The Neurological Examination

Stephen Deputy, MD

Introduction

The Neurological Examination is, by necessity, long and cumbersome. That does not mean that every patient with a neurological chief complaint needs to undergo a "complete" Neurological exam. (Can you imagine testing each and every sensory dermatome for all modalities of primary sensation and doing a Cremaster reflex on a patient presenting with headaches?). The purpose of the Neuro Exam is to answer questions gleaned from the History, to identify any neurological deficits, and to localize those deficits on the basis of pertinent findings. With this in mind, it is essential to "touch base" within each of the fundamental realms of the Neurological Examination (Mental Status, Cranial Nerves, Motor, Coordination, Sensory, and Gait) in order to cover the entire neuroaxis, which ranges from the cortex, subcortical white matter, deep grey matter structures, brainstem, cerebellum, spinal cord, peripheral nerves, neuromuscular junction and muscles. What one does within each of these realms with regards to examination depends on the clinical situation. The redundancy of the Neuro Exam helps one to confirm deficits. Pertinent "negatives" are just as important as "positive" findings to aid in localization.

With that said, consider doing a "screening" Neuro Exam on every patient that you encounter to touch base within each of the fundamental realms. Next, expand the Neuro Exam to answer questions gleaned from the history. Finally, take advantage of the redundancy of the Neuro Exam to confirm or refute any abnormal findings.

Don't just do the Neurological Exam to please or impress your resident or attending with your "completeness". Rather, always use the Neuro Exam as a tool to answer questions and to localize deficits.

Mental Status

The Mental Status exam is really looking at cognition, which is a cortical function. The following examination techniques are based on localization of discreet deficits. Neurocognitive testing is not included here, though it does have a value in localization and diagnosis for disorders of higher cognitive functioning. Likewise, the field of psychiatry uses essentially an expanded mental status exam to evaluate mood disorders, anxiety disorders, and abnormalities of thought process, which, while very important to patient care, do not have the same discreet localization as the following Mental Status techniques provide. The Mini Mental Status Exam can be a useful tool to quantify a patient's overall mental status when dementia is a clinical consideration.

Alertness and Orientation:

"A and O x 3" is often seen on physical exam statements. The patient's level of alertness is a subjective impression by the examiner. Is the patient fully alert and responsive? If not, using terms such as "somnolent", "sleepy", "obtunded" etc. are not very helpful as they are difficult to quantify. It is better to use descriptions such as "the patient appears to be awake, though he is agitated and often provides confused responses to questions", or "the patient has closed eyes, but opens them to verbal stimulation", or "the patient only opens his eyes briefly to noxious, painful stimulation". Coma has a very specific definition which is that of "unarousable unresponsiveness". Other, lesser degrees of impaired consciousness should be described more descriptively.

Orientation should be listed as to the specific items for which the patient is oriented to. Common useful questions include: "What is your name?" "Do you know where you are right now?" "What is today's date?" "What day of the week is it?" "How old are you?" At the end of these questions, one can write in the chart, "The patient is alert and oriented to name, place and date" which is more specific than "A and O x 3".

The patient's level of alertness and orientation needs to be established before any other aspect of the MS (Mental Status) is tested. This is because, if the patient is not fully alert and oriented, then the localizing value of the rest of the MS exam no longer becomes valid. (For example, disoriented, confused patients who cannot properly identify a watch do not necessarily suffer from anomia, which may otherwise be localized to the Broca's region of the dominant hemisphere).

Concentration

The evaluation of concentration require that a patient "remain on task" during a sustained mental activity. Some of the more common tasks include "serial sevens" (where the patient is asked to mentally subtract sevens from one hundred in a sequential manner) as well as to spell the word "World" backwards. Of course, the patient needs to have the mental capabilities in basic math and spelling to validate these as "concentration" tasks. One challenging task that I like to use is to ask the patient to state the months of the year forward and backwards. As with any neuro exam task, start with the most difficult task and if the patient cannot complete it, try to quantify the deficit by asking them to try and complete easier tasks (for example, if the patient cannot complete serial 7's, then try serial 3's. Or, if they cannot do the months of the year backwards, then try the days of the week backwards, etc.).

