NEUROLOGICAL EXAMINATION
Professor Yasser Metwally

www.yassermetwally.com
Preface

Thank you for using my publication, this publication is directed to undergraduate students and neurologist in training. Its primary aim is how to elicit neurological signs and symptoms and how to group these signs and symptoms into a single clinical diagnosis for each patient. This publication is simple a guide and not a substitute for clinical experience. Postgraduate neurologists get their information from the patients they attend and not from papers, for them patients are the primary source of information and from them, they get their clinical experience. I hope you will find this publication as useful as I truly wish. This publication is free of charge and can be distributed on the following conditions

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Professor Yasser Metwally

Professor of neurology, Ain Shams University

Cairo, Egypt

www.yassermetwally.com

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INDEX

1-A guide to neurological examination
2-Skills of eliciting clinical neurological signs
3-Neurological examination slides
4-Papilledema versus papillitis
5-Fundus examination

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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 circumscript 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 1 - The Glasgow Coma Score (GCS)**

<table>
<thead>
<tr>
<th>Eye opening</th>
<th>Spontaneous (4)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>To speech (3)</td>
</tr>
<tr>
<td></td>
<td>To pain (2)</td>
</tr>
<tr>
<td></td>
<td>Remain closed (1)</td>
</tr>
<tr>
<td>Best verbal response</td>
<td>Oriented (5)</td>
</tr>
<tr>
<td></td>
<td>Confused (4)</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words (3)</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible words (2)</td>
</tr>
<tr>
<td></td>
<td>Mute (1)</td>
</tr>
<tr>
<td>Best motor response</td>
<td>Obeying commands (6)</td>
</tr>
<tr>
<td></td>
<td>Localizing pain (5)</td>
</tr>
<tr>
<td></td>
<td>Quick withdrawal to pain (4)</td>
</tr>
<tr>
<td></td>
<td>Flexion (coordinated movement to the chest; decorticate) to pain (3)</td>
</tr>
<tr>
<td></td>
<td>Extension (endo rotation and stiffening; decerebrate) to pain (2)</td>
</tr>
<tr>
<td></td>
<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>
<thead>
<tr>
<th>Table 2 - Mental Status Examination (MSE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. <strong>Orientation:</strong> Patient needs to state her full name, address, building, city, and state and current date.</td>
</tr>
<tr>
<td>2. <strong>Attention:</strong> is tested by giving a patient a series of numbers. The patient needs to repeat seven digits forward, usually using a full phone number.</td>
</tr>
<tr>
<td>3. <strong>Learning:</strong> the patient is asked to repeat and memorize four words that are unconnected to each other, such as, apple, shoe, crying, and Mr. Murphy.</td>
</tr>
<tr>
<td>4. <strong>Calculation:</strong> is tested by subtracting 7 from 100 including other arithmetic problems. For example, asking to multiply 5 x 13 or add 11 and 29.</td>
</tr>
<tr>
<td>5. <strong>Abstraction:</strong> 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.</td>
</tr>
<tr>
<td>6. <strong>Judgment:</strong> can also be investigated by asking the patient what to do if he would see a person collapse while crossing the street.</td>
</tr>
<tr>
<td>7. <strong>Construction:</strong> is tested by having a patient draw a cube and a clock showing the hands directed at a certain time.</td>
</tr>
</tbody>
</table>

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 3 – Aphasia**

<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 3 – Dysarthria**

<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 odored 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/Y 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 be 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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<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>
</tr>
<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>
</tr>
<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 findings. 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 indicate 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)

• **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, pronunciation 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 tympani. 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.

• 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.
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>

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 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>Region</td>
<td>Nerve Distribution</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>
</tbody>
</table>
| C8 - T1 | Thenar (median nerve)  
           | Hypothenar (ulnar nerve)  
           | Interossei (ulnar nerve) |
| L2-3-4 | Iliopsoas (femoral nerve)  
           | Adductor thigh (obturator nerve) |
| L4-5 - S1 | Hamstrings (sciatic nerve)  
           | Toe extensors (peroneus nerve) |
| L2-3-4 | Quadriceps (femoral nerve) |
| L4-5  | Anterior tibial (peroneal nerve) |
| L5 - S1 | Extensor hallucis longus (peroneal nerve)  
           | Peronei (peroneal nerve)  
           | Posterior tibial (tibial nerve)  
           | Toe flexors (tibial nerve) |
| L5 - S1 | Gluteus maximus (inferior gluteal nerve)  
           | Gastrocnemius / Soleus (tibial nerve) |
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 (Gastrocnemius). 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 Ulner 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). Brachioradialis 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 maneuver), 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.
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 his 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>Type</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebellar ataxia</td>
<td>Wide based, staggering steps, leans forward. Abnormal turning</td>
</tr>
<tr>
<td>Sensory ataxia</td>
<td>Romberg (+); wide based; high stepping.</td>
</tr>
<tr>
<td>Frontal lobe ataxia</td>
<td>Hesitating start, shuffling, freezing with corners.</td>
</tr>
<tr>
<td>Spastic</td>
<td>Bouncing wide - based, “tin-man”-like</td>
</tr>
<tr>
<td>Akinetic</td>
<td>Shuffling, no arm swing, small steps</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

<table>
<thead>
<tr>
<th>Steppage Gait</th>
<th>Hemiplegic Gait</th>
<th>Parkinsonian Gait</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retropulsion</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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 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>Eye opening</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>Best verbal response</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>Best motor response</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

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetative state</td>
<td>No cerebral cortical function that can be judged by behavior</td>
</tr>
<tr>
<td>Severe disability</td>
<td>Conscious but dependent</td>
</tr>
<tr>
<td>Moderate disability</td>
<td>Independent but disabled</td>
</tr>
<tr>
<td>Good recovery</td>
<td>Able to participate in normal social life and able to return to work</td>
</tr>
</tbody>
</table>

### 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>

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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>Scoring of Deep Tendon Reflexes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>0</td>
</tr>
<tr>
<td>1+</td>
</tr>
<tr>
<td>2+</td>
</tr>
<tr>
<td>3+</td>
</tr>
<tr>
<td>4+</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:

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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 examine 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 goods 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
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>Minimal Distances for Two Point Discrimination by Location</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Body part</strong></td>
</tr>
<tr>
<td>Tongue</td>
</tr>
<tr>
<td>Fingertips</td>
</tr>
<tr>
<td>Toes</td>
</tr>
<tr>
<td>Palms of hands</td>
</tr>
<tr>
<td>Chest and forearms</td>
</tr>
<tr>
<td>Back</td>
</tr>
<tr>
<td>Upper arms and thighs</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


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Index

- Equipment Needed
- General Considerations
- Mental Status
- Cranial Nerves
- Observation
  - I - Olfactory
  - II - Optic
  - III - Oculomotor
  - IV - Trochlear
  - V - Trigeminal
  - VI - Abducens
  - VII - Facial
  - VIII - Acoustic
  - IX - Glossopharyngeal
  - X - Vagus
  - XI - Accessory
  - XII - Hypoglossal
- Motor
  - Observation
  - Muscle Tone
  - Muscle Strength
  - Pronator Drift
- Coordination and Gait
  - Rapid Alternating Movements
  - Point-to-Point Movements
  - Romberg
  - Gait
- Reflexes
  - Deep Tendon Reflexes
  - Clonus
  - Plantar Response (Babinski)
- Sensory
  - General
  - Vibration
  - Subjective Light Touch
  - Position Sense
  - Dermatomal Testing
  - Pain
  - Temperature
  - Light Touch
  - Discrimination
- Meningeal signs
- Decerebrate & Decorticate posturing
- Notes

Click to download the publication "CNS examination" in PDF format
General Considerations

- Always consider left to right symmetry
- Consider central vs. peripheral deficits
- Organize your thinking into seven categories:
  1. Mental Status
  2. Cranial Nerves
  3. Motor
  4. Coordination and Gait
  5. Reflexes
  6. Sensory
  7. Special Tests

Mental Status

The [Mini Mental Status Examination](#) is a useful screening tool.

Cranial Nerves

Observation

- Ptosis (III)
- Facial Droop or Asymmetry (VII)
- Hoarse Voice (X)
- Articulation of Words (V, VII, X, XII)
- Abnormal Eye Position (III, IV, VI)
- Abnormal or Asymmetrical Pupils (II, III)

**I - Olfactory [1]**

Evaluate the patency of the nasal passages bilaterally by asking the patient to breathe in through their nose while the examiner occludes one nostril at a time. Once patency is established, ask the patient to close their eyes. Occlude one nostril, and place a small bar of soap near the patent nostril and ask the patient to smell the object and report what it is. Making certain the patient's eyes remain closed. Switch nostrils and repeat. Furthermore, ask the patient to compare the strength of the smell in each nostril.
II - Optic

- Examine the Optic Fundi

- Test Visual Acuity
  1. Allow the patient to use their glasses or contact lens if available. You are interested in the patient's best corrected vision.
  2. Position the patient 20 feet in front of the Snellen eye chart (or hold a Rosenbaum pocket card at a 14 inch "reading" distance).
  3. Have the patient cover one eye at a time with a card.
  4. Ask the patient to read progressively smaller letters until they can go no further.
  5. Record the smallest line the patient read successfully (20/20, 20/30, etc.) [2]
  6. Repeat with the other eye.

- Screen Visual Fields by Confrontation
  1. Stand two feet in front of the patient and have them look into your eyes.
  2. Hold your hands about one foot away from the patient's ears, and wiggle a finger on one hand. [3]
  3. Ask the patient to indicate which side they see the finger move.
  4. Repeat two or three times to test both temporal fields.
  5. If an abnormality is suspected, test the four quadrants of each eye while asking the patient to cover the opposite eye with a card. [4]

- Test Pupillary Reactions to Light
  1. Dim the room lights as necessary.
  2. Ask the patient to look into the distance.
  3. Shine a bright light obliquely into each pupil in turn.
  4. Look for both the direct (same eye) and consensual (other eye) reactions.
  5. Record pupil size in mm and any asymmetry or irregularity.
  6. If abnormal, proceed with the test for accommodation.

- Test Pupillary Reactions to Accommodation [5] [4]
  1. Hold your finger about 10cm from the patient's nose.
  2. Ask them to alternate looking into the distance and at your finger.
  3. Observe the pupillary response in each eye.

III - Oculomotor

- Observe for Ptosis

The olfactory nerve is part of our ability to smell. Loss of the sense of smell is called anosmia. Most patients with anosmia can still smell harsher smells (sweet and sour) but have difficulty with flavors like cinnamon and peppermint. Patients with anosmia often complain that they’ve lost their sense of taste. Much of the pleasure derived from eating is due to smell, not taste (think of sniffing a glass of fine wine before drinking it). There are many causes for anosmia:

1. Trauma
2. Surgery
3. Masses affecting the orbitofrontal region or cribiform plate
4. Destruction of the neuroepithelium due to inflammation, as in chronic rhinitis or viral infection
- Test Extraocular Movements
  1. Stand or sit 3 to 6 feet in front of the patient.
  2. Ask the patient to follow your finger with their eyes without moving their head.
  3. Check gaze in the six cardinal directions using a cross or "H" pattern.
  4. Pause during upward and lateral gaze to check for nystagmus. [6]
  5. Check convergence by moving your finger toward the bridge of the patient's nose.

- Test Pupillary Reactions to Light (See Above)

**Examination of ocular motility**

1. Observe for ptosis.

2. **Test Extraocular Movements**

Stand or sit 3 to 6 feet in front of the patient. Ask the patient to follow your finger with their eyes without moving their head. Check gaze in the six cardinal directions using a cross or "H" pattern. Pause during upward and lateral gaze to check for nystagmus.

1. Check convergence by moving your finger toward the bridge of the patient's nose.

IV - Trochlear

Test Extraocular Movements (Inward and Down Movement, See Above)

V - Trigeminal  
(Click for an online video)

- Test Temporalis and Masseter Muscle Strength
  1. Ask patient to both open their mouth and clench their teeth.
  2. Palpate the temporal and masseter muscles as they do this.

- Test the Three Divisions for Pain Sensation
  1. Explain what you intend to do.
  2. Use a suitable sharp object to test the forehead, cheeks, and jaw on both sides. [7]
  3. Substitute a blunt object occasionally and ask the patient to report "sharp" or "dull."

- If you find and abnormality then:
  1. Test the three divisions for temperature sensation with a tuning fork heated or cooled by water. ++
  2. Test the three divisions for sensation to light touch using a wisp of cotton. ++

- Test the Corneal Reflex ++
  1. Ask the patient to look up and away.
  2. From the other side, touch the cornea lightly with a fine wisp of cotton.
  3. Look for the normal blink reaction of both eyes.
  4. Repeat on the other side.
  5. Use of contact lens may decrease this response.

