Neurological Assessment Tips

- If a patient develops any decrease in level of consciousness, the priority is to promptly identify and treat any alterations in ABCGS (Airway, Breathing, Circulation, Glucose or Seizures) that may be causing the deterioration.

- If the neurological change persists despite normalization of the ABCGS, a detailed neurological assessment should be performed. The examination should attempt to determine if focal findings are present (suggesting a structural abnormality, such as stroke, bleed, tumour, etc.) or absent (suggesting generalized neurological depression, as seen with sedation or septic encephalopathy, etc.). Bilateral findings may also suggest cord injury.

- Change is the most important finding in any neurological assessment and should be reported promptly to ensure timely medical intervention if warranted. To ensure that neurological findings are communicated effectively at change of shift, exiting nurses should perform a neurological examination with the oncoming shift.

- Propofol may be used to sedate patients with neurological impairment to facilitate rapid awakening and assessment. Remember that propofol does not provide analgesia, and pain can raise intracranial pressure. In patients with catastrophic brain injury who are being sedated for raised intracranial pressure, deep analgesia and sedation is provided to promote brain rest. Analgesia and sedation should not be stopped for routine neurological assessment unless approved by neurosurgery.

Steps to Neurological Assessment in the Critical Care Unit:

1. Assess mental status/higher function:

A. Conscious patient:

1) Talk to patient and ask questions that avoid yes/no answers if possible.
   - Evaluate orientation, attention, coherence, comprehension, memory/recall
   - Screen for delirium
   - Identify symptoms such as headache, nausea or visual problems
2) Determine Glasgow Coma Scale (GCS)

B. Altered patient:

1) Assess for response to:
   a) Normal voice
   b) Loud voice (if no response to normal voice)
   c) Light touch (if no response to loud voice)
   d) Central pain (if no response to light touch)

   Differentiate between higher function of “awareness” (e.g., purposeful movement, recognition of family) versus arousability (grimacing or eye opening to pain only)
2) Determine Glasgow Coma Scale (GCS)

2. Consider whether seizures could be present

Look for evidence of seizures (non-convulsive seizures should be considered in patients with an unexplained decrease in level of consciousness, particularly in the setting of injury, stroke, tumour etc)

3. Test Cranial Nerves (see next page)

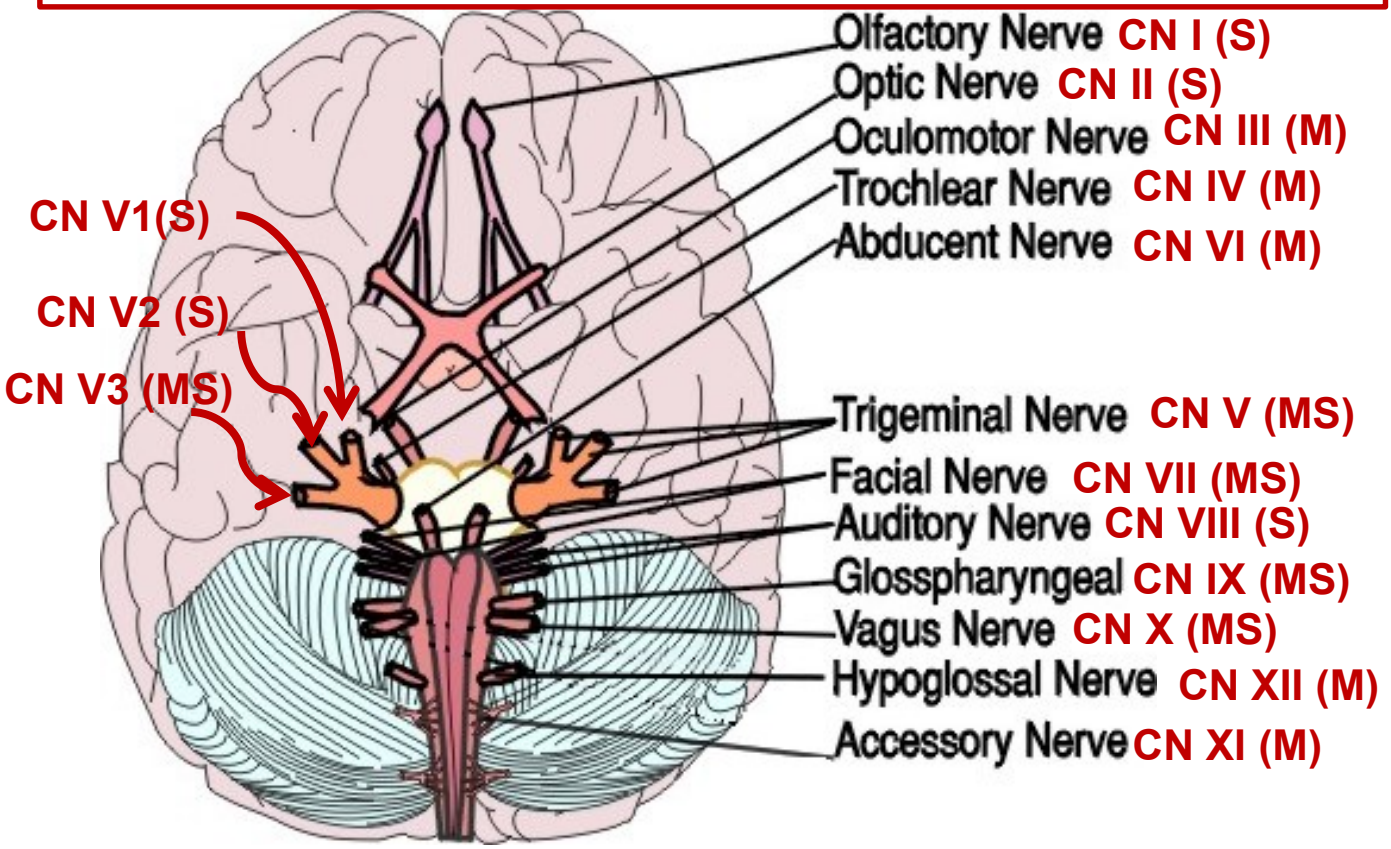
4. Assess motor function (look for asymmetry)

