Basic Neurologic Life Support
Tips on the Neurologic Examination

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The goal of this chapter is to familiarize the clinician with the neurologic examination and to provide some helpful tips in obtaining or interpreting data from the examination. It is not intended as a thorough review of all details of a complete and comprehensive neurologic examination. We will try to direct some of our comments to the specific conditions discussed in more detail in the other chapters of this book. Guidelines for a comprehensive neurologic examination and an examination on a patient with an altered level of consciousness have been proposed by the American Academy of Neurology (AAN) and are outlined in Tables 1 and 2.

General Examination

A general physical examination certainly complements a neurologic examination when evaluating patients with neurologic diseases.

Head and Neck Examination: In patients with altered levels of consciousness it is important to inspect and palpate the patient's head for signs of trauma including looking for blood behind the ear (Battle's sign) and around the eyes (raccoon eyes). The nose should be inspected for blood or CSF drainage. Otoscopy should be done to look for blood behind the eardrum. Auscultation for bruits should be done as part of the general cardiovascular examination. A stethoscope bell can be placed along the anterior border of the sternocleidomastoid muscle in the region of the upper border of the thyroid cartilage to listen for cervical carotid artery bruits and placed along the posterior border of the sternocleidomastoid to listen for vertebral artery bruits. The bell can also be gently placed over closed eyes to listen for ocular bruits. Auscultation of the mastoid processes, temporal, frontal, or parietal head regions may be helpful in certain circumstances. Irritation of the subarachnoid space, by infection or blood, can lead to nuchal rigidity, a

resistance to active and passive neck flexion. No neck manipulations should be done until a cervical spinal cord lesion or a fracture or dislocation of cervical vertebrae has been ruled-out with appropriate radiologic tests. Brudzinski's neck sign is noted if passive flexion of the head is followed by flexion of both thighs and legs. Kernig's sign can also be used to test for meningeal irritation. The hip and knee are flexed and then the knee/leg is slowly and gently passively extended looking for pain and resistance to leg extension. In patients with suspected giant cell arteritis, the superficial temporal arteries should be palpated for tenderness or induration. The temporal areas and scalp should be palpated for tenderness.

Chest/respiration: In patients with neuromuscular respiratory difficulties there are some bedside tests of function. The simplest method is to ask the patient to count aloud on a single deep breath. If the patient can count to 10, the forced vital capacity (FVC) is approximately 1 liter. If the patient can count to 25, the FVC is approximately 2 liters. Other respiratory patterns may be helpful in localizing lesions in patients who are comatose. These will briefly be discussed in the section on examining the comatose patient.

Cardiovascular: An irregularly irregular pulse may be indicative of atrial fibrillation, multifocal atrial tachycardia, premature atrial contractions, or premature ventricular contractions.

Abdomen: In encephalopathic patients, hepatomegaly and splenomegaly may be a sign of hepatic disease with portal hypertension. Massive splenomegaly may be seen in malaria. Abdominal striae may be a sign of Cushing's syndrome.

Extremities / spine: In patients with spinal cord or spinal column trauma, the spine, especially the cervical spine, should be immobilized until appropriate radiologic studies are done. In patients with potential spinal cord lesions the spinous processes at the appropriate level may be tender to palpation.

Skin: Observation of the skin may be helpful. Skin temperature and color may help lead to a diagnosis. One should inspect the skin for ticks, needle marks or characteristic rashes. Characteristic rashes may give away the diagnosis in infectious diseases such as Rocky Mountain Spotted Fever or meningococcal meningitis. Livedo reticularis may be seen in younger patients with ischemic stroke due to hypercoagulable states such as antiphospholipid antibody syndrome. Kaposi's sarcoma may be seen in HIV positive patients.

A Comprehensive Neurologic Examination

Guidelines for a comprehensive neurologic examination have been published by the AAN and are noted in table 1. The major sections of the neurologic examination include mental status, cranial nerves, motor function, reflexes, and sensation. We will discuss these individually and give some helpful hints on interpreting observations made during the examination.

Much information regarding a patient's mental status will be noted while obtaining the history. The patient's level of alertness is noted when speaking with the patient. Asking the patient simple orientation questions such as their name, the date, and their location will give you quick information on their sensorium. Having them recite the months or spelling world backwards can test their attention span. The fluency of speech and ability to comprehend spoken language are noted when the patient answers questions.

