

- 1. Neurological and Neurosurgical services. History of Neurology and neurosurgery.**
- 2. Disorders of pyramidal system and voluntary movements.**
- 3. Disturbances of reflexes.**
- 4. Sensory disorders.**

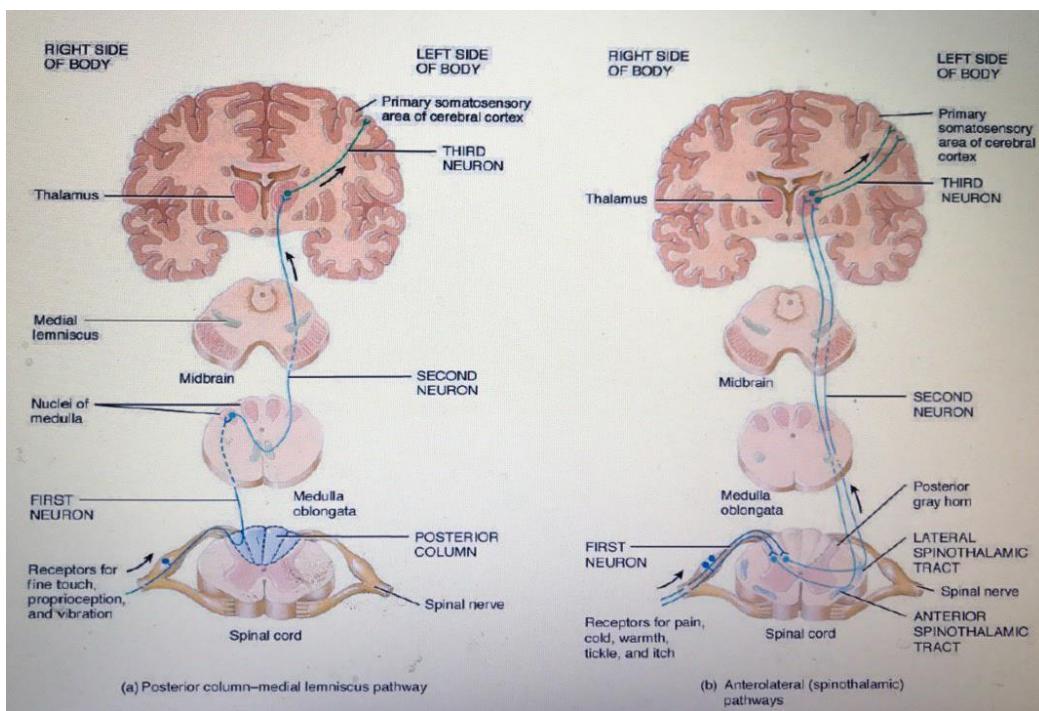
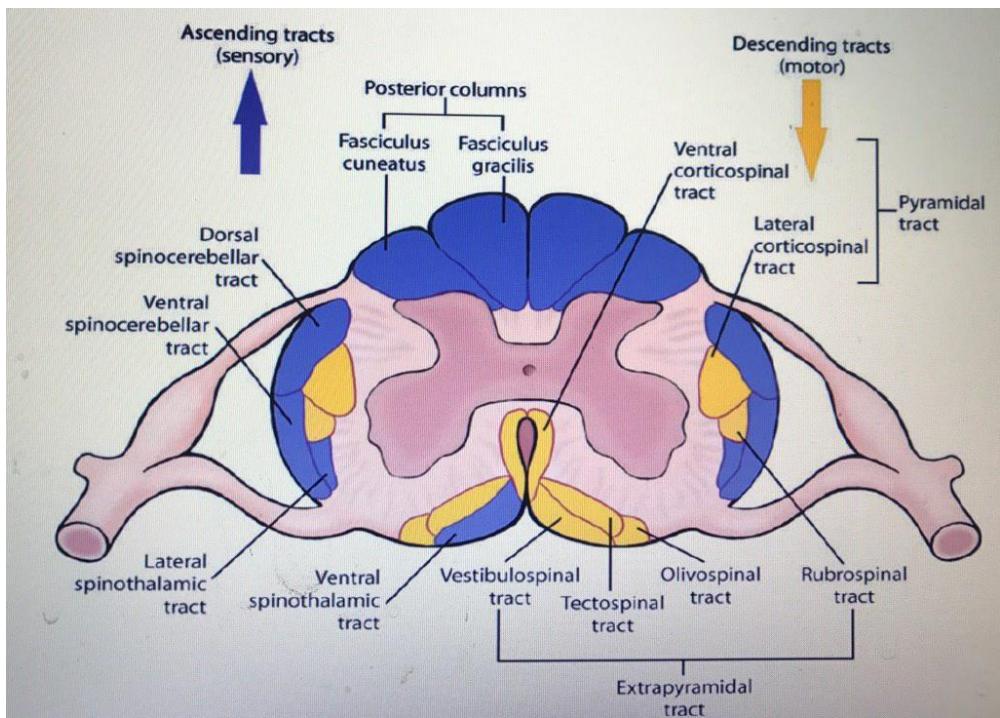
 the answers of question 1 to 4 are on the site(2 lectures)

1. <http://etest.bsmu.by/mod/page/view.php?id=151386&forceview=1>
2. <http://etest.bsmu.by/mod/page/view.php?id=151387&forceview=1>

more information

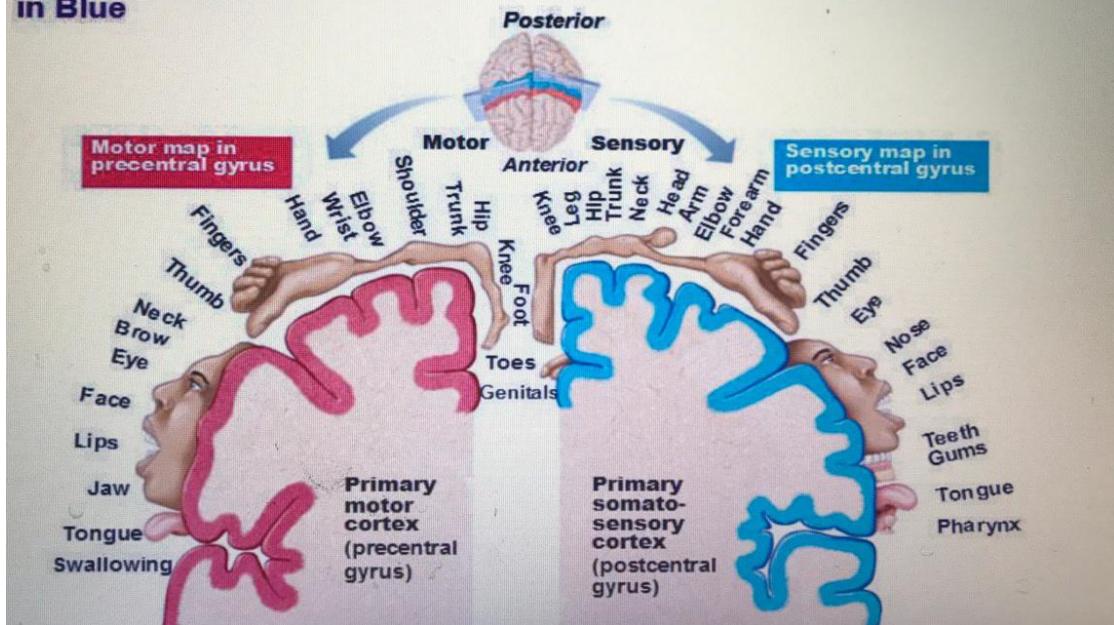
Main ascending pathways carrying sensory information to the brain

- Spinothalamic tracts
 - anterior spinothalamic tract(crude touch)
 - lateral spinothalamic tract(temperature, pain)
- Dorsal column–medial lemniscus pathway (position sense, vibration, two-point discrimination, fine touch)



Homunculus of Primary Somatosensory Cortex in Blue

Note that each hemisphere receives info from the opposite side of the body



6. *Sensory function:*

- Sensory assessment involves testing for touch, pain, vibration & discrimination.
- A complete sensory examination is possible only on a conscious & co-operative patient.
- Always test sensation with patient's eye closed.
- Help the patient relax & keep warm.
- Conduct sensory assessment systematically.
- Test a particular area of the body, & then test the corresponding area on the other side.

Physical Examination

Sensory Function Tests:

- Touch
 - Light touch 1st then Pain & Temperature
- Vibration
- Proprioception: Position sense
- Stereognosis
- Graphesthesia
- 2-point discrimination

Sensory Function- Spinothalamic Tract

Superficial sensation

- Pain
 - Assess sharp and dull
 - Allow at least 2 sec between
- Temperature
 - Test only when pain sensation abnormal
- Light (crude) touch
 - Apply wisp of cotton to arms, forearms, hands, chest, thigh, and legs

Sharp and dull and light touch intact



Vibration examination

Sensory Function Tests:

Sensory Exam: Vibration

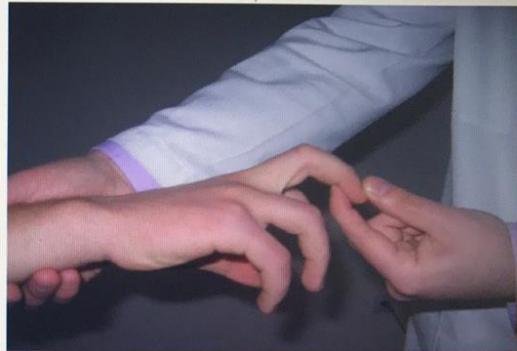


- use a 128 hertz vibration fork
- apply the stimulus over the distal phalanx of the index finger or large toe.
- ask the patient to report whether they feel vibration sense and then to report when it stops in order to assess the minimal threshold to perceive the stimulus.
- compare to your own extremities.



Position sense

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



Stereognosis

- ability to perceive and recognize the form of an object in the absence of visual and auditory information, by using tactile information to provide cues from texture, size, spatial properties, and temperature, etc.
- mediated by the posterior column-medial lemniscus pathway of the central nervous system.
- determine whether or not the parietal lobe of the brain is intact.

Typically, these tests involved having the patient identify common objects (e.g. keys, comb, safety pins) placed in their hand without any visual cues. Stereognosis is a higher cerebral associative cortical function.

Sensory Function Tests:

Stereognosis



Sensory Function Tests:

Graphesthesia

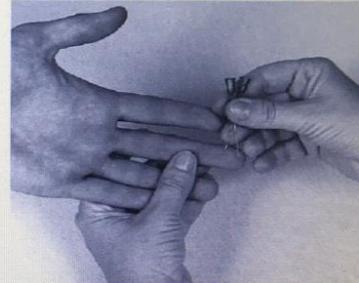


Two-point discrimination

- ability to discern that two nearby objects touching the skin are truly two distinct points, not one
- tested with two sharp points
- person should be able to recognize two points separated by as little as 2–4 mm on the lips and finger pads, 8–15 mm on the palms and 30–40 mm on the shins or back (assuming the points are at the same dermatome)
- impaired by damage to dorsal column pathway or to a peripheral nerve

Sensory Function Tests:

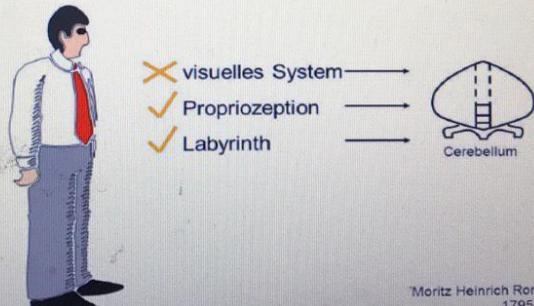
Two-point discrimination



Romberg test

- based on the premise that a person requires at least two of the three following senses to maintain balance while standing: proprioception (the ability to know one's body in space); vestibular function (the ability to know one's head position in space); and vision (which can be used to monitor [and adjust for] changes in body position).

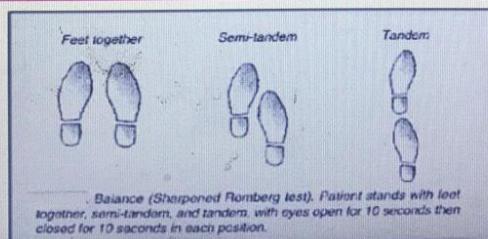
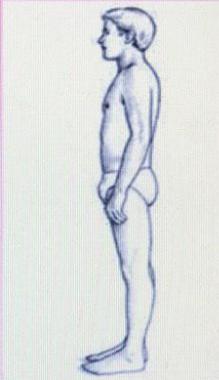
Romberg*-Test



ASSESSMENT TIP

Assessing Romberg's sign

Observe the patient's balance as he stands with his eyes open, feet together, and arms at his sides. Then ask him to close his eyes. Hold your arms out on either side of him to protect him if he sways. If he falls to one side, the result of the Romberg's test is positive.



SENSORY ATAXIA

“Disturbances in the sensory input to the cerebellum”

- Tests of proprioception- Joint sense, passive movement

“The corrective effects of the Visual system”

- Classical Sensory Ataxic Gait
- Romberg's sign
- Loss of tendon reflexes
- Features of Peripheral neuropathy



- 
- 5. Syndromes of the cervical part of the spinal cord lesions.**
 - 6. Syndromes of the thoracic part of the spinal cord lesions.**
 - 7. Syndromes of the lumbar part of the spinal cord lesions.**
 - 8. Syndromes of cauda equina and sacral part of the spinal cord lesions.**

The answers of questions 5 to 8 are together

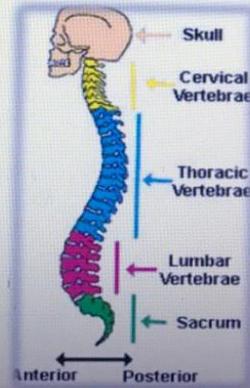


Introduction

- The spinal cord is a collection of nerves that travels from the bottom of the brain down your back. There are 31 pairs of nerves that leave the spinal cord and go to arms, legs, chest and abdomen. These nerves allow your brain to give commands to your muscles and cause movements of your arms and legs.

