly touch the nose and becomes shaky when reaching target.
Figure 19. Ask the patient to alternately reach out and touch your extended finger and his own nose. Test both hands. Ask the (supine) patient to touch his heel to the opposite knee, and slide it smoothly down he shin of that leg. Test both legs. Normal: Patient can touch the target and perform movements in a smooth, coordinated manner. Heel to knee to shin are performed smoothly and accurately.

**Gait**

A favorite pastime of neurologists is to investigate gait. Typically the patient is asked to walk on the hallway and several components are investigated - stability, stride, initiation of gait, and turns. Typical abnormalities in a Parkinson’s patient is stooped gait, reduced arm swing, fragmentary turning. In a patient with a hemiplegia the affected leg is swung outwards with a tendency for the foot to catch on the ground. Profound loss of sensory information from the feet is actually heard due a stamping gait.

<table>
<thead>
<tr>
<th>Table 8 - Gait</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type</strong></td>
</tr>
<tr>
<td>Cerebellar ataxia</td>
</tr>
<tr>
<td>Sensory ataxia</td>
</tr>
<tr>
<td>Frontal lobe ataxia</td>
</tr>
<tr>
<td>Spastic</td>
</tr>
<tr>
<td>Akinetic</td>
</tr>
</tbody>
</table>
Figure 20. Ask the patient to walk back and forth across the room. Observe for equality of arm swing, balance and rapidity and ease of turning. Next, ask the patient to walk on his tiptoes, then on heels. Ask the patient to tandem walk. Test patient's ability to stand with feet together with eyes open and then closed. (Romberg's test). Reassure patient that you will support him, in case he becomes unsteady. Normal: Person can walk in balance with the arms swinging at sides and can turn smoothly. Person should be able to stand with feet together without falling with eyes open or closed.
Some abnormal gaits

Steppage Gait  |  Hemiplegic Gait  |  Parkinsonian Gait

Retropulsion

Localization Principles

This document is not designed to give a complete evaluation of the localization techniques and the reader should be referred to the book by Brazis, Masdeu, and Biller, Localization in Clinical Neurology, Lippincott Raven, 3rd Edition. Some generalities should be mentioned. Lesions of the upper motor neuron will give paralysis, distally more involved in than the proximal muscles as well as increased reflexes and clonus, and loss of cutaneous reflexes and a Babinski sign. Lesions of the lower motor neuron involve atrophy, flaccid paralysis, fasciculations and weakness is segmental in character. The segmental distribution is noticeable. Specific muscles are innervated through a single cord segment because the spinal cord is arranged through separate reflex arcs. Absence of sensory abnormalities are seen when the lesion is entirely anterior horn. Lesions of the extrapyramidal system will produce bradykinesia with slowness of movement, tremor, shuffling walking with small steps, slow movements, resting tremor, and slowing of mentation as well as initiating movements. Posterior column syndromes involve ataxia, dysmetria with impossibility of coordinated smooth movements, overshooting the mark and increase in symptoms after elimination of vision. Syndromes of cerebellar abnormalities involve decomposition of movement, inability to perform movements smoothly, hypertonia, excessive rebound, and intention tremor.
Clinical skills in Neuro Evaluation

Learning Objectives

Upon completion of this course the learner will be able to:

- Describe the major components of the neurological exam.
- Identify 3 common neurological disorders tested during a neurological evaluation.
- Identify the major components of the motor exam.
- Describe the two sensory modalities tested using the sensory exam.
- Describe three common tests used to assess cerebellar dysfunction.

Introduction

The purpose of this course is to summarize the main parts of the neurological exam. Familiarity with this material will allow you to diagnose common neurological disorders, identify neurological emergencies and make referrals to appropriate specialists.

Any health professional faced with the task of assessing a person with an emergent, acute or even long-standing neurological deficit knows the importance of a quick and reliable neurological exam. Your knowledge of neurological evaluation techniques will allow you to gather accurate information about your client's medical condition and help you to create an accurate plan of care. Once you are familiar with the neurological exam, you should be able to complete the exam in 10 to 15 minutes. Additionally you will learn some of the tests that are used to identify certain types of common neurological dysfunctions. The course will cover the following parts of the neurological evaluation:

Index

- Tools
- Patient History
- Physical Exam
- Cognitive Assessment/Mental Status
- Cranial Nerve Assessment
- Motor Exam
- Sensory Exam
- Coordination Exam

Tools

The following tools will be used during the neurological exam:

- Reflex hammer (tomahawk model)
- Penlight
- Tongue blade
- Safety pin
Patient History

As with all other nursing examinations, the neurological exam begins with the gathering of an accurate patient history and information about the course of the present injury. This will help to create a baseline as well as providing you with valuable information about the course and characteristics of the present illness. The following information is gathered during the patient history portion of the neurological exam:

- Personal and family history
- Description of the current problem
- Past medical history
- Prior level of function
- Medication review
- Review of other major systems

Personal and family history.