**Examination of the motor part of the trigeminal nerve**

1. **Test Temporalis and Masseter Muscle Strength**

2. **Ask the patient to open their mouth and clench their teeth.**

3. **Palpate the temporalis and masseter muscles as they do this.**
Table 1. Examination of the sensory part of the trigeminal nerve

- **Test the Three Divisions for Pain Sensation**
  - Explain what you intend to do then ask the patient to close their eyes.
  - Use a clean, slightly sharp, disposable object to test the forehead, cheeks, and jaw on both sides.
  - Substitute a blunt object (cotton swab) occasionally and ask the patient to report "sharp" or "dull."
  - The ophthalmic, maxillary, and mandibular divisions of the fifth cranial nerve are usually denoted as V1, V2, and V3.
  - Test for Temperature and Light Touch
  - If an abnormality is suspected, proceed with more detailed testing.
  - Test the three divisions for temperature sensation with a tuning fork heated or cooled by water.
  - Test the three divisions for sensation to light touch using a wisp of cotton.

- **Test the Corneal Reflex**
  - The Corneal Reflex is not necessary unless an abnormality of the trigeminal (V) or facial (VII) nerve is suspected.
  - Remove contact lenses, if present, as these may decrease this response.
  - Ask the patient to look up and away.
  - From the other side, touch the cornea lightly with a fine wisp of cotton.
  - Look for the normal blink reaction of both eyes.
  - Repeat on the other side.

VI - Abducens **Click for online video**
Test Extraocular Movements (Lateral Movement, See Above)

VII - Facial **Click for the online topic "examination of the facial nerve**

- Observe for Any Facial Droop or Asymmetry
- Ask Patient to do the following, note any lag, weakness, or asymmetry:
  1. Raise eyebrows [8]
  2. Close both eyes to resistance
  3. Smile
4. Frown
5. Show teeth
6. Puff out cheeks
- Test the Corneal Reflex (See Above) ++

**Figure 3.** A, Examination of the facial nerve. B,C, Facial palsy

**Figure 4.** Intracranial facial nerve connection

### Table 2. Principle of facial nerve examination

Observe for any facial droop or asymmetry.

Ask the patient to do a few of the following. (It is not necessary to do them all.):
- Raise eyebrows
- Close both eyes to resistance
- Smile
- Frown
- Show teeth
VIII - Acoustic

- **Screen Hearing** [9]
  1. Face the patient and hold out your arms with your fingers near each ear.
  2. Rub your fingers together on one side while moving the fingers noiselessly on the other.
  3. Ask the patient to tell you when and on which side they hear the rubbing.
  4. Increase intensity as needed and note any asymmetry.
  5. If abnormal, proceed with the Weber and Rinne tests.

- **Test for Lateralization (Weber)** ++
  1. Use a 512 Hz or 1024 Hz tuning fork.
  2. Start the fork vibrating by tapping it on your opposite hand.
  3. Place the base of the tuning fork firmly on top of the patient's head.
  4. Ask the patient where the sound appears to be coming from (normally in the midline).

- **Compare Air and Bone Conduction (Rinne)** ++
  1. Use a 512 Hz or 1024 Hz tuning fork.
  2. Start the fork vibrating by tapping it on your opposite hand.
  3. Place the base of the tuning fork against the mastoid bone behind the ear.
  4. When the patient no longer hears the sound, hold the end of the fork near the patient's ear (air conduction is normally greater than bone conduction).

- **Vestibular Function:** Use the past pointing test

---

**The Corneal Reflex** is not necessary unless an abnormality of the trigeminal (V) or facial (VII) nerve is suspected. Tease out a fine wisp from the end of a cotton ball or swab. Warn the conscious patient what you are about to do. If necessary, hold the patient's eyelid open to expose the cornea. Touch the cornea with the wisp by approaching from the side and avoiding the area of central vision. The patient should spontaneously shut both eyes in response to corneal stimulation. This is a monosynaptic reflex between the sensation to the cornea provided by the ophthalmic nerve (V1) and the muscles of the eyelids, innervated by the facial nerve (VII).

With CN VII recall that the type of finding relates to the presence of a central versus a peripheral nervous system lesion. With a unilateral central nervous system lesion (e.g., stroke), recall this would involve the corticobulbar pathway. Function is preserved over the upper part of the face (forehead, eyebrow, eyelid). If the lesion involves the peripheral seventh nerve (Bell's palsy), the entire face is involved.

---

![Figure 5. Examination of the acoustic nerve](image)

![Figure 6. A, Rinne test, B, Weber](image)
Table 3. Principle of Rinne test and Weber tests

### Compare Air and Bone Conduction (Rinne)

1. Start with a 512 Hz tuning fork vibrating by tapping it on your hand.
2. Place the base of the tuning fork against the mastoid process on one side.
3. Ask the patient to tell you when the sound goes away.
4. When the patient no longer hears the sound, bring the end of the tuning fork near the patient's ear.
5. Air conduction is normally greater than bone conduction so they should hear the sound again for several more seconds.

### Test for Lateralization (Weber)

1. Start with a 512 Hz tuning fork vibrating by tapping it on your hand.
2. Place the base of the tuning fork firmly in the center on top of the patient's head.
3. Ask the patient where they hear the sound.
4. They should hear the sound "in the center" if they have normal hearing.

The Weber and Rinne tests are used to differentiate conductive from sensorineural hearing loss.

- **Conductive Hearing Loss (Middle Ear Deafness)**

   The finger rubbing test is abnormal on the affected side. The Rinne test will indicate bone conduction better than air on that side. The sound is transmitted to the cochlea through bone rather than through the middle ear. Paradoxically the Weber test will lateralize to the affected ear. This is because sounds arriving via bone conduction appear louder when air conduction is decreased (masking effect).

   Etiology: Usually due to a structural defect (blocked canal, trauma, scarred or torn tympanic membrane. Otosclerosis occurs when the stapes bone in the middle ear is scared and immobile inhibiting the transfer of sound vibrations.

- **Sensorineural Hearing Loss**

   The finger rubbing test is abnormal on the affected side. The Rinne test would indicate that air conduction better than bone (normal if heard at all). The Weber test would lateralize to the unaffected ear. Due to the damage to the auditory neural pathway neither bone or air will conduct well and for this reason the sound lateralizes to the unaffected (better) ear.

   Etiology: Caused by damage to the nerve transmitting the sound (VIII) or its associated neurons and receptors (hair cells). This can be caused by tumors (acoustic neuromas, meningiomas), stroke, trauma, children born to mothers who had Rubella during pregnancy, aminoglycoside toxicity, atrophy of the cochlea seen in aging and various genetic causes.

### IX - Glossopharyngeal

See Vagus Nerve

### X - Vagus

**Click for online video clips**

- Listen to the patient's voice, is it hoarse or nasal?
- Ask Patient to Swallow
- Ask Patient to Say "Ah"
  - Watch the movements of the soft palate and the pharynx.
- Test Gag Reflex (Unconscious/Uncooperative Patient) ++
  1. Stimulate the back of the throat on each side.
  2. It is normal to gag after each stimulus.

**Principle of vagus nerve examination**

- Listen to the patient's voice, is it hoarse or nasal?
- Ask the patient to swallow.
- Ask the patient to say "Ah".
  - Do the soft palate and the other pharyngeal structures move? Is the movement symmetrical?

**Gag Reflex**

- It is usually not necessary to perform this test on a conscious, cooperative patient. It is very useful in cases of coma.
- Stimulate the back of the throat on each side.
- It is normal to gag after each stimulus.
- This reflex protects the lungs from food and liquid contamination. A diminished gag reflex greatly increases the risk of aspiration.

**XI - Accessory**

- From behind, look for atrophy or asymmetry of the trapezius muscles.
- Ask patient to shrug shoulders against resistance.
- Ask patient to turn their head against resistance. Watch and palpate the sternomastoid muscle on the opposite side.

**Examination of the accessory nerve**

- From behind, look for atrophy or asymmetry of the trapezius muscle.
- Ask the patient to shrug their shoulders against resistance.
- Ask the patient to turn their head against resistance. Watch and palpate the sternocleidomastoid muscle on the opposite side.

**XII - Hypoglossal**

- Listen to the articulation of the patient's words.
- Observe the tongue as it lies in the mouth
- Ask patient to:
  1. Protrude tongue [10]
  2. Move tongue from side to side

**Examination of the hypoglossal nerve**

1. Listen to the articulation of words as the patient speaks.

**Click for an online video**
Motor

Observation

- Involuntary Movements
- Muscle Symmetry
  - Left to Right
  - Proximal vs. Distal
- Atrophy
  - Pay particular attention to the hands, shoulders, and thighs.
- Gait

Muscle Tone

1. Ask the patient to relax.
2. Flex and extend the patient's fingers, wrist, and elbow.
3. Flex and extend patient's ankle and knee.
4. There is normally a small, continuous resistance to passive movement.
5. Observe for decreased (flaccid) or increased (rigid/spastic) tone.

Muscle Strength

- Test strength by having the patient move against your resistance.
- Always compare one side to the other.
- Grade strength on a scale from 0 to 5 "out of five":

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0/5</td>
<td>No muscle movement</td>
</tr>
<tr>
<td>1/5</td>
<td>Visible muscle movement, but no movement at the joint</td>
</tr>
<tr>
<td>2/5</td>
<td>Movement at the joint, but not against gravity</td>
</tr>
<tr>
<td>3/5</td>
<td>Movement against gravity, but not against added resistance</td>
</tr>
<tr>
<td>4/5</td>
<td>Movement against resistance, but less than normal</td>
</tr>
<tr>
<td>5/5</td>
<td>Normal strength</td>
</tr>
</tbody>
</table>

Test the following:
1. Flexion at the elbow (C5, C6, biceps)
2. Extension at the elbow (C6, C7, C8, triceps)
3. Extension at the wrist (C6, C7, C8, radial nerve)
4. Squeeze two of your fingers as hard as possible ("grip," C7, C8, T1) [10]
5. Finger abduction (C8, T1, ulnar nerve)
6. Opposition of the thumb (C8, T1, median nerve)
7. Flexion at the hip (L2, L3, L4, iliopsoas)
8. Adduction at the hips (L2, L3, L4, adductors)
9. Abduction at the hips (L4, L5, S1, gluteus medius and minimus)
10. Extension at the hips (S1, gluteus maximus) [12]
11. Extension at the knee (L2, L3, L4, quadriceps) [10]
12. Flexion at the knee (L4, L5, S1, S2, hamstrings)
13. Dorsiflexion at the ankle (L4, L5)
14. Plantar flexion (S1) [12]

Table 4.

<table>
<thead>
<tr>
<th>Muscle Action</th>
<th>Major Muscles Involved</th>
<th>Nerves/Roots Involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip flexion</td>
<td>Iliopsoas</td>
<td>L2-L4</td>
</tr>
<tr>
<td>Hip extension</td>
<td>Gluteus maximus</td>
<td>S1</td>
</tr>
<tr>
<td>Hip adduction</td>
<td>Adductors</td>
<td>L2-L4</td>
</tr>
<tr>
<td>Hip abduction</td>
<td>Gluteus medius and minimus</td>
<td>L4, L5, S1</td>
</tr>
<tr>
<td>Knee extension</td>
<td>Quadriceps</td>
<td>L2-L4</td>
</tr>
<tr>
<td>Knee flexion</td>
<td>Hamstrings</td>
<td>L4, L5, S1, S2,</td>
</tr>
<tr>
<td>Ankle dorsiflexion</td>
<td>Anterior and lateral leg muscles</td>
<td>L4, L5</td>
</tr>
</tbody>
</table>

Pronator Drift

1. Ask the patient to stand for 20-30 seconds with both arms straight forward, palms up, and eyes closed.
2. Instruct the patient to keep the arms still while you tap them briskly downward.
3. The patient will not be able to maintain extension and supination (and "drift into pronation") with upper motor neuron disease.

Coordination and Gait

Rapid Alternating Movements

1. Ask the patient to strike one hand on the thigh, raise the hand, turn it over, and then strike it back down as fast as possible.
2. Ask the patient to tap the distal thumb with the tip of the index finger as fast as possible.
3. Ask the patient to tap your hand with the ball of each foot as fast as possible.

Point-to-Point Movements  Click for the video-enriched online topic "Classification of tremors"

1. Ask the patient to touch your index finger and their nose alternately several times. Move your finger about as the patient performs this task.
2. Hold your finger still so that the patient can touch it with one arm and finger outstretched. Ask the patient to move their arm and return to your finger with their eyes closed.
3. Ask the patient to place one heel on the opposite knee and run it down the shin to the big toe. Repeat with the patient's eyes closed.

Finger to Nose  Click for online video Click for online video

- Ask the patient to touch your index finger and their nose alternately several times.
- Move your finger to a new position each time the patient returns to their nose.
- Hold your finger still so that the patient can touch it with one arm and finger outstretched.
- With their eyes closed, ask the patient to touch their nose and return to your finger twice.

Heel to Shin
Romberg

1. Be prepared to catch the patient if they are unstable.

2. Ask the patient to stand with the feet together and eyes closed for 5-10 seconds without support.

3. The test is said to be positive if the patient becomes unstable (indicating a vestibular or proprioceptive problem).

Rapid alternating movement is used to assess cerebellar function in the upper and lower extremities.

- Ask the patient to strike one hand on the thigh, raise the hand, turn it over, and then strike it back down as fast as possible.
- Ask the patient to tap their thumb with the tip of the index finger as fast as possible.
- Ask the patient to tap your hand with the ball of each foot as fast as possible.
- Slow or uncoordinated alternating movement is known as dysdiadochokinesis.

