The Precise Neurological Exam

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http://edinfo.med.nyu.edu/courseware/neurosurgery/index.html

The objective of the neurological examination is threefold:

1. To identify an abnormality in the nervous system.
2. To localize any lesion present to the extent possible, i.e differentiate peripheral from central lesions.
3. To establish internal consistency, i.e. does the patient cooperate fully? Are the findings a variant of normal?

The 6 tools required to perform a neurological exam include: a Reflex hammer, Tuning Fork, Ophthalmoscope, Visual acuity Card, Q-tip, and Soap.

General Appearance

Active observation – level of consciousness, personal Hygiene and Dress, Posture and motor activity (involuntary activity?), dysmorphic features, check vital signs.

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), choreoathetotic 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.

If a history suggests 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 release 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.
Examination of the Cranial Nerves

When testing the cranial nerves one must be cognizant of asymmetry.

- 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,VI,IX,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**

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.

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

**Cranial Nerve II**

In a well lit room (make certain that if the patient wears glasses), hold the chart 14 inches from the patient's face, ask the patient to cover one of their eyes completely and read the lowest line on the chart possible.
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. When 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. 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 from 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 ophthalmoscope 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 ophthalmoscope 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 extra-ocular 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 adduction 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.

Cranial Nerve V

First, palpate the masseter muscles while you instruct the patient to bite down hard (also note wasting). 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.

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.

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**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 dampens 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.

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**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. Then ask the patient if the sound was the same in both ears, or louder in a specific ear. If there are hearing abnormalities perform the Rinne/Weber tests using 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 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, 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 possible 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.

The Motor System Examination

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. Watch for involuntary actions such as tics, tremors, or fasciculations.

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.

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.

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<th>Score</th>
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<tr>
<td>0</td>
<td>No muscle contraction is detected</td>
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<td>1</td>
<td>A trace contraction is noted in the muscle by palpating the muscle while the patient attempts to contract it.</td>
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<td>2</td>
<td>The patient is able to actively move the muscle when gravity is eliminated.</td>
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<td>3</td>
<td>The patient may move the muscle against gravity but not against resistance from the examiner.</td>
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<td>4</td>
<td>The patient may move the muscle group against some resistance from the examiner.</td>
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<td>5</td>
<td>The patient moves the muscle group and overcomes the resistance of the examiner. This is normal muscle strength.</td>
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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, where supination is weaker than pronation in the upper extremity, leading to a pronation of the affected arm. This is also excellent for verification of internal consistency, because if a patient fakes the weakness, they usually drop their arm without pronating it.

The Deltoid muscle is innervated by the C5 nerve root via the axillary nerve. The biceps muscle is innervated by the C5
and C6 nerve roots via the musculocutaneous nerve. The triceps muscle is innervated by the C6 and C7 nerve roots via the radial nerve. 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. Finger flexion is innervated by the C8 nerve root via the median nerve. Finger abduction or "fanning" is innervated by the T1 nerve root via the ulnar nerve. Thumb opposition is innervated by the C8 and T1 nerve roots via the median nerve.

Hip flexion is innervated by the L2 and L3 nerve roots via the femoral nerve. Adduction of the hip is mediated by the L2, L3 and L4 nerve roots. Abduction of the hip is mediated by the L4, L5 and S1 nerve roots. Hip extension is innervated by the L4 and L5 nerve roots via the gluteal nerve. Knee extension by the quadriceps muscle is innervated by the L3 and L4 nerve roots via the femoral nerve. The hamstrings are innervated by the L5 and S1 nerve roots via the sciatic nerve. Ankle dorsiflexion is innervated by the L4 and L5 nerve roots via the peroneal nerve. Ankle plantar flexion is innervated by the S1 and S2 nerve roots via the tibial nerve. The extensor hallucis longus muscle is almost completely innervated by the L5 nerve root.

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.

**Sensory System**

The sensory exam includes testing for: pain sensation (pin prick), light touch sensation (brush), position sense, stereognosia, graphesthesia, and extinction. Diabetes mellitus and thiamine deficiency 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.

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.

**Stereognosis**

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".

**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.

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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.

The reflex is elicited by placing your thumb on the tendon and striking your thumb with the reflex hammer and observing motor activity. Repeat and compare with the other side. The biceps and brachioradialis reflexes are mediated by the C5 and C6 nerve roots. The triceps reflex is mediated by the C6 and C7 nerve roots, predominantly by C7. The knee jerk reflex is mediated by the L3 and L4 nerve roots, mainly L4. 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.

**Coordination, Gait and Romberg Test**

**Coordination**

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

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.

Dysdiadochokinesia 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**

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. 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.

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.
Romberg 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 this is indicative of pathology in the proprioceptive pathway. This is a positive Romberg.