As with Alertness and Orientation, if a patient's Concentration is off, then the localizing value of the rest of the Mental Status exam comes into question. Therefore, one should perform tasks of Alertness and Orientation as well as tasks of Concentration before moving on to the rest of the Mental Status exam.

Memory

While there are many different types of memory which can be better settled with neurocognitive testing, we will focus on short-term memory and long-term memory here. Long-term memory is the most ingrained with any sort of encephalopathy and is therefore, the most resilient and "last to go". Asking historical questions about where the patient went to high school, their birth date, their wedding anniversary, the names of their parents, etc. is the best way to evaluate long-term memory.

Short-term memory, on the other hand, is evaluated by having a patient recall 3 objects after several minutes. Each of you should pick your own unique set of 3 objects for use in this task. These objects should be readily familiar to most patients and come from different "categories". For example, I use "piano" "baseball bat" and the color "blue" (These are my unique 3 objects. Pick your own!) Whatever objects you choose to use, stick with them (so you won't forget the objects that you asked the patient to recall!). Make sure that the patient can accurately and immediately repeat the objects and tell them that you will be asking them to recall them in a few minutes. If the patient cannot immediately accurately recall each of the three objects, then this is a deficit of registration (not short-term memory) which is really a task of concentration and not of short-term memory. Assuming registration is intact, if the patient cannot recall one or more of the objects after several minutes, then give them a clue. The patient's performance on the short-term memory task can be reported as "Short-term memory intact with 3/3 objects recalled after 5 minutes" or "STM = 3/3" or "STM = 2/3 plus 3/3 with prompting" etc.

Language

As with memory, language is complex and best "broken down" through neurocognitive testing. For the purposes of a general neurological examination however, we will stick to "expressive language", "receptive language", and "repetition". Language function is mostly confined to the dominant hemisphere. This is essentially the left hemisphere for right handed individuals and may be the right hemisphere, left hemisphere or have bilateral representation for left handed individuals. This is why it is important to note whether an individual is right or left handed in their HPI description. Recall that "language" itself is not confined to spoken language. Patients with "aphasias" will have difficulty not only with spoken language, but also with reading, writing, sign language, and facial expressions or hand gestures that are used to communicate meaning. "Dysarthria" is a term that describes intact "language" along with a motor production deficiency of clear speech. Dysarthria does not localize to the language areas of the brain but may be secondary to motor functions in the frontal lobe, deep sub-cortical basal ganglia regions, brainstem or their connections.

"Expressive language" is a function of Broca's area cortex. This is localized to the dominant hemisphere insular frontal cortex. Patients with a "Broca's aphasia" or an "expressive aphasia" will have fluency problems as well as word finding deficits (non-fluent aphasia). Once again, try to quantify the deficit by having the patient try to name easier objects (such as a watch or thumb) then move on to more difficult names (such as a watch band or index finger).

"Receptive language" is a function of Wernicke's area (angular gyrus region of the parietal lobe) of the dominant hemisphere. As a "language" function, injury to Wernicke's area results in deficits of understanding language

(whether that be spoken language, reading, or understanding gestures). Good ways to test expressive language include asking a patient to perform a multi-step task. Remember that a 3-step task is more difficult than a one-step task and that midline tasks (such as "open your mouth") are easier than appendicular task (such as "show me the thumb on your left hand"). Having the patient perform a task that they need to read is also helpful in distinguishing alexia from aphasia. Patients with Wernicke's aphasia not only have a difficult time understanding others' language, but also with understanding their own language. The result is a fluent aphasia, where the patient rambles on in a incoherent fashion. Neologisms and inappropriate word substitutions are also part of Wernicke's aphasia.