Asking the patient to name objects, repeat a phrase, read and write will complete a quick screen of language function. Asking the patient to remember 3 objects can test short-term memory. Simple calculations can be done on a piece of paper. Visuospatial processing can be tested by asking the patient to reproduce drawn objects such as interlocking pentagons or perhaps drawing the face of a clock. Asking the patient to interpret the meaning of proverbs can test abstract reasoning.

Testing cranial nerve (CN) function is most easily done by remembering and testing them in numerical order. The olfactory nerve (CN I) is not often tested in a routine neurologic examination. However, having the patient identify a common smell such as coffee or wintergreen with each nostril separately is a reasonable first test. A noxious or irritating odor, such as ammonia, should not be used to test olfaction as these odors also activate the fibers of the trigeminal nerve (CN V). The optic nerve (CN II) plays a major role in vision and is the afferent limb of the pupillary reflex. Visual acuity is best tested using a distance chart but can be tested with a pocket visual chart. When testing visual acuity patients should wear their appropriate corrective lenses. Reading glasses should be worn if necessary for near vision when using a pocket visual chart. Confrontation visual fields should be done with the patient covering one eye with a hand. A number of the examiner's fingers can be presented in each of the four quadrants of each eye and the patient is asked to state how many fingers were presented. Visual field testing is often not done or forgotten by those not experienced in the neurologic examination but often provides useful information. For example, a homonymous hemianopia may be the most dramatic sign of an occipital lobe infarction. The pupillary light reflex is one of the most important parts of the neurologic examination. Normally,

both pupils will promptly constrict when light is focused on either retina giving both the direct and consensual response. The alternating light test is the standard clinical technique to look for a relative afferent pupillary defect (RAPD). Swinging the light from eye to eye will elicit a brisker reaction to light in the unaffected eye and a less brisk reaction or dilatation of the pupil in the affected eye. A RAPD is a sensitive indicator of a unilateral injury to the afferent pupillary pathway, typically large retinal lesions or damage to the optic nerve, which is most common. For example, an ischemic optic neuropathy associated with giant-cell arteritis may give an RAPD. A funduscopic examination is extremely important in establishing the appearance of the optic disc, retina and macula. Ideally, the fundus should be viewed after pharmacologic mydriasis to ensure the best view. Papilledema, or a swollen optic disc, is often a sign of increased intracranial pressure and makes funduscopy one of the most important parts of the examination of patients with headaches. Papilledema can be seen in conditions that globally increase intracranial pressure such as meningitis, subarachnoid hemorrhage, or intracranial mass lesions. A pallid swollen disc is often seen in giant-cell arteritis. Cholesterol emboli (Hollenhorst plaque) may be indicative of ulcerating atheromatous plaque in the ipsilateral carotid artery. The oculomotor nerve (CN III), trochlear nerve (CN IV) and the abducens nerve (CN VI) innervate the muscles that move the eyes or extraocular muscles. The abducens nerve innervates the lateral rectus muscle. The trochlear nerve innervates the superior oblique muscle. The oculomotor nerve innervates the rest of the extraocular muscles which include the medial rectus, inferior rectus, superior rectus, and inferior oblique muscles. The oculomotor nerve also innervates the muscles that constrict the pupil and the levator palpebrae superioris that helps elevate the

eyelid. The patient should be asked to follow an object that tests all directions of movement of both eyes. In a complete CN III palsy there is ptosis or drooping of the eyelid, dilatation of the pupil, and the eye is deviated laterally and a bit downwards. The trigeminal nerve (CN V) innervates the muscles of mastication or chewing and is responsible for facial sensation. The 3 main branches of the trigeminal nerve, the ophthalmic branch, maxillary branch, and mandibular branch, provide sensation for the forehead, cheek, and chin, respectively. Facial sensation can be tested much the same way as sensation in other parts of the body is tested. This is outlined below. The facial nerve (CN VII) innervates the muscles of facial expression. The most easily tested facial muscles include the frontalis which wrinkles the forehead, the orbicularis oculi that close the eye tightly, and the buccinator which helps one smile. The distribution of facial weakness will help localize lesions. A lesion of the facial nucleus, the facial nerve fascicles within the brainstem or of the facial nerve itself can lead to a lower motor neuron type of facial weakness where all ipsilateral facial muscles will be equally weak. If the corticobulbar fibers are affected, an upper motor neuron lesion, then the lower face will be much more affected than the upper face as the neurons guiding upper facial muscles receive bilateral innervation. For example, the entire ipsilateral face will be weak in a typical idiopathic facial nerve palsy or Bell's palsy. In a patient with a hemispheric infarction, typically the smile is much weaker than forehead wrinkling and in fact the frontalis function may be normal. The vestibulo-cochlear nerve (CN VIII) is essentially two fiber systems which participate in hearing (cochlear nerve) and balance, equilibrium, and orientation in space (vestibular nerve). Hearing can be tested in several ways. The ability of the patient to hear two fingers rustled together, hear whispered