5. Assess sensory function (look for asymmetry)

6. Assess cerebellar function

CRANIAL NERVES:

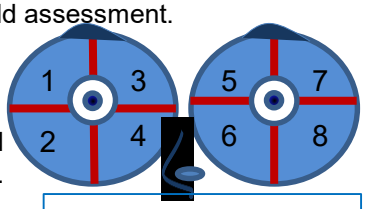
- The cranial nerves are arranged in pairs in descending order along the brainstem.
- There are 3 sensory (S) nerves (CN I, II and VIII), 5 motor (M) nerves (CN III, IV, VI, XI and XII) and 4 mixed motor and sensory (MS) nerves (CN V, VII, IX and X). CN IX (accessory is actually a spinal nerve that arises upward toward the brainstem.
- Cranial nerves control the same side of the body (ipsilateral response). For example, an expanding lesion that compresses on the R CN III would cause a loss of the R CN III function. This would mean that the right pupil would lose its ability to constrict to light (or become fixed and dilated). Exception: CN IV controls contralateral funtion (moves eye downward toward the nose).
- All cranial nerves can be tested in alert patients who are able to participate in the examination.
- Only some of the cranial nerves can be tested if the patient is unconscious. Pairs of cranial nerves can be tested by stimulating a sensory nerve and watching for an automatic motor nerve response. Light reflex is an example; light is carried toward the brain via CN II (optic, sensory nerve). This automatically causes reflex stimulation of both CN IIIs (oculomotor, motor), making both pupils constrict to light.
- When brainstem herniation syndromes occur, cranial nerve function can be lost in descending order (if the origin of the injury is above the tentorium and compression moving downward).
- CN I and II are located above the brainstem; CN III through XII are located along the brainstem.
- CN III is located at the level of the tentorium; sudden change in CN III function (decreased reactivity, droopy eye, dilated pupil) suggests herniation through the tentorium/top of brainstem.
- Asymmetrical loss of any CN function suggests unilateral compression
- Because of their arrangement along the brainstem, most brainstem reflex tests are tests of cranial nerve function.



CN	Name	Main Function	Testing in ICU (assess symmetry)
I	Olfactory (S)	• Sense of smell (may be injured with anterior basal skull #)	• Block one nare and test ability to smell equally from each nare (cloves, coffee) • Dysfunction causes food to lose its taste
II	Optic (S)	• Sense of sight Each of the 4 quadrants of vision in each eye (visual fields) take a unique pathway between the retina and brain. One or more visual fields can be lost due to damage to the retina, a branch of the optic nerve or the occipital lobe (visual cortex).	• Recognition of objects or people. • Eye chart, reading • If alert, ability to see objects in all 8 fields. • Light reflex tests CN II and III • Remember to test with glasses on
III	Oculomotor (M)	• Pupil constriction • Eyelid opening • Eye movement (all directions ***except*** those of CN IV and VI; CN III, IV and VI tested together for eye movement)	• Light reflex • Eye opening against resistance • Ability to follow an object upward, horizontally toward nose, straight down and downward/laterally
IV	Trochlear (M)	• Contralateral downward and nasal rotation of eye. • Arises posteriorly. Crosses behind brainstem and emerges anteriorly. R CN IV is between L CN III and V.	• Ability to look down toward nose. • The only CN that arises posteriorly and controls contralateral function • Compression usually occurs after it crosses causing ipsilateral findings.
V	Trigeminal (SM)	• Primary Sensory: feeling to face in three branches: V1(forehead, cornea, nose), V2 (cheeks), V3 (jaw) • Motor: Chewing or mastication (V3)	• Sensory: assess each region with tissue (light touch) and pin (pain). Assess for symmetry. • Motor: ability to lift cheeks • Corneal reflex tests V1 branch of CN V (sensation) and CN VII (blink)
VI	Abducens (M)	• Horizontal and lateral movement of the eye	• Ability to follow an object in the horizontal/temporal gaze
VII	Facial (M/S)	• Primarily Motor: face movement, eyelid closure, lacrimation, salivation • Sensation/taste to front 2/3 tongue	• Face symmetry for eye closure against resistance, face movement (smile, nasolabial fold, show teeth) • Loss of forehead wrinkle on paralyzed side suggests CN VII dysfunction versus stroke
VIII	Auditory or vestibulocochlear (S)	• Hearing and balance (cochlear) • Vestibular system sends information about head movement to pons; makes CN III/VI move eyes together for horizontal movement	• Response to voice, tuning fork • Balance during mobilization • Detailed testing by audiology post ICU discharge • Included in Doll's eyes/Cold Calorics
IX	Glossopharyngeal (MS)	• Sensation to back of tongue/tonsils • Parotid secretion • Stylopharyngeus muscle	• CN IX and X collectively tested by touching each side of the back of the throat and observing for gag
X	Vagal (MS)	• Contraction larynx/pharynx • Parasympathetic function all organs except adrenal glands • Sensation pharynx, taste	• Barro/chemo receptors important for BP regulation • Cough reflex during suctioning
XI	Accessory/ spinal (M)	• Shoulder shrug • Head rotation	• Ability to shrug or turn cheek against resistance
XII	Hypoglossal (M)	• Tongue movement	• Ability to move tongue side to side