Finally, the task of "repetition" requires that language be comprehended (through Wernicke's area of the parietal lobe), successfully transmitted (through the angular gyrus and the arcurate fasiculus), and reach Broca's area (in the insular cortex) to be spoken. Therefore, a good screening task for all of these areas is to ask the patient to repeat a complex sentence. I always use "today is a sunny day in the city of New Orleans", though other phrases such as "there are no ifs, ands, or buts about it" are commonly used. Make sure the sentence is somewhat complex and if the patient cannot repeat it accurately, move towards an easier sentence.

<u>Cranial Nerves</u>

The cranial nerve exam reflects the function of the brainstem and can help to co-localize long track findings. It is best to go "in order" when examining the cranial nerves (I-XII) in order to look into midbrain, pons, and medullary function sequentially.

CN # I: The Olfactory Nerve.

This nerve is rarely tested except in cases of closed head injury where shearing forces can sever branches coming off the olfactory nerves as they transverse the skull base at the cribiform plate. There is a direct air communication in the superior nasal passageways so that it is difficult to isolate deficits of the olfactory nerve to the right or left side exclusively. Noxious stimulation (such as smelling ammonium salts) actually irritates the nasal mucosa which is supplied by the Maxillary division of the Trigeminal Nerve. Therefore, to test olfactory function, the patient should be asked to distinguish between smells such as coffee and cinnamon or other spices.

CN #2: The Optic Nerve

The optic nerve relays signals derived from retinal cells to the Lateral Geniculate Nucleus of the Thalamus. From there, visual information is transmitted to the Visual Cortex. The Optic Chiasm sends fibres reflecting visual input from the lateral visual fields from each eye to the contralateral Lateral Geniculate Nucleus. Nasal fields remain ipsilateral.

There are four ways to examine the Optic Nerve.

1) Visual Acuity: Make sure to test each eye individually with a Snellen Eye Chart and provide a quantitative visual assessment for each eye (i.e. 20/25 OD, 20/40 OS at near corrected).
2) Visual Fields: Compare the patient's visual fields against your own through direct confrontational testing. "VFFTC" stands for "Visual Fields Full to Confrontation".
3) Pupillary Light Reflex: Recall that the afferent portion of the pupillary light reflex runs through the Optic Nerve and jumps off at the Edinger-Westphal Nucleus. From there, the efferent component (parasympathetic nervous system) travels along the Oculomotor Nerve to reach the pupillary constrictor muscles. An "afferent pupillary defect" suggests dysfunction of the Optic Nerve with an intact Oculomotor Nerve. A patient with an "APD" will demonstrate an intact consensual pupillary light reflex but neither pupil will constrict when light is shined in the affected eye. "PEERLA" stands for "Pupils Equal and Equally Reactive to Light and Accommodation". When patients are asked to look at an object very near to their eyes, their eyes will converge and their pupils will constrict (Accommodation).
4) Fundoscopic Exam: Guess what. You can actually directly see the head of the optic nerve with fundoscopic examination. Observation should note any swelling or elevation (papilledema) or pallor or atrophy of the optic nerve head.

CN's III, IV, and VI: The Oculomotor, Trochlear, and Abducens Nerves

These nerves are responsible for controlling eye movements. The Trochlear Nerve innervates the Superior Oblique muscle for each eye which results in medial downward deviation and intortion of the eye. The Abducens Nerve innervates the Lateral Rectus muscle which abducts the eye. The Oculomotor Nerve innervates the rest of the eye muscles, provides parasympathetic fibres responsible for pupillary constriction, and aids with eyelid opening. Injury to the Trochlear Nerve results in an extorted eye which is accommodated for by the patient tilting their head towards the opposite side. Injury to the Abducens Nerve results in medial deviation (adduction) of the eye. Injury to the Oculomotor Nerve results in an eye that is "Down and Out" (laterally deviated and depressed), a "Blown Pupil", and ptosis.

The extraocular muscles are tested for by having the patient look in all of the 6 Cardinal directions of gaze. Ask about diplopia during this part of the exam. Remember that the degree of diplopia will be greatest when the patient is looking into the direction corresponding to the action of the impaired muscle.