words, or detect the sound of a vibrating tuning fork can be used. The glossopharyngeal nerve (CN IX) and the vagus nerve (CN X) can be quickly tested by asking the patient to raise the soft palate. In a unilateral lesion, the uvula deviates to the opposite side and the ipsilateral soft palate will not elevate. The gag reflex is discussed below. Dysarthria, or difficulty with articulation of speech due to disturbances of muscular control, can be seen in both central and peripheral nervous system disorders and therefore can be seen in many lesions of the nervous system. The spinal accessory nerve (CN XI) innervates the sternocleidomastoid (SCM) muscles and the rostral or proximal parts of the trapezii muscles. It can be tested by having the patient shrug the shoulders (trapezii) and having them attempt to return the head to the midline position against resistance after it has been turned to the right and left. It is best to actually palpate the SCM muscle being tested so that activation is felt. It is important to realize that the left SCM helps turn the head to the right and the right SCM helps turn the head to the left. The hypoglossal nerve (CN XII) innervates the tongue musculature. The tongue should be observed for atrophy and fasciculations as a clue to lower motor neuron lesions. The patient is then asked to protrude the tongue. If there is a unilateral weakness then the tongue will protrude towards the side of the lesion.

Examination of motor function is the next step in the neurologic examination. While the testing of power or strength is important in examining the motor system there are certainly other techniques that should not be missed. Inspection of overall and individual muscle bulk looking for atrophy or hypertrophy may be helpful in formulating a diagnosis. Testing of muscle tone, or resistance to passive manipulation, is also important and may be normal, decreased, or abnormally increased. Muscles being tested

for tone should be relaxed. The most common forms of increased muscle tone are spasticity and rigidity. Neuroleptic malignant syndrome is associated with severe generalized muscle rigidity. The patient should be observed for involuntary hyperkinetic movements such as tremor, chorea, myoclonus, hemiballismus, athetosis, and dystonia. The testing of muscle power or strength is the most used part of the examination of the motor system and is the part most familiar to all physicians. While there are a multitude of muscles that can be tested for power the main movements tested in the routine neurologic examination include shoulder abduction, elbow flexion and extension, wrist flexion and extension, finger flexion, extension and abduction, hip flexion and extension, knee flexion and extension, and ankle dorsiflexion and plantar flexion. Muscle power can be rated on a five-point scale as listed in table 3. A subtler test of upper extremity power is asking the patient to hold their arms and hands outstretched in front of them with eyes closed. If there is mild weakness, there may be slow pronation of the hand, slight flexion of the wrist and elbow and downward drift of the arm. The testing of patients with suspected hysteria or malingering may show give-away weakness. The muscle being tested may not sustain contraction and may give way abruptly rather than gradually. There may also be inconsistency in the amount of power exerted for the individual muscle groups. Coordination is the smooth execution of motor movements. While cerbellar lesions often cause incoordination or ataxia, it can also be seen in lesions of the pyramidal system, extrapyramidal system and sensory system. The most common methods of detecting incoordination are the finger-to-nose test in the upper extremities and the heel-to-shin test in the lower extremities. Rapid alternating movements can also be done to test coordination and dexterity. Having the patient tap on the thigh with the