Romberg Test

- Be prepared to catch the patient if they are unstable.
- First, test when the patient's eyes are open to get a general sense of balance and proprioception.
- Ask the patient to stand with their feet together and eyes closed for five to ten seconds without support.
- The test is said to be positive if the patient becomes unstable (indicating a vestibular or proprioceptive problem).

Gait

(Click for an online video clip) (Click for an online video clip) (Click for an online video clip)

Ask the patient to:

1. Walk across the room, turn and come back
2. Walk heel-to-toe in a straight line
3. Walk on their toes in a straight line
4. Walk on their heels in a straight line
5. Hop in place on each foot
6. Do a shallow knee bend
7. Rise from a sitting position

Reflexes

Deep Tendon Reflexes

- The patient must be relaxed and positioned properly before starting.
Reflex response depends on the force of your stimulus. Use no more force than you need to provoke a definite response.
Reflexes can be reinforced by having the patient perform isometric contraction of other muscles (clenched teeth).
Reflexes should be graded on a 0 to 4 "plus" scale:

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Absent</td>
</tr>
<tr>
<td>1+ or +</td>
<td>Hypoactive</td>
</tr>
<tr>
<td>2+ or ++</td>
<td>&quot;Normal&quot;</td>
</tr>
<tr>
<td>3+ or +++</td>
<td>Hyperactive without clonus</td>
</tr>
<tr>
<td>4+ or ++++</td>
<td>Hyperactive with clonus</td>
</tr>
</tbody>
</table>

- **Biceps (C5, C6)**
  1. The patient's arm should be partially flexed at the elbow with the palm down.
  2. Place your thumb or finger firmly on the biceps tendon.
  3. Strike your finger with the reflex hammer.
  4. You should feel the response even if you can't see it.

- **Triceps (C6, C7)**
  1. Support the upper arm and let the patient's forearm hang free.
  2. Strike the triceps tendon above the elbow with the broad side of the hammer.
  3. If the patient is sitting or lying down, flex the patient's arm at the elbow and hold it close to the chest.

- **Brachioradialis (C5, C6)**
  1. Have the patient rest the forearm on the abdomen or lap.
  2. Strike the brachioradialis tendon where it passes over the radius about two to four centimeters above the wrist.
  3. Watch and feel for flexion and supination of the forearm.

- **Abdominal (T8, T9, T10, T11, T12)**
  1. Use a blunt object such as a key or tongue blade.
  2. Stroke the abdomen lightly on each side in an inward and downward direction above (T8, T9, T10) and below the umbilicus (T10, T11, T12).
  3. Note the contraction of the abdominal muscles and deviation of the umbilicus towards the stimulus.

- **Knee (L2, L3, L4)**
  1. Have the patient sit or lie down with the knee flexed.
  2. Strike the patellar tendon just below the patella.
  3. Note contraction of the quadriceps and extension of the knee.

- **Ankle (S1, S2)**
  1. Dorsiflex the foot at the ankle.
  2. Strike the Achilles tendon.
  3. Watch and feel for plantar flexion at the ankle.

## Classical tendon jerk reflexes in the upper limb

### Biceps reflex (C5, C6)
- The patient's arm should be partially flexed at the elbow with the palm down resting on their lap.
- Place your thumb or finger firmly on the biceps tendon.
- Strike your finger with the reflex hammer.
- You should feel the response even if you can't see it.

### Brachioradialis reflex (C5, C6)
- Have the patient rest the forearm on the abdomen or lap.
- Strike the brachioradialis tendon where it passes over the radius about two to four centimeters above the wrist.
**Classical tendon jerk reflexes in the lower limb**

**Clonus**

If the reflexes seem hyperactive, test for ankle clonus:

1. Support the knee in a partly flexed position.
2. With the patient relaxed, quickly dorsiflex the foot.
3. Observe for rhythmic oscillations.

**Plantar Response (Babinski)**

**Triceps reflex (C6, C7)**

- The patient's arm should be partially flexed at the elbow with the palm down resting on their lap. Alternatively, support the upper arm and let the patient's forearm hang free.
- Strike the triceps tendon above the elbow with the broad side of the hammer.
- Watch for extension of the forearm.

**Knee reflex (L2, L3, L4)**

- Have the patient sit or lie down with the knee flexed.
- Strike the patellar tendon just below the patella.
- Note contraction of the quadriceps and extension of the knee.

**Ankle reflex (S1, S2)**

- Dorsiflex the foot at the ankle.
- Strike the Achilles tendon.
- Watch and feel for plantar flexion at the ankle.

**Eliciting clonus**

- Clonus occurs when there is a lack of normal cortical inhibition of a deep tendon reflex, resulting in rapid, strong, oscillating muscular contractions. This occurs when sustained tension is placed on one of the muscles controlling a joint, such as the wrist or ankle.

- If the reflexes seem hyperactive, test for ankle clonus:
  1. Support the knee in a partly flexed position.
  2. With the patient relaxed, quickly dorsiflex the foot.
  3. Observe for rhythmic oscillations.

- Up to 8-10 contractions of ankle clonus is considered normal in newborns, but contractions sustained beyond this are evidence of a central nervous system deficit.
1. Stroke the lateral aspect of the sole of each foot with the end of a reflex hammer or key.

2. Note movement of the toes, normally flexion (withdrawal).

3. Extension of the big toe with fanning of the other toes is abnormal. This is referred to as a positive Babinski.

**Planter response**

- This test is used to assess upper motor neuron lesions.
- Stroke the lateral aspect of the sole of each foot with the blunt end of a reflex hammer or key.
- Note movement of the toes, normally flexion (withdrawal).
- Extension of the big toe with fanning of the other toes is abnormal. This is referred to as a positive plantar response.
- Symmetry is particularly important for interpretation. A positive Babinski on one side and not the other is an important clue to the location of a lesion.
- The other plantar tests are equivalent.
- The term "Babinski reflex" is the abnormal response to plantar stimulation. Therefore, it is incorrect to make the statement that a patient has a "normal" or "down-going Babinski." Rather, if the response is normal, one should simply state that the "plantar reflex is normal" or that the "toes are down-going."
- The presence of a Babinski response in infants may be normal (sometimes up to 2 years of age), and should not be taken as conclusive evidence of neurological disease.

**Abdominal reflex (T8, T9, T10, T11, T12)**

- Use a blunt object such as a key or tongue blade.
- Stroke the abdomen lightly on each side in an inward and downward direction above (T8, T9, T10) and below the umbilicus (T10, T11, T12).
- Note the contraction of the abdominal muscles and deviation of the umbilicus towards the stimulus.
- Babies do not exhibit the abdominal reflex until about 6 months, but if spinal cord lesions are suspected the anal reflex is present at birth and can be tested.

**Sensory**

**General**

- Explain each test before you do it.
- Unless otherwise specified, the patient's eyes should be closed during the actual testing.
- Compare symmetrical areas on the two sides of the body.
- Also compare distal and proximal areas of the extremities.
- When you detect an area of sensory loss map out its boundaries in detail.

**Vibration**

- Use a low pitched tuning fork (128Hz).
  1. Test with a non-vibrating tuning fork first to ensure that the patient is responding to the correct stimulus.
  2. Place the stem of the fork over the distal interphalangeal joint of the patient's index fingers and big toes.
  3. Ask the patient to tell you if they feel the vibration.
- If vibration sense is impaired proceed proximally: ++

15 Click for an online video
Examination of vibration sense

- Use a low pitched (128 Hz) tuning fork.
- Test with a non-vibrating tuning fork first to ensure that the patient is responding to the correct stimulus.
- Place the stem of the fork over the distal interphalangeal joint of the patient's index fingers and great toes.
- Ask the patient to tell you if they feel the vibration.
- If the patient consistently detects vibration at these four points their vibratory sensation is intact.
- If vibration sense is impaired proceed proximally:
  - Wrists, Elbows, Medial malleoli, Patellae, Anterior superior iliac spines, Spinous processes, Clavicles
- Vibratory sensation uses the same receptors as proprioception. These receptors are only sensitive to lower frequencies. Your exam will be inaccurate if you use a tuning fork with a pitch higher than 128 Hz.

Subjective Light Touch

- Use your fingers to touch the skin lightly on both sides simultaneously. [13]
- Test several areas on both the upper and lower extremities.
- Ask the patient to tell you if there is difference from side to side or other "strange" sensations.

Position Sense

1. Grasp the patient's big toe and hold it away from the other toes to avoid friction. ++
2. Show the patient "up" and "down."
3. With the patient's eyes closed ask the patient to identify the direction you move the toe.
4. If position sense is impaired move proximally to test the ankle joint. ++
5. Test the fingers in a similar fashion.
6. If indicated move proximally to the metacarpophalangeal joints, wrists, and elbows. ++

Examination of sense of position

- Use this test when an abnormality is suspected, for instance if a patient has an uncoordinated gait or positive Romberg Test.
- Grasp the patient's big toe on the sides and hold it away from the other toes to avoid friction.
- Move the distal joint with your other hand.
- Demonstrate "up" and "down" while the patient watches.
- With the patient's eyes closed ask the patient to identify the direction you move the toe.
- If position sense is impaired move proximally to test the ankle joint.
Dermatomal Testing

If vibration, position sense, and subjective light touch are normal in the fingers and toes you may assume the rest of this exam will be normal.

Pain

- Use a suitable sharp object to test "sharp" or "dull" sensation. [7]
- Test the following areas:
  1. Shoulders (C4)
  2. Inner and outer aspects of the forearms (C6 and T1)
  3. Thumbs and little fingers (C6 and C8)
  4. Front of both thighs (L2)
  5. Medial and lateral aspect of both calves (L4 and L5)
  6. Little toes (S1)

Temperature

- Often omitted if pain sensation is normal. ++
- Use a tuning fork heated or cooled by water and ask the patient to identify "hot" or "cold."
- Test the following areas:
  1. Shoulders (C4)
  2. Inner and outer aspects of the forearms (C6 and T1)
  3. Thumbs and little fingers (C6 and C8)
  4. Front of both thighs (L2)
  5. Medial and lateral aspect of both calves (L4 and L5)
  6. Little toes (S1)

Light Touch

- Use a fine whisp of cotton or your fingers to touch the skin lightly.
- Ask the patient to respond whenever a touch is felt.
- Test the following areas:
  1. Shoulders (C4)
  2. Inner and outer aspects of the forearms (C6 and T1)
  3. Thumbs and little fingers (C6 and C8)
  4. Front of both thighs (L2)
  5. Medial and lateral aspect of both calves (L4 and L5)
  6. Little toes (S1)
Discrimination

Since these tests are dependent on touch and position sense, they cannot be performed when the tests above are clearly abnormal.

- Graphesthesia
  1. With the blunt end of a pen or pencil, draw a large number in the patient's palm.
  2. Ask the patient to identify the number.

- Stereognosis
  1. Use as an alternative to graphesthesia.
  2. Place a familiar object in the patient's hand (coin, paper clip, pencil, etc.).
  3. Ask the patient to tell you what it is.

- Two Point Discrimination
  1. Use in situations where more quantitative data are needed, such as following the progression of a cortical lesion.
  2. Use an opened paper clip to touch the patient's finger pads in two places simultaneously.
  3. Alternate irregularly with one point touch.
  4. Ask the patient to identify "one" or "two."
  5. Find the minimal distance at which the patient can discriminate.

Meningeal signs

Signs of meningeal irritation indicate inflammation of the dura; these signs are as follows:

1. Nuchal rigidity or neck stiffness is tested by placing the examiner's hand under the patient's head and gently trying to flex the neck. Undue resistance implies diffuse irritation of the cervical nerve roots from meningeal inflammation.

2. Brudzinski sign is flexion of both knees during the maneuver to test nuchal rigidity. This indicates diffuse meningeal irritation in the spinal nerve roots.

3. Kernig sign is elicited by flexing the hip and knee on one side while the patient is supine, then extending the knee with the hip still flexed. Hamstring spasm results in pain in the posterior thigh muscle and difficulty with knee extension. With severe meningeal inflammation, the opposite knee may flex during the test.

4. Lasegue or straight leg raising (SLR) sign is elicited by passively flexing the hip with the knee straight while the patient is in the supine position. Limitation of flexion due to hamstring spasm and/or pain indicates local irritation of the lower lumbar nerve roots. Reverse SLR is elicited by passively hyperextending the hip with the knee straight while the patient is in the prone position. Limitation of extension due to spasm and/or pain in the anterior thigh muscles indicates local irritation of the upper lumbar nerve roots.
Decerebrate & Decorticate posturing

These are often assessed in patients who present comatose and non-responsive, often requiring respiratory support. There are two classic reflexive postures: decorticate and decerebrate.

Decerebrate posturing

- Decerebrate posturing is seen in patients with lesions of the brainstem itself. These patients will exhibit extension of the arms, flexion of the wrists, jaw-clenching, back-arching, plantar flexion, and neck extension, either spontaneously or in response to a sternal rub.

- A way to remember the difference between the two postures is that in the decorticate posture, the patient's arms will point to the cortex.

Decorticate posturing

- Decorticate posturing is seen when there is a lesion of the corticospinal tract superior to the level of the brainstem. This is indicated in the comatose patient who responds to a sternal rub by full flexion of the elbows, wrists, and fingers, as well as plantar flexion of the feet with

Straight leg raising test

- This is a test for nerve root compression in the lower back.