Cranial Nerve Testing: Awake Patient

1. **Assess sense of smell (CN I [Olfactory]):**
   - Block each nare individually and test ability to smell a strong aroma such as cloves or coffee. Assess for symmetrical sensation (omitted in most critical care assessments)
2. **Evaluate ability to see (CN II [Optic]):**
   - If patient wears glasses, test with glasses on.
   - Assess ability to identify objects or the number of digits held up by examiner. If unable to speak, have them mimic actions or number of digits. Does patient recognize family members?
   - Observe response to visual stimulation from either side of bed; occipital lobe stroke causes loss of vision to the opposite visual field of one or both eyes (e.g., a left occipital lobe stroke can cause blindness to the right visual field of the right and/or left eye).
   - Assess ability to read
   - While looking ahead, ask patient to indicate when he/she can see a pen that is wiggled into each of the 8 visual fields shown. Deficits require follow-up for proper visual field assessment.
3. **Light Reflex (CN II [Optic] and CN III [Oculomotor]):**
   - Conduct 4 point assessment: a) direct light response in L eye; b) direct light response in R eye; c) consensual response in L eye; and d) consensual response in R eye. Record pupil size when exposed to ambient room light. Hold both lids open to determine size/symmetry.



Visual fields of each eye

4. **Eye Opening (CN III [Oculomotor]):**
   - Ask patient to open eyes wide; observe for upward movement of lids.
   - Look at the white portion of each eye. Ptosis (eyelid droop) may be present if there is less white showing on the affected side.
5. **Eye Movement (EOM) (CN III [Oculomotor], IV [Trochlear] and VI [Abducens]):**
   - Hold a pen in front of the patient. Stand at least a couple of feet away.
   - Ask patient to follow the pen as you SLOWLY move it horizontally, vertically and diagonally, in both directions. Follow eye movements into extreme vertical and horizontal positions.
   - Eye movements should be conjugate (together). Dysconjugate gaze causes diplopia and may be due to CN III, IV or VI dysfunction, disorders of one of the muscles involved in eye movement or during recovering from coma (not awake enough).
   - Observe for nystagmus (extra eye movements). Nystagmus can be normal in the extreme horizontal gaze but never in vertical gaze.
5. **Facial Sensation (CN V [Trigeminal]; test 3 branches [V1, V2 and V3] independently):**
   - Sensory: Preferably with patient's eyes closed, touch each side of the forehead (V1), cheek (V2) and jaw (V3) with a whisp of tissue (light touch). Repeat with a blunt needle (pin).
   - Ask patient to identify when they perceive the stimulus; assess for symmetry.
   - Motor (V3): Place two fingers on each of the patient's cheeks and ask him/her to raise them.
6. **Facial Movement (CN VII [Facial]):**
   - Have patient smile, show teeth and wrinkle forehead. Observe nasal labial fold and symmetry.
   - Ask patient to close eyelids tightly; assess ability to keep eyes closed against resistance. CN VII protects the eye (eyelid closure/lacrimation). CN VII produces saliva/sensation to anterior tongue.
7. **Hearing (CN VIII [Auditory]):**
   - Arise in pons. Critical care screening includes response to voice or loud noises. Comprehensive testing following critical care discharge by audiologist.
   - Tinnitus, vertigo and nausea with upright positioning, or impaired horizontal eye movement may indicate CN VIII or pons disorders.
8. **Gag Reflex (CN IX [Glossopharyngeal] and X [Vagus]).**
   - Touch back of throat (on each side) and assess for gag. Assess for cough during suctioning.
9. **Assess Shoulder Shrug and Face Turning (CN XI [Accessory]).**
   - Ask patient to raise both shoulders and hold up against resistance; observe symmetry.
   - Have patient turn head side-to-side. Repeat while you apply resistance to cheek.
10. **Assess Tongue Movement (CN XII [Glossopharyngeal]).**
    - Ask patient to stick out tongue and move it side to side, can test against resistance.