palm and dorsum of the hand alternately or tapping the toes on the ground can test for impairment of rapid alternating movements. The testing of muscle stretch reflexes is also an important part of the motor exam. The most commonly tested muscle stretch reflexes are the biceps, triceps, brachioradialis, patellar (or knee jerk), and Achilles (or ankle jerk). The part of the body to be tested should be relaxed and the best position is usually intermediate between full extension and flexion. Muscle stretch reflexes can be graded on the scale presented in table 4. Often it is not the absolute number assigned to the reflex but the degree of symmetry or asymmetry noted. The most important superficial reflex is the plantar response. With the patient supine and relaxed, the lateral sole of the foot is stimulated starting near the heel and moving towards the toes. The toes will flex in a normal plantar response. If the corticospinal tract is interrupted or diseased, the great toe will dorsiflex and the other toes often fan out. This great toe extension to plantar stimulation is the Babinski sign. When there is muscle weakness noted, the most fundamental question to be answered by the examination is whether a lower motor neuron or upper motor neuron lesion is present. Chronic upper motor neuron lesions (those affecting the corticospinal tract) classically show weakness, increased tone/spasticity, hyperreflexia, and a Babinski sign in the appropriate distribution based on the anatomic location of the lesion. Chronic lower motor neuron lesions classically show weakness, decreased tone, hypo- or areflexia, and flexor or neutral plantar response again in the appropriate distribution based on the anatomic location of the lesion.

The sensory examination is the most subjective part of the neurologic examination because it relies on the responses of the patient and not on the examiner's observations. It is often also the most tedious and time-consuming part of the

examination. At times, it seems unreliable and confusing. However, much information can be gained by an appropriate sensory examination. The most common modalities tested are light touch, pain or temperature sensation, proprioception, and vibration. Light touch can be tested with a cotton swab or a gentle touch with the examiner's finger. Pain sensation can be tested with a sharp object such as a safety pin. It is important to use sharp objects only once and dispose of them appropriately because of the possibility of transmitting pathogens, especially viruses, between patients. Temperature sensation can be tested using the cool end of a tuning fork and comparing to the examiner's warmer finger. Vibration is tested with a tuning fork. Proprioception is tested by moving an isolated toe or finger up or down, often by only a few millimeters, and asking the patient which direction the digit was moved. It is important to hold the digit on the lateral portions or sides and not the top or bottom as the patient may sense the pressure direction and correctly state the direction of movement based on the pressure sensation and not a true awareness of the joint position. Certainly not every square inch of the skin is examined completely in every examination as this would be impractical. However, a detailed examination of the appropriate anatomy should be done in any patient with a complaint of sensory changes such as numbness, pain, or tingling. It is important to test the main sensory modalities as they take different anatomic paths within the central nervous system, especially the spinal cord. Pain and temperature sensations are carried by the spinothalamic tracts and vibration and proprioception are carried in the dorsal columns of the spinal cord. In testing sensation of all types, the most accurate results are obtained by proceeding from the area of less sensation to the area of more or normal sensation. The pattern of sensory loss often guides in localizing a lesion within the

nervous system and some specific patterns are noteworthy. For example, in hemispheric brain infarcts the pattern is typically one of loss of all modalities of sensation on the contralateral face, body and extremities. In peripheral neuropathies the sensory changes are most often distal in the extremities with the legs often being more affected than arms. With lesions compressing the spinal cord there is most often loss of sensation distal to the lesion. Some noteworthy anatomic landmarks include the nipple level which is at the T4 sensory level and the umbilicus which is at the T10 sensory level. Sensory loss may occur in the perianal and genital areas, a "saddle distribution", in lesions of the cauda equina or conus medullaris.

Examination of gait should be done in every person capable of walking. In fact, if given only one part of the examination with which to make a diagnosis, most neurologists would choose the station and gait examination. An integration of most parts of the nervous system is necessary to walk normally. The patient should be observed walking with as little assistance as necessary to keep them safe. If possible, they should be asked to walk on their heels and toes. Tandem walking, or walking along a straight line one foot directly in front of the other, should be attempted in all patients not a significant fall risk. Certainly some gait patterns can give away a diagnosis. Easy examples being the hemiparetic gait of patient who has had a stroke, the shuffling gait of a patient with Parkinsonism, or the ataxic gait of a patient with cerebellar disease.

Examination of the patient in coma or altered level of consciousness

Guidelines for the examination of patients with altered levels of consciousness are presented in Table 2. In general any part of the comprehensive neurologic examination listed previously that can be performed should be performed. However, many aspects of

the comprehensive neurologic examination cannot be performed in patients unable to follow commands or follow a course of action. Other reflex testing is often done in comatose patients that is not routinely performed on alert patients. These will be highlighted in this section.

A general physical examination should be performed in all patients with an altered level of consciousness and may be helpful in arriving at a cause. Some examples are listed above in the general examination section. Simple methods such as smelling the patient's breath may prove helpful in determining a cause, especially toxic ingestions. For example, a garlic breath odor may be seen in arsenic poisoning and cyanide poisoning may cause a bitter almond odor. Ethanol ingestions can often be diagnosed by the characteristic breath odor.