CN # V: The Trigeminal Nerve

The Trigeminal Nerve is composed of a Branchial Motor component and a general sensory component. The motor component supplies innervations to the muscles of mastication (Temporalis and Masseters) and the Lateral Pterygoid muscles (keeps jaw open). The sensory nerve is broken down into the Ophthalmic (V1), Maxillary (V2), and Mandibular (V3) divisions. Each of these three divisions can be tested for all modalities of primary sensation (light touch, position sense, vibration, and pain and temperature). The afferent component of the Corneal Blink Reflex comes from V1 stimulation of the corneal nerve.

CN VII: The Facial Nerve

The Facial Nerve supplies not only innervation to all of the facial muscles, but also has visceral motor branches to the submandibular and sublingual glands as well as to the nasal mucosa (parasympathetic innervations). It has a little known sensory division supplying input for primary sensory modalities from the skin from part of the auricle, a patch of skin behind the ear and from the external surface of the tympanic membrane. A "lower motor neuron" deficit of CN VII (e.g. Bell's Palsy) will affect the upper and lower facial muscles ipsilateral to the site of injury. An "upper motor neuron" lesion affecting the contralateral corticobulbar tracts will result in weakness of the lower part of the face with sparing of the upper part of the face (secondary to bilateral innervation of the forehead muscles). Bifacial paresis is more difficult to pick up due to the lack of asymmetry of deficits. These patients will have difficulty with forced eyelid closure and difficulty holding air in puffed out cheeks.

CN VIII: The Vestibulocochlear Nerve

This nerve sends signal from the cochlear gland (hearing) and vestibular gland (balance) to their respective brainstem nuclei where processed signals are sent via way of the Thalamus to the superior temporal gyrus (hearing) and cerebellum (balance). The "Vestibulocochlear Reflex" aka the "Vestibulo-Ocular Reflex" aka "Doll's Eye Maneuver" aka "Cold Calorics" attains its afferent input from stimulation to the vestibular apparatus with efferent outputs going to the muscles affecting lateral eye movements. Hearing can be assessed by asking if the patient can hear a soft sound (such as rubbing fingers together) in each ear. The Weber Test (where the tuning fork is placed in the middle of the vertex of the skull) will result in the patient hearing the loudest sound to the ear that has ipsilateral conduction deficits or contralateral sensory-neural deficits. The Renee´ Test will tell if Air Conduction is louder than Bone Conduction (which it should be). If Bone Conduction is louder, this suggests a conduction deficit to the ipsilateral ear.

CN IX and X: The Glossopharyngeal and Vagus Nerves

While the Glossopharyngeal Nerve has Branchial Motor branches (to the stylopharyngeus muscle), Visceral Motor branches (parasympathetic innervation to the Parotid Gland), Visceral Sensory branches (input from the carotid body and carotid sinus), General Sensory branches (primary sensory inputs from the posterior 1/3 of tongue, skin of the external ear and the inner surface of the tympanic membrane) and Special Sensory branches (taste from the posterior 1.3 of the tongue) it is usually only tested for integrity through the afferent component of the gag reflex.

The Vagus Nerve, likewise has many functions such as Branchial Motor branches to several of the striated muscles of the pharynx, tongue and larynx, Visceral Motor input to smooth muscle and glands of the pharynx, larynx, and

abdominal viscera (down to the middle of the transverse colon), Visceral Sensory branches from the pharynx, larynx, and abdominal viscera as well as from stretch receptors and chemoreceptors from the aortic arch, and a minimal General Sensory input from part of the ear and external ear canal.

Despite the complex functions of these nerves, their integrity is assessed primarily through assessment of the gag reflex (Afferent = Glossopharyngeal Nerve and Efferent = Vagus Nerve innervation to the palate).