Neurological Assessment Tips

Brainstem Testing: Unconscious Patient:

Light reflex (CN II [Optic], III [Oculomotor]):

- Light impulse is carried to CN III via CN II.
- Light shone into either eye causes bilateral CN III stimulation (pupils constrict). Both pupils constrict to light that is shone into either eye (direct and consensual response).
- If the pupil reacts to light from either direction, it is probably not a CN III problem.
- Record pupil size and symmetry when both lids are open in response to ambient room light.

Corneal reflex (V1 branch of CN V [Trigeminal] and CN VII [Facial]):

- Touching the cornea causes blink response. The sensation is detected by the first branch of CN V (V1 branch), which stimulates CN VII bilaterally to cause both eyes to blink.
- Be careful to "sneak in from the side" when touching the cornea (with a whisp of tissue). If the patient blinks because they see you, you have tested CN II and VII. If they blink because they hear you, you have tested CN VIII (Acoustic) and VII.
- Blinking of only one eye suggests weakness on the side of the face where the blink is absent.

Doll's Eyes or Oculocephalic reflex (CN III [Oculomotor], VI [Abducens] and VIII [Acoustic] and pons)

- Normally, when the head is turned, the vestibular apparatus (CN VIII) is activated, causing the eyes to move in the opposite direction. CN VIII communicates in the pons to activated both CN III and VI to produce horizontal eye movement.
- Doll's eyes CONTRAINDICATED IF C-SPINE UNCLEARED or vertebral vessel disease.
- Vertical eye movement is located at top of brainstem (CN III) and frontal eye fields. This loop ensures that both CN III look upward together.

Cold Caloric or Oculovestibular reflex (CN III [Oculomotor], VI [Abducens] and VIII [Auditory] and pons)

- If done in an awake patient, will cause vertigo, nausea and nystagmus (involuntary and erratic eye movement)
- Integrity of eardrum should be assessed first, HOB elevated to 30 degrees
- Cold water instilled into ear of an awake person causes nystagmus with the fast beating toward the opposite side. Warm water causes fast beating toward same side (COWS: Cold Opposite Warm Same). Lack of nystagmus on one side in awake person suggests CN VIII disorder.
- Cold water instilled into the ear of an unconscious patient will cause eyes to deviate slowly toward irrigated ear. Eyes will remain in this position until the irrigation stops, and then quickly return to mid position.
- Observe for 1 minute after completion of test, wait 5 minutes before testing other ear
- Delayed or absent movement indicates abnormality; fixed position in brain death.

Gag Reflex (CN IX [Glossopharyngeal] and X [Vagus]):

- Test one side at a time

Coughing and Breathing (CN X and Medulla):

- Assess for cough reflex during suctioning.
- Elevated PCO_2 must be confirmed before apnea can be verified.

Pupillary Dilation

- Sympathetic control of the pupil is located in the pons; pons damage is associated with pinpoint non-reactive pupils (loss of sympathetic dilation) and loss of horizontal eye movement.

Motor Assessment:

- Observe patients for symmetry of movements. Observe response to command and spontaneous/localizing movements. Apply central pain if no response to verbal command.
- If the patient is able to obey commands, describe motor response using the 0-5/5 Motor Scoring Scale.
- The single best test to identify a mild upper motor neuron weakness in a patient who is able to obey commands is the pronator drift test. Have the patient hold their arms forward, 90 degrees to his/her body (modify position as tolerated). Have the hands positioned palms up with eyes closed (if possible). Mild weakness is noted if the palm rotates toward the floor. This is more sensitive than waiting for the arm to drift downward. Shoulder injuries may make this difficult to perform.
- Asymmetrical weakness may indicate that the weakness is due to a lesion in a specific area of the brain (focal finding)
- When motor weakness is identified, the area of weakness should be evaluated to determine whether is cause is likely due to a problem in the upper (brain or cord) or lower (peripheral) motor nerve pathway.
- Summary: When motor weakness is identified, look for focal findings (asymmetrical findings). When focal findings are present, evaluate weakness for signs of upper or lower motor neuron problems. Symmetrical weakness due to a brain lesion is uncommon and usually metabolic in nature, however, spinal cord injury can cause symmetrical (or asymmetrical) weakness.
- Document a clear description of the method of stimulation and specific patient response. A detailed description provides more information than the GCS/graphic entries alone.

Upper versus Lower Motor Neuron Weakness

The upper motor neuron (UMN) pathway begins in the motor strip of the contralateral cerebral hemisphere, terminating in the spinal cord. Following impulse transmission to the end of the UMN pathway, the impulse synapses with the lower motor neuron (LMN) of the spinal nerve to activate the muscle.

Motor weakness can occur as a result of UMN pathway damage (such as stroke or cord injury), or LMN injury (e.g., injury to the brachial plexus or disc protrusion into a spinal nerve). Increased tone and deep tendon reflexes (2+ is normal, 3+ or 4+ is increased) are characteristics of an UMN cause for weakness. Upgoing toe following Babinski testing in conjunction with lower extremity weakness suggests an UMN cause for the weakness. Clonus may also be present (>5 sustained involuntary contractions following muscle stretching).