The personal history should include a brief personal profile and description of the patient. A brief family history should be included, the source of the information indicated and the mental status of the patient noted. Included in this section are the following items:

- Date
- Age
- Gender
- Racial background
- Place of birth
- Marital status
- Occupation
- Religion

Description of the current problem.

A description of the current problem or "chief complaint" and the reason the patient is seeking medical care should be noted. Ask for an explanation of current signs and symptoms including any physical or psychological changes. Ask about the presence of dizziness, headaches, visual disturbances, speech or motor control problems. Inquire about the onset and duration of symptoms and remember that in an emergent neurological event the progression of symptoms may help to identify the part of the brain that has been affected. The following items are included in this section
• Present illness including onset of the problem, the setting it developed in, manifestations and past treatment for the problem.
• Analysis of the "main symptom" including location, quality, severity, onset, duration, frequency and factors that aggravate or alleviate the condition.

Past medical history.

Ask about the person's past medical history, previous illnesses and psychological history. Include educational background and any recent change in personality or behavior. Included in this section are the following items:

• Childhood illnesses
• Psychological illnesses
• Past accidents and injuries
• Operations
• Previous hospitalizations
• Current health
• Allergies
• Family history

Prior level of function.

Prior level of function is a critical piece of information that helps to establish the extent of the current neurological damage and helps you to differentiate between longstanding and emergent signs and symptoms. Ask about the person's level of daily activities and use of assistive devices prior to the onset of the current medical problem.

Medication review.

Ask the patient for a list of over-the-counter and prescription medications as well as use of recreational drugs and alcohol.

Review of other major systems.

Ask the patient about any problems with the other major systems of the body including heart, lungs and abdomen.

The Physical Exam

The physical exam includes inspection of the skin and neck, carotid and heart sounds, blood pressure, heart rate and respiratory rate. The following items are inspected in this section:

• Presence of weakness, fatigue or fever
• The condition of the skin - color, sores, rash, lumps
• Eyes - visual changes, spots, double vision, cataracts, blurred vision, glasses or contact lenses
• Ears - pain, tinnitus, vertigo, discharge or infection
• Nose and sinuses - presence of cold, stuffiness, discharge or bleeding
• Mouth and throat - general condition of the teeth and gums, sore throat, bleeding, sores, hoarseness or dryness
• Neck - presence of stiffness, pain, lumps or swollen glands
• Respiratory and cardiac systems
• Gastrointestinal urinary systems
• Genitals - presence of pain, discharge, swelling, sensory changes

Cognitive Assessment/Mental Exam

The mental exam starts when the patient enters the medical setting and includes your observations and conversations with the patient. If the patient demonstrates cognitive functioning that is grossly intact including level of consciousness, alertness, speech, memory and judgement, there is no need to do a more formal cognitive assessment. If it appears that any of the cognitive functions are impaired it will be necessary to do a more detailed cognitive assessment. The following items are included in the cognitive assessment:

• Level of consciousness
• Orientation
• Speech and language
• Memory
• Fund of information
• Insight and judgement
• Abstract thought
• Calculations

Level of consciousness.

There are many acceptable methods to determine level of consciousness including the Glasgow coma Scale, the Mini-Mental State or by categorizing the level of consciousness using descriptive cognitive scales.