- Ask the patient to lie supine on the exam table with knees straight.

- Grasp the leg near the heel and raise the leg slowly towards the ceiling.

- Pain in an L5 or S1 distribution suggests nerve root compression or tension (radicular pain).

- Dorsiflex the foot while maintaining the raised position of the leg.

- Increased pain strengthens the likelihood of a nerve root problem.

- Repeat the process with the opposite leg.

- Increased pain on the opposite side (a positive crossed straight leg raise) indicates a high probability of nerve root compression on that side.

Brudzinski’s Sign

- Flex the patient's neck and observe the hips and knees.

- If the hips and knees flex in response, this suggests meningeal irritation.

Kernig’s Sign

- With the patient supine, flex the leg 90 degrees at the hip and knee.

- Keeping the hip flexed, straighten the leg slowly at the knee.

- Some discomfort is normal, but bilateral pain and increased resistance to extension suggest meningeal irritation.
Decorticate posturing

Notes


2. Visual acuity is reported as a pair of numbers (20/20) where the first number is how far the patient is from the chart and the second number is the distance from which the "normal" eye can read a line of letters. For example, 20/40 means that at 20 feet the patient can only read letters a "normal" person can read from twice that distance.

3. You may, instead of wiggling a finger, raise one or two fingers (unilaterally or bilaterally) and have the patient state how many fingers (total, both sides) they see. To test for neglect, on some trials wiggle your right and left fingers simultaneously. The patient should see movement in both hands.

4. Additional Testing - Tests marked with (++) may be skipped unless an abnormality is suspected.

5. PERRLA is a common abbreviation that stands for "Pupils Equal Round Reactive to Light and Accommodation." The use of this term is so routine that it is often used incorrectly. If you did not specifically check the accommodation reaction use the term PERRL. Pupils with a diminished response to light but a normal response to accommodation (Argyll-Robertson Pupils) are a sign of neurosyphilis.

6. Nystagmus is a rhythmic oscillation of the eyes. Horizontal nystagmus is described as being either "leftward" or "rightward" based on the direction of the fast component.

7. Testing Pain Sensation - Use a new object for each patient. Break a wooden cotton swab to create a sharp end. The cotton end can be used for a dull stimulus. Do not go from patient to patient with a safety pin. Do not use non-disposable instruments such as those found in certain reflex hammers. Do not use very sharp items such as hypodermic needles.

8. Central vs Peripheral - With a unilateral central nervous system lesion (stroke), function is preserved over the upper part of the face (forehead, eyebrows, eyelids). With a peripheral nerve lesion (Bell's Palsy), the entire face is involved.

9. The hearing screening procedure presented by Bates on page 181 is more complex than necessary. The technique presented in this syllabus is preferred.

10. Deviation of the tongue or jaw is toward the side of the lesion.

11. Although it is often tested, grip strength is not a particularly good test in this context. Grip strength may be omitted if finger abduction and thumb opposition have been tested.

12. The "anti-gravity" muscles are difficult to assess adequately with manual testing. Useful alternatives include: walk on toes (plantarflexion); rise from a chair without using the arms (hip extensors and knee extensors); step up on a step, once with each leg (hip extensors and knee extensors).

13. Subjective light touch is a quick survey for "strange" or asymmetrical sensations only, not a formal test of dermatomes.
NEUROLOGICAL EXAMINATION SLIDES

INDEX

General Appearance

Preparing the patient and the physician for the neurological examination.

Cognition

Sensory System

Motor system

Deep Tendon Reflexes

Coordination, Gait and Rhomberg Test

Examination of the Cranial Nerves

The Examination of a Comatose or Stuporous Patient

General Appearance

Have the patient sit facing you on the examining table. Take a few seconds to actively observe the patient, and continue to actively observe the patient during the exam.

Level of consciousness.

Always begin the exam by introducing yourself to the patient as a tool to evaluate the patient's gross level of consciousness. Is the patient awake, alert and responsive? If not, then the exam may have to be abbreviated or urgent actions may have to be taken.
Personal Hygiene and Dress.

Note the patient's dress. Is it appropriate for the environment, temperature, age or social status of the patient? Is the patient malodorous or disheveled?

Posture and Motor Activity.

What posture does the patient assume when instructed to sit on the table? Are there signs of involuntary motor activity, including tremors (resting versus intention, also note the frequency in hertz of the tremor), choreathetotic movements, fasciculations, muscle rigidity, restlessness, dystonia or early signs of tardive dyskinesia?

Chorea refers to sudden, ballistic movements, and athetosis refers to writhing, repetitive movements. Fasciculations are fine twitching of individual muscle bundles, most easily noted on the tongue. Dystonia refers to sudden tonic contractions of the muscles of the tongue, neck (torticollis), back (opisthotonos), mouth, or eyes (oculogyric crisis). Early signs of tardive dyskinesia are lip smacking, chewing, or teeth grinding.

Damage to the substantia nigra may produce a resting tremor. This tremor is prominent at rest and characteristically abates during volitional movement and sleep. Damage to the cerebellum may produce a volitional or action tremor that usually worsens with movement of the affected limb. Spinal cord damage may also produce a tremor, but these tremors do not follow a typical pattern and are not useful in localizing lesions to the spinal cord.

Height, Build and Weight.

Is the patient obese or cachectic? If cachectic, note any wasting of the temporalis muscles. Note the general body proportions and look for any gross deformities. Also check for dysmorphic features, including low set ears, wide set eyes, small mandible, mongoloid facies, etc.

Vital Signs.

These include temperature, pulse, respiratory rate and blood pressure. It is essential that the vitals always be taken as an initial assessment of a patient. Emergency measures may have to be taken for drastically abnormal vital signs.
Follow this vital sign acquisition routine:

Place the thermometer under the patient's tongue and instruct the patient to keep it there. Wait 20-30 seconds for the results.

Next, find the radial pulse in the patient's right arm with your first two fingertips of your right hand. Look at your watch and count the pulses over 15 seconds and then multiply by 4. Note the quality of the pulse. Is it bounding or thready, weak or prominent, regular or irregular, slow or rapid? Once you are finished with the pulse measurement, keep your fingers on the pulse and secretly look at the patient's chest and count respirations for 15 seconds and also multiply this number by 4. Keeping your hand on the patient's pulse prevents the patient from becoming conscious of you watching them breath, preventing a likely adjustment in their respiratory rate.

Next, take the blood pressure. If it is high repeat the measurement later in the examination.
Finally, if a high temperature is present, or a previous history was taken suggesting meningeal irritation, test the patient for meningismus. Ask the patient to touch their chin to their chest to evaluate neck stiffness (a person with meningeal inflammation can only do this with pain). A positive Brudzinski's test is when the patient lifts their legs off the table in an effort to relieve pain felt when the neck is flexed.

Next, have the patient lie flat on the examining table. Keeping the lower leg flexed, raise the upper leg until it is perpendicular to the floor. Slowly extend the lower leg while keeping the upper leg stationary. If meningeal irritation is present, this maneuver will be painful for the patient. Sometimes the patient will raise their head off the table and/or scream if pain is present, this is considered a positive Kernig's test.

Meningismus consists of fever, clouding of consciousness, photophobia (bright light being painful to look at), nuchal rigidity, a positive Brudzinski’s test, and possibly a positive Kernig's test.

Special Topic: Classic Cerebrospinal Fluid Characteristics

<table>
<thead>
<tr>
<th>Condition</th>
<th>CSF Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic Seizures</td>
<td>Clear CSF with normal protein, normal glucose, no WBC's, no RBC's, normal opening pressure and normal % Gamma globulin.</td>
</tr>
<tr>
<td>Bacterial Meningitis:</td>
<td>Milky CSF with increased protein, decreased glucose, high WBC's (PMN predominate), few RBC's, mildly increased opening pressure and normal % Gamma globulin.</td>
</tr>
<tr>
<td>Guillain-Barre Syndrome:</td>
<td>Yellow CSF with very high protein (up to a gram), normal glucose, no WBC's, no RBC's, normal opening pressure and normal % Gamma globulin.</td>
</tr>
<tr>
<td>Subarachnoid Hemorrhage:</td>
<td>Yellow CSF with increased protein, normal glucose, few WBC's, innumerable RBC's, mildly increased opening pressure and normal % Gamma globulin.</td>
</tr>
<tr>
<td>Herpes Simplex Encephalitis:</td>
<td>Cloudy CSF with increased protein, normal glucose, increased WBC's (lymphocyte predominate), few RBC's, increase in opening pressure and normal % Gamma globulin.</td>
</tr>
<tr>
<td>Viral Meningitis:</td>
<td>Cloudy CSF with increased protein, normal glucose, increased WBC's (lymphocyte predominate), no RBC's,</td>
</tr>
</tbody>
</table>
Preparing the patient and the physician for the neurological examination.

The patient should be awake and alert, sitting on the examining table facing the examiner. The room should be quiet and adequately illuminated. It is imperative that the patient is naked, save the patient's underwear and hospital gown, to perform a full, initial neurological screening of the patient. Future or serial examinations may be more directed and may not require removal of clothing. It is also important that the patient is cooperative, non-intoxicated and is able to follow commands during the examination. If this is not the case, then errors may occur. Eyeglasses, if required, should be worn by the patient and are a requisite to perform a full ophthalmologic examination.

It is key for the physician to be prepared as well. When the physician is well versed with the organization of the neurological examination, is equipped with the correct tools, and is constantly anticipating the next part of the examination, the exam will run smoothly and rapidly with minimal patient discomfort. The physician should have a basic expectation, derived from the history taken previously, of what neurological findings may be present in a patient before an examination. These expectations must be tested and confirmed during the exam to establish a working diagnosis. One must remember to fully examine a patient and be very objective when results are documented, because initial expectations are often incorrect. In summary, it is important to fully examine a new patient and keep an open mind.

The tools required to perform a neurological exam. There are six basic tools required.

1. The reflex hammer and accessories.

The reflex hammer is used to illicit deep tendon reflexes throughout the body. This important item may come with detachable pin and brush accessories that are used to test for the sensory modalities of pin prick and light touch, respectively. If your reflex hammer does not have these items, then a common safety pin will be an adequate replacement.

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Multiple Sclerosis: Clear CSF with mild increase in protein, normal glucose, few WBC's (lymphocytic predominate), no RBC's, normal opening pressure, increased % Gamma globulin.

Benign Intracranial Hypertension: Clear CSF with normal protein, normal glucose, no WBC's, no RBC's, increased opening pressure and normal % Gamma globulin.
The 256 hertz tuning fork.

This item has multiple uses during the exam. It is used to test vibration sense throughout the body, to evaluate conductive versus neurological hearing loss, and may be placed under either warm or cold water (remember to dry it off before use) and then utilized for temperature sensation evaluation. Although one may purchase 126 and 512 hertz tuning forks (the 512 is better for auditory evaluation and the 126 is optimal for vibratory examination) the 256 hertz fork is adequate for an initial examination of both modalities. To cause the fork to vibrate, wrap it sharply on the palm of your hand before each time you touch its base to the patient's skin.

The ophthalmoscope.

This instrument is used to observe the optic disc, fovea and retina vessels. The light projected by the ophthalmoscope can also be used to test pupil light responses and then the scope itself may be followed in space to assess extra-ocular muscle movements. If one is without an ophthalmoscope, a light pen may substitute for pupil light response and extra-ocular muscle examinations. The ophthalmoscope is the most commonly underused tool in the neurological exam.

Visual acuity card.

This card is placed approximately 14 inches from the patient's face, and while the patient is wearing their glasses, if required, gross visual acuity is assessed. A reference for pupil diameter is also found on these cards.

A large Q-tip.
This item may be manipulated so the cotton tip is teased out to a fine point. The tip is used to test the corneal reflex by placing the end of the cotton wisp on the cornea, not the sclera, and checking for the eye to respond by blinking. These Q-tips may be pilfered from any hospital storage room.

### Soap.

It is important that a small bar of soap is carried by the physician in a small, convenient box. The soap is used to test the olfactory nerve. Some physicians prefer to carry a small vial of coffee grounds instead of soap.

Clinical Tip!

Soap is essential in evaluating the patient with possible head trauma or a history of inhalation of toxic fumes. Head trauma may lead to a fracture of the cribriform plate causing possible leakage of CSF fluid out of the nose. Noting a loss of olfaction (due to a disruption of the olfactory nerve roots where they cross the cribriform plate) with CSF rhinorrhea, in a patient with a history of head trauma, helps establish a diagnosis cribriform plate fracture.

### Cognition

The mini mental status exam is an important diagnostic tool used to evaluate a patient's orientation, concentration and memory (i.e., cognition). Although it is not imperative to perform a mini mental exam each time you evaluate a patient neurologically, a baseline score should be established when a patient is first examined. Serial mini mentals may be performed if deficits in cognition are noted at a later date or discovered upon the initial examination. The mini mental exam should be photocopied from any standard psychiatry text and carried with the student until it is administered frequently enough to be committed to memory. Once the student becomes proficient at instructing the patient to perform this battery, it will become a quick and efficient part of the neurological exam. More indepth neurocognitive tests may be necessary if deficits are discovered. Furthermore, it is important to assess a patient's ability to follow commands to perform a comprehensive, meaningful neurological exam.