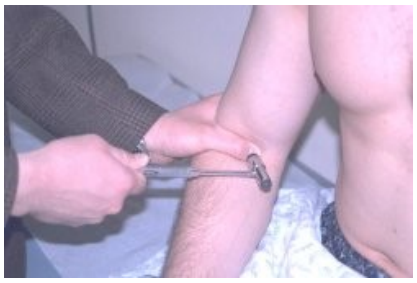
Flaccid paralysis with decreased deep tendon reflexes (0-1+) suggests a LMN cause. Fasciculations may be present. Note that during the early spinal shock phase of an acute spinal cord injury, the temporary loss of reflexes can produce a paralysis similar to that of a LMN injury.

While UMN causes for hemiplegia are far more common in CCTC than LMN lesions, LMN injury can also be seen in critical care. Examples include:

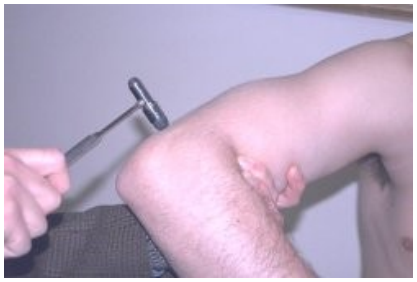
- Brachial plexus injury: the brachial plexus is a network of motor nerves from the cervical spine, that join together to form a plexus (group of nerves) that pass below the collar bone. These nerves, which include C5-8 and T1 are collectively responsible for all arm and hand movement. Flaccid paralysis of the arm with decreased upper extremity deep tendon reflexes, particularly in conjunction with a shoulder injury, may indicate brachial plexus injury. This is an example of a LMN cause for the arm weakness.
- Cranial nerves are LMNs. Injury to CN VII causes facial paralysis (motor weakness) on the same side. This includes an inability to close the eyelid or wrinkle the forehead on the affected side. Examples of CN VII injuries are Bell's Palsy and injury due to middle fossa basal skull fracture (with CN VII impingement).
- Contralateral facial weakness following stroke or brain injury (inability to stimulate the contralateral CN VII) is an example of an UMN cause for facial weakness. Forehead wrinkle and at least weak eyelid closure is generally preserved in UMN facial weakness. The preservation of the forehead wrinkle occurs because stimulation of the forehead wrinkle response from either side of the brain automatically stimulates the upper branches of CN VII bilaterally.
- Any spinal cord injury that causes disc protrusion onto the spinal nerve can cause LMN weakness. LMN weakness is associated with decreased reflexes.

Deep Tendon Reflexes

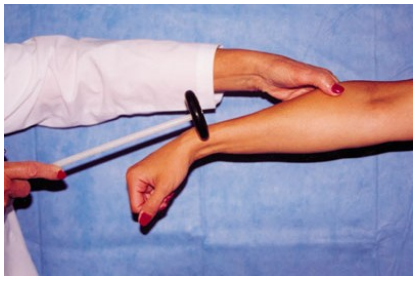
- Motor weakness associated with increased tone and deep tendon reflexes (3 or 4+), upgoing great toe, and/or with clonus suggests an UMN cause for the weakness.
- Motor weakness associated with flaccid paralysis and decreased deep tendon reflexes (< 2+) suggests a LMN cause for the weakness.



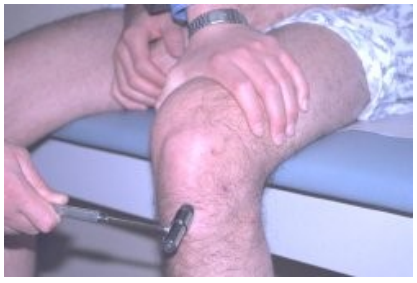
Biceps Brachii Tendon
C5, C6



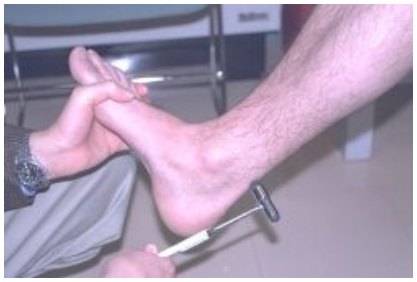
Triceps Tendon
C7, C6



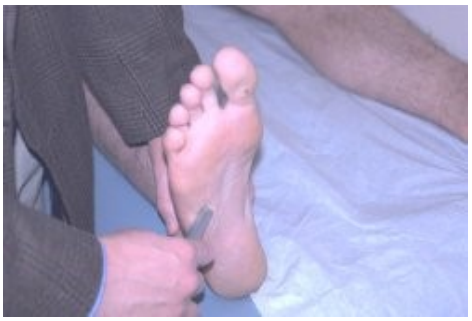
Brachioradialis Tendon
C6, C5



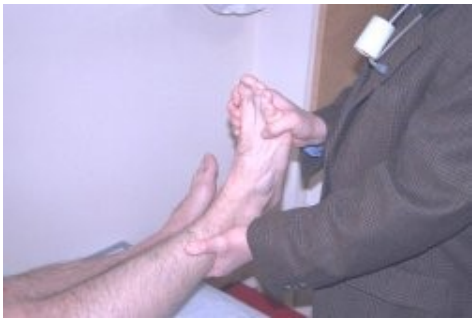
Quadriceps Tendon (knee jerk)
L4, L3, L2