Glasgow Coma Scale

A coma is defined by Jennett and Teasdale as "not obeying commands, not uttering words and not opening the eyes". The Glasgow Coma (table 1) scale was first developed in 1974 as a way to assess and monitor levels of consciousness. A Glasgow score of 8 or less out of a possible score of 15 defines coma in 90% of cases. The Glasgow Coma scale may be used in conjunction with the Glasgow Outcome scale (table 2) to determine level of recovery.
### Table 1: Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Score</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>To voice</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>No response</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>Best verbal response</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oriented, converses</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Disoriented, converses</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>No response or intubated</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>Best motor response</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follows commands</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Localizes response</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Withdraws</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Abnormal flexion</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Abnormal extension</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>No response</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Highest score = 15 Lowest score = 3

### Table 2: Glasgow Outcome Scale

- **Vegetative state**: No cerebral cortical function that can be judged by behavior
- **Severe disability**: Conscious but dependent
- **Moderate disability**: Independent but disabled
- **Good recovery**: Able to participate in normal social life and able to return to work

### Table 3: Glasgow Coma Scale Scores vs. Glasgow Outcome Scale

<table>
<thead>
<tr>
<th>Glasgow coma scale at 24 hours</th>
<th>Good recovery or moderate disability by %</th>
<th>Vegetative or dead by %</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-15</td>
<td>91%</td>
<td>6%</td>
</tr>
<tr>
<td>8-10</td>
<td>59%</td>
<td>27%</td>
</tr>
<tr>
<td>5-7</td>
<td>28%</td>
<td>54%</td>
</tr>
<tr>
<td>3-4</td>
<td>13%</td>
<td>80%</td>
</tr>
</tbody>
</table>

[Professor Yasser Metwally](www.yassermetwally.com)
Another scale used to descriptively assess the level of cognitive functioning in a person with a brain injury is as follows:

- **Alert**
- **Obtunded or confused** - a slight but noticeable decrease in alertness with decreased interest in what is happening in the environment, decreased attention span and memory.
- **Stupor** - the person appears to be in a deep sleep but can be aroused by noxious or vigorous stimuli.
- **Coma** - eyes closed, no directed motor or verbal activity and unarousable.

**Orientation.**

Orientation is generally determined by asking the patient to answer a few common questions such as the name, place and time. Other questions might include the year, date, day or the name of the president or vice president. Time is often the first part of orientation that is affected. The inability to remember one's name may be evidence of a psychiatric condition.

**Speech and language.**

A detailed assessment of speech and language function is the job of a specialist. During the initial neurological assessment, however you will be trying to establish the presence of a speech disorder that did not exist prior to the onset of the current medical problem. During this gross assessment, it is most common to look for the presence of aphasia, a problem with the understanding of speech or the inability to communicate via speech. Additionally, it is important to note the quality, clarity and fluency of speech. The following items are contained within a speech and language assessment:

- **Articulation** - look for difficulty with the pronunciation of words, especially words containing "p", "l" and "ch" sounds.
- **Rate and rhythm** - look for changes in the rate and rhythm of speech.
- **Prosody** - aprosodia occurs due to a lesion in the right parietal lobe - the part of the brain that is involved with the tone and musicality of speech. A patient with a lesion to this part of the brain will have flat intonation and a loss of pitch. There will also be a change in the accentuation and stress of words and syllables.
- **Aphasia** - aphasia is an acquired communication disorder often caused by vascular insult that affects a person's ability to speak and/or comprehend the spoken word. Aphasia can affect modalities other than speaking such as writing, gesturing and other non-verbal aspects of communication. For the purpose of a basic neurological exam it is sufficient to classify the aphasias as receptive, conductive or expressive.
Receptive (Wernicke's aphasia).

Speech is often clear and fluent and language is normal in rate, rhythm and melody but there may be errors in words as well as the presence of added syllables. Language may be excessive and convey little meaning. Comprehension is usually severely affected.

Conductive aphasia.

Speech is clear, but the patient is unable to repeat words. There is the ability to follow commands because comprehension is usually preserved. Naming and repeating is severely impaired. Reading aloud is impaired but reading silently is conserved. Speech is fluent but with many incorrect words or sounds substituted for correct words.

Expressive (Broca's aphasia).

Comprehension is usually well preserved but speech is unclear and non-fluent. Patients tend to use only key words and omit many nouns and verbs. Neurological damage may extend to the frontal lobe motor control areas adjoining Broca's area.

Memory.

The portion of the exam that tests memory skills is usually divided into three parts - immediate, recent and past memory.

1. Immediate - ask the patient to recall a few objects over the span of 3 to 5 minutes.
2. Recent - ask the patient to recall events within the last several hours to several days. Common questions might include, "What did you have for breakfast?" "Where do you live?" and "When did you start to feel ill?"
3. Past - ask about events from childhood or long ago events.

Fund of information.

Ask about current events, name of the president, geography, etc.

Insight and judgement.

Ask about the patient's understanding and awareness of the current illness.

Abstract thought.