CN XI: The Accessory Nerve

The Accessory Nerve supplies Motor branches to the Trapezius and Sternocleidomastoid muscles. Shoulder shrug and lateral head rotation are ways to check these muscles. Remember that there is bilateral neck flexion weakness in several myopathies so check for neck flexion weakness if indicated.

CN XII: The Hypoglossal Nerve

The Hypoglossal Nerve supplies Somatic Motor innervation to all intrinsic and extrinsic muscles of the tongue. A CN XII deficit will result in ipsilateral deviation of the protruded tongue (along with ipsilateral weakness and atrophy).

Motor Examination

The Motor Examination consists of several different components (Strength, Tone, Posture, Fine Motor Coordination, DTR's, Plantar Responses, and Involuntary Movements). Each will be reviewed in their turn.

Strength can best be assessed by isolating an individual muscle group and comparing your power with that of the patient. A standardized Medical Research Council (MRC) score can be noted for any muscle group tested. A score of 5/5 is full strength. A score of 4/5 (or 4+ or 4-) implies weakness with enough strength to overcome varying degrees of physical resistance. 3/5 means that the patient can overcome gravity but not resistance. 2/5 implies that the limb can be moved only when gravity is taken out of the equation. 1/5 is muscle activation without limb movement. 0/5 is no muscle activation whatsoever.

Tone is the resistance that the examiner subjectively experiences when moving a patient's limb passively. It can be normal, increased, or decreased (not "good" or "bad"). Causes of increased tone include Spasticity (Due to Corticospinal Tract dysfunction), Rigidity (Basal Ganglia dysfunction), Dystonia (Basal Ganglia) or Paratonia (inability to relax due to cognitive processing difficulties). Spasticity has a velocity and directional component. In other words, rapid changes in limb position will result in a "spastic catch" (the Jack-Knife phenomenon). Extension of the arms (at the elbow) and flexion of the legs (at the knees) is more likely to bring out increased resistance. Rigidity has increased tone in all directions that is not velocity-dependent. Dystonia is caused by over activation of agonist and antagonist muscles of the same joint concurrently (and hence is not really a "tone"issue). Patients with Dystonia tend to keep their limbs forcefully twisted in an uncomfortable position. Paratonia is inconsistent changes in tone secondary to a patient's inability to relax fully when being examined.

Posture is best observed and is important in pediatric neurology. A "Frog leg" position of the legs shows abducted, flexed, and externally rotated legs and suggests hypotonia. "Scissoring" of the legs with vertical suspension suggests increased adductor tone which is usually caused by spasticity.

Fine Motor Coordination is assessed by the patient's dexterity and speed in performing fine motor tasks. Injury to the Corticospinal Tract results in distal slowing and incoordination of fine finger movements. Likewise, many children with cognitive delays will have immature fine motor skills (fine motor dyspraxia) due to a more global "hardwiring" problem of the motor system.

Deep Tendon Reflexes (DTR's) can be graded on a 4 point scale with 1 and 2 being normal, 3 suggesting pathological briskness (with "spread") and 4 suggesting clonus. Exaggerated DTR's suggests corticospinal tract dysfunction causing disinhibition of the locally-mediated spinal reflex. Absent or suppressed DTR's may be a sign of an underlying peripheral neuropathy or myopathy.

Plantar Responses are normal if the great toe moves in a plantar (downward) fashion following an irritating stimulus to the foot. An "Extensor Plantar Response" results in extension of the great toe followed by abduction "fanning" of the rest of the toes. An "Extensor Plantar Response" suggests dysfunction to the Corticospinal Tract.

Involuntary Movements are usually due to dysfunction of the Basal Ganglia or their connections. A useful website for more information about movement disorders can be found at www.mdvu.org.