Achilles Tendon (ankle jerk)
S1



Plantar Reflex (Babinski)

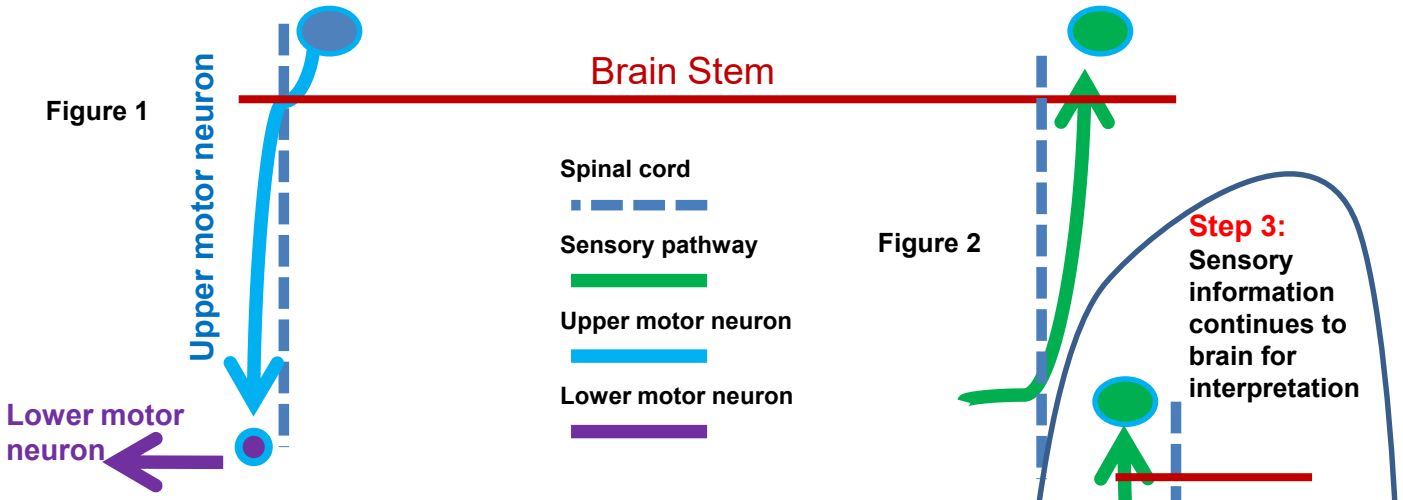


Clonus: Oscillations between flexion and extension

Spinal Cord Function

Information between the brain and spinal cord are carried via one of several tracts. Each tract has a unique channel and crossing point. Consequently, incomplete spinal cord injuries can produce a variety of motor and sensory deficits, depending upon the location of the lesion.

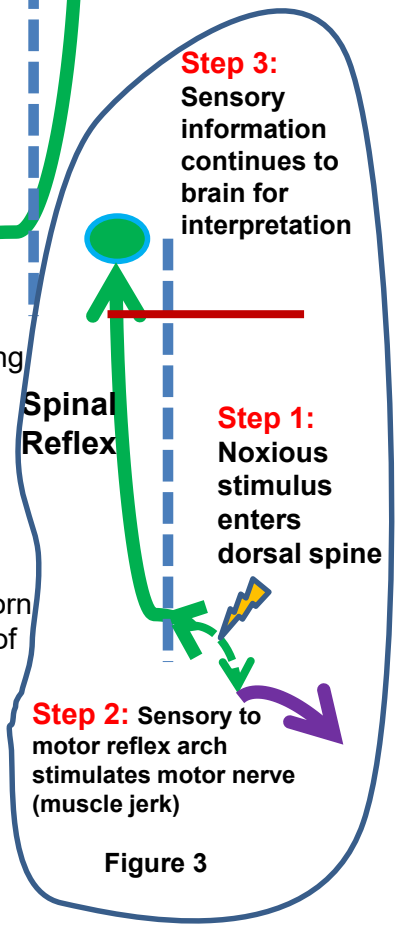
Motor Pathways (Corticospinal Tract): **Pain/Temperature (Spinothalamic)**



Motor pathways originate in the motor strip of the cerebral cortex, descending to cross at the brainstem before traveling down the contralateral cord (Figure 1). At the end of the upper motor neuron pathway, the impulse activates the lower motor nerve that causes the muscle activity.
The right side of the brain makes the left side of the body work.

Figure 2 above shows the pathway for pain and temperature interpretation (Spinothalamic). Painful stimuli enter the sensory nerve root in the dorsal horn of the spinal cord (back of cord). This impulse crosses to the opposite side of the cord, ascending to the contralateral parietal lobe for interpretation.