Ask the patient to compare and contrast two objects such as a car and a bus or a cucumber and an apple. Ask the patient to interpret a complex concept or political event.
Calculations.

Ask the patient to do a calculation such as counting backwards from 100 by increments of 7 or count upwards by threes. Ask how many dimes are in a dollar or how many weeks in two years.

The Cranial Nerves

Assessment of the cranial nerves provides information about the function of the nerves in the head and neck region. With practice this part of the neurological exam can be completed in just a few minutes. Testing is usually done in numerical order starting with CNI and proceeding to CNXII. The cranial nerves are arranged along the brainstem in descending order from 12 to 1. (CN I is located just above the olfactory epithelium on the inferior surface of the frontal lobe. CN II is located on the inferior surface of the cerebrum behind the eyes.)

Cranial nerves 12 - 9 are located in the medulla oblongata, the part of the brainstem contiguous with the spinal cord. Cranial nerves 8 - 5 are located along the pons, the next portion of the brainstem. Cranial nerves 4 and 3 are located in the area of the midbrain, the uppermost portion of the brainstem.

CN I - The olfactory nerve.

The olfactory nerve is a sensory nerve responsible for smell.

Assessment of CNI is often omitted unless it is suspected that there is damage to the inferior frontal lobe - the area where the olfactory nerve is located. First make sure the nostrils are patent. CN I is usually tested by holding coffee, rubbing alcohol or some other pungent or aromatic substance under the nose of the patient. Compare one side to the other.

CN II - The optic nerve.

The optic nerve is a sensory nerve responsible for vision. Always test this cranial nerve because it will give you information about visual acuity and visual fields deficits. Test visual acuity, visual fields and fundi.

1. Visual Acuity. Use the eye chart from your toolkit and test the patient's corrected vision in good light. Have the patient stand 20 feet from the eye chart then read the smallest line possible. Test both eyes. Compare the results of this test to the patient's prior level of function.

2. Visual fields. It is important to test for the presence of visual field deficits if you suspect a disorder that is located in front of the optic chiasm. The visual fields are tested by positioning your finger or a pencil beside the patient's temple in the area of the peripheral vision. Slowly bring the object forward and ask the patient to say when the object becomes visible. Move the pencil or finger up, down, right, left and
diagonally to the upper right, lower right, upper left and lower left. Keep your movements small and slow - it is easier for the eye to detect motion and will make the test less sensitive to visual field defects. Common visual field deficits include: 1) homonymous hemianopsia in which 1/2 of the visual field on the same side is affected i.e. the nasal side of the right eye and the temporal side of the left eye, 2) bitemporal hemianopsia in which either the nasal side or the temporal sides of both eyes are affected, and 3) unilateral blindness in which one eye is blinded.

3. Fundi. Look closely at each eye and check for symmetry, clarity, color, contour, retinal abnormalities and the condition of the blood vessels in the eye.

**CN III, IV and VI - The oculomotor, trochlear and abducens nerves.**

The oculomotor, trochlear and abducens are motor nerves responsible for control of all eye movements and innervation of all the extraocular eye muscles. CN III controls most of the extraocular eye muscles, eye opening and pupillary constriction. CN IV controls downward and inward eye movements. CN VI controls lateral eye movements.

These cranial nerves are responsible for motor control of the eye muscles, eyelids and the pupils. They are tested in a group because they work together to control eye movement. Check the eyelids for drooping and symmetry. An eyelid drooping over the pupil (CN III) may indicate the presence of myasthenia gravis or 3rd cranial nerve palsy. Hold your finger in front of the patient and ask her to follow your finger as you move it through the 6 cardinal fields. The cardinal fields are: 1) lateral and medial along the horizontal plane and 2) superior and inferior in far lateral gaze.

Check pupil function for response to light. With the room darkened shine a penlight into one eye and look for pupil constriction - this is called a direct response. The opposite pupil should also constrict - this is known as a consensual response.

Next ask the patient to look at a distant object and then at your finger which is held about 4 inches in front of the patient's nose. The pupils should constrict and the eyes converge when the eyes shift from the distant object to your finger. Dilated or constricted pupils may indicate neurological disease, glaucoma, drug abuse or reaction to certain medications.

Finally check for the position of the eyes in primary gaze by having the patient look straight ahead, then shine a bright light into the pupils. The reflection of the light should be the same in each pupil. A deviation suggests the presence of a strabismus - the inability to align the visual axes so they are directed at the same point.