The mini mental exam is scored out of a total of 30 points. A score of 24 or higher is considered within normal range, although specific deficits may be noted and investigated further. A score of below 24 is indicative of dementia.
Performance of the mini mental examination.

- **Orientation.**
  Date: Ask the patient to state the date. The patient achieves one point for each correct answer of the following: year, month, day, season and numerical date (a total of five points). If the patient does not volunteer all of these, ask specifically for the parts omitted.
  Location: Ask the patient to state where they are. The patient achieves one point for state, country, town, hospital and floor (a total of five points). Once again, if parts are omitted, ask for them specifically.

- **Registration.**
  Ask the patient if you can test their memory. State the name of three unrelated items (dog, pencil, ball) and then ask the patient to repeat the three items. The patient gets one point for each item repeated, i.e. registered (a total of three points). Ask the patient to remember these items, because you will ask for the patient to repeat them again later in the examination. In order to evaluate recent memory later in the exam, make certain all three objects have been registered. You may have to repeat them 5-6 times.

- **Attention and Calculation.**
  Ask the patient to begin with 100 and count backwards by subtracting 7's. The patient receives 1 point for each correct answer with a maximum of five points. If the patient is unable to subtract, have them spell the word WORLD in reverse, getting 1 point for each correct letter.

- **Recall.**
  Ask the patient to repeat the three words that they were asked to remember. Score 0-3.

- **Language.**
  Naming: Show the patient a wrist watch and then ask them to name the object shown to them. Repeat this question showing the patient a pen or pencil. Score 0-2. Repetition: Ask the patient to repeat the following sentence: "No ifs, ands, or buts." Score 0-1.

  Three step command: Hand the patient a piece of paper and state the following command: "hold this piece of paper, fold the paper in half, place the paper on the floor". One point for each correct movement, maximum of 3.
  Reading: On a blank sheet of paper write clearly the following: CLOSE YOUR EYES. Show this paper to the patient and ask them to read it to themselves and do what it says. Score 0-1.
  Writing: Give the patient a piece of paper and pen and
ask the patient to write any sentence they would like. The sentence must contain a noun and a verb, yet correct punctuation is not required. Score 0-1. Copying: On a sheet of paper draw two intersecting pentagons and then ask the patient to copy these objects. Score 0-1. All ten angles must be present with appropriate intersection points. Ignore tremor.

- Consciousness.
  Estimate the patient's level of consciousness: alert, lethargic, obtunded, stuporous, or comatose.

An alert patient is vigilantly attentive and keen. A lethargic patient is dull, sluggish and appears half asleep. An obtunded patient opens their eyes, responds slowly to questions, is somewhat confused, and has a decreased interest in their environment. A stuporous patient is near unconscious with apparent mental inactivity and reduced ability to respond to stimulation. Comatose patients are unconscious and unresponsive.

Add the up the total score and note the level of consciousness. Some clinicians defer the mini mental status examination until the end of the neurological examination for patients who are likely to have a normal mini mental status examination. The rationale for this change is that many patient's find some questions offensive and pointless and often become irritated, thus jeopardizing cooperation during the rest of the neurological examination.

Sensory System

The Sensory System Examination

The sensory exam includes testing for: pain sensation (pin prick), light touch sensation (brush), position sense, stereognosia, graphesthesia, and extinction. Diabetes mellitus, thiamine deficiency and neurotoxin damage (e.g. insecticides) are the most common causes of sensory disturbances. The affected patient usually reports paresthesias (pins and needles sensation) in the hands and feet. Some patients may report dysesthesias (pain) and sensory loss in the affected limbs also.

Pain and Light Touch Sensation

Initial evaluation of the sensory system is completed with the patient lying supine, eyes closed. Instruct the patient to say "sharp" or "dull" when they feel the respective object. Show the patient each object and allow them to touch the needle and brush prior to beginning to alleviate any fear of being hurt during the examination.
With the patient's eyes closed, alternate touching the patient with the needle and the brush at intervals of roughly 5 seconds. Begin rostrally and work towards the feet.

Make certain to instruct the patient to tell the physician if they notice a difference in the strength of sensation on each side of their body.

Alternating between pinprick and light touch, touch the patient in the following 13 places. Touch one body part followed by the corresponding body part on the other side (e.g., the right shoulder then the left shoulder) with the same instrument. This allows the patient to compare the sensations and note asymmetry.

The corresponding nerve root for each area tested is indicated in parenthesis.

1. posterior aspect of the shoulders (C4)
2. lateral aspect of the upper arms (C5)
3. medial aspect of the lower arms (T1)
4. tip of the thumb (C6)
5. tip of the middle finger (C7)
6. tip of the pinky finger (C8)
7. thorax, nipple level (T5)
8. thorax, umbilical level (T10)
9. upper part of the upper leg (L2)
10. lower-medial part of the upper leg (L3)
11. medial lower leg (L4)
12. lateral lower leg (L5)
13. sole of foot (S1)

If there is a sensory loss present, test vibration sensation and temperature sensation with the tuning fork. Also concentrate the sensory exam in the area of deficiency.
Position Sense

Test position sense by having the patient, eyes closed, report if their large toe is "up" or "down" when the examiner manually moves the patient's toe in the respective direction. Repeat on the opposite foot and compare. Make certain to hold the toe on its sides, because holding the top or bottom provides the patient with pressure cues which make this test invalid.

Fine touch, position sense (proprioception) and vibration sense are conducted together in the dorsal column system. Rough touch, temperature and pain sensation are conducted via the spinothalamic tract. Loss of one modality in a conduction system is often associated with the loss of the other modalities conducted by the same tract in the affected area.

Stereognosia

Test stereognosis by asking the patient to close their eyes and identify the object you place in their hand. Place a coin or pen in their hand. Repeat this with the other hand using a different object.

Astereognosis refers to the inability to recognize objects placed in the hand. Without a corresponding dorsal column system lesion, these abnormalities suggest a lesion in the sensory cortex of the parietal lobe.

Graphesthesia

Test graphesthesia by asking the patient to close their eyes and identify the number or letter you will write with the back of a pen on their palm. Repeat on the other hand with a different letter or number.

Apraxias are problems with executing movements despite intact strength, coordination, position sense and comprehension. This finding is a defect in higher intellectual functioning and is associated with cortical damage.
Extinction

To test extinction, have the patient sit on the edge of the examining table and close their eyes. Touch the patient on the trunk or legs in one place and then tell the patient to open their eyes and point to the location where they noted sensation. Repeat this maneuver a second time, touching the patient in two places on opposite sides of their body, simultaneously. Then ask the patient to point to where they felt sensation. Normally they will point to both areas. If not, extinction is present.

With lesions of the sensory cortex in the parietal lobe, the patient may only report feeling one finger touch their body, when in fact they were touched twice on opposite sides of their body, simultaneously. With extinction, the stimulus not felt is on the side opposite of the damaged cortex. The sensation not felt is considered "extinguished".

Motor system

The motor system evaluation is divided into the following: body positioning, involuntary movements, muscle tone and muscle strength.

Upper motor neuron lesions are characterized by weakness, spasticity, hyperreflexia, primitive reflexes and the Babinski sign. Primitive reflexes include the grasp, suck and snout reflexes. Lower motor neuron lesions are characterized by weakness, hypotonia, hyporeflexia, atrophy and fasciculations.

Fasciculations are fine movements of the muscle under the skin and are indicative of lower motor neuron disease. They are caused by denervation of whole motor units leading to acetylcholine hypersensitivity at the denervated muscle. Atrophy of the affected muscle is usually concurrent with fasciculations. Fibrillations are spontaneous contractions of individual muscle fibers and are therefore not observed with the naked eye.
Note the position of the body that the patient assumes when sitting on the examination table.

Paralysis or weakness may become evident when a patient assumes an abnormal body position. A central lesion usually produces greater weakness in the extensors than in the flexors of the upper extremities, while the opposite is true in the lower extremities: a greater weakness in the flexors than in the extensors.

Next, examine the patient for tics, tremors and fasciculations. Note their location and quality. Also note if they are related to any specific body position or emotional state.

Muscle Power examination

Systematically examine all of the major muscle groups of the body. For each muscle group:

1. Note the appearance or muscularity of the muscle (wasted, highly developed, normal).
2. Feel the tone of the muscle (flaccid, clonic, normal).
3. Test the strength of the muscle group.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No muscle contraction is detected</td>
</tr>
<tr>
<td>1</td>
<td>A trace contraction is noted in the muscle by palpating the muscle while the patient attempts to contract it.</td>
</tr>
<tr>
<td>2</td>
<td>The patient is able to actively move the muscle when gravity is eliminated.</td>
</tr>
<tr>
<td>3</td>
<td>The patient may move the muscle against gravity but not against resistance from the examiner.</td>
</tr>
<tr>
<td>4</td>
<td>The patient may move the muscle group against some resistance from the examiner.</td>
</tr>
<tr>
<td>5</td>
<td>The patient moves the muscle group and overcomes the resistance of the examiner. This is normal muscle strength.</td>
</tr>
</tbody>
</table>

- Since this rating scale is skewed towards weakness, many clinicians further subclassify their finding by adding a + or -, e.g., 5- or 3+. 
Starting with the deltoids, ask the patient to raise both their arms in front of them simultaneously as strongly as then can while the examiner provides resistance to this movement. Compare the strength of each arm.

The deltoid muscle is innervated by the C5 nerve root via the axillary nerve.

Next, ask the patient to extend and raise both arms in front of them as if they were carrying a pizza. Ask the patient to keep their arms in place while they close their eyes and count to 10. Normally their arms will remain in place. If there is upper extremity weakness there will be a positive pronator drift, in which the affected arm will pronate and fall. This is one of the most sensitive tests for upper extremity weakness.

Pronator drift is an indicator of upper motor neuron weakness. In upper motor neuron weakness, supination is weaker than pronation in the upper extremity, leading to a pronation of the affected arm. This test is also excellent for verification of internal consistency, because if a patient fakes the weakness, they almost always drop their arm without pronating it.

The patient to the left does not have a pronator drift.

Test the strength of lower arm flexion by holding the patient’s wrist from above and instructing them to “flex their hand up to their shoulder”. Provide resistance at the wrist. Repeat and compare to the opposite arm. This tests the biceps muscle.

The biceps muscle is innervated by the C5 and C6 nerve roots via the musculocutaneous nerve.
Now have the patient extend their forearm against the examiner's resistance. Make certain that the patient begins their extension from a fully flexed position because this part of the movement is most sensitive to a loss in strength. This tests the triceps. Note any asymmetry in the other arm.

The triceps muscle is innervated by the C6 and C7 nerve roots via the radial nerve.

Test the strength of wrist extension by asking the patient to extend their wrist while the examiner resists the movement. This tests the forearm extensors. Repeat with the other arm.

The wrist extensors are innervated by C6 and C7 nerve roots via the radial nerve. The radial nerve is the "great extensor" of the arm: it innervates all the extensor muscles in the upper and lower arm.

Examine the patient's hands. Look for intrinsic hand, thenar and hypothenar muscle wasting.

Test the patient's grip by having the patient hold the examiner's fingers in their fist tightly and instructing them not to let go while the examiner attempts to remove them. Normally the examiner cannot remove their fingers. This tests the forearm flexors and the intrinsic hand muscles. Compare the hands for strength asymmetry.

Finger flexion is innervated by the C8 nerve root via the median nerve.
Test the intrinsic hand muscles once again by having the patient abduct or "fan out" all of their fingers. Instruct the patient to not allow the examiner to compress them back in. Normally, one can resist the examiner from replacing the fingers.

Finger abduction or "fanning" is innervated by the T1 nerve root via the ulnar nerve.

To complete the motor examination of the upper extremities, test the strength of the thumb opposition by telling the patient to touch the tip of their thumb to the tip of their pinky finger. Apply resistance to the thumb with your index finger. Repeat with the other thumb and compare.

Thumb opposition is innervated by the C8 and T1 nerve roots via the median nerve.

Proceeding to the lower extremities, first test the flexion of the hip by asking the patient to lie down and raise each leg separately while the examiner resists. Repeat and compare with the other leg. This tests the iliopsoas muscles.

Hip flexion is innervated by the L2 and L3 nerve roots via the femoral nerve.
Test the adduction of the legs by placing your hands on the inner thighs of the patient and asking them to bring both legs together. This tests the adductors of the medial thigh.

Adduction of the hip is mediated by the L2, L3 and L4 nerve roots.

Test the abduction of the legs by placing your hands on the outer thighs and asking the patient to move their legs apart. This tests the gluteus maximus and gluteus minimus.

Abduction of the hip is mediated by the L4, L5 and S1 nerve roots.

Next, test the extension of the hip by instructing the patient to press down on the examiner's hand which is placed underneath the patient's thigh. Repeat and compare to the other leg. This tests the gluteus maximus.

Hip extension is innervated by the L4 and L5 nerve roots via the gluteal nerve.
Test extension at the knee by placing one hand under the knee and the other on top of the lower leg to provide resistance. Ask the patient to "kick out" or extend the lower leg at the knee. Repeat and compare to the other leg. This tests the quadriceps muscle.

Knee extension by the quadriceps muscle is innervated by the L3 and L4 nerve roots via the femoral nerve.