Both motor and pain pathways are oriented toward the anterio-lateral cord, and are vulnerable to compromised anterior spinal artery flow. A common cervical cord injury is a flexion injury at C5-6. This can cause anterior spinal artery occlusion and incomplete spinal cord injury with a loss of motor and pain function.

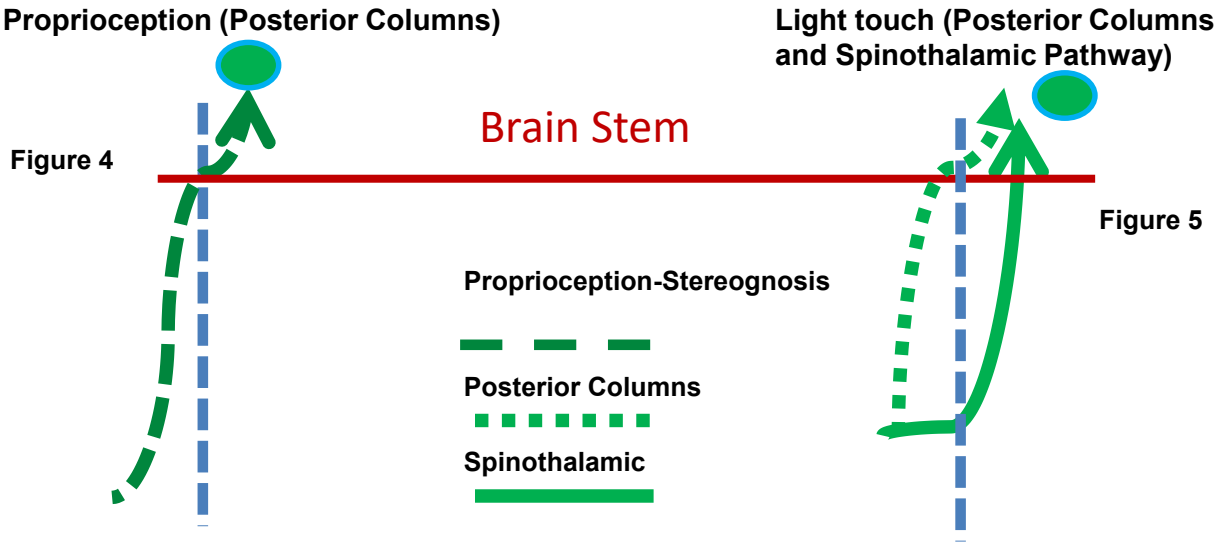


Spinal Reflex

The spinal reflex arch provides a rapid and protective motor response to noxious stimuli (Figure 3). A noxious stimulus enters the sensory nerve via the dorsal root (Step 1). Sensory information is immediately transmitted to the lower motor nerve at the same cord level (Step 2). This sensory to motor communication is called the reflex arch, and triggers the muscle activity or jerk. The sensory information continues to travel up the cord via the spinothalamic tract for interpretation of the sensation by the brain (Step 3). Consequently, when you get a paper cut, you jerk your hand back first and recognize the painful message after you have your protective motor response has already taken place. As long as the spinal cord is intact, pain is perceived immediately after the muscle jerks. In the setting of spinal cord injury, the jerk remains intact below the level of the lesion, but the pain is not perceived. Preservation of this spinal reflex also occurs in brain death. It is temporarily lost during the early spinal shock phase of an acute spinal cord injury.

Spinal Cord Function

Pathways for light touch (Figure 5) are carried up both the spinothalamic tract and the posterior columns (up the back of the cord, Figure 4). Proprioception (position sense) is also carried up the posterior columns. Many spinal cord injuries are incomplete with preservation of some function in one or more pathways.



Incomplete Spinal Cord Syndromes

Central Cord Syndrome:

A central cord syndrome occurs when the worst cord damage is in the centre of the cord. Because lower extremity motor pathways are located more laterally and upper extremity motor pathways are closer to the centre of the cord, upper extremity deficits are usually worse than lower extremity deficits in a central cord syndrome (patient can walk but not move their arms). Bladder dysfunction is usually present, while vibration and proprioception may be spared. There is variable sensory loss below the injury. Cervical level central cord injuries are often caused by extreme hyperextension of the neck (hitting chin on the pavement and extending head backward).

Brown-Sequard Syndrome:

This type of injury involves damage to one half of the cord, and may be due to penetrating trauma or unilateral cord compression from tumour or hematoma. Because pain and motor function for a given limb travel via opposite sides of the cord (Figure 1 and 2), Brown-Sequard Syndrome is characterized by a loss of motor function below the level of the injury on the side of the lesion, with preservation of pain and temperature. On the side opposite the lesion, pain and temperature is lost but motor function is preserved below the injury.

Anterior Cord Injury:

This type of injury is often due to disruption of the anterior spinal artery, causing the worst cord damage toward the front of the cord. Motor function and pain sensation are often the most impaired, while preservation of the posterior columns may be present (light touch, vibration and proprioception). Bladder dysfunction is usually present. This type of cord damage may l due to injuries that cause flexion of the neck (chin to chest).