In addition to checking these functions remember to look for the presence of horizontal and vertical nystagmus. Have the patient hold a lateral gaze for 5 to 10 seconds. If a brainstem lesion is suspected, vertical nystagmus is often present and is almost always an indication of central nervous system damage as opposed to vestibular dysfunction.
**CN V - The trigeminal nerve.**

The trigeminal nerve is a mixed motor and sensory nerve that innervates the muscles of mastication and sensation from the skin, muscles and joints in the face, mouth and teeth and lateral jaw movement. Both motor and sensory components should be assessed.

The motor portion of the trigeminal nerve is tested by palpating the masseter muscles while the patient clenches the jaw and by testing the strength of the jaw while opening and closing the mouth. If a weakness is present the jaw will deviate to the weak side.

The sensory portion of the trigeminal nerve can be tested for temperature sensation using the metal surface of your tuning fork or using test tubes filled with warm and cold water.

The corneal reflex should be tested in a patient with suspected brainstem or hemispheric lesions by having the patient look up and away from the examiner. Approaching the eye from the side, lightly touch the cornea of one eye with a piece of cotton. Repeat in the other eye. A symmetric blink reflex should be present. Absence of the blink reflex indicates a 5th or 7th cranial nerve lesion.

**CN VII - The facial nerve.**

The facial nerve is mixed motor and sensory. The motor portion innervates the muscles of facial expression, the lacrimal and salivary glands. The sensory portion is responsible for taste sensation from the anterior 2/3rds of the tongue and the skin of the external ear.

The facial nerve can be tested by asking the patient to smile, wrinkle the forehead, puff out the cheeks and close the eyes tightly. Look for symmetry from side to side and differences between the upper two-thirds and the lower one-third of the face. Weakness in the upper part of the face may indicate a central nervous system lesion while weakness in the lower one third may indicate a peripheral nervous system problem.

**CN VIII - The vestibulocochlear nerve.**

The vestibulocochlear nerve is sensory for hearing, balance and orientation in space.

Cranial nerve VIII is responsible for two main functions - control of vestibular function and hearing. It is usually only tested in patients with symptoms of vertigo, imbalance or suspected lesions in the cerebellopontine angle or brainstem.

Hearing can be screened initially by asking whether of not the patient hears your questions. To test hearing acuity, cover one ear and test the other with a watch held close to the ear or by whispering.

The vestibular portion of CN VIII is more complicated but should be tested if the patient reports symptoms of nausea, vertigo, anxiety or if there are signs of nystagmus, sweating, hypotension, vomiting, hypotension or postural deviations. Use the caloric test in which the
patient is placed in supine with the head elevated to 30 degrees. Irrigate the ear canal with cold water. If the vestibular pathways are intact and the patient is awake the caloric test will cause nausea, horizontal nystagmus and vertigo to the irrigated side.

**CN IX and X - The glossopharyngeal and vagus nerves.**

The glossopharyngeal and vagus nerves are both mixed motor and sensory nerves. CN IX is involved with control of swallowing, sensation to the posterior 2/3rds of the tongue and innervation of the parotid gland. CN X innervates the smooth muscle of the heart, blood vessels, trachea, bronchi, esophagus, stomach and intestines. The motor portion innervates the muscles of the larynx, pharynx, palate and muscles of speech. The sensory portion innervates visceral sensation from the pharynx, larynx, thorax and abdomen.

These cranial nerves supply motor control to the larynx as well as sensory control to the tongue. Observe the position and symmetry of the palate at rest and as the patient pants of says "ahhh". Test the gag reflex by lightly touching the posterior wall of the pharynx on each side with a tongue blade - this is an indication of both glossopharyngeal and vagus function. In addition, listen for a soft breathy voice and ask if the patient has had difficulty with swallowing. Hoarse sounding speech may indicate vocal cord paralysis while nasal speech indicates paralysis of the palate.

**CN XI - The spinal accessory nerve.**

The spinal accessory nerve is a motor nerve that innervates the trapezius and sternocleidomastoid muscles.

To test the trapezius muscles, ask the patient to shrug the shoulders upward while the examiner applies a downward pressure. To test the sternocleidomastoid muscles, have the patient turn her head to the right while the examiner applies a gentle pressure in the opposite direction. During both of these tests look for symmetry of motion from side to side and palpate the muscles to look for atrophy, abnormal tone or fasciculations in the muscle. Also note the strength of the contractions.

Slow, alternating motion may indicate the presence of a central nervous system lesion while atrophy and fasciculations may indicate the presence of a peripheral nervous system lesion.

**CN XII - The hypoglossal nerve.**

The hypoglossal nerve is responsible for motor control of the intrinsic muscles of the tongue.

Check the position of the tongue within the mouth then look for smoothness of movement and symmetry as the patient protrudes the tongue and moves it from side to side.

**The Motor Exam**
The motor portion of the neurological exam includes observation of gross motor functions such as gait, extremity strength, reflexes, abnormal movement and abnormal tone - especially weakness and floppiness in the distal extremities. Begin by checking the general appearance of the patient - preferably with the clothes removed. Inspect the muscles visually and by palpation and note the presence of muscle fasciculations, abnormalities in muscle bulk and tenderness.

**Tone.**

The term muscle tone refers to the force with which a muscle resists lengthening. Tone is present because of two factors - the mechanical elasticity of the muscle fibers and a neural component called the stretch reflex. Both of these factors resist uncontrolled lengthening of the muscle and contribute to the natural tone of the muscle.

Tone is assessed clinically by passively flexing and extending the patient's limbs and noting the resistance of the muscles. Look for the presence of hypotonicity, hypertonicity, rigidity or spasticity. When checking for tone, make sure the patient is fully relaxed then take hold of the wrist and shake it back and forth. Raise the arm and drop it onto a soft surface. Grasp the ankle and shake it from side to side. Finally, with the patient in supine, raise the knee quickly and observe the movement of the ankle. Normally, when no abnormal tone is present, the foot will slide along the bed without lifting into the air.

**Strength.**

Muscle strength is usually assessed on a scale of 0-5 with 0 being no movement and 5 full movement against strong resistance. The following muscle groups are typically tested:

- Ask the patient to squeeze your pointer and middle fingers
- Elbow flexion
- Elbow extension
- Thumb opposition
- Hip flexion
- Knee flexion
- Knee extension
- Ankle dorsiflexion (L4, L5)
- Ankle plantarflexion (S1)
Use the following scale to rate strength:

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No movement, no contraction of the muscle</td>
</tr>
<tr>
<td>1</td>
<td>Trace, evidence of muscle contraction but no joint movement</td>
</tr>
<tr>
<td>2</td>
<td>Poor, complete range of motion with gravity eliminated</td>
</tr>
<tr>
<td>3</td>
<td>Fair, complete range of motion against gravity</td>
</tr>
<tr>
<td>4</td>
<td>Good, complete range of motion against gravity with moderate resistance</td>
</tr>
<tr>
<td>5</td>
<td>Normal, complete range of motion against gravity with maximal resistance without evidence of fatigue</td>
</tr>
</tbody>
</table>

**Neurological dysfunction.**

If neurological dysfunction is suspected it is more important to look for patterns of weakness and changes in tone rather than the strength of individual muscles. Depending upon the findings a more detailed strength exam may be needed. The general patterns to be aware of are as follows:

- Pyramidal weakness (corticospinal tract) - look for the presence of weakness in the arm extensors and leg flexors commonly seen in stroke.
- Proximal weakness - look for the presence of hip and shoulder musculature weakness commonly seen in muscular dystrophies.
- Distal weakness - look for the presence of weakness in the small muscles of the hands and feet commonly seen in peripheral neuropathies.

**Abnormal movements.**

During the motor portion of the neurological screen the examiner should note the presence of any of the following abnormal movement patterns:

- Bradykinesia - excessively slow movement or difficulty initiating movement. Bradykinesia is a common symptom of Parkinson's disease.
- Athetosis - large amplitude uncontrolled movements. Athetosis occurs because of damage to the basal ganglia (extrapyramidal system). It is common with certain types of cerebral palsy, head injury, stroke, long-term use of antipsychotic medications and other disorders that damage the basal structures of the brain.
- Chorea and ballism - smaller amplitude abrupt, uncontrolled movements. Also due to damage to the extrapyramidal system.
- Tremors - small amplitude movements that may be present at rest or with movement. A wide range of neurological disorders may exhibit some type of tremor including cerebral stroke, cerebellar stroke, Parkinson's disease, basal ganglia disorders as well as various drug reactions.
Reflexes

Two categories of reflexes are tested:

1. The stretch or deep tendon reflexes
2. The superficial or cutaneous reflexes

Stretch reflexes.