As implied by their name, evaluation of vital signs is important in the examination of a comatose patient. Hypertension may be due to increased intracranial pressure or just be due to chronic primary hypertension. Hypotension may be due to hypovolemia, hemorrhagic shock or myocardial depression. Hyperthermia can be seen in infections such as meningitis, encephalitis or sepsis. It can also be seen in disruption of autonomic pathways, neuroleptic malignant syndrome or a serotonin syndrome. Hypothermia may be due to exposure (environmental), hypoglycemia, or hypothyroidism. Wernicke's encephalopathy and sedative overdose can also lower body temperature. Respiratory patterns may also be helpful in diagnosing the cause of the altered level of consciousness or the location of a lesion in the brain. Hyperventilation can be seen in metabolic acidosis, pulmonary edema, primary respiratory alkalosis, or from central neurogenic hyperventilation. Hypoventilation may be due to pulmonary insufficiency, central

alveolar hypoventilation or other causes. An abnormal breathing pattern may point to a lesion in a specific location within the brain. Cheyne-Stokes respiration or periodic breathing is probably the most common pattern is typically caused by bilateral cerebral hemisphere dysfunction or dysfunction of the diencephalon. Central neurogenic hyperventilation is seen in low midbrain or upper pontine lesions. Dysfunction or lesions of the mid-caudal pons often cause Apneustic breathing. Ataxic breathing is caused by medullary lesions.

The patient's state of consciousness is measured by their awareness and responsiveness to the environment. There are many levels or states of awareness and they have been identified by many descriptors such as stupor, lethargy, somnolence, confusion, delirium, etc. The definitions for these states are a bit variable and are likely a continuum from normal to coma. Coma is defined as a state of complete loss of consciousness or a state of unarousable unresponsiveness. In practice it is most helpful to describe the patients response to stimuli. These include auditory stimuli such as a voice or loud clap, visual stimuli such as light or threatening gesture, or noxious stimuli applied in central locations (an example being the sternum) and each limb. Responses such as arousal, eye opening, or grimacing should be noted.

There are several cranial nerve reflexes that can be done to test the integrity of the brainstem from the midbrain to the medulla. The pupillary light reflex is relatively resistant to metabolic insults and is one of the most important signs in distinguishing structural from metabolic causes of coma. Again, the afferent limb of this reflex is some optic nerve fibers and the efferent limb is the oculomotor nerve (CN III). For example, a structural lesion compressing CN III will typically cause an ipsilateral fixed and dilated

pupil. A pontine lesion may cause pinpoint pupils that require a magnifying glass to see the preserved trace light reflex. Any eye movements present and their pattern should be noted. Roving conjugate spontaneous eye movements imply that the nuclei responsible for ocular motility in the midbrain and pons are communicating with each other. Eye movements can be induced by the oculocephalic (doll's eyes) and vestibulo-ocular (caloric testing) reflexes. The oculocephalic reflex is easier to accomplish but is less of a stimulus to eye movements than the vestibulo-ocular reflex. In a comatose patient lying supine who may be intubated, performing horizontal oculocephalics is simply easier but vertical oculocephalics can also be done. Before performing the oculocephalic reflex one must be sure there is no abnormalities of the cervical spine. The head is then quickly turned from side to side. If the brainstem is intact then the eyes will move in the direction opposite to the skull movement and stay looking forward. The corneal reflex is done by touching the lateral portion of the cornea with a non-abrasive stimulus such as a wisp of cotton. A positive corneal reflex occurs when both eyes blink to the stimulus. The afferent limb of this reflex is the trigeminal nerve (CN V) while the efferent limb is the facial nerve (CN VII). The gag reflex is produced by stimulating the posterior tongue or pharynx and looking for elevation and constriction of the pharyngeal muscles which often produces a cough, gag, or swallow. This reflex may be difficult to do in intubated patients or in those with an altered level of consciousness. It can also lead to aspiration pneumonia. The gag reflex may be an overrated test and given its potential for causing problems it is often skipped in the evaluation of comatose patients.