Spinal Cord Injury

Spinal Shock

Following acute spinal cord injury, all reflexes above the level of injury are typically lost for a period of hours to days or weeks. During this period known as spinal cord shock, the patient typically has flaccid paralysis with a loss of deep tendon reflexes and bladder and bowel tone. Anal sphincter reflex is one of the first reflexes to return after the spinal shock phase ends. Reflex contraction of the anal sphincter (motor) following sensory stimulation suggests resolution of the spinal shock phase. A gentle tug on the Foley catheter can provide the sensory stimulus (bulbocavernosus reflex) that should automatically trigger anal sphincter contraction.

The end of the spinal shock period is significant for the following reasons. One hopes that any paralysis or sensory deficit immediately following an acute injury will be at least partially due to swelling and spinal cord shock. When the shock period ends, continued absence of sensation during a rectal exam and/or inability to voluntarily “squeeze” the anal sphincter is a bad sign.

During spinal shock, the loss of the bladder and anal sphincter reflex is associated with incontinence. Because relaxation to void or defecate is a voluntary function, the end of the spinal shock phase is usually associated with urinary and fecal retention. Early and aggressive bowel routine is important to facilitate future ADLs. Conversion to intermittent catheterization should begin as soon as it is no longer necessary to measure hourly urine output for other reasons and diuretic use is not required. Over distension of the bladder should be avoided (500 ml per catheterization optimal); over distension can lead to overflow incontinence and ureteral reflux.

An aggressive bowel routine that ensures a minimum of q 2 day bowel evacuation should be instituted immediately, even before the spinal shock phase ends. Diarrhea may be present during early training. The goal for bowel function is to have a soft stool q 2 days with evacuation aided by digital stimulation, suppository and fleet if required.

Neurogenic Shock

Neurogenic shock usually mirrors the spinal shock phase (loss of spinal reflexes). It is characterized by vasodilation, hypotension and bradycardia, due to disruption of autonomic fibres below the level of the injury. Neurogenic shock usually improves or resolves with time, however, it may remain an ongoing problem for individuals with complete and high cervical cord injuries. Turning, head of bed elevation and suctioning can precipitate bradycardia and hypotension. Cardiac arrest can also occur. Gradual and careful position changes and the use of TED stockings/abdominal binders to prevent positional hypotension, can help. Preoxygenation with 100% oxygen and abrupt termination of suctioning with return to mechanical ventilation will usually resolve bradycardias induced by suctioning. Atropine should be available at the bedside. Temporary pacemakers are occasionally required, less frequently, patients may need permanent cardiac pacing.

Other causes for shock (e.g., sepsis, myocardial infarction, hypovolemia may be masked by the loss of sympathetic response.

Autonomic Dysreflexia

Following resolution of the spinal shock phase with return of spinal cord reflexes, patients with spinal cord injury are at risk for the development of autonomic dysreflexia. The higher the cord injury, the great the potential. Virtually all quadriplegics and most individuals with injuries at or above T6 can experience this problem. Thus, patients with chronic spinal cord injury or those with acute spinal cord injury and prolonged critical care admission should be monitored for and taught to recognize signs of autonomic dysreflexia.

Autonomic dysreflexia is a life-threatening event that is triggered by a noxious stimulus. Sensory input causes a release of catecholamines, with vasoconstriction and hypertension. The rise in blood pressure stimulates carotid and aortic receptors, causing inhibitory messages to be sent down the cord. Because inhibitory messages can only transcend as far as the level of the injury, vasoconstriction (and hypertension) continues below the cord injury. Vasodilation above the lesion causes facial flushing, profuse sweating, bounding headache, nasal congestion and on occasion, Horner's syndrome. The higher the injury, the greater the hypertension. Vagal stimulation (which exits above the lesion) causes reflex slowing of the heart. Signs and symptoms of autonomic dysreflexia include:

- Hypertension (may only be 20-30 mmHg above baseline)
- Vasoconstriction below lesion
- Vasodilation with flushing above lesion and bounding headache
- Profuse sweating above lesion
- Bradycardia
- Goose bumps above and sometimes below lesion
- Visual disturbance; spots may be visible by patient in visual fields
- Horner's Syndrome: constriction of the pupil, mild eyelid droopiness, loss of sweating on one side of face.

The most common trigger for autonomic dysreflexia is a full bowel or bladder. Inadequate bowel routine, delayed intermittent catheterization or urinary tract infection are important and common causes. Any painful situation, including procedures or physical therapy, can cause this syndrome. Pregnancy, especially during labour and delivery in a patient with spinal cord injury can precipitate autonomic dysreflexia.

The treatment priority is to remove the cause of the autonomic dyreflexia (e.g., bladder catheterization, fecal disimpaction). Sitting the patient up can cause orthostatic lowering of the blood pressure. If antihypertensives are needed, use rapid onset agents with a short duration of action. Nitrates can be used, but are contraindicated if patients are receiving sildenafil or other medications for erectile dysfunction. Calcium channel blockers such as nifedipine can be useful; labetolol should be used with caution as it may worsen bradycardia.

Brenda Morgan
Clinical Nurse Specialist
Critical Care Trauma Centre
London Health Sciences Centre
Last Revision: April 23, 2014
Reviewed January 29, 2020