- **Chorea** comes from the Greek word "Khoreia" which means literally "to dance". Patients with chorea have rapid, near-continual, activation of regional muscle groups that move around in an unpredictable fashion. Even when sitting in a relaxed position, the erratic muscle contractions and limb movements can be felt by the examiner. Voluntary activation of the skeletal muscles of a limb (such as in performing a specific movement) will exaggerate the "choreaform" movements.
- **Athetosis** suggests a more distal "snake-like" writhing movement with twisting and incoordination. Pathological processes causing chorea will also result in athetoid movements.
- **Tremor** is an oscillatory back-and-fourth movement with equal amplitudes and frequencies in both directions of movement. Tremor can be broken down into "postural", "ataxic" and "resting" subtypes.
    - Postural Tremor (4-11 Hz) needs to be distinguished from an "exaggerated" physiological tremor. Causes include autosomal dominant Familial Essential Tremor, caffeine or sympathomimetic drug use, and hyperthyroidism. Familial Essential Tremor likely localizes to dysfunction of the Subthalamic Nucleus. Postural Tremor is brought out by voluntary activation of the skeletal muscles (by having the patient extend their arms in the air) and does not necessarily become more prominent with pointing toward a target. Action tremor should disappear when the limbs are completely rested.
    - Cerebellar Tremor (or "Action Tremor" or "Intention Tremor") (3-10 Hz) is caused by dysfunction to the ipsilateral cerebellar hemisphere. The quality of this tremor is that it increases as the patient is approaching an object with an extended index figure. Patients may also have truncal ataxia and "titubation" (back-and-fourth rocking movements when trying to stand or sit) with midline or "pan-cerebellar" dysfunction). Nystagmus may also be a clue to cerebellar dysfunction.
    - Resting Tremor (3-6Hz) is a much slower tremor that is exaggerated when the patient is awake and the limbs are at rest. It dampens during voluntary movements. It is localized to dysfunction of the Basal Ganglia (e.g. Parkinson's Disease).
- **Tics** are "un-voluntary" movements that tend to be stereotypic and affect localized groups of muscles or result in vocalizations. The can be differentiated from chorea by their stereotypic nature and their tendency to wax and wane over time (with new tics replacing older ones and older ones returning at future dates). Tourette Syndrome is diagnosed in individuals having both motor and vocal tics (though not necessarily concurrently) for greater than one year duration.
- **Parkinsonism** is defined by the symptom complex of bradykinesia, rigidity, resting tremor, and postural instability. These symptoms can be seen in Parkinson's Disease (due to degeneration of the Substantia Nigral Dopaminergic innervations into the Corpus Striatum) or from other pathological processes affecting dopaminergic transmission in the Basal Ganglia. Medication-induced Parkinsonism is most common cause of Parkinsonism not due to Parkinson's Disease. Bradykinesia is defined as a paucity of movement. Bradykinetic patients will have "masked facies", a decreased spontaneous blink rate (closer to 12 per minute compared to 24 per minute in normal individuals), a "magnetic gait", and be slow to get up when sitting in a chair. Rigidity differs from spasticity in that the increased resistance to passive range of motion is felt equally in flexion and extension and does not have a velocity-dependent component. Postural instability is a major cause of falls in patients with Parkinson's disease. It can be elicited by having a patient stand upright and then applying mild backward pressure on their shoulders and seeing how well they retain their upright posture (verses falling backwards into the arms of the examiner).
- **Dystonia** is really a misnomer as it is not a disorder of tone as much as an over action of antagonist and agonist muscles concurrently at an individual joint. Patients with dystonia tend to keep their limbs fixed in a twisted uncomfortable posture. As with other movement disorders (except for myoclonus) dystonia disappears with sleep and is more pronounced when a patient is agitated or uncomfortable. Dystonia can be further broken down into focal (a regional group of muscles), segmental (muscles from contiguous regional groups), and generalized (at least one foot/leg plus trunk or upper extremity involvement) distributions. Dystonia is often action-induced and many focal dystonias (such as writer's cramp) are induced by very specific actions.

- **Myoclonus** is a brief, shock-like contraction of a group of muscles. It may be generalized or involve focal muscle groups. Some causes of myoclonus are epileptic, though other causes may be due to metabolic disturbances (hepatic encephalopathy), or structural causes (palatal myoclonus due to injury of Mollaret's triangle).
- **Hemiballismus** is a violent, large amplitude flinging movement with arm abduction and extension. It is usually caused by ipsilateral damage to the Subthalamic Nucleus.