In testing the motor function of a comatose patient, inspection of muscle bulk and testing of muscle tone should not be forgotten. Obviously, the comatose patient cannot

cooperate with formal testing of the power of all muscle groups but some information regarding motor function can be deduced from some simple maneuvers. Watching for voluntary movements may show an asymmetry between the two sides. Reflexive posturing, either decerebrate or decorticate, may indicate fairly severe brain injury. Reflexive withdrawal of the extremities to painful stimulation such as nail-bed pressure may be the only way to see movement of the extremities. Testing the muscle stretch reflexes and evaluating plantar responses is necessary. Asterixis, which is commonly sought in the upper extremities, may be indicative of a variety of metabolic encephalopathies or focal brain lesions. Testing of sensation is often extremely limited except for gross evaluation of responses to pain.

Despite what appears to be a limited neurologic examination in the comatose patient, quite a bit of useful information can be gained. Often a tentative diagnosis can be made on the basis of the examination alone. For example, a patient with intact brainstem reflexes, symmetric spontaneous and induced movements of the face and extremities, and symmetric reflexes the most likely cause of their coma would be a metabolic derangement that is affecting the brain diffusely. If a comatose patient with bilateral papilledema, a unilateral CN III palsy with a fixed and dilated pupil and no movement of the contralateral body one must be terribly suspicious of a mass or structural lesion.

Table 1: Guidelines for a Comprehensive Neurologic Examination

1. Mental Status
    a. Level of Alertness
    b. Language function
        i. Fluency
        ii. Comprehension
        iii. Repetition
        iv. Naming
    c. Memory (short-term and long-term)
    d. Calculation
    e. Visuospatial processing
    f. Abstract reasoning
2. Cranial Nerves
    a. Vision
        i. Visual Fields
        ii. Visual Acuity
        iii. Funduscopic examination
    b. Pupillary light reflex
    c. Eye movements
    d. Facial sensation
    e. Facial strength – muscles of facial expression
    f. Hearing
    g. Palatal movement
    h. Speech
    i. Neck movements
        i. Head rotation
        ii. Shoulder Elevation
    j. Tongue movements
3. Motor Function
    a. Gait
        i. Casual
        ii. On toes
        iii. On heels
        iv. Tandem
    b. Coordination
        i. Fine finger movements
        ii. Rapid alternating movements
        iii. Finger-to-nose
        iv. Heel-to-shin
    c. Involuntary movements
    d. Pronator drift
    e. Tone (resistance to passive manipulation)
    f. Bulk
    g. Strength
        i. Shoulder Abduction
        ii. Elbow flexion and extension

iii. Wrist flexion and extension
iv. Finger flexion, extension, and abduction
v. Hip flexion and extension
vi. Knee flexion and extension
vii. Ankle dorsiflexion and plantar flexion

4. Reflexes
    a. Muscle stretch reflexes
        i. Biceps
        ii. Triceps
        iii. Brachioradialis
        iv. Patellar
        v. Achilles
    b. Plantar responses
5. Sensation
    a. Light touch
    b. Pain or temperature
    c. Proprioception
    d. Vibration

Table 2: Guidelines for the Neurologic Examination in Patients with Altered Levels of Consciousness

1. Mental Status
    a. Level of Arousal
    b. Response to auditory stimuli (including voice)
    c. Response to visual stimuli
    d. Response to noxious stimuli applied both centrally and each limb
2. Cranial Nerves
    a. Response to visual threat
    b. Pupillary light reflex
    c. Oculocephalic (doll's eyes) reflex
    d. Vestibulo-ocular (caloric testing) reflex
    e. Corneal reflex
    f. Gag reflex
3. Motor Function
    a. Voluntary movements
    b. Reflex withdrawal
    c. Spontaneous and involuntary movements
    d. Tone (resistance to passive manipulation)
4. Reflexes
    a. Muscle stretch reflexes
    b. Plantar responses
5. Sensation (to noxious stimulation)

Table 3: Grading of Muscle Power

0	No contraction
1	Flicker or trace of contraction
2	Active movement with gravity eliminated
3	Active movement against gravity
4	Active movement against gravity and resistance
5	Normal power

Table 4: Grading of Muscle Stretch Reflexes

0+	Absent
1+	Present but diminished
2+	Normal
3+	Increased but not necessarily to a pathologic degree
4+	Markedly hyperactive, often with associated clonus

Recommended readings:

1. DeMyer WE. Technique of the Neurologic Examination – A Programmed Text – 4th edition. 1994. McGraw-Hill. New York
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