The stretch reflex is a type of spinal reflex also referred to as the deep tendon or myotatic reflex. Stretch reflexes trigger a contraction when the muscle is stretched or lengthened. Typically, the following stretch reflexes are tested with the patient in a seated or supine position:

- Biceps reflex (C5, C6)
- Triceps reflex (C6, C7)
- Brachioradialis reflex (C5, C6)
- Patellar reflex (L2,3,4)
- Achilles reflex (S1)
- Plantar or Babinski reflex (L5, S1)

The Babinski or plantar reflex is a non-specific reflex but is a useful screen for the presence of a central nervous system lesion. In a normal response there will be a downward flexing of the toes. If the great toe lifts and the other toes fan upward, the Babinski is positive and may indicate the presence of upper motor neuron damage. If the Babinski reflex is positive, a more thorough motor exam should be done.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No response</td>
</tr>
<tr>
<td>1+</td>
<td>Sluggish or diminished</td>
</tr>
<tr>
<td>2+</td>
<td>Active or expected response</td>
</tr>
<tr>
<td>3+</td>
<td>More brisk than expected, slightly hyperactive</td>
</tr>
<tr>
<td>4+</td>
<td>Brisk, hyperactive, with intermittent or transient clonus</td>
</tr>
</tbody>
</table>

A hyperactive reflex response suggests upper motor neuron damage while a decreased response suggest lower motor neuron, spinal damage or a disease of the neuromuscular junction or muscles.

Superficial reflexes.

The superficial reflexes are also referred to as cutaneous reflexes and include the following:
• Abdominal reflex. The abdominal reflex is tested by scratching the skin near the umbilicus in a diagonal manner. In a normal response the umbilicus will pull in the direction of the stimulation. Loss of this reflex may indicate a central nervous system lesion or an injury to the nerves that supply the umbilical region (T7 to L1).
• Cremasteric reflex. The cremasteric reflex is tested by stroking the skin along the inner thigh and looking for a lifting of the scrotum on the stimulated side.
• Anal wink. The anal wink is tested by stroking the perianal area and looking for a contraction of the anal sphincter.
• Pharyngeal or gag reflex. Test the gag reflex by touching the posterior wall of the pharynx with a tongue blade - this is an indication of both glossopharyngeal and vagus function.

Gait

Gait is the single most important part of the motor exam because it allows to examiner to assess muscle strength, coordination, balance and timing - all vital higher cortical functions. Gait is also a vital functional skill and gives the examiner an idea of the ability of the patient to perform a complex motor task.

Ask the patient to walk down a hallway. Observe for symmetry, rhythm and speed while walking. Look for limping, scissoring, staggering, weight bearing and foot clearance during the swing phase. Ask the patient to walk on the heels and then on the toes - these are good tests for peroneal/tibialis and gastrocnemius muscle function. Observe the person walking toe to heel and note any abnormalities with balance and coordination during this task. If the patient has difficulty with any of these tasks involving gait or balance a more thorough balance evaluation should be done by a physical therapist.

Note any of the following common gait disorders:

• Ataxic gait - a possible indication of cerebellar dysfunction.
• Festinating gait - a possible sign of Parkinson's disease.
• Hemiplegic gait with one-sided weakness - a possible sign of cerebral stroke.
• Spastic gait - a possible sign of cerebral palsy.

The Sensory Exam

The sensory portion of the neurological exam is very useful if the patient is cooperative and alert and able to give accurate responses to your questions. If the patient is uncooperative or unreliable the sensory exam is often skipped. In general keep the following principles in mind during the sensory portion of the exam:

• Compare distal sensation to proximal sensation

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• Look for symmetry by comparing one side to the other
• Test each of the dermatomes by moving the stimulation

The examiner will need a standard dermatome chart to accurately map sensory function. Begin by applying a light sensation to one side of the body, then compare to the other side. Gradually increase the intensity of the stimulation. The exam should focus on the suspected lesion. For example if a central nervous system lesion is suspected the tests should be directed towards central nervous system sensory tests. Sensory testing is usually divided into two parts:

1. Primary sensory modalities such as pain, light touch, temperature, vibration and joint position sense.
2. Cortical sensory modalities such as stereognosis, two-point discrimination, graphesthesia and double simultaneous stimulation.

Primary sensory modalities.

The purpose of this part of the neurological exam is to test the sensory pathways that ascend via the spinal cord from the periphery to the sensory processing centers of the brain. The following sensory modalities are commonly tested:

• Pain - have the patient identify a sharp or dull sensation by alternating the point of a sterile needle with the end of a blunt object such as the rounded end of a paper clip. The patient should be able to differentiate between sharp and dull.
• Light touch - lightly touch an area of the skin with your finger or a small piece of cotton. The patient should be able to identify the location of the touch.
• Vibration - place the stem of the tuning fork on the bony prominence of the ankle, shin, wrist, elbow, shoulder and sternum. The patient should be able to name the location of the vibration. Loss of vibration sense suggests the presence of a peripheral neuropathy.
• Joint position sense - hold the joint lightly in a neutral position between your two fingers. Raise or lower the digit or extremity and ask the patient to identify the direction of the movement.

Cortical sensory modalities.

Cortical sensory input is processed by the parietal lobes. Processing of sensory input at the cortical level involves some degree of cognitive processing and control. Because the parietal lobes receive their sensory input from the primary sensory modalities, cortical modalities should be tested only if primary modalities are intact. The following sensory modalities should be tested:

• Stereognosis - ask the patient to identify an object placed in the hand. Compare the results of one hand to the other.
• Two-point discrimination - the ability to identify two closely placed stimuli. Stimulate the skin using an object with two points and ask the patient to identify the
when two points of stimulus are felt versus one point of stimulus. Different parts of the body will have different thresholds for two-point discrimination.

- Double simultaneous discrimination - with the eyes closed, touch one or both hands and have the patient identify the location of the stimulation. If a parietal lobe lesion is present the patient will be unable to identify simultaneous stimulation on the contralateral side of the body.

<table>
<thead>
<tr>
<th>Body part</th>
<th>Minimal distance (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tongue</td>
<td>1 mm</td>
</tr>
<tr>
<td>Fingertips</td>
<td>2-8 mm</td>
</tr>
<tr>
<td>Toes</td>
<td>3-8 mm</td>
</tr>
<tr>
<td>Palms of hands</td>
<td>8-12 mm</td>
</tr>
<tr>
<td>Chest and forearms</td>
<td>40 mm</td>
</tr>
<tr>
<td>Back</td>
<td>40 to 70 mm</td>
</tr>
<tr>
<td>Upper arms and thighs</td>
<td>75 mm</td>
</tr>
</tbody>
</table>

**Coordination**

Coordination refers to the ability of the nervous system to organize multiple systems into organized and efficient patterns of movement. Coordination is affected by a variety of medical and physiologic factors including medical condition, medications, strength, alignment, timing and scaling of movements. Alignment refers to the arrangement of body segments on one another and is affected by both musculoskeletal and neurological factors. Timing is the ability to apply force with the appropriate speed and precision to prevent loss of balance or dyscoordination. Scaling a movement properly means that the force output of the muscles is appropriate to the amplitude of the instability.

The following items are commonly assessed to determine if there are problems with coordination:

- Observe the patient at rest and note the presence of abnormal postures, tremor, chorea, athetosis or dystonia.
- Have the patient hold both arms outstretched with eyes closed and note any abnormal movement such as tremor, weakness or posturing.
- Have the patient perform a simple functional task such as buttoning a shirt or writing and observe the smoothness and rhythm of the movement.
- Have the patient move from sit to stand without use of the hands and observe for postural instability and loss of balance.

Finally, a series of simple coordination tests should be performed to evaluate cerebellar dysfunction:
• Finger tap - tap the index finger against the thumb or a firm surface. Look for smoothness of movement.
• Finger to nose - ask the patient to touch her nose and then touch the examiner's outstretched finger. Observe the movement for accuracy, smoothness and presence of tremor or oscillations.
• Rapid alternating hand movement - have the patient pronate and supinate her hands as quickly as possible. Observe the rhythm and accuracy of the movement as well as the patient's ability to stop the movement and change direction.
• Heel to shin test - while in sitting ask the patient to run the heel of one foot up and down along the shin of the opposite leg. The patient should be able to perform this movement smoothly and repeat it easily.

Conclusion

Completion of a neurological evaluation on a person with a suspected neurological injury will give you a picture of the extent and possible location of the nervous system damage. This is especially important with sudden onset or life-threatening diseases in which the outcome is dependent on an accurate referral or timely treatment. With practice you will refine your evaluation skills and find that you will be able to complete the neurological exam quickly and efficiently.

References