Test flexion at the knee by holding the knee from the side and applying resistance under the ankle and instructing the patient to pull the lower leg towards their buttock as hard as possible. Repeat with the other leg. This tests the hamstrings.

The hamstrings are innervated by the L5 and S1 nerve roots via the sciatic nerve.

Test dorsiflexion of the ankle by holding the top of the ankle and have the patient pull their foot up towards their face as hard as possible. Repeat with the other foot. This tests the muscles in the anterior compartment of the lower leg.

Ankle dorsiflexion is innervated by the L4 and L5 nerve roots via the peroneal nerve.
Holding the bottom of the foot, ask the patient to "press down on the gas pedal" as hard as possible. Repeat with the other foot and compare. This tests the gastrocnemius and soleus muscles in the posterior compartment of the lower leg.

Ankle plantar flexion is innervated by the S1 and S2 nerve roots via the tibial nerve.

To complete the motor exam of the lower extremity ask the patient to move the large toe against the examiner's resistance "up towards the patient's face". The extensor hallucis longus muscle is almost completely innervated by the L5 nerve root. This tests the extensor hallucis longus muscle.

Patients with primary muscle disease (e.g. polymyositis) or disease of the neuromuscular junction (e.g. myasthenia gravis), usually develop weakness in the proximal muscle groups. This leads to the greatest weakness in the hip girdle and shoulder girdle muscles. This weakness usually manifests as difficulty standing from a chair without significant help with the arm musculature. Patients often complain that they can't get out of their cars easily or have trouble combing their hair.

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Root Levels</th>
<th>Test Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trapezius</td>
<td>C3-C4</td>
<td>Shrug shoulders</td>
</tr>
<tr>
<td>Deltoid</td>
<td>C5-C6</td>
<td>Abduct shoulder</td>
</tr>
<tr>
<td>Biceps</td>
<td>C5-C6</td>
<td>Flex elbow</td>
</tr>
<tr>
<td>Triceps</td>
<td>C6-C8</td>
<td>Extend elbow</td>
</tr>
<tr>
<td>Wrist extensors</td>
<td>C6-C7</td>
<td>Extend wrist</td>
</tr>
</tbody>
</table>
Table 2: MRC Scale for Grading Muscle Strength

<table>
<thead>
<tr>
<th>Score</th>
<th>Muscle Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No Movement</td>
</tr>
<tr>
<td>1</td>
<td>Muscle belly moves but the joint does not move</td>
</tr>
<tr>
<td>2</td>
<td>Joint moves with gravity eliminated</td>
</tr>
<tr>
<td>3</td>
<td>Joint moves against gravity</td>
</tr>
<tr>
<td>4</td>
<td>Joint moves against gravity and some resistance</td>
</tr>
<tr>
<td>5</td>
<td>Full strength</td>
</tr>
</tbody>
</table>

**Deep Tendon Reflexes**

Using a reflex hammer, deep tendon reflexes are elicited in all 4 extremities. Note the extent or power of the reflex, both visually and by palpation of the tendon or muscle in question.
Rate the reflex with the following scale:

<table>
<thead>
<tr>
<th>Grade</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>5+</td>
<td>Sustained clonus</td>
</tr>
<tr>
<td>4+</td>
<td>Very brisk, hyperreflexive, with clonus</td>
</tr>
<tr>
<td>3+</td>
<td>Brisker or more reflexive than normally.</td>
</tr>
<tr>
<td>2+</td>
<td>Normal</td>
</tr>
<tr>
<td>1+</td>
<td>Low normal, diminished</td>
</tr>
<tr>
<td>0.5+</td>
<td>A reflex that is only elicited with reinforcement</td>
</tr>
<tr>
<td>0</td>
<td>No response</td>
</tr>
</tbody>
</table>

Reinforcement is accomplished by asking the patient to clench their teeth, or if testing lower extremity reflexes, have the patient hook together their flexed fingers and pull apart. This is known as the Jendrassik maneuver. It is key to compare the strength of reflexes elicited with each other. A finding of 3+, brisk reflexes throughout all extremities is a much less significant finding than that of a person with all 2+, normal reflexes, and a 1+, diminished left ankle reflex suggesting a distinct lesion. Have the patient sit up on the edge of the examination bench with one hand on top of the other, arms and legs relaxed. Instruct the patient to remain relaxed.

<table>
<thead>
<tr>
<th>Reflex</th>
<th>Root Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaw Jerk</td>
<td>CN5 (Mandibular)</td>
</tr>
<tr>
<td>Biceps</td>
<td>C5</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>C6</td>
</tr>
<tr>
<td>Triceps</td>
<td>C7</td>
</tr>
<tr>
<td>Quadriceps</td>
<td>L4</td>
</tr>
<tr>
<td>Achilles</td>
<td>S1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grade</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None</td>
</tr>
<tr>
<td>1</td>
<td>Reduced, requires reinforcement to obtain</td>
</tr>
<tr>
<td>2-3</td>
<td>Normal</td>
</tr>
<tr>
<td>4</td>
<td>Hyperreflexia, clonus, pathological spread</td>
</tr>
</tbody>
</table>
The biceps reflex is elicited by placing your thumb on the biceps tendon and striking your thumb with the reflex hammer and observing the arm movement. Repeat and compare with the other arm. The brachioradialis reflex is observed by striking the brachioradialis tendon directly with the hammer when the patient's arm is resting. Strike the tendon roughly 3 inches above the wrist. Note the reflex supination. Repeat and compare to the other arm.

The biceps and brachioradialis reflexes are mediated by the C5 and C6 nerve roots.

The triceps reflex is measured by striking the triceps tendon directly with the hammer while holding the patient's arm with your other hand. Repeat and compare to the other arm.

The triceps reflex is mediated by the C6 and C7 nerve roots, predominantly by C7.

With the lower leg hanging freely off the edge of the bench, the knee jerk is tested by striking the quadriceps tendon directly with the reflex hammer. Repeat and compare to the other leg.

The knee jerk reflex is mediated by the L3 and L4 nerve roots, mainly L4. Insult to the cerebellum may lead to pendular reflexes. Pendular reflexes are not brisk but involve less damping of the limb movement than is usually observed when a deep tendon reflex is elicited. Patients with cerebellar injury may have a knee jerk that swings forwards and backwards several times. A normal or brisk knee jerk would have little more than one swing forward and one back. Pendular reflexes are best observed when the patient's lower legs are allowed to hang and swing freely off the end of an examining table.
The ankle reflex is elicited by holding the relaxed foot with one hand and striking the Achilles tendon with the hammer and noting plantar flexion. Compare to the other foot.

The ankle jerk reflex is mediated by the S1 nerve root.

The plantar reflex (Babinski) is tested by coarsely running a key or the end of the reflex hammer up the lateral aspect of the foot from heel to big toe. The normal reflex is toe flexion. If the toes extend and separate, this is an abnormal finding called a positive Babinski's sign.

A positive Babinski's sign is indicative of an upper motor neuron lesion affecting the lower extremity in question.

The Hoffman response is elicited by holding the patient's middle finger between the examiner's thumb and index finger. Ask the patient to relax their fingers completely. Once the patient is relaxed, using your thumbnail press down on the patient's fingernail and move downward until your nail "clicks" over the end of the patient's nail. Normally, nothing occurs. A positive Hoffman's response is when the other fingers flex transiently after the "click". Repeat this maneuver multiple times on both hands.

A positive Hoffman response is indicative of an upper motor neuron lesion affecting the upper extremity in question.

Finally, test clonus if any of the reflexes appeared hyperactive. Hold the relaxed lower leg in your hand, and sharply dorsiflex the foot and hold it dorsiflexed. Feel for oscillations between flexion and extension of the foot indicating clonus. Normally nothing is felt.

Special Topic: Lower Back Syndromes

Sciatica is the clinical description of pain in the leg that occurs due to lumbrosacral nerve root compression usually secondary to lumbar disc prolapse or extrusion. L5/S1 disc level is the
most common site of disc herniation. The following are the characteristic "lower back syndromes" associated with nerve root compression. Note that disc herniations are mostly in the posterolateral direction, thus compression of the nerve root exiting from the vertebral foramen at one level below is affected. (The nerve root at the same level of the herniation is already within the vertebral foramen and therefore not compressed)

L5/S1 Disc Prolapse

- Pain along posterior thigh with radiation to the heel
- Weakness on plantar flexion (may be absent)
- Sensory loss in the lateral foot
- Absent ankle jerk reflex

L4/L5 Disc Prolapse

- Pain along the posterior or posterolateral thigh with radiation ot the top of the foot
- Weakness of dorsiflexion of the great toe and foot
- Paraesthesia and numbness of top of foot and great toe
- No reflex changes noted

L3/L4 Disc Prolapse

- Pain in front of thigh
- Wasting of quadriceps muscles may be present
- Diminished sensation on the front of the thigh and medial lower leg
- Reduced knee jerk reflex

Coordination, Gait and Rhomberg Test

Coordination
Coordination is evaluated by testing the patient's ability to perform rapidly alternating and point-to-point movements correctly.

Rapidly Alternating Movement Evaluation

Ask the patient to place their hands on their thighs and then rapidly turn their hands over and lift them off their thighs. Once the patient understands this movement, tell them to repeat it rapidly for 10 seconds. Normally this is possible without difficulty. This is considered a rapidly
alternating movement.

Dysdiadochokinesis is the clinical term for an inability to perform rapidly alternating movements. Dysdiadochokinesia is usually caused by multiple sclerosis in adults and cerebellar tumors in children. Note that patients with other movement disorders (e.g. Parkinson's disease) may have abnormal rapid alternating movement testing secondary to akinesia or rigidity, thus creating a false impression of dysdiadochokinesia.

Point-to-Point Movement Evaluation

Next, ask the patient to extend their index finger and touch their nose, and then touch the examiner's outstretched finger with the same finger. Ask the patient to go back and forth between touching their nose and examiner's finger. Once this is done correctly a few times at a moderate cadence, ask the patient to continue with their eyes closed. Normally this movement remains accurate when the eyes are closed. Repeat and compare to the other hand.

Dysmetria is the clinical term for the inability to perform point-to-point movements due to over or under projecting ones fingers.

Next have the patient perform the heel to shin coordination test. With the patient lying supine, instruct him or her to place their right heel on their left shin just below the knee and then slide it down their shin to the top of their foot. Have them repeat this motion as quickly as possible without making mistakes. Have the patient repeat this movement with the other foot. An inability to perform this motion in a relatively rapid cadence is abnormal.

The heel to shin test is a measure of coordination and may be abnormal if there is loss of motor strength, proprioception or a cerebellar lesion. If motor and sensory systems are intact, an abnormal, asymmetric heel to shin test is highly suggestive of an ipsilateral cerebellar lesion.
Gait

Gait is evaluated by having the patient walk across the room under observation. Gross gait abnormalities should be noted. Next ask the patient to walk heel to toe across the room, then on their toes only, and finally on their heels only. Normally, these maneuvers possible without too much difficulty. Be certain to note the amount of arm swinging because a slight decrease in arm swinging is a highly sensitive indicator of upper extremity weakness. Also, hopping in place on each foot should be performed.

Walking on heels is the most sensitive way to test for foot dorsiflexion weakness, while walking on toes is the best way to test early foot plantar flexion weakness.

Abnormalities in heel to toe walking (tandem gait) may be due to ethanol intoxication, weakness, poor position sense, vertigo and leg tremors. These causes must be excluded before the unbalance can be attributed to a cerebellar lesion. Most elderly patients have difficulty with tandem gait purportedly due to general neuronal loss impairing a combination of position sense, strength and coordination. Heel to toe walking is highly useful in testing for ethanol inebriation and is often used by police officers in examining potential "drunk drivers".
**Rhomberg Test**

Next, perform the Romberg test by having the patient stand still with their heels together. Ask the patient to remain still and close their eyes. If the patient loses their balance, the test is positive. To achieve balance, a person requires 2 out of the following 3 inputs to the cortex: 1. visual confirmation of position, 2. non-visual confirmation of position (including proprioceptive and vestibular input), and 3. a normally functioning cerebellum. Therefore, if a patient loses their balance after standing still with their eyes closed, and is able to maintain balance with their eyes open, then there is likely to be lesion in the cerebellum. This is a positive Rhomberg.

To conclude the gait exam, observe the patient rising from the sitting position. Note gross abnormalities.
Examination of the Cranial Nerves

When testing the cranial nerves one must be cognizant of asymmetry. The following is a summary of the cranial nerves and their respective functioning.

- I - Smell
- II - Visual acuity, visual fields and ocular fundi
- II,III - Pupillary reactions
- III,IV,VI - Extra-ocular movements, including opening of the eyes
- V - Facial sensation, movements of the jaw, and corneal reflexes
- VII - Facial movements and gustation
- VIII - Hearing and balance
- IX,X - Swallowing, elevation of the palate, gag reflex and gustation
- V,VII,X,XII - Voice and speech
- XI - Shrugging the shoulders and turning the head
- XII - Movement and protrusion of tongue

Lesions of the nervous system above the spinal cord are often classified as peripheral or central in location. Peripheral lesions are lesions of the cranial nerve nuclei, the cranial nerves or the neuromuscular junctions. Central lesions are lesions in the brainstem (not involving a cranial nerve nucleus), cerebrum or cerebellum. If there is a lesion in the brainstem involving a cranial nerve nucleus along with other areas of the brain stem, then the lesion is considered both central and peripheral.

Cranial Nerve I

Evaluate the patency of the nasal passages bilaterally by asking the patient to breath in through their nose while the examiner occludes one nostril at a time. Once patency is established, ask the patient to close their eyes. Occlude one nostril, and place a small bar of soap near the patent nostril and ask the patient to smell the object and report what it is. Making certain the patient's eyes remain closed. Switch nostrils and repeat. Furthermore, ask the patient to compare the strength of the smell in each nostril.

Very little localizing information can be obtained from testing the sense of smell. This part of the exam is often omitted, unless their is a reported history suggesting head trauma or toxic inhalation.
Cranial Nerve II

First test visual acuity by using a pocket visual acuity chart. Perform this part of the examination in a well lit room and make certain that if the patient wears glasses, they are wearing them during the exam. Hold the chart 14 inches from the patient's face, and ask the patient to cover one of their eyes completely with their hand and read the lowest line on the chart possible. Have them repeat the test covering the opposite eye. If the patient has difficulty reading a selected line, ask them to read the one above. Note the visual acuity for each eye.

Next evaluate the visual fields via confrontation. Face the patient one foot away, at eye level. Tell the patient to cover their right eye with their right hand and look the examiner in the eyes. Instruct the patient to remain looking you in the eyes and say "now" when the examiner's fingers enter from out of sight, into their peripheral vision. Once this is understood, cover your left eye with your left hand (the opposite eye of the patient) and extend your arm and first 2 fingers out to the side as far as possible. Beginning with your hand and arm fully extended, slowly bring your outstretched fingers centrally, and notice when your fingers enter your field of vision. The patient should say now at the same time you see your own fingers. Repeat this maneuver a total of eight times per eye, once for every 45 degrees out of the 360 degrees of peripheral vision. Repeat the same maneuver with the other eye.

Using an ophthalmoscope, observe the optic disc, physiological cup, retinal vessels and fovea. Note the pulsations of the optic vessels, check for a blurring of the optic disc margin and a change in the optic disc's color form its normal yellowish orange. The initial change in the ophthalmoscopic examination in a patient with increased intracranial pressure is the loss of pulsations of the retinal vessels. This is followed by blurring of the optic disc margin and possibly retinal hemorrhages.

Cranial Nerves II and III

Ask the patient to focus on an object in the distance. Observe the diameter of the pupils in a dimly lit room. Note the symmetry between the pupils. Next, shine the
penlight or opthalmoscope light into one eye at a time and check both the direct and consensual light responses in each pupil. Note the rate of these reflexes. If they are sluggish or absent, test for pupillary constriction via accommodation by asking the patient to focus on the light pen itself while the examiner moves it closer and closer to their nose. Normally, as the eyes accommodate to the near object the pupils will constrict. The test for accommodation should also be completed in a dimly lit room. End the evaluation of cranial nerves II and III by observing the pupils in a well lit room and note their size and possible asymmetry.

Anisocoria is a neurological term indicating that one pupil is larger than another. Yet which pupil is abnormal? For example, if the right pupil is of a greater diameter than the left pupil in room light, is their a sympathetic lesion in the left eye or a parasympathetic lesion in the right eye? To determine this, observe and compare the asymmetry of the pupils in both bright and dim light. If the asymmetry is greatest in dim light than the sympathetic system is disrupted in the left eye, not allowing it to dilate in dim light, while the functioning right eye dilates even further in the dim light causing an increase in asymmetry. Conversely, if the asymmetry is greatest in bright light, then there is a parasympathetic lesion in the right eye. If the asymmetry remains the same in dim and bright light, then the anisocoria is physiologic.

Ptosis is the lagging of an eyelid. It has 2 distinct etiologies. Sympathetics going to the eye innervate Muller's muscle, a small muscle that elevates the eyelid. The III cranial nerve also innervates a much larger muscle that elevates the eye lid: the levator palpebrae. Thus, disruption of either will cause ptosis. The ptosis from a III nerve palsy is of greater severity than the ptosis due to a lesion of the sympathetic pathway, due to the size of the muscles innervated. As an aside, the parasympathetics run with the III cranial nerve and are usually affected with an abnormal III cranial nerve.

Anisocoria can only be produced if the efferent pathway of the pupillary light reflex is disrupted. A lesion of the afferent pathway along the II cranial does not yield anisocoria. To test for a lesion of the afferent pathway one must perform a "swinging light test". To interpret this test one must understand that the level of pupillary constriction is directly related to the total "perceived" illumination the brain appreciates from both eyes. If, for example, their is a 90% decrease in the afferent pathway in the left eye, shining a bright light in this eye will produce less constriction in both eyes (remember, the efferent pathways are functioning), compared to a bright light shining in the normal eye. Therefore with an afferent lesion, "swinging" the light back and forth between the eyes rapidly will cause the pupils to change diameter when the light goes from the normal eye (brain perceiving increased illumination) to the abnormal eye (brain perceiving less illumination). If both eyes are normal, no change would occur, because the total perceived illumination remains constant. This is called an afferent pupillary defect (APD) or Marcus-Gunn pupil.

Cranial Nerves III, IV and VI

Instruct the patient to follow the penlight or opthalmoscope with their eyes without moving their
head. Move the penlight slowly at eye level, first to the left and then to the right. Then repeat this horizontal sweep with the penlight at the level of the patient's forehead and then chin. Note extraocular muscle palsies and horizontal or vertical nystagmus.

The limitation of movement of both eyes in one direction is called a conjugate lesion or gaze palsy, and is indicative of a central lesion. A gaze palsy can be either supranuclear (in cortical gaze centers) or nuclear (in brain stem gaze centers). If the gaze palsy is a nuclear gaze palsy, then the eyes can't be moved in the restricted direction voluntarily or by reflex, e.g. oculocephalic reflex. If the lesion is cortical, then only voluntary movement is absent and reflex movements are intact.

Disconjugate lesions, where the eyes are not restricted in the same direction or if only one eye is restricted, are due to more peripheral disruptions: cranial nerve nuclei, cranial nerves or neuromuscular junctions. One exception to this rule is an isolated impairment of abduction of one eye, which is commonly due to an ipsilateral median longitudinal fasciculus (MLF) lesion. This lesion is also called an internuclear ophthalmoplegia (INO). In INO, nystagmus is often present when the opposite eye is abducted.

Gaze-evoked nystagmus (nystagmus that is apparent only when the patient looks to the side or down) may be caused by many drugs, including ethanol, barbiturates, and phenytoin (Dilantin). Ethanol and barbiturates (recreational or therapeutic) are the most common cause of nystagmus. Dilantin may evoke nystagmus at slight overdoses, and ophthalmoplegia at massive overdoses.

Abnormal patterns of eye movements may help localize lesions in the central nervous system. Ocular bobbing is the rhythmical conjugate deviation of the eyes downward. Ocular bobbing is without the characteristic rapid component of nystagmus. This movement is characteristic of damage to the pons.

Downbeat nystagmus (including a rapid component) may indicate a lesion compressing on the cervicomedullary junction such as a meningioma or chordoma.

An electronystagmogram (ENG) may be ordered to characterize abnormal eye movements. The basis of this test is that there is an intrinsic dipole in each eyeball (the retina is negatively charged compared to the cornea. During an ENG, recording electrodes are placed on the skin around the eyes and the dipole movement is measured and eye movement is accurately characterized.
Cranial Nerve V

First, palpate the masseter muscles while you instruct the patient to bite down hard. Also note masseter wasting on observation. Next, ask the patient to open their mouth against resistance applied by the instructor at the base of the patient's chin.

Next, test gross sensation of the trigeminal nerve. Tell the patient to close their eyes and say "sharp" or "dull" when they feel an object touch their face. Allowing them to see the needle before this examination may alleviate any fear of being hurt. Using the needle and brush from your reflex hammer or the pin from a safety pin, randomly touch the patient's face with either the needle or the brush. Touch the patient above each temple, next to the nose and on each side of the chin, all bilaterally. Ask the patient to also compare the strength of the sensation of both sides. If the patient has difficulty distinguishing pinprick and light touch, then proceed to check temperature and vibration sensation using the vibration fork. One may warm it or cool it under a running faucet.

Finally, test the corneal reflex using a large Q-tip with the cotton extended into a wisp. Ask the patient to look at a distant object and then approaching laterally, touch the cornea (not the sclera) and look for the eye to blink. Repeat this on the other eye.

Some clinicians omit the corneal reflex unless there is sensory loss on the face as per history or examination, or if cranial nerve palsies are present at the pontine level.

Cranial Nerve VII

Initially, inspect the face during conversation and rest noting any facial asymmetry including drooping, sagging or smoothing of normal facial creases. Next, ask the patient to raise their eyebrows, smile showing
their teeth, frown and puff out both cheeks. Note asymmetry and difficulty performing these maneuvers. Ask the patient to close their eyes strongly and not let the examiner pull them open. When the patient closes their eyes, simultaneously attempt to pull them open with your fingertips. Normally the patient’s eyes cannot be opened by the examiner. Once again, note asymmetry and weakness.

When the whole side of the face is paralyzed the lesion is peripheral. When the forehead is spared on the side of the paralysis, the lesion is central (e.g., stroke). This is because a portion of the VII cranial nerve nucleus innervating the forehead receives input from both cerebral hemispheres. The portion of the VII cranial nerve nucleus innervating the mid and lower face does not have this dual cortical input.

Hyperacusis (increased auditory volume in an affected ear) may be produced by damage to the seventh cranial nerve. This is because the seventh cranial nerve innervates the stapedius muscle in the middle ear which damps ossicle movements which decreases volume. With seventh cranial nerve damage this muscle is paralyzed and hyperacusis occurs. Furthermore, since the branch of the seventh cranial nerve to the stapedius begins very proximally, hyperacusis secondary to seventh cranial nerve dysfunction indicates a lesion close to seventh cranial nerve's origin at the brainstem.

Cranial Nerve VIII

Assess hearing by instructing the patient to close their eyes and to say "left" or "right" when a sound is heard in the respective ear. Vigorously rub your fingers together very near to, yet not touching, each ear and wait for the patient to respond. After this test, ask the patient if the sound was the same in both ears, or louder in a specific ear. If there is lateralization or hearing abnormalities perform the Rinne and Weber tests using the 256 Hz tuning fork.
The Weber test is a test for lateralization. Wrap the tuning fork strongly on your palm and then press the butt of the instrument on the top of the patient's head in the midline and ask the patient where they hear the sound. Normally, the sound is heard in the center of the head or equally in both ears. If there is a conductive hearing loss present, the vibration will be louder on the side with the conductive hearing loss. If the patient doesn't hear the vibration at all, attempt again, but press the butt harder on the patient's head.

The Rinne test compares air conduction to bone conduction. Wrap the tuning fork firmly on your palm and place the butt on the mastoid eminence firmly. Tell the patient to say "now" when they can no longer hear the vibration. When the patient says "now", remove the butt from the mastoid process and place the U of the tuning fork near the ear without touching it. Tell the patient to say "now" when they can no longer hear anything. Normally, one will have greater air conduction than bone conduction and therefore hear the vibration longer with the fork in the air. If the bone conduction is the same or greater than the air conduction, there is a conductive hearing impairment on that side. If there is a sensineuronal hearing loss, then the vibration is heard substantially longer than usual in the air. Make certain that you perform both the Weber and Rinne tests on both ears. It would also be prudent to perform an otoscopic examination of both eardrums to rule out a severe otitis media, perforation of the tympanic membrane or even occlusion of the external auditory meatus, which all may confuse the results of these tests. Furthermore, if hearing loss is noted an audiogram is indicated to provide a baseline of hearing for future reference.

Because of the extensive bilateral connections of the auditory system, the only way to have an ipsilateral hearing loss is to have a peripheral lesion, i.e. at the cranial nerve nucleus or more peripherally. Bilateral hearing loss from a single lesion is invariably due to one located centrally.
Cranial Nerves IX and X

Ask the patient to swallow and note any difficulty doing so. Ask the patient if they have difficulty swallowing. Next, note the quality and sound of the patient's voice. Is it hoarse or nasal? Ask the patient to open their mouth wide, protrude their tongue, and say "AHH". While the patient is performing this task, flash your penlight into the patient's mouth and observe the soft palate, uvula and pharynx. The soft palate should rise symmetrically, the uvula should remain midline and the pharynx should constrict medially like a curtain. Often the palate is not visualized well during this maneuver. One may also try telling the patient to yawn, which often provides a greater view of the elevated palate. Also at this time, use a tongue depressor and the butt of a long Q-tip to test the gag reflex. Perform this test by touching the pharynx with the instrument on both the left and then on the right side, observing the normal gag or cough.

Some clinicians omit testing for the gag reflex unless there is dysarthria or dysphagia present by history or examination, or if cranial nerve palsies are present at the medullary level.

Roughly 20% of normal individuals have a minimal or absent gag reflex.

Dysarthria and dysphagia are due to incoordination and weakness of the muscles innervated by the nucleus ambiguus via the IX and X cranial nerves. The severity of the dysarthria or dysphagia is different for single versus bilateral central lesions. The deficiency is often minor if the lesion is centrally located and in only one cortical hemisphere, because each nucleus ambiguus receives input from both cerebral hemispheres. In contrast, bilateral central lesions, or "pseudobulbar palsies", often produce marked deficits in phonation and swallowing. Furthermore, on examination the quality of the dysarthria is distinct for central versus peripheral lesions. Central lesions produce a strained, strangled voice quality, while peripheral lesions produce a hoarse, breathy and nasal voice.
Cranial Nerve XI

This cranial nerve is initially evaluated by looking for wasting of the trapezius muscles by observing the patient from the rear. Once this is done, ask the patient to shrug their shoulders as strong as they possibly can while the examiner resists this motion by pressing down on the patient's shoulders with their hands. Next, ask the patient to turn their head to the side as strongly as they possibly can while the examiner once again resists with their hand.

Repeat this maneuver on the opposite side. The patient should normally overcome the resistance applied by the examiner. Note asymmetry.

Peripheral lesions produce ipsilateral sternocleidomastoid (SCM) weakness and ipsilateral trapezius weakness. Central lesions produce ipsilateral SCM weakness and contralateral trapezius weakness, because of differing sources of cerebral innervation. This is a common clinical misunderstanding.
Cranial Nerve XII

The hypoglossal nerve controls the intrinsic musculature of the tongue and is evaluated by having the patient "stick out their tongue" and move it side to side. Normally, the tongue will be protruded from the mouth and remain midline. Note deviations of the tongue from midline, a complete lack of ability to protrude the tongue, tongue atrophy and fasciculations on the tongue.

The tongue will deviate towards the side of a peripheral lesion, and to the opposite side of a central lesion.

Special Topic: Pathology found on opthalmologic examination

Papilledema. Note swelling of the disc, hemorrhages, and exudates, with preservation of the physiologic cup.

Optic Atrophy. Note the chalky white disc with discrete margins. Optic atrophy is a late finding with increased intracranial pressure.
Central Retinal Artery Occlusion. Note the diffusely pale retina and prominent central fovea which is usually blended in with the normal, pink retina.

Central Retinal Vein Occlusion. The disc is massively swollen with diffuse hemorrhages and cotton-wool spots.

Proliferative Diabetic Retinopathy. Note the multiple hemorrhages, exudates and neovascularization throughout the retina. Chorioretinal striae extend towards the area of fibrovascular proliferation in the lower portion of the photograph.

Cytomegalovirus Retinitis. Note the area of retinal necrosis and hemorrhage along the lower portion of the photograph. Common in patients with immunodeficiency, especially AIDS.

The Examination of a Comatose or Stuporous Patient

Because the comatose patient cannot understand and follow commands, the examination of the comatose patient is a modified version of the neurological examination of an alert patient. If a patient is comatose, it is safe to assume that the nervous system is being affected at the brainstem.
level or above. The goal of a neurological examination in a comatose patient is to determine if the coma is induced by a structural lesion or from a metabolic derangement, or possibly from both.

Two findings on exam strongly point to a structural lesion: 1. consistent asymmetry between right and left sided responses, and 2. abnormal reflexes that point to specific areas within the brain stem.

Mental status is evaluated by observing the patient's response to visual, auditory and noxious (i.e., painful) stimuli. The three main maneuvers to produce a noxious stimulus in a comatose patient are: 1. press very hard with your thumb under the bony superior roof of the orbital cavity, 2. squeeze the patient's nipple very hard, and 3. press a pen hard on one of the patient's fingernails.

Comatose patients may demonstrate motor responses indicative of more generalized reflexes. Decorticate posturing consists of adduction of the upper arms, flexion of the lower arms, wrists and fingers. The lower extremities extend in decorticate posturing. Decerebrate posturing consists of adduction of the upper arms, extension and pronation of the lower arms, along with extension of the lower extremities.

In general, patients with decorticate posturing have a better prognosis than patients who exhibit decerebrate posturing. Posturing does not have any localizing utility in humans.

Visual acuity cannot be tested in a comatose patient, but pupillary responses may be tested as usual. Visual fields may be partially evaluated by noting the patient's response to sudden objects introduced into the patient's visual field. Extra-ocular muscles may be evaluated by inducing eye movements via reflexes. The doll's eyes reflex, or oculocephalic reflex, is produced by moving the patient's head left to right or up and down. When the reflex is present, the eyes of the patient remain stationary while the head is moved, thus moving in relation to the head. Thus moving the head of a comatose patient allows extra-ocular muscle movements to be evaluated.

An alert patient does not have the doll's eyes reflex because it is suppressed. If a comatose patient does not have a doll's eyes reflex, then a lesion must be present in the afferent or efferent loop of this reflex arc. The afferent arc consists of the labyrinth, vestibular nerve, and neck proprioceptors. The efferent limb consists of cranial nerves III, IV and VI and the muscles they innervate. Furthermore, the pathways that connect the afferent and efferent limbs in the pons and medulla may also be disrupted and cause a lack of the doll's eyes reflex in a comatose patient.

If the patient is being examined in the emergency department or if there is a history of potential cervical spine injury, the doll's eyes reflex should not be elicited until after a cervical spine injury is ruled out.

The oculovestibular reflex, or cold calorics, is produced by placing the patient's upper body and head at 30 degrees off horizontal, and injecting 50-100cc of cold water into an ear. The water has the same effect on the semicircular canal as if the patient's head was turned to the opposite side of the injection. Therefore, the patient's eyes will look towards the ear of injection. This eye
deviation lasts for a sustained period of time. This is an excellent maneuver to assess extra-ocular muscles in the comatose patient with possible cervical spine injury.

If the oculovestibular reflex is absent, a lesion of the pons, medulla, or less commonly the III, IV, IV or VIII nerves is present. Unlike the oculocephalic reflex, the oculovestibular reflex is present in awake patients. In alert patients, this reflex not only induces eye deviation, it also produces nystagmus in the direction of the non-injected ear. The slow phase is towards the injected ear and the fast phase is away.

Cranial nerve V may be tested in the comatose patient with the corneal reflex test. Cranial nerve VII may be examined by observing facial grimacing in response to a noxious stimulus. Cranial nerves IX an X may be evaluated with the gag reflex.

The motor system is assessed by testing deep tendon reflexes, feeling the resistance of the patient's limbs to passive movements, and testing the strength of posturing and local withdrawal movements. Local withdrawal movements may be elicited by pressing a pen hard on the patient's fingernail and observing if the patient withdraws the respective limb from the noxious stimulus.

Upper motor neuron lesions are characterized by spasticity. Spasticity is increased muscle tone leading to resistance of the limbs to passive manipulation. This spasticity classically results in the clasp-knife response. The clasp-knife response is when the spastic limb is passively moved with great resistance, when suddenly the limb "gives", becoming very easy to move. The clasp knife response is most prominent in the muscle groups least affected by the upper motor lesion, e.g., flexors in the upper extremities or extensors in the lower extremities.

The sensory system can only be evaluated by observing the patient's response, or lack of response, to noxious stimuli in different parts of the body.

In addition to withdrawing from noxious stimuli, patient's may localize towards noxious stimuli. Localization indicates a shallower coma compared to the patient that withdraws.

A common prognostic assessment, called the Glasgow Coma Scale, is often used to measure the depth of coma. The Glasgow Coma Scale is often used serially as a means to follow a comatose patient clinically. It has 3 sections: I. best motor response, II. best verbal response, and III. eye opening.
### Glasgow Coma Scale:

<table>
<thead>
<tr>
<th>I. Motor Response</th>
<th>II. Verbal Response</th>
<th>III. Eye Opening</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 - Obeys commands fully</td>
<td>5 - Alert and Oriented</td>
<td>4 - Spontaneous eye opening</td>
</tr>
<tr>
<td>5 - Localizes to noxious stimuli</td>
<td>4 - Confused, yet coherent, speech</td>
<td>3 - Eyes open to speech</td>
</tr>
<tr>
<td>4 - Withdraws from noxious stimuli</td>
<td>3 - Inappropriate words, and jarbled phrases consisting of words</td>
<td>2 - Eyes open to pain</td>
</tr>
<tr>
<td>3 - Abnormal flexion, i.e. decorticate posturing</td>
<td>2 - Incomprehensible sounds</td>
<td>1 - No eye opening</td>
</tr>
<tr>
<td>2 - Extensor response, i.e. decerebrate posturing</td>
<td>1 - No sounds</td>
<td></td>
</tr>
<tr>
<td>1 - No response</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Glascow Coma Scale = I + II + III.  
A lower score indicates a deeper coma and a poorer prognosis.

Patients with a Glasgow Coma Scale of 3-8 are considered comatose. Patients with an initial score of 3-4 have a >95% incidence of death or persistent vegetative state.
Papilledema is an optic disc swelling that is secondary to elevated intracranial pressure. In contrast to other causes of optic disc swelling, vision usually is well preserved with acute papilledema. Papilledema almost always presents as a bilateral phenomenon and may develop over hours to weeks. The term should not be used to describe optic disc swelling with underlying infectious, infiltrative, or inflammatory etiologies.

The disc swelling in papilledema is the result of axoplasmic flow stasis with intra-axonal edema in the area of the optic disc. The subarachnoid space of the brain is continuous with the optic nerve sheath. Hence, as the cerebrospinal fluid (CSF) pressure increases, the pressure is transmitted to the optic nerve, and the optic nerve sheath acts as a tourniquet to impede axoplasmic transport. This leads to a buildup of material at the level of the lamina cribrosa, resulting in the characteristic swelling of the nerve head. Papilledema may be absent in cases of prior optic atrophy. This absence of papilledema is most likely because of a decrease in the number of physiologically active nerve fibers.

**Characteristic of papilledema**

1. Congestion of retinal veins – loss of venous pulsation,

2. Hyperemia of disc – filling of physiologic cup,

3. Disc edges blurred – first upper and lower margins, then nasal margin, finally temporal margin;

4. Elevation of nerve head (3 – 10 Dioptres),

5. Spread of edema to retina – macular fan,

6. Hump of vessels leaving and entering disc more marked,

7. Vessels appear and disappear as they course near the disc,

8. When Disc swelling reduces – disc becomes yellowish white – arteries become constricted, veins remain congested,

- **Optic neuritis/papillitis**

Optic neuritis implies an inflammatory process involving the optic nerve. In children, most cases are due to an immune-mediated process. These cases may be associated with a viral or other
infection or with immunization. Less commonly, optic neuritis may be the first manifestation of multiple sclerosis (MS) or part of a more diffuse demyelinating disorder, including acute disseminated encephalomyelitis or Devic disease. Optic neuritis may be related to specific infections, diseases of the adjacent sinuses or orbital structures, and infectious and infiltrative diseases of the brain or meninges that involve the optic nerves.

Optic neuritis involving the optic disc with disc edema is called Papillitis. Bilateral simultaneous optic neuritis Refers to Optic neuritis in both eyes occurring within 3 weeks of each other

Table 1. Papilledema versus papillitis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Optic Neuritis</th>
<th>Papilloedema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Side</td>
<td>Unilateral</td>
<td>Bilateral (rare exceptions)</td>
</tr>
<tr>
<td>Pain on eye movement</td>
<td>Usually present</td>
<td>No pain (rare exceptions)</td>
</tr>
<tr>
<td>Onset of visual loss</td>
<td>Acute</td>
<td>Gradual</td>
</tr>
<tr>
<td>Degree of visual loss as compared to degree of disc swelling</td>
<td>Gross</td>
<td>Slight</td>
</tr>
<tr>
<td>Color vision</td>
<td>Impaired (especially red and green)</td>
<td>Defective only at late stage</td>
</tr>
<tr>
<td>Field defect</td>
<td>Central or centrocecal scotoma</td>
<td>Enlarged blind spot, Peripheral constriction</td>
</tr>
<tr>
<td>Pupillary reaction</td>
<td>Ill sustained</td>
<td>Normal</td>
</tr>
<tr>
<td>Degree of disc swelling</td>
<td>Less than 3 Dioptres</td>
<td>More than 3 Dioptres</td>
</tr>
<tr>
<td>Venous engorgement</td>
<td>Less marked</td>
<td>More marked</td>
</tr>
<tr>
<td>Venous pulsation</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Hemorrhages</td>
<td>Uncommon</td>
<td>May be present</td>
</tr>
<tr>
<td>Slit lamp examination for cells in vitreous</td>
<td>Positive</td>
<td>Negative</td>
</tr>
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<td>Onset of visual loss</td>
<td>Sudden</td>
<td>Gradual</td>
</tr>
<tr>
<td>Degree of visual loss as compared to degree of disc swelling</td>
<td>Gloss</td>
<td>Slight (except PPOA)</td>
</tr>
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<td>Color vision</td>
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<td>Negative</td>
</tr>
</tbody>
</table>
Figure 1. Papilledema

Figure 2. Papilledema

Figure 3. A, Optic neuritis, B, Papillitis
References

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Anterior ischemic optic neuropathy
Dot, flame, Boat Retinal Hemorrhages
Optic neuritis
Optic neuritis
papilledema
papilledema
Postpapilledemalic atrophy
Primary optic atrophy
Primary optic atrophy