Definition of spinal cord injuries (SCI)

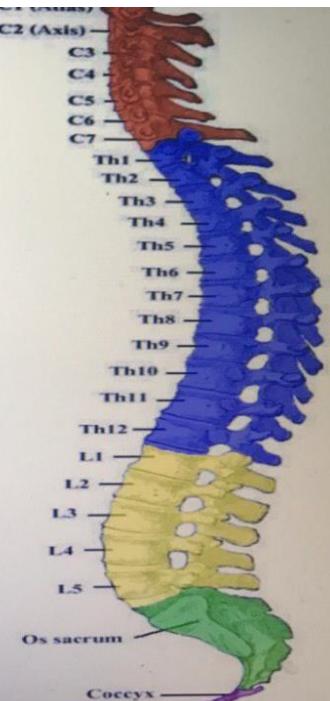
- abuse to spinal cord resulting in a change, in the normal motor, sensory or autonomic function. This change is either temporary or permanent.



Anatomy

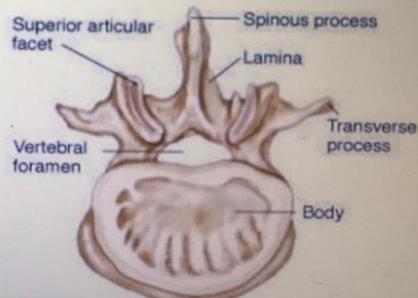
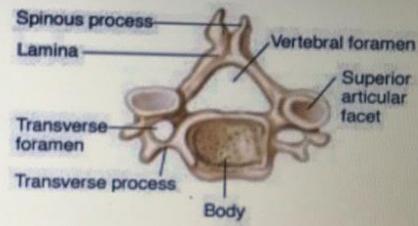
Spinal Column

- Composed of 33 vertebrae
 - 7 cervical
 - 12 thoracic
 - 5 lumbar
 - 5 sacrum (fused)
 - 4 coccyx (fused)
- Spinal cord lies in the spinal canal
- Spinal nerve roots pass out through
- the vertebral foramen



Spinal Column

- Each vertebra consists of:
 - Solid body
 - Posterior and anterior arch
 - Posterior spinous process
 - Transverse process (in some vertebrae)



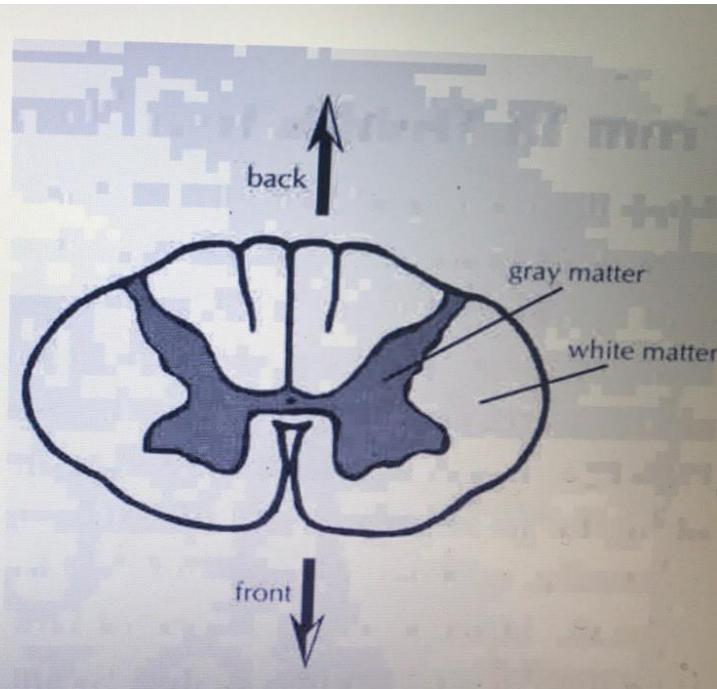
Anatomy cont....

Adult skull

- Sits on top of first cervical vertebra (C1) (atlas)
- Second cervical vertebra (C2) (axis) and its Odontoid process allow the head to move with about 180-degree range of motion

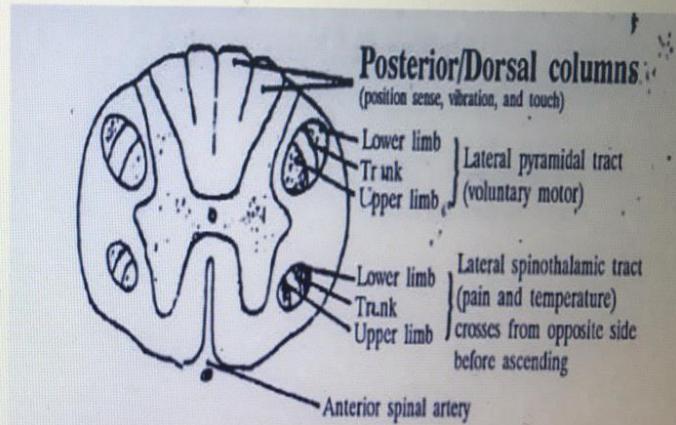
Spinal Cord

- Gray matter- cell bodies of voluntary and autonomic motor neurons
- White matter axons of ascending and descending motor fibers



Spinal Cord

- White tracts send messages to and from the brain
- Ascending Tracts-
 - carry into higher levels of CNS
 - touch, deep pressure, vibration, position, temperature
- Descending Tracts
 - impulses for voluntary muscle movement



Segmental Spinal Cord Level and Motor Function

Level	Motor Function
<u>C1-C6</u>	<u>Neck flexors</u>
<u>C1-T1</u>	<u>Neck extensors</u>
<u>C3, C4, C5</u>	Supply <u>diaphragm</u> (mostly <u>C4</u>)
<u>C5, C6</u>	<u>Shoulder</u> movement, raise <u>arm</u> (<u>deltoid</u>); flexion of <u>elbow</u> (<u>biceps</u>); <u>C6</u> externally rotates the <u>arm</u> (<u>supinates</u>)
<u>C6, C7</u>	Extends <u>elbow</u> and <u>wrist</u> (<u>triceps</u> and <u>wrist extensors</u>); <u>pronates</u> <u>wrist</u>
<u>C7, T1</u>	Flexes <u>wrist</u>

<u>C7, T1</u>	Supply small muscles of the <u>hand</u>
<u>T1 -T6</u>	<u>Intercostals</u> and <u>trunk</u> above the <u>waist</u>
<u>T7-L1</u>	<u>Abdominal</u> muscles
<u>L1, L2, L3, L4</u>	<u>Thigh flexion</u>
<u>L2, L3, L4</u>	<u>Thigh adduction</u>
<u>L4, L5, S1</u>	<u>Thigh abduction</u>
<u>L5, S1, S2</u>	<u>Extension of leg at the hip</u> (<u>gluteus maximus</u>)
<u>L2, L3, L4</u>	<u>Extension of leg at the knee</u> (<u>quadriceps femoris</u>)

<u>L4, L5, S1, S2</u>	<u>Flexion of leg at the knee (hamstrings)</u>
<u>L4, L5, S1</u>	<u>Dorsiflexion of foot (tibialis anterior)</u>
<u>L4, L5, S1</u>	<u>Extension of toes</u>
<u>L5, S1, S2</u>	<u>Plantar flexion of foot</u>
<u>L5, S1, S2</u>	<u>Flexion of toes</u>

Etiology of Traumatic SCI

- MVA . motor vehicle accidents- most common cause
- Other: falls, violence, sport injuries
- SCI typically occurs from indirect injury from vertebral bones compressing cord
- SCI frequently occur with head injuries
- Cord injury may be caused by direct trauma from knives, bullets, etc

Etiology of Traumatic SCI

- 78% people with SCI are male
- Typically young men – 16-30
- Number of older adults rising (>61 yr)
- Greater complications
- Life Expectancy 5 years less than same age without injury
- 90% go home

Mechanism of injury

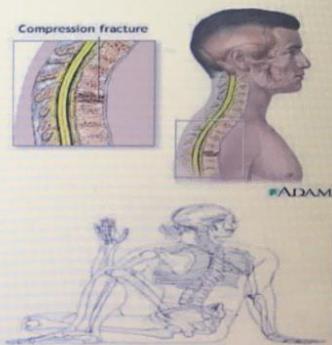
Types of movements that can cause spinal injury are:

- Hyperextension: the head is forced back.
- Hyperflexion: the head is forced forward.
- Axial loading: a severe blow to the top of the head.



Mechanism of injury cont...

- **Compression:** forces from above and below compress the vertebrae.
- **Lateral bend:** the head and neck are bent to one side beyond the range of motion.
- **Overrotation and distraction:** the head turns to one side, and the cervical vertebrae are forced beyond normal limits.



Classifications:

- **Complete spinal injury:**

When complete injury occurs, motor and sensory function cease below the level of injury, pain, touch, temperature and inhalation are evaluated as part of sensory evaluation.

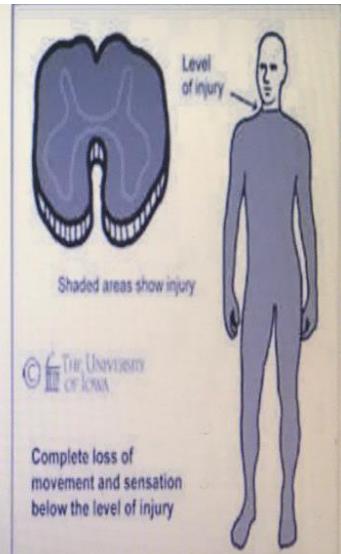


Figure 9. Complete Spinal Cord Injury

Incomplete spinal injury

1-Central Cord Syndrome

- Characteristics: Motor deficits (in the upper extremities compared to the lower extremities; sensory loss varies but is more pronounced in the upper extremities); bowel/bladder dysfunction is variable, or function may be completely preserved.

- Cause: Injury or edema of the central cord, usually of the cervical area.

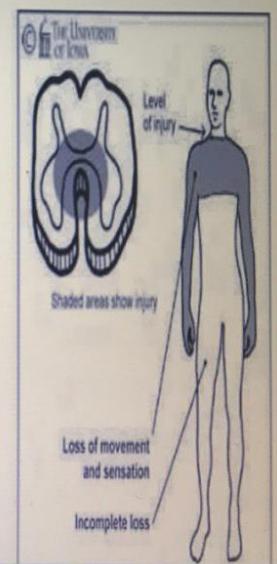


Figure 11. Central Cord Syndrome

Incomplete spinal injury cont...

2-Anterior Cord Syndrome

- Characteristics: Loss of pain, temperature, and motor function is noted below the level of the lesion; light touch, position, and vibration sensation remain intact.
- Cause: The syndrome may be caused by acute disk herniation or hyperflexion injuries associated with fracture-dislocation of vertebra. It also may occur as a result of injury to the anterior spinal artery, which supplies the anterior two thirds of the spinal cord.

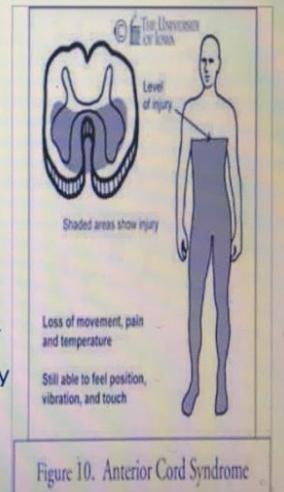


Figure 10. Anterior Cord Syndrome

Incomplete spinal injury cont...

3-Brown-Séquard Syndrome (Lateral Cord Syndrome)

- Characteristics: Ipsilateral paralysis or paresis is noted, together with ipsilateral loss of touch, pressure, and vibration and contralateral loss of pain and temperature.
- Cause: The lesion is caused by a transverse hemisection of the cord (half of the cord is transected from north to south), usually as a result of a knife or missile injury, fracture dislocation of a unilateral articular process, or possibly an acute ruptured disk.

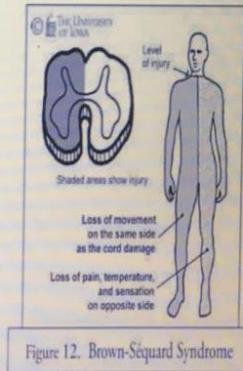


Figure 12. Brown-Séquard Syndrome

Clinical Manifestations

- “Neurologic level” refers to the lowest level at which sensory and motor functions are normal.
- ❖ Below the neurologic level
 - loss of bladder and bowel control
 - (usually with urinary retention and bladder distention)
 - loss of sweating and vasomotor tone
 - marked reduction of blood
 - pressure from loss of peripheral vascular resistance.
 - A complete spinal cord lesion can result in paraplegia (paralysis of the lower body) or quadriplegia (paralysis of all four extremities).

Assessment and diagnostic finding

- A detailed neurologic examination is performed.
- Diagnostic x-rays (lateral cervical spine x-rays)
- CT scanning are usually performed initially.
- An MRI scan may be ordered as a further workup if a ligamentous injury is suspected

Management

Emergency Management

- a rapid assessment, immobilization, extrication, stabilization or control of life-threatening injuries, and transportation to the most appropriate medical facility



Management cont...

Acute Phase

- PHARMACOLOGIC THERAPY
- high-dose corticosteroids,

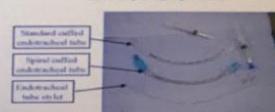


RESPIRATORY THERAPY

Oxygen is administered to maintain a high arterial PO₂

If Endotracheal intubation is necessary, extreme care is taken to avoid flexing or extending the patient's

Airway Equipment



Management cont...

Surgery

- Depending on the circumstances, when surgery is required, it may be performed within 8 hours following injury. Surgery may be considered if the spinal cord is compressed and when the spine requires stabilization. The surgeon decides the procedure that will provide the greatest benefit for the patient.

Management cont...

Surgery Management

- Early surgery, within 12 - 24 hours of the injury, is done when all body systems are stable, for:
 - Evidence of cord compression
 - Progressive neurological deficit
 - Compound fracture of the vertebrae
 - Penetrating wounds of the spinal cord
 - Bony fragment in the spinal canal

Types of surgery include:

- **Decompression laminectomies** using anterior cervical and thoracic approaches with fusion, in which one or more laminae are removed to allow for cord expansion due to edema
- **Posterior laminectomy** using interspinous wiring and fusion with an autologous iliac bone graft, to immobilize the neck and prevent further damage to the spinal column from hypermobility of the vertebrae
- **Posterior approach** using an autologous fusion graft or the insertion of rods or other instruments, to correct and stabilize thoracic deformities

Halo & Orthotic devices:

- Some patients may have Halo devices applied by surgeons, or a brace made by orthotics to maintain correct alignment of the spine. These devices are fixed to the child's chest.
- Ensure you know how to open devices to perform chest compressions in the event of a cardiac arrest, and that spinal immobilization is maintained manually throughout any resuscitation

Complication of SCI

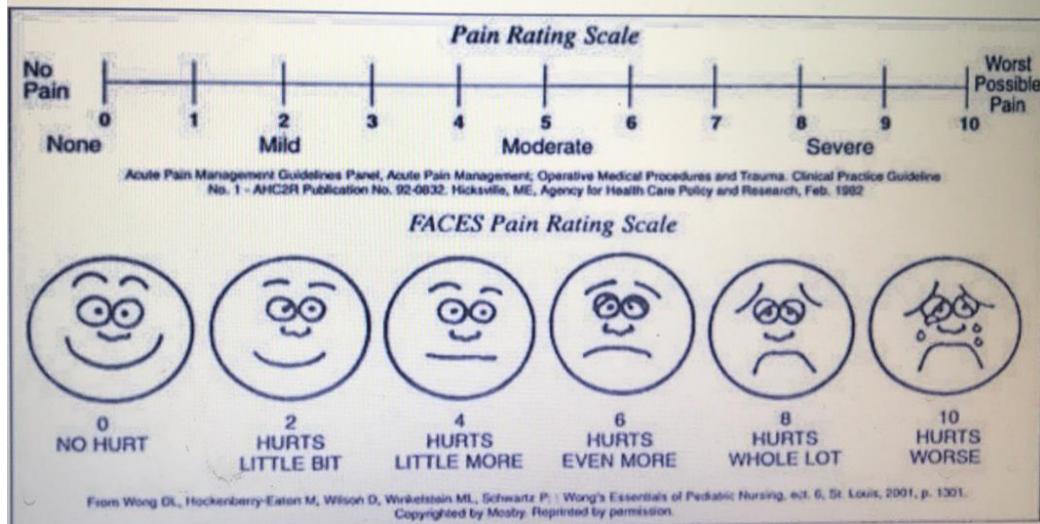
These complications include:

- urinary tract infections or urinary incontinence (inability to control the flow of urine),
- bowel incontinence (inability to control bowel movements),
- pressure sores,
- infections in the lungs (pneumonia),
- blood clots,
- muscle spasms,
- chronic pain, and depression.

Pain Management

- Assess for pain
- Use a self-reported numeric rating if possible
- Ask about characteristics, location, onset
- Minimize evoked pain through careful handling

Pain Management



Rehabilitation in spinal cord injury

Level of Injury	Physical Abilities	Functional Goals	Equipment Used
C1-C3	C3—Limited movement of head and neck.	<p>Breathing: Depends on ventilator for breathing.</p> <p>Communication: Talking is sometimes difficult, very limited, or impossible. If the ability to talk is limited, communication can be accomplished independently, with adaptive equipment.</p> <p>Daily tasks: Full assistance from caregiver for turning in bed, transfers, and all self-care (including bowel and bladder management). Assistive technology can allow for independence in such tasks as reading a book or newspaper, using a telephone, and operating lights and appliances.</p> <p>Mobility: Can operate an electric wheelchair by using a head control, mouth stick, sip and puff, or chin control. Can also operate a power tilt wheelchair also for independent pressure relief.</p>	<p>Suction equipment to clear secretions, two ventilators with backup generator and battery</p> <p>Mouth stick and assistive technology (e.g., computer, communication board) for speech or typing</p> <p>Mouth stick, environmental control unit (ECU)</p> <p>Power or manual lift, electric or semi-electric hospital bed, power wheelchair with pressure-relieving cushion</p>

Level of Injury	Physical Abilities	Functional Goals	Equipment Used
C3-C4	Usually has head and neck control. At C4 level, may shrug shoulders.	<p>Breathing: May initially require a ventilator for breathing; usually adjusts to breathing full time without ventilator assistance.</p> <p>Communication: Normal.</p> <p>Daily Tasks: Individual requires full assistance from a caregiver for turning in bed, transfers, and all self-care (including bowel and bladder management). Individual may be able to use adaptive equipment to eat independently. May also be able to operate an adjustable bed and perform other tasks, such as painting, writing, typing, and using a telephone with assistive technology.</p> <p>Mobility: Can operate a power wheelchair by using head control, a mouth stick, sip and puff, or chin control. Power tilt function on wheelchair allows for independence with pressure relief.</p>	Cough-assist device Eating: Sandwich holder on a gooseneck, feeder, long straw for liquids Other Activities: ECU for operating bed (e.g., head or voice activated, mouth stick controller), hands-free devices, mouth stick for typing, etc. Power or manual lift, electric or semi-electric hospital bed, power wheelchair with pressure-relieving cushion

Level of Injury	Physical Abilities	Functional Goals	Equipment Used
C5	Typically has head and neck control, can shrug shoulders, and has some shoulder control. Can bend elbows and turn palms face up.	<p>Daily Tasks: Individual can be independent with eating and grooming (e.g., face washing, oral care, shaving, make-up application) after setup from caregiver, with specialized equipment. Individuals will require total assistance from caregiver for bed mobility, transfers, and all other self-care. May be able to assist caregiver with upper body dressing and some bathing, with adaptive equipment.</p> <p>Health Care: Individual will require assistance from caregiver for cough assist. Can perform pressure relief with power tilt in power wheelchair.</p> <p>Mobility: May have strength to push a manual wheelchair for short distances over level surfaces; however, a power wheelchair with hand controls will be required for daily activities. At this level, the individual may be able to drive with specialized hand controls in a modified van with a lift, but still may require attendant to assist with transportation.</p> <p>Bowel and Bladder Management: Individual requires total assistance from caregiver for bowel and bladder management. Individual may have indwelling catheter or the caregiver may perform intermittent catheterization for bladder management. Bowel management can be performed with use of specialized equipment or medication.</p>	Eating: Universal cuff for attachment of utensils, scoop plate, plate guard, long straw Grooming: Universal cuff for attachment of tooth brush, comb or brush, adapted or electric razor, makeup applicators; wash mitt for face Bathing: Roll-in padded shower and commode chair, or padded transfer tub bench; wash mitt; adapted loofah Cough-assist device Wheelchair: Power or manual lift, electric or semi-electric hospital bed, power wheelchair with pressure-relieving cushion Bed: Bed ladder, thigh straps, and bed rails used for bed mobility Bowel: Roll-in padded shower and commode chair, or padded transfer tub bench Bladder: Leg-bag emptier

Level of Injury	Physical Abilities	Functional Goals	Equipment Used
C6	<p>Has movement in head, neck, shoulders, arms, and wrists. Can shrug shoulders, bend elbows, turn palms up and down, and extend wrists.</p>	<p>Daily Tasks: With use of some specialized equipment and setup from a caregiver, an individual can be independent with most feeding, grooming, and upper body dressing. Will still require some assistance for lower body dressing and will be able to assist with upper body during bathing. Can perform sliding board transfers to padded shower commode chair and/or tub bench for toileting and bathing, with some to total assist from caregiver. Can perform some light meal preparation tasks.</p> <p>Health Care: Can independently perform pressure relief with power tilt and may require some to no assist for forward or lateral lean pressure relief.</p> <p>Mobility: An individual may require some to no assist for turning in bed, with use of special equipment. May be able to perform sliding board transfers on level surfaces with some to no assistance from caregiver. Can use a ultra-lightweight manual wheelchair for mobility, but some may use a power wheelchair for greater ease over uneven terrain. Can be independent driving a vehicle from power or manual wheelchair with specialized equipment.</p> <p>Bowel and Bladder Management: Some to total assist with adaptive equipment for management of bowel and bladder.</p>	<p>Feeding: Universal cuff, built-up utensils, scoop plate, long straw, plate guard</p> <p>Grooming: Universal cuff, adapted electric razor, or toothbrush</p> <p>Dressing: Dressing stick, leg lifter, thigh straps, dressing hook splints; adapted or specialized clothing</p> <p>Bathing: Adapted loofah, long-handled sponge with universal cuff</p> <p>Transfers: Power or manual lift, sliding board, padded drop-arm bedside commode, padded tub bench with cutout, padded shower and commode chair</p> <p>Bed: Bed ladder, thigh straps, bed rails</p> <p>Wheelchair: Wheelchair pegs, specialized wheelchair gloves, and rubber tubing on wheels. Also, power-assist wheels can be used for independence with manual wheelchair propulsion.</p> <p>Transportation: Modified van with lift, specialized hand controls, tie-downs</p> <p>Bowel: Digital stimulation splint device, enema insertion device</p> <p>Bladder: Catheter inserter, penis positioner, thigh spreader with mirror</p>

C7-T1	<p>Has movement similar to C6 level, with the added ability to straighten elbows.</p> <p>At the C8-T1 level, has added strength and precision of hands and fingers.</p>	<p>Daily Tasks: Independent with all feeding, grooming, and upper body dressing, with equipment. Individuals may require some to no assistance with lower body dressing and bathing with equipment. Can perform sliding board transfers with some to no assistance to padded shower commode chair and/or tub bench for toileting and bathing.</p> <p>Health Care: Independent with wheelchair pushup or lateral lean for pressure relief.</p> <p>Mobility: Independent with manual wheelchair propulsion and level surface sliding board transfers. Some assistance may be required from caregiver for uphill transfers. Can be independent with driving if able to load and unload wheelchair.</p> <p>Bowel and Bladder Management: Depending on hand function, some to total assist for bowel management, with use of adaptive equipment or medication. Can be independent or need some assist for bladder management with ICP or condom catheter.</p>	<p>Feeding: Universal cuff, built-up handles, curved utensils, long straw, plate guard, adapted techniques for grasp</p> <p>Grooming: Universal cuff, splint material to adapt devices</p> <p>Dressing: Leg lifter, dressing stick, zipper pull, hooks on shoes</p> <p>Bathing: Adapted loofah, long-handled sponge with universal cuff</p> <p>Transfers: Sliding board, padded drop-arm bedside commode, padded tub bench with cutout, padded shower and commode chair</p> <p>Wheelchair: Rigid or folding lightweight wheelchair, wheelchair pegs, wheelchair gloves</p> <p>Transportation: Hand controls, modified van if unable to perform transfer or load-unload chair</p> <p>Bowel: Digital stimulation splint device, enema insertion device, toileting aid</p> <p>Bladder: Catheter inserter house hold (for men), thigh spreader with mirror (for women)</p>
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Level of Injury	Physical Abilities	Functional Goals	Equipment Used
L1-L5	Has additional return of motor movement in the hips and knees.	Mobility: Independent with all bed mobility and transfers with or without use of equipment. Independent with wheelchair propulsion on uneven and even surfaces and up and down curbs. Ambulation possible with use of specialized leg braces and walking devices. Functionality of ambulation depends on strength and movement in legs. Individuals' ability to ambulate depends primarily on their level household distances. Individuals may use a wheelchair for community mobility. Able to load and unload wheelchair independently for driving with hand controls.	Wheelchair: Ultra-lightweight wheelchair if necessary. Walking: Leg braces that extend to the hip, the knee, or just the ankle/foot and varying assistive devices Transportation: Hand controls
S1-S5	Depending on level of injury, various degrees of return of voluntary bladder, bowel, and sexual function.	Mobility: Increased ability to walk with fewer to no bracing or assistive devices.	Walking: Braces that support the ankle/foot
T2-T12	Has normal motor function in head, neck, shoulders, arms, hands, and fingers. Has increased use of rib and chest muscles, or trunk control. At the T10-T12 level, more improvements in trunk control due to increase in abdominal strength.	<p>Daily Tasks: Independent with all self-care, including bowel and bladder management, with adaptive equipment if necessary.</p> <p>Health Care: Independent with wheelchair pushup for pressure relief.</p>	Dressing: Thigh straps, reacher, dressing stick, sock aid Bathing: Long-handled sponge Transfers: Sliding board, padded drop-arm bedside commode, padded tub bench with cutout, padded shower/commode chair Bowel/Bladder: Mirror
		Mobility: Independent with all bed mobility and transfers, with or without use of equipment. Independent with wheelchair propulsion on uneven and even surfaces and up and down curbs. Able to load and unload wheelchair independently for driving with hand controls.	Wheelchair: Ultra-lightweight wheelchair Transfers: Sliding board, leg straps Transportation: Hand controls

Spinal Cord Injury Infographic

Types of Spinal Cord Injury

Prepared and designed by

www.apparelyzed.com
spinal cord injury peer support

Complete Spinal Cord Injury

Complete loss of motor and sensory function below the spinal cord injury.



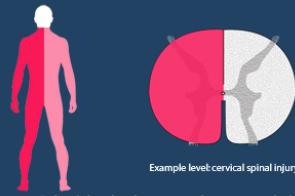
Incomplete Spinal Cord Injury

Partial random preservation of motor or sensory function below the spinal cord injury.



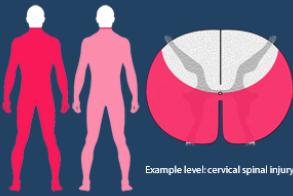
Common Types of Incomplete Spinal Cord Injuries

Brown-Séquard Syndrome



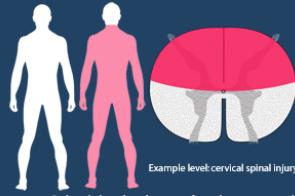
Example level: cervical spinal injury
Below injury level, motor weakness or paralysis on one side of the body (hemiparaplegia). Loss of sensation on the opposite side (hemianesthesia).

Anterior Cord Syndrome



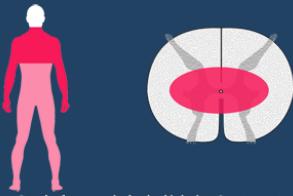
Example level: cervical spinal injury
Below injury level, motor paralysis and loss of pain and temperature sensation. Proprioception (position sense), touch and vibratory sensation preserved.

Posterior Cord Syndrome



Example level: cervical spinal injury
Below injury level, motor function preserved. Loss of sensory function: pressure, stretch, and proprioception (position sense).

Central Cord Syndrome



Results from cervical spinal injuries. Greater motor impairment in upper body compared to lower body. Variable sensory loss below the level of injury.

Cervical Nerves:

Diaphragm
Deltoids
Biceps
Wrist extensors
Rotates arm
Triceps
Bends fingers

Cervical
Vertebrae

Thoracic
Vertebrae

Thoracic Nerves:
Spread fingers
Chest muscles
Abdominal muscles
Muscles in the back

Lumbar
Vertebrae

Lumbar Nerves:
Hip muscles
Thigh muscles
Knee Muscles
Foot muscles

Sacral
Vertebrae

Sacral Nerves:
Bladder and bowel
Sexual function

Sources: www.apparelyzed.com
www.wikipedia.org
sci.rutgers.edu

Key:

- Normal Function
- Impaired Motor Function
- Impaired Sensory Function

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Cauda equina syndrome

- Cauda equina syndrome is caused by a significant narrowing of the spinal canal that compresses the spinal cord and causes nerve problems below the level of the compression
- Condition is a surgical emergency

Symptoms

- Leg pain, weakness, anaesthesia
- Saddle anaesthesia
- Bladder dysfunction
- Decreased anal tone
- Sexual dysfunction
- Absence of ankle reflex

Causes

■ Congenital

- Meningomyelocoele
- Congenital Dermoid sinus
- Congenital Midline Tumours: dermoid, epidermoid, teratoma, lipoma

■ Acquired

- **Infectious:** Neurosarcoïdosis, Schistosomiasis, Abscess formation
- **Traumatic:** Road traffic accident, Fall from height, Penetrating injuries – gunshot, stabbing
- **Degenerative:** Central disc prolapse
- **Neoplastic:** Primary – Ependymoma, Neurofibroma, Meningioma, Secondary metastasis
- **Vascular:** Arteriovenous malformations
- **Iatrogenic:** Anaesthetic, Orthopaedic and Neurosurgical procedures, Lumbar arachnoiditis following radiculogram

Investigation

- **Bloods**
 - FBC, U&E, LFT, CRP
- CXR
- Spinal x-rays
- ECG
- MRI spine



Treatment - Medical

- Treat any underling cause of compression
 - Infection – antibiotics
 - Inflammation – anti-inflammatories
 - Tumours – Radio/ chemotherapy

Treatment - Medical

However

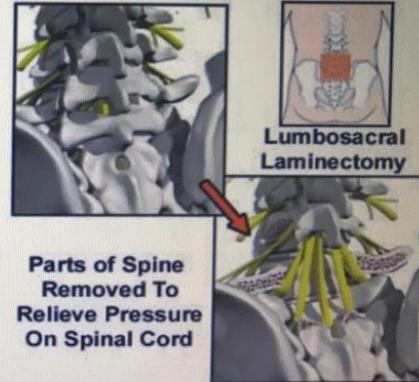
- If symptoms do not begin to improve within 24hr then surgery.

Or

- If symptoms continue to deteriorate immediate surgery.

Treatment - Surgical

- Surgical decompression
 - Drainage
 - Laminectomy
 - Fixation of fracture
 - Removal of foreign body



Prognosis

- Patient who have bilateral leg pain have a poorer prognosis than those with unilateral pain.
- Patient with complete groin numbness are more likely to have permanent bladder paralysis.
- The extent of saddle anesthesia is one of the most important prognostic indicators.

Prevention

- Control of any predisposing underlying pathology.
- Early diagnosis and treatment are crucial

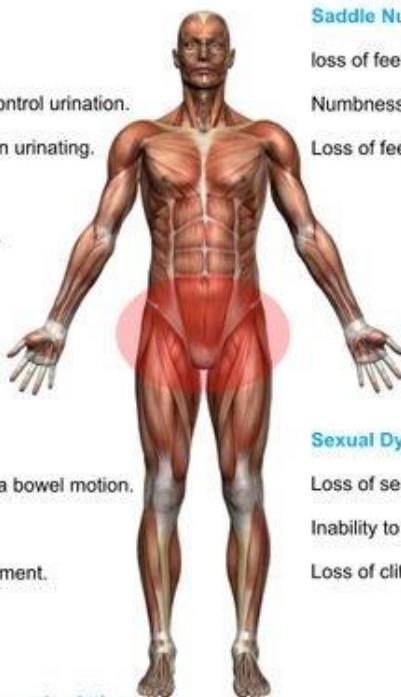
Cauda Equina Syndrome Symptom Chart

Bladder disturbances

Urination different to normal.
Inability to start, stop and/or control urination.
Loss of normal sensation when urinating.
Loss of full bladder sensation.
Inability to empty bladder fully.

Saddle Numbness

loss of feeling between the legs.
Numbness in and around the genitals/anus.
Loss of feeling of toilet paper when wiping.



Bowel function affected

Loss of feeling when passing a bowel motion.
Constipation.
Loss of control of bowel movement.

Sexual Dysfunction

Loss of sensation during sexual intercourse.
Inability to achieve an erection or ejaculate.
Loss of clitoral sensation.

Low Back pain/leg weakness and sciatica

A combination of these problems may be present. Keep a look out for bilateral toe extensor/flexor weakness, this can occur before other muscle weakness. Marked inability to bend forward with back pain/sciatica and leg weakness may indicate a large disc prolapse. Anal sphincter reflex maybe affected. Look out for bilateral achilles reflex absence.

Sacral part

Lumbosacral Injuries

Spinal cord injuries in the lower back region can affect **one or both legs**, as well as the **muscles controlling bladder and bowel functions**.

- Numbness and sensory changes.
- Loss of bladder and bowel control.
- Weakness
- Paraplegia
- Pain
- Increased muscle tone.

Diagnosis of Spinal Cord Injury

physical examination, including a neurological examination should be done to identify the level of injury.

Tests may include :

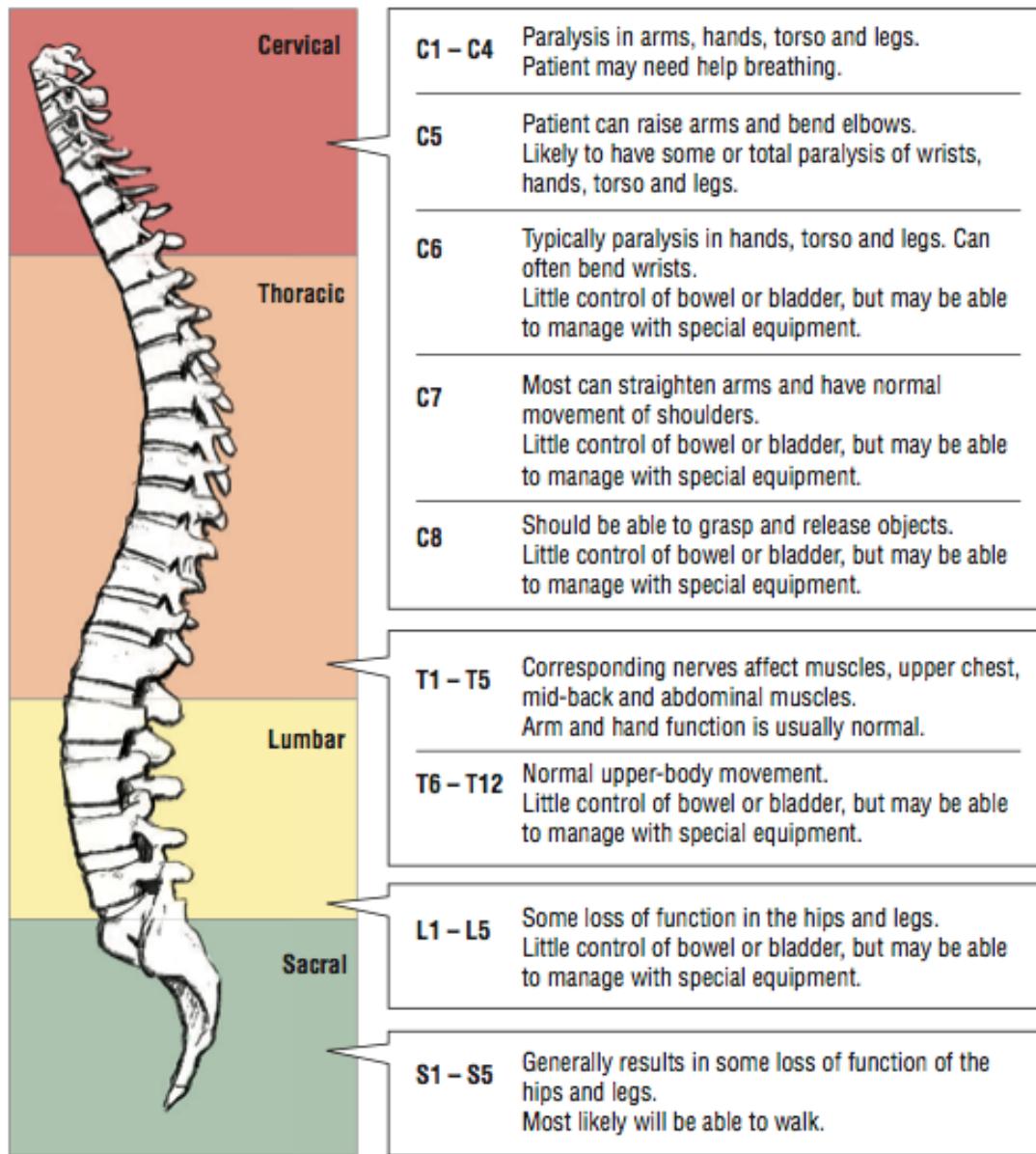
- Plain x-ray – lateral, oblique, and antero-posterior view to detect vertebral damage.
- CT scan or MRI – for location and extent of damage.
- Myelogram – x-ray of the spine following injection of dye.
- Somatosensory evoked potential (SSEP) testing or magnetic stimulation – to test for nerve signals.

Treatment of a Spinal Cord Injury

- Corticosteroids, such as methylprednisolone or dexamethasone, if started within 8 hours of trauma, can help to reduce swelling that may damage the spinal cord and thus significantly improve subsequent recovery.
- Early surgical decompression includes surgical removal of damaged bone, disc fragments, and hematoma.
- Skeletal traction for cervical injuries.
- Rehabilitation therapies, including physical therapy and occupational therapy may be started after healing of the acute injury.

Results of spinal cord injuries

Injuries to different regions of the spinal cord can cause different levels of severity in loss of motion or sensation.



SOURCE: Shepherd Center

Arkansas Democrat-Gazette/NIKKI DAWES

Effects of Spinal Injury

Level of Injury	Effect*
C E R V I C A L	Between C2 and C5 Between C5 and C6 Between C6 and C7 Between C7 and C8 C8 to T1 T2 to T4 T5 to T8 T9 to T11 T11 to L1 L2 to S2 S3 to S5
T H O R A C I C	Paralysis of some or all muscles used for breathing and all arm and leg muscles Typically, fatal unless a ventilator is used
	Paralysis of the legs, trunk, hand, and wrist Weakness of the muscles that move the shoulder and elbow
	Paralysis of the legs, trunk, and part of the wrists and hands Normal movement of the shoulders and elbows
	Paralysis of the legs, trunk, and hands
	Paralysis of the legs and trunk Weakness of the muscles that move fingers and hands Horner syndrome (with a drooping eyelid, a constricted pupil, and reduced sweating on one side of the face) Possibly normal movement of the shoulders and elbows
	Paralysis of the legs and trunk Loss of sensation below the nipples Normal movement of the shoulders and elbows
	Paralysis of the legs and lower trunk Loss of sensation below the rib cage
	Paralysis of the legs Loss of sensation below the navel
	Paralysis of and loss of sensation in the hips and legs
	Various patterns of leg weakness and numbness, depending on the precise level of injury Numbness in the perineum

* At any level of the spinal cord, severe injury can cause loss of bladder and bowel control.

9. Disorders of smell.

Abbreviations

- Olfactory nerve: CN1
- Optic nerve: CN2
- Oculomotor nerve: CN3
- Trochlear nerve: CN4
- Trigeminal nerve: CN5
- Abducens nerve: CN6
- Facial nerve: CN7
- Vestibulocochlear nerve: CN8
- Glossopharyngeal nerve: CN9
- Vagus nerve: CN10
- Accessory nerve: CN11
- Hypoglossal nerve: CN12

Overview

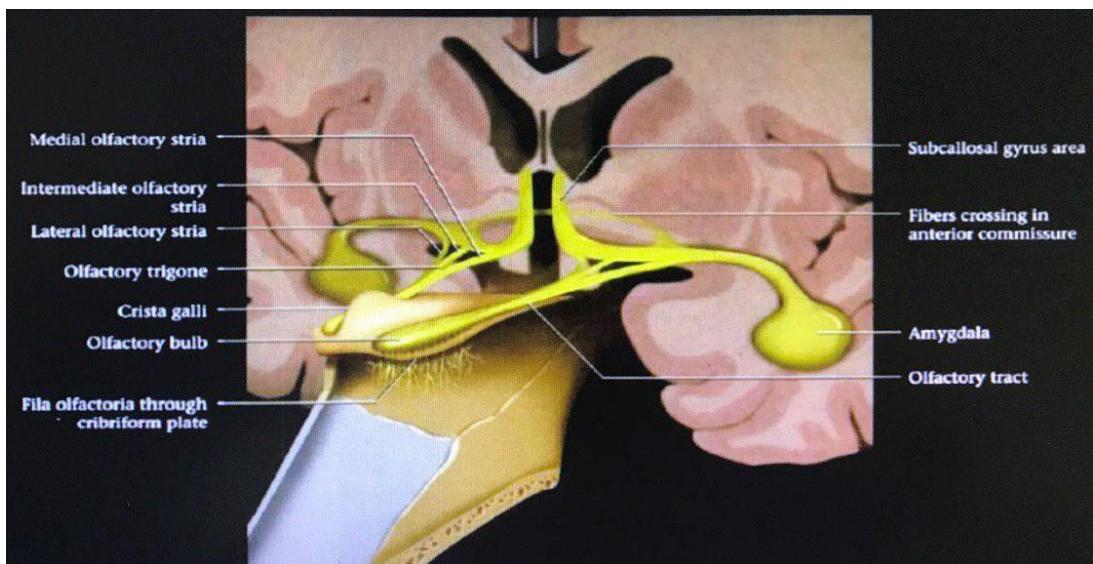
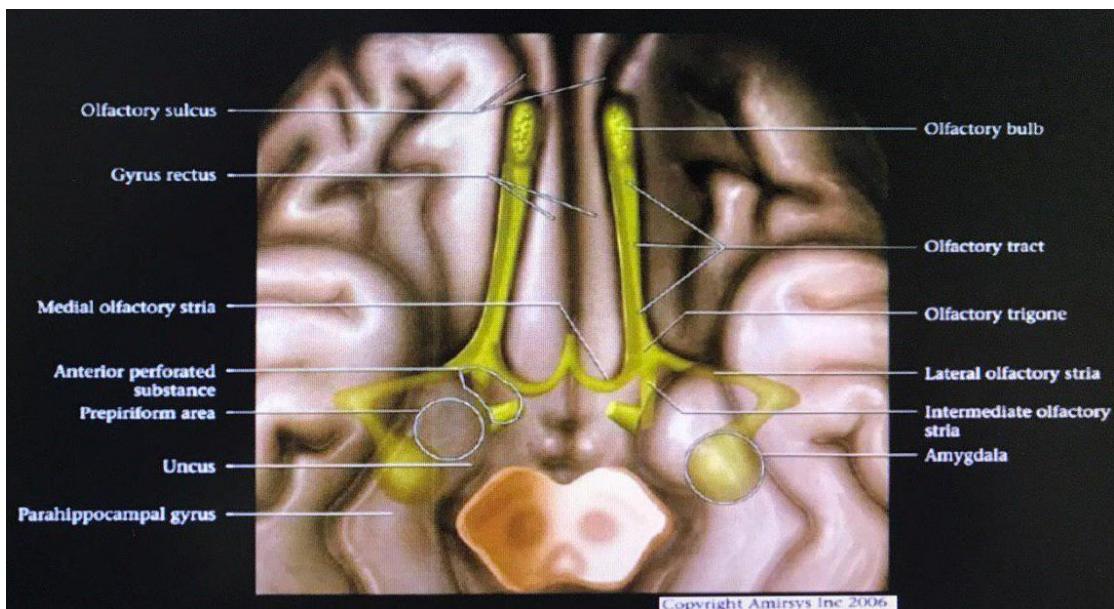
- Cranial nerve groupings based on area of brainstem origin
 - Diencephalon: CN2
 - Mesencephalon (midbrain): CN3 and CN4
 - Pons: CN5, CN6, CN7, and CN8
 - Medulla: CN9, CN10, CN11 and CN12

Imaging Approaches

- CN1, 2, 3, 4 and 6: Include focused orbital sequences
- CN5: Include entire face to inferior mandible if V3 affected
- CN7: Include CPA, temporal bone and parotid space
- CN8: Include CPA-IAC and inner ear
- CN9-12: Include basal cistern, skull base, nasopharyngeal carotid space
- CN10: Follow carotid space to aortopulmonic window on left, cervicothoracic junction on right
- CN12: Remember to reach hyoid bone to include distal loop as it rises into sublingual space

Olfactory nerve: CN1

- First cranial nerve
- Special visceral afferent cranial nerve for olfaction (sense of smell)
- Olfactory nerve segments
 - End receptor in olfactory epithelium in nasal vault
 - Transeethmoidal segment through cribriform plate
 - Intracranial olfactory bulb, tract and cortex



CN1: Imaging Recommendations

- Coronal sinus CT is best study for isolated anosmia
 - Identifies nasal vault and cribriform plate lesions
- MR of brain, anterior cranial fossa and sinonasal region used in complex anosmia cases
 - Identifies intracranial dural and parenchymal lesions

CN1: Clinical Importance

- CN1 dysfunction produces unilateral anosmia
 - Each side of nose must be tested individually
- Esthesioneuroblastoma arises from olfactory epithelium in nasal vault
- Head trauma may cause anosmia: Cribriform plate fracture or shear forces; anterior temporal lobe injury
- Seizure activity in lateral olfactory area may produce "uncinate fits", imaginary odor, oroglossal automatisms and impaired awareness

Cranial Nerve I: The Olfactory Nerve

- These secondary axons in the olfactory nerve eventually terminate in the inferomedial temporal lobe, uncus and entorhinal cortex
- To avoid confusing the olfactory nerve with the gyrus rectus on axial images, it is important to remember that the olfactory nerve is situated deep in the olfactory groove, inferior to the gyrus rectus
- Coronal images are easiest to interpret because the nerves are seen in cross section

From another source:

Cranial Nerves

- Indicated by Roman numerals I-XII from anterior to posterior
- May have one or more of 3 functions
 - Sensory (special or general)
 - Somatic motor (skeletal muscles)
 - Parasympathetic (regulation of glands, smooth muscles, cardiac muscle)

12 pairs of Cranial Nerves arise from the forebrain and the brain stem.

I Olfactory **Telencephalon**

II Optic **Diencephalon**

Cranial Nerves III through XII
arise from the Brain Stem:

Midbrain

III Oculomotor

IV Trochlear

Pons

V Trigeminal

VI Abducens

VII Facial

Medulla

VIII Vestibulocochlear

IX Glossopharyngeal

X Vagus

XII Hypoglossal

Cranial Nerve XI -

Spinal Accessory Nerve

arises from the **Spinal Cord and medulla**

Olfactory nerve

Origin: cerebral hemisphere, doesn't have nuclei

Innervation: nasal mucous membranes

Function: sensory -sense of smell

Dysfunction: anosmia, hyposmia, parosmia, smell hallucinations

Clinical evaluation:

-Test each nostril separately

-Use non-noxious aromatic substances

-Mark any abnormality

10. Visual disturbances.

Abbreviations

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CN2

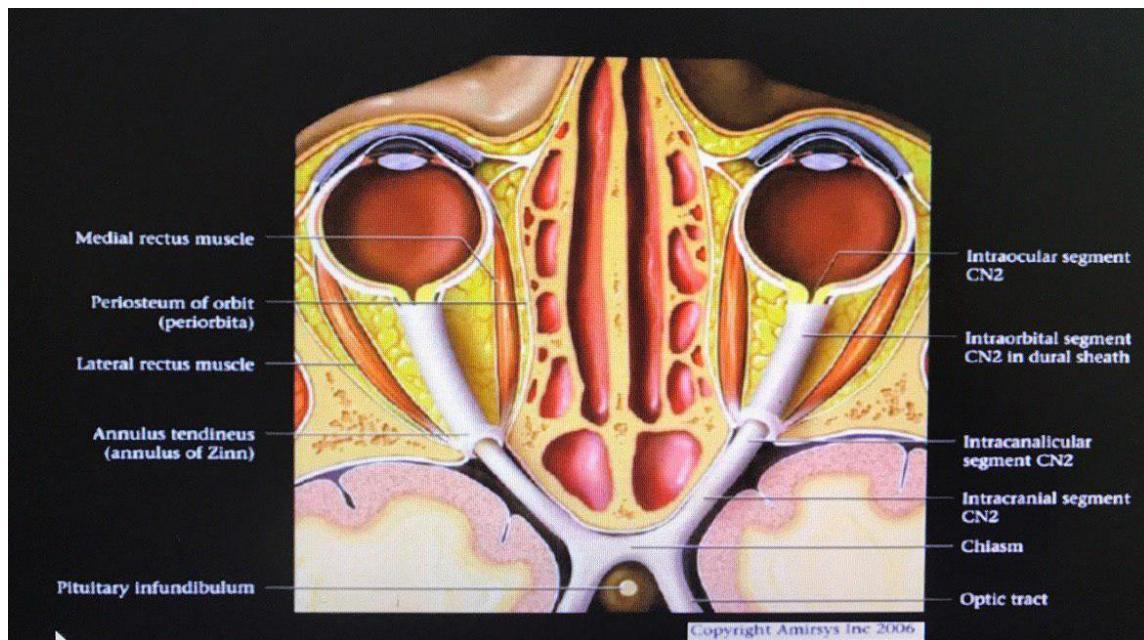
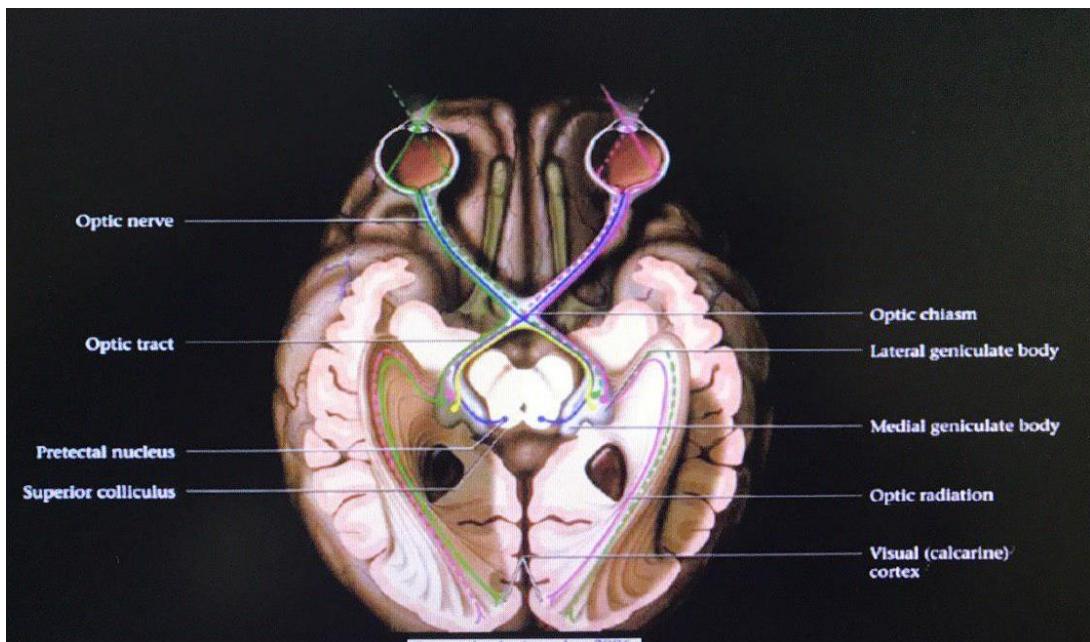
- Second cranial nerve
- Nerve of sight
- Visual pathway consists of optic nerve, optic chiasm and retrochiasmal structures

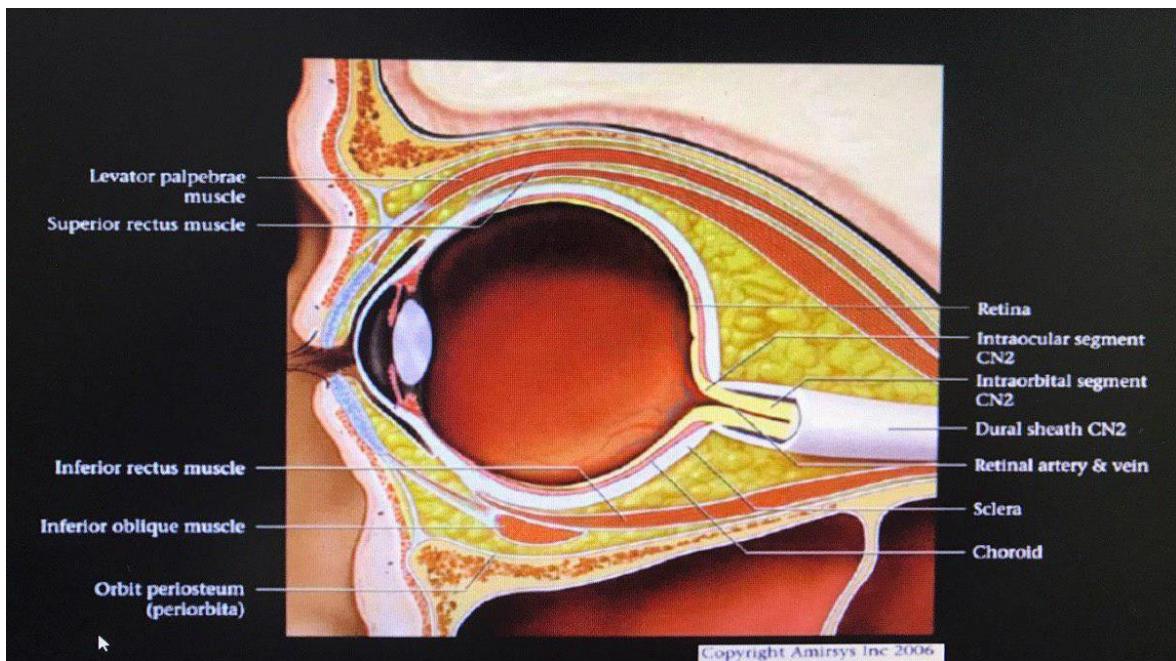
Overview

- Optic nerve not true cranial nerve but rather extension of the brain
 - Represents collection of retinal ganglion cell axons
 - Myelinated by oligodendrocytes not by Schwann cells as with true cranial nerves
 - Enclosed by meninges
 - Throughout its course to visual cortex nerve fibers are arranged in retinotopic order

Overview

- Optic nerve has four segments
 - *Intraocular, intraorbital, intracanalicular and intracranial*
- Partial decussation CN2 fibers within optic chiasm
 - Axons from medial portion of each retina cross to join those from lateral portion of opposite retina
- Retrochiasmal structures: Optic tract, lateral geniculate body, optic radiation and visual cortex





Optic tracts

- Posterior extension of optic chiasm
- Fibers pass posterolaterally curving around cerebral peduncle and divide into medial and lateral bands
 - Lateral band (majority of fibers) ends in lateral geniculate body of the thalamus
 - Medial band goes by medial geniculate body to pretectal nuclei deep to superior colliculi

Cranial Nerve II: The Optic Nerve

- Like the olfactory nerve, the optic nerve is a white-matter tract without surrounding Schwann cells.
- It includes four anatomic segments: **retinal, orbital, canicular, and cisternal**.
- The retinal segment leaves the ocular globe through the lamina cribrosa sclerae (the optic foramen of the sclera).
- The orbital segment, which is surrounded by a dural sheath containing CSF, travels through the center of the fat-filled orbit.
- The canicular segment is the portion that lies in the optic canal, below the ophthalmic artery. This segment of the nerve is frequently overlooked on radiologic images, so it should be specifically sought when imaging for vision loss.
- Finally, the cisternal segment of the nerve can be visualized in the suprasellar cistern, where the nerve leads to the optic chiasm. The anterior cerebral artery passes over the superolateral aspect of the cisternal segment of the nerve.

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VII Facial

Medulla

VIII Vestibulocochlear

IX Glossopharyngeal

X Vagus

XII Hypoglossal

Cranial Nerve XI -

Spinal Accessory Nerve

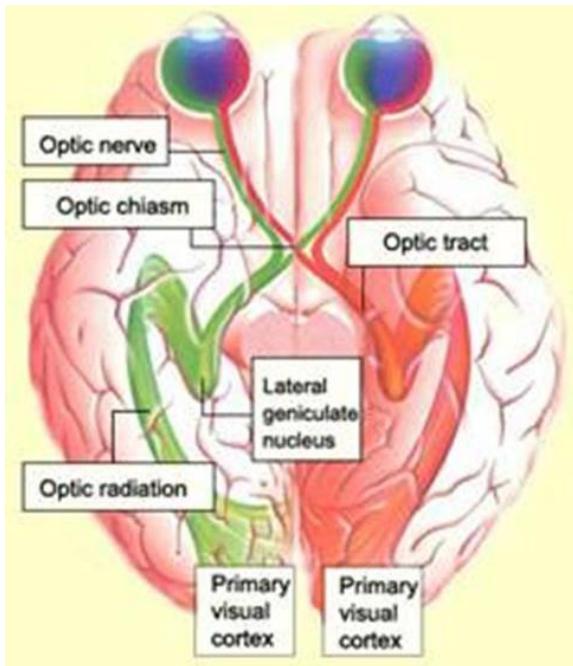
arises from the **Spinal Cord and medulla**

Optic nerve

Origin: cerebral hemisphere, doesn't have nuclei

Function: sensory –vision

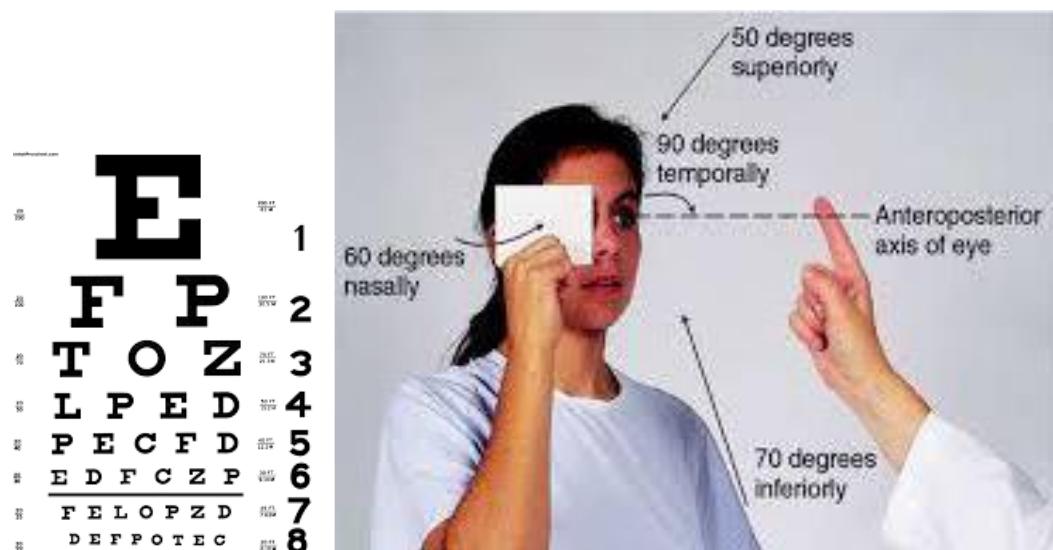
Dysfunction: vision loss (blindness) –unilateral or bilateral, hemianopsia, cortical blindness, scotomas, optic disc edema or atrophy



Visual acuity: Snellen chart for distant vision, fingers count for near vision

Visual fields: confrontation

Fundi and optic discs: funduscopy



Types of hemianopsia

Heteronymous

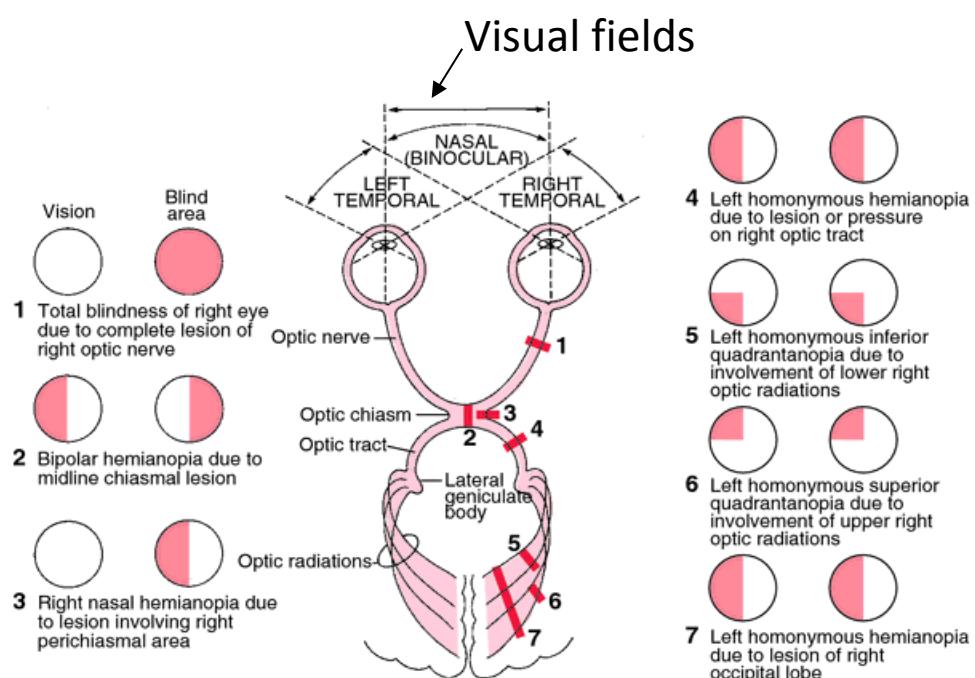
-Bitemporal

-Binasal

Homonymous

-left/right hemianopsia

-superior or inferior quadrantanopsia



11. III, IV, VI cranial nerves disorders.

Overview

- Third cranial nerve
- Motor nerve to extraocular muscles except lateral rectus (CN6) and superior oblique muscles (CN4); parasympathetic to pupillary sphincter and ciliary muscle
- Mixed cranial nerve (motor and parasympathetic)
- Four anatomic segments: ***Intra-axial, cisternal, cavernous and extracranial***

Clinical Importance and Findings

- ***Uncal herniation pushes CN3 on petroclinoid ligament***
- ***During trauma downward shift of brainstem upon impact can stretch CN3 over petroclinoid ligament***
- ***CN3 susceptible to compression by PCA aneurysms***
- CN3 neuropathy divided into simple if isolated and complex if with other CN involvement (CN4 & CN6)
 - Simple CN3 with pupillary involvement
- Must exclude PCA aneurysm as cause
- Explanation: Parasympathetic fibers are peripherally distributed
 - Simple CN3 with pupillary sparing
 - . Presumed microvascular infarction involves vessels supplying core of nerve with relative sparing of peripheral pupillary fibers
- ***Oculomotor ophthalmoplegia:*** Strabismus, ptosis, pupillary dilatation, downward abducted globe and paralysis of accommodation

Cranial Nerve III: The Oculomotor Nerve

- The oculomotor nerve originates from nuclei deep to the superior colliculus, ventral to the cerebral aqueduct, and inferior to the pineal gland.
- The nerve then travels across the midbrain from posterior to anterior.
- The oculomotor nerve root emerges into the interpeduncular cistern, and this root entry zone in the cistern is a good way to identify the oculomotor nerve on axial SSFP MR images.
- In the prepontine cistern, the nerve travels between the superior cerebellar and posterior cerebral arteries, which makes it easy to identify on coronal SSFP images.

Overview

- Fourth cranial nerve
- Motor nerve to superior oblique muscle
- CN4 is a pure motor nerve
- *Four segments: Intra-axial, cisternal, cavernous and extracranial*

Overview

- Sixth cranial nerve
- Motor nerve to lateral rectus muscle only
- CN6 is a pure motor nerve
- *Five segments can be defined: Intra-axial, cisternal, interdural, cavernous and extracranial (intra-orbital)*

Extracranial (Intra-Orbital) Segment

- CN6 enters orbit through superior orbital fissure together with CN3 and CN4
- Passes through annulus of Zinn
- Supplies motor innervation to lateral rectus muscle

Imaging Recommendations

- MR for intra-axial, cisternal, interdural & cavernous segments
 - Thin-section high-resolution T2 and contrast-enhanced TI in axial and coronal planes
- Depicts small structures including cranial nerves surrounded by CSF with high contrast & high spatial resolution
- Bone CT best for skull base and its bony foramina
- Dorello canal, cavernous sinus and orbital CN6 not visualized on routine MR imaging

Imaging "Sweet Spots"

- Axial and coronal MR sequences should include brainstem, fourth ventricle, cavernous sinus and orbit
- CN6 nucleus and intra-axial segment not directly visualized
 - Position of CN6 inferred by identifying facial colliculus in floor of fourth ventricle on high-resolution thin-section T2 MR
- Cisternal segment routinely visualized on high-resolution T2
- CN6 entrance into Dorello canal may be visualized due to invagination of cerebrospinal fluid into proximal canal

Imaging Pitfalls

- Use of fat-saturation on post-contrast T1 MR sequences can amplify blooming (susceptibility) artifact around a well aerated sphenoid sinus
 - Cavernous sinus & orbital apex subtle lesions may be obscured by this artifact
 - Remove fat-saturation and repeat T1 post-contrast MR if this artifact obscures key areas of interest

Clinical Importance

- In abducens neuropathy, affected eye will not abduct (rotate laterally)
- CN6 neuropathy divided into simple if isolated & complex if associated with other CN involvement (CN3, 4 and 7)
 - Simple CN6 neuropathy most common ocular motor nerve palsy
 - Usually presents as complex cranial neuropathy:
*Pontine lesions affect CN6 with CN7
Cavernous sinus, superior orbital fissure
lesions affect CN6 with CN3, 4 and CNV1*

Cranial Nerve VI: The Abducens Nerve

- The abducens nerve emerges from nuclei anterior to the fourth ventricle, then courses anteriorly through the pons to the pontomedullary junction and into the prepontine cistern.
- After crossing the prepontine cistern in a posterior-to-anterior direction, the abducens nerve runs vertically along the posterior aspect of the clivus, within a fibrous sheath called the Dorello canal.
- The nerve then continues over the medial petrous apex and through the medial cavernous sinus, entering the orbit through the superior orbital fissure to innervate the lateral rectus muscle.

Cranial Nerve VI: The Abducens Nerve

- It is important to note that the abducens nerve runs almost the entire length of the clivus.
- Radiologists should be vigilant for clivus and petrous apex abnormalities in the setting of abducens nerve palsy.
- Although the abducens nerve lies near the anterior inferior cerebellar artery and has a similar caliber, the two structures course in orthogonal directions and are thus easily distinguished.

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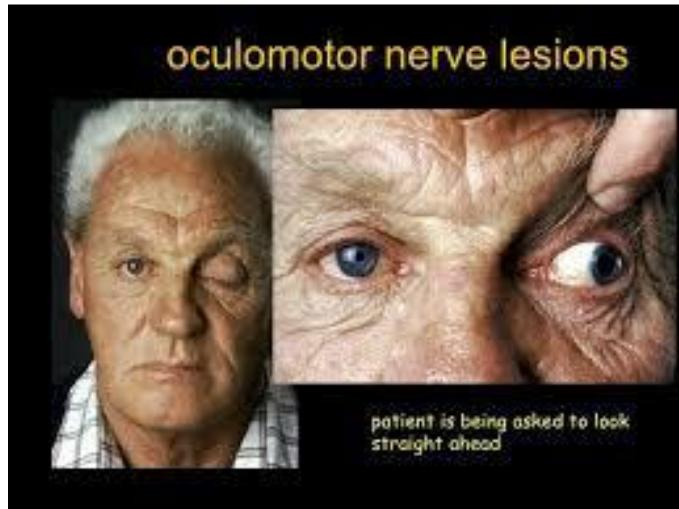
Oculomotor nerve

Origin: midbrain

Innervation: EOM's, eyelid, ciliary and sphincter of iris

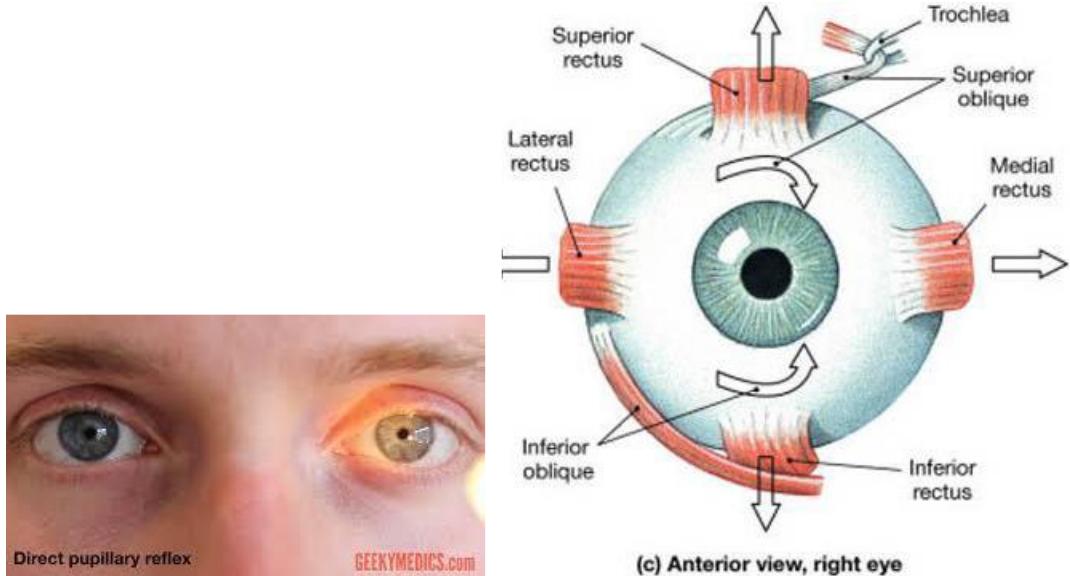
Function: motor and parasympathetic - eye movement medially, upward, downward, pupil constriction, upper eyelid elevation, accommodation reflex

- Dysfunction:** unable to look up, down and medially, eyelidptosis, pupil dilatation, and loss of accomodation, diplopia

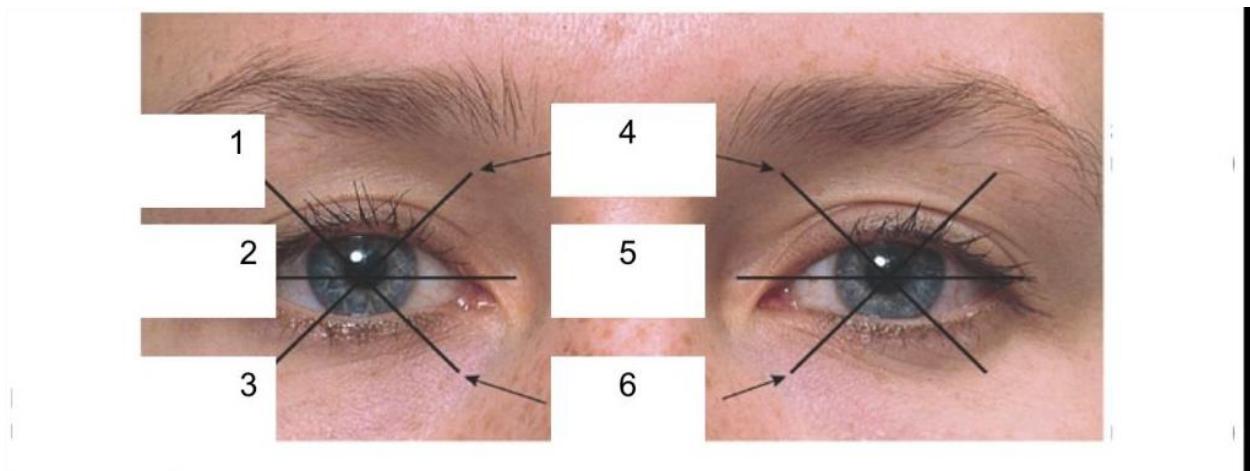


Oculomotor nerve

- Observe for eye opening and symmetry
- Pupil size and shape
- Direct light response
- Consensual response
- Accommodation
- EOM's



(c) Anterior view, right eye

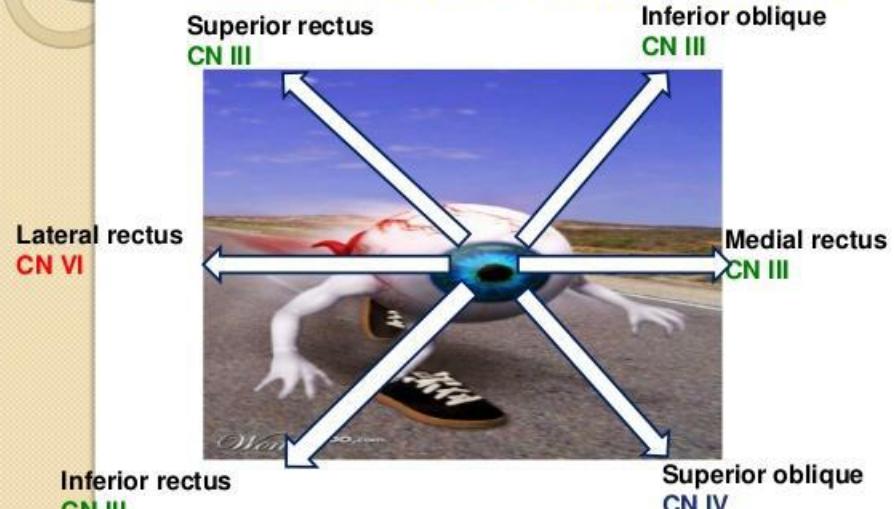


Cardinal Directions of Gaze

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CRANIAL NERVE FUNCTION & MUSCLE INNERVATION RELATIVE TO EYE MOVEMENT



Oculomotor nerve lesion



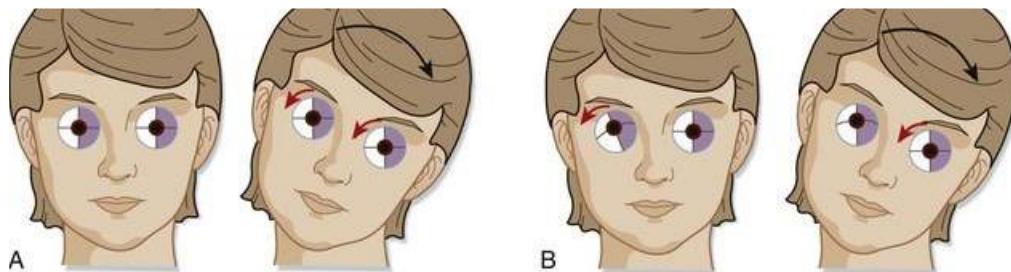
Trochlear nerve

Origin: Midbrain

Innervation: superior oblique muscle

Function: motor -down and inward movement of the eye

Dysfunction: loss of downward inner movement of eye, diplopia
“looking down”



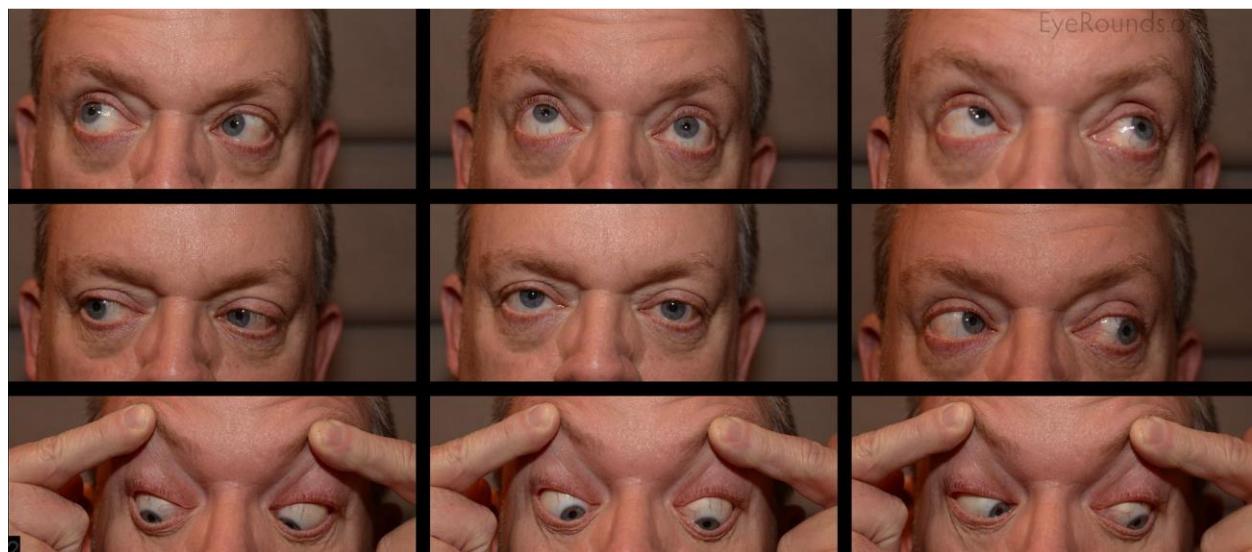
Normal eye rotation

When the head tilts to the left, both eyes rotate in the opposite direction (right eye extorts, left eye intorts)

Cranial nerve IV palsy (right eye)

Right eye extorted and slightly elevated causing double vision. To compensate, the patient tilts her head to the left

Trochlear nerve lesion



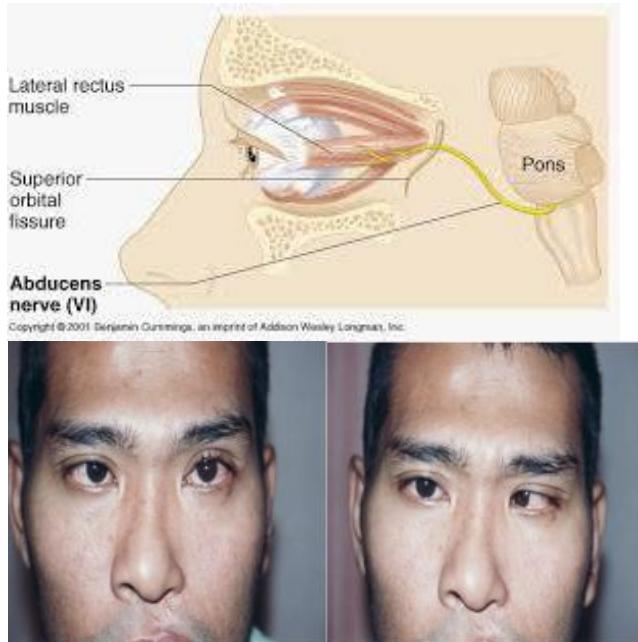
Abducens nerve

Origin: Pons

Innervation: lateral rectus muscle

Function: motor -outward lateral movement of eye

Dysfunction: loss of lateral eye movement, diplopia



Abducens nerve lesion



12. Disorders of trigeminal nerve.

Overview

- Trigeminal nerve: CN5, CNV
- Ophthalmic division, trigeminal nerve: CNV1
- Maxillary division, trigeminal nerve: CNV2
- Mandibular division, trigeminal nerve: CNV3
- Fifth cranial nerve, nervus trigeminus
- Great sensory cranial nerve of head and face; motor nerve for muscles of mastication
- Mixed nerve (both sensory, motor components)
- *Four segments: Intra-axial, cisternal, interdural and extracranial*

Intra-Axial Segment

Four nuclei (3 sensory, 1 motor)

Located in brainstem, upper cervical cord

Mesencephalic nucleus CNS

- Slender column of cells projecting cephalad from pons to level of inferior colliculus
- Found anterior to upper fourth ventricle/aqueduct near lateral margin of central gray
- Afferent fibers for facial proprioception (teeth, hard palate and temporomandibular joint)
- Sickle-shaped mesencephalic tract descends to motor nucleus, conveys impulses that control mastication and bite force

Divisions (Post-Ganglionic) of CNS

Maxillary nerve (CNV2)

- Courses in cavernous sinus lateral wall below CNV 1
- Exits skull through foramen rotundum
- Traverses roof of pterygopalatine fossa
- Continues as infraorbital nerve in floor of orbit
- Exits orbit through infraorbital foramen
 - Sensory to cheek and upper teeth

Divisions (Post-Ganglionic) of CNS

Mandibular nerve (CNV3)

- Does not pass through cavernous sinus
- Exits directly from Meckel cave, passing inferiorly through foramen ovale into masticator space
- Carries both motor and sensory fibers
 - Motor root bypasses TG, joins V3 as it exits through foramen ovale
 - Divides into masticator (muscles of mastication) and mylohyoid nerves (mylohyoid and anterior belly of digastric muscles)
 - Masticator nerve take off just below skull base
 - Mylohyoid nerve take off at mandibular foramen
 - Main sensory branches include inferior alveolar, lingual and auriculotemporal nerves

Clinical Importance

- Sensory complaints: Pain, burning, numbness in face
- Motor (V3 only): Weakness in chewing
 - Proximal V3 injury causes motor atrophy of masticator muscles within 6 weeks to 3 months
 - Distal V3 injury (above mylohyoid nerve takeoff) affects only anterior belly of digastric & mylohyoid
- Tic douloureux (trigeminal neuralgia)
 - Sharp, excruciating pain in V2-3 distributions
 - Look for vascular compression at REZ (on MR)

Cranial Nerve V: The Trigeminal Nerve

- The trigeminal nerve is the largest cranial nerve.
- It is composed of a ***large sensory root that runs medial to a smaller motor root.***
- The roots emerge from the lateral midpons and travel anteriorly through the prepontine cistern and the porus trigeminus to the Meckel (trigeminal) cave, a CSF-containing pouch in the middle cranial fossa.
- Because the trigeminal nerve is large and its course proceeds straight forward from the lateral pons, it is easy to recognize on most MR images.

Cranial Nerve V: The Trigeminal Nerve

- In the Meckel cave, the nerve forms a meshlike web that can be visualized only with high-resolution imaging.
- Along the anterior aspect of the cavity, the trigeminal nerve forms the trigeminal (gasserian) ganglion before splitting into three subdivisions.
- The ophthalmic (V1) and maxillary (V2) divisions of the nerve move medially into the cavernous sinus and exit the skull through the superior orbital fissure and foramen rotundum, respectively.
- The mandibular division (V3), which includes the motor branches, exits the skull inferiorly through the foramen ovale.

From another source:

Cranial Nerves

- Indicated by Roman numerals I-XII from anterior to posterior
- May have one or more of 3 functions
 - Sensory (special or general)
 - Somatic motor (skeletal muscles)
 - Parasympathetic (regulation of glands, smooth muscles, cardiac muscle)

12 pairs of Cranial Nerves arise from the forebrain and the brain stem.

I Olfactory Telencephalon

II Optic Diencephalon

Cranial Nerves III through XII
arise from the Brain Stem:

Midbrain

III Oculomotor

IV Trochlear

Pons

V Trigeminal

VI Abducens

VII Facial

Medulla

VIII Vestibulocochlear

IX Glossopharyngeal

X Vagus

XII Hypoglossal

Cranial Nerve XI -

Spinal Accessory Nerve

arises from the **Spinal Cord and medulla**

Trigeminal nerve

Origin: pons. The sensory nuclei extend from the pons to midbrain, and also to the medulla and spinal cord

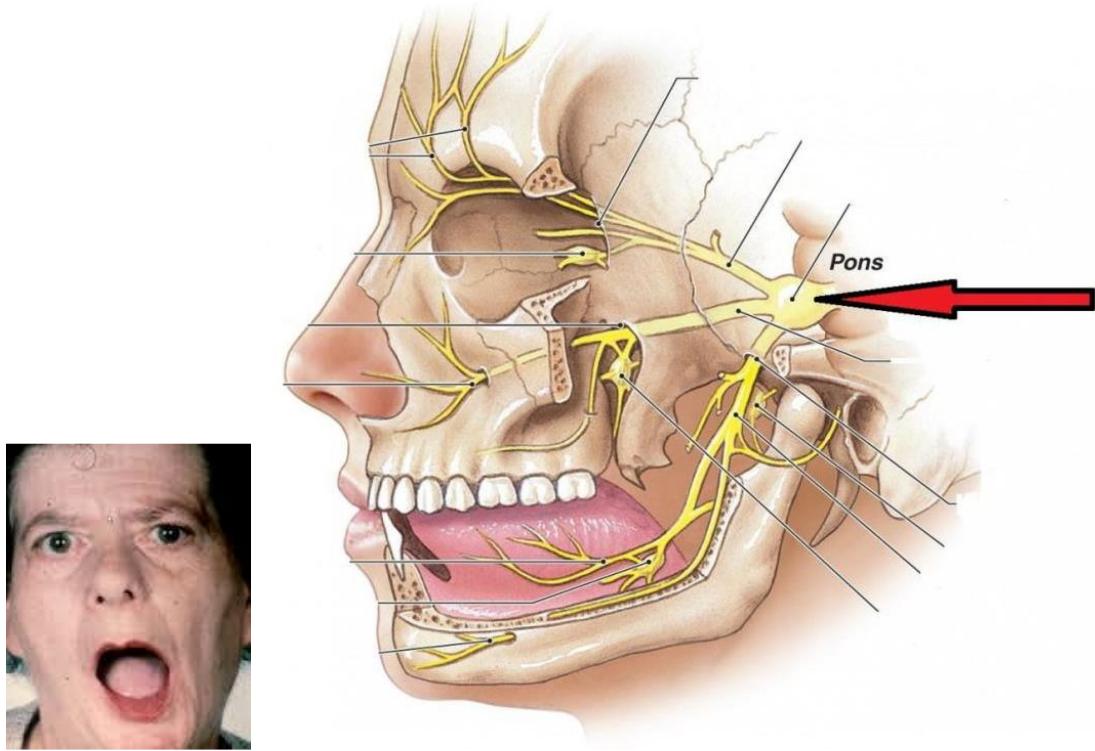
Innervation: sensory innervation to head's skin and mucous membranes, teeth, tongue, external auditory canal, cornea etc.
Motor innervation of masseter and temporal muscles

Three branches: ophthalmic, maxillary and mandibular

Trigeminal nerve

Function: sensory and motor -sensation of pain, touch, temperature; movement of masseter and temporal muscles

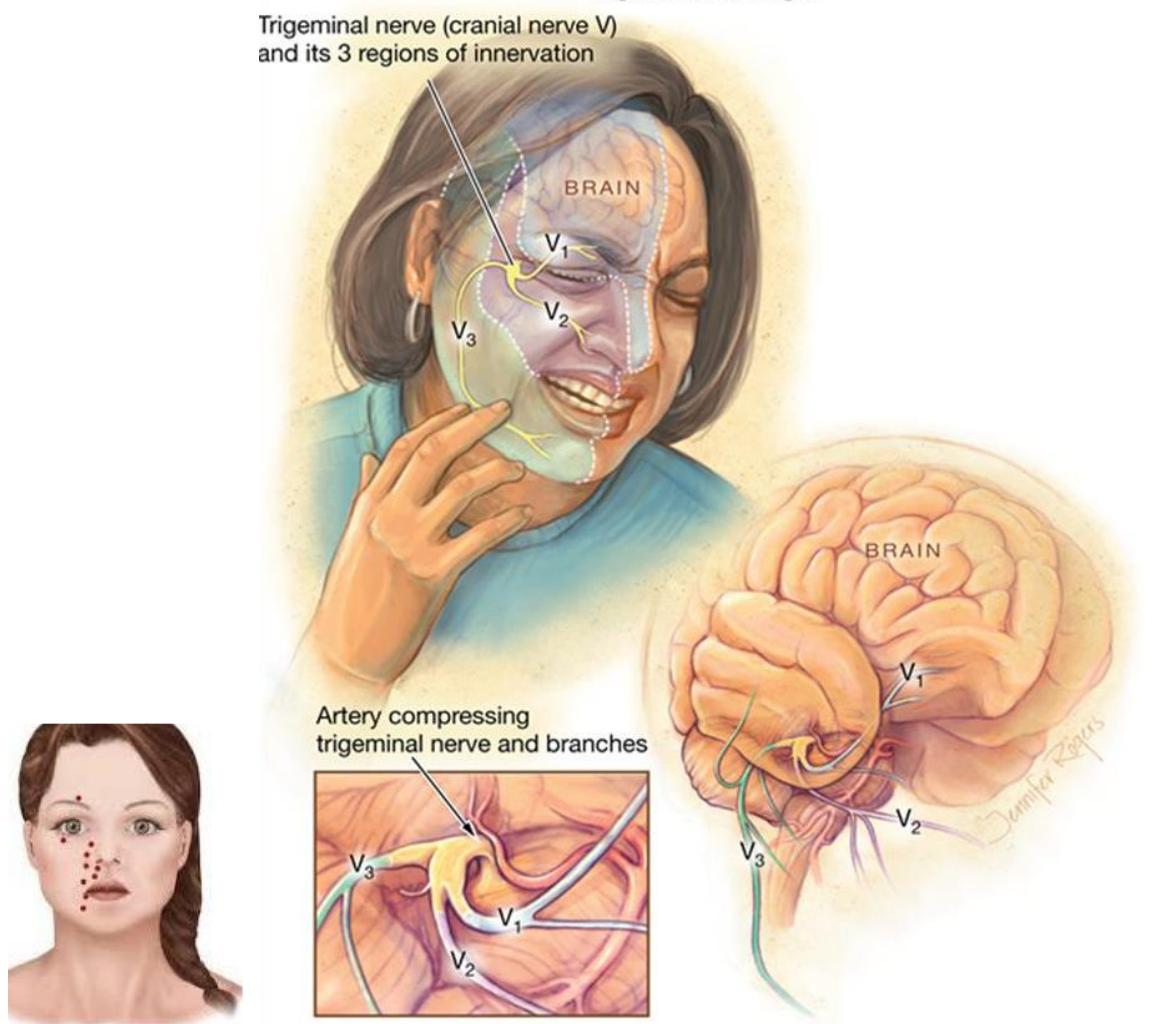
Dysfunction: severe pain, paraesthesia, loss of sensation on face, weakness of chewing, deviation of jaw



Trigeminal neuralgia

- Paroxysmal attack of severe, short, sharp, stabbing pain affecting one or more branch of the nerve
- Chewing, speaking, washing face, cold water, air flow may precipitate pain attack -TRIGGER POINT

Trigeminal neuralgia



Trigeminal nerve examination

- Sensation: light touch, superficial pain and temperature sensation on forehead, cheeks and jaw
- Muscle power of masseter and temporal muscle.





- Corneal response

Light touch cornea with wisp of wet cotton. Response: closing of both eyes

-Afferent –ophthalmic branch of trigeminal nerve

-Efferent –facial nerve

- Jaw jerk

Ask patient to relax jaw. Place finger on the chin and tap with hammer.

Response: closing of mouth

-Afferent –mandibular branch of trigeminal nerve

-Efferent -Motor root of trigeminal nerve



13. Facial nerve disorders.

VII Facial n.	nucleus of the facial n. (pons, motor fibers for the muscles of facial expression), superior salivatory nucleus (secretory fibers for the lacrimal, nasal, and palatal glands), nucleus of the tractus solitarius (gustatory fibers), nerve	innervation of the muscles of facial expression and the stapedius m.; lacrimation and salivation; taste on the anterior two-thirds of the tongue
---------------	---	--

Table 11-2 Cranial Nerves (Continued)

Number	Name	Components ^a	Function	Opening in Skull
VII	Facial	Motor (SVE)	Muscles of face and scalp, stapedius muscle, posterior belly of digastric and stylohyoid muscles	Internal acoustic meatus, facial canal, stylomastoid foramen
		Sensory (SVA)	Taste from anterior two-thirds of tongue, from floor of mouth and palate	
		Secretomotor (GVE) parasympathetic	Submandibular and sublingual salivary glands, the lacrimal gland, and glands of nose and palate	

Table 11-1 The Letter Symbols Commonly Used to Indicate the Functional Components of Each Cranial Nerve

Component	Function	Letter Symbols
Afferent Fibers	Sensory	
General somatic afferent	General sensations	GSA
Special somatic afferent	Hearing, balance, vision	SSA
General visceral afferent	Viscera	GVA
Special visceral afferent	Smell, taste	SVA
Efferent Fibers		
General somatic efferent	Somatic striated muscles	GSE
General visceral efferent	Glands and smooth muscles (parasympathetic innervation)	GVE
Special visceral efferent	Branchial arch striated muscles	SVE

Facial nerve

Origin: pons, medulla

Innervation: facial muscle, anterior 2/3 of tongue (taste), lacrimal and salivatory glands (sublingual, submandibular)

Function: motor, parasympathetic, sensory

- control of facial movement (expressions)
- motor realization of corneal and blink reflex
- secretion of lacrimal and salivatory glands
- taste sensation of anterior 2/3 of tongue

Facial nerve examination

Motor function

-Observe facial symmetry

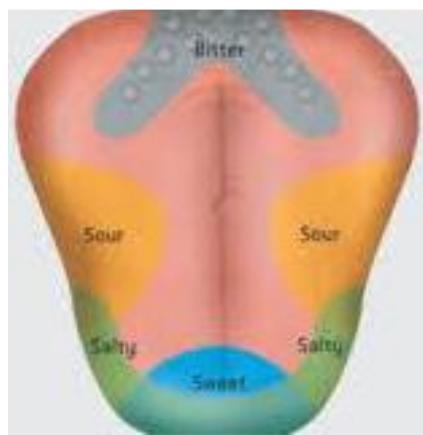