Sensory Examination

The sensory exam should be broken down into primary sensory modalities and cortical sensory modalities. Primary sensory modalities include light touch, vibration perception, joint position sense, and pain and temperature sensation. Deficits of primary sensory modalities are due to injury/dysfunction anywhere from the skin receptors for these modalities, the peripheral sensory nerves, the ascending spinal sensory tracts all the way up to the Thalamus. The posterior columns carry ipsilateral vibration and joint position sensation. Position sense can be tested by gently moving the patient's big toe in either flexion or extension and asking them which way the toe moved. Likewise, the Romberg sign (which is assessed by having the patient stand with arms extended and eyes closed to see if they keep their balance when vision is removed) is a function of perception of where the body is in space. Vibration sensation is assessed using a 128 Hz tuning fork and comparing your own sense with that of the patient as the vibration fades. Pain and temperature sensation are carried by small myelinated and unmyelinated sensory nerves which enter the spinal cord via the dorsal root ganglia to travel up and down 2 to 3 segments in Lissauer's Tract. From there, second order neurons originating in dorsal lamina I, IV, and V cross the midline at the anterior commissure to travel up the contralateral lateral spinothalamic tract (pain and temperature sensation) and anterior spinothalamic tract (light touch sensation) to finally reach the Thalamus. Pain sensation should be distinguished from light touch by using a sharp pin (one per patient) and cold sensation can be differentiated from warm sensation using a cold object.

Cortical processing of primary sensory input results in further processing of the "raw data" primary sensory modalities. Deficits in cortical sensory function will result in contralateral difficulties in two-point discrimination, asterognosis, agraphesthesia, and extinction. Two-point discrimination is tested by providing either a single stimulus or two side-by-side stimuli concurrently. Stereognosis is tested by placing a coin or another familiar object into the contralateral hand of the affected patient and then by asking them to identify it by touch only. Graphesthesia is tested by writing numbers (without using ink) on the contralateral palm of the patient. Extinction occurs when patients can identify stimuli individually on the right or left side of their body, but not when stimuli are simultaneously presented bilaterally.

Coordination

Coordination essentially looks at cerebellar function. From a simplistic perspective, the cerebellum can be broken down into midline functions and hemisphere functions. The cerebellar midline structures provide balance input to the truncal musculature. Midline cerebellar deficits result in truncal ataxia, an ataxic speech, impairments with tandem gait, and truncal titubation. Cerebellar hemisphere dysfunction results in ipsilateral intention tremor, dysdiadochokinesis, hypotonia, excessive rebound, and nystagmus. Intention tremor can be evaluated by having the patient point their finger from their nose to the examiner's finger. The "heel-knee-shin" test examines this function in the lower extremities. Dysdiadochokinesis is defined as difficulty with alternating supination/pronation hand movements. Rebound is the patient's ability to keep their limb in a fixed position when the examiner suddenly releases their grip on the limb. Nystagmus is commonly seen with disorders of the cerebellum or it's inputs/outputs.

Gait

Gait essentially puts together a lot of different neurological functions including vision, balance, joint position sense, strength, and postural tone. Ask the patient to walk back and forth across the room a few times. With each pass, check on a different aspect of their gait. Note the width of the base of the gait. Look for steadiness with balance. Look for fluidity in arm swing. Note if there is any crouching to the gait. Look for a well-placed heel strike with each foot and for equal amounts of time spent with each foot on the ground. When this is complete, one may wish to have the patient walk on their heels and toes and to perform an attempt at tandem walking. Classic gait abnormalities include the hemiparetic gait, the spastic diplegic gait, the high stepping gait, the waddling gait, the shuffling gait, and the ataxic gait. Try to imitate the patient's "limp" and figure out exactly what mechanics lie behind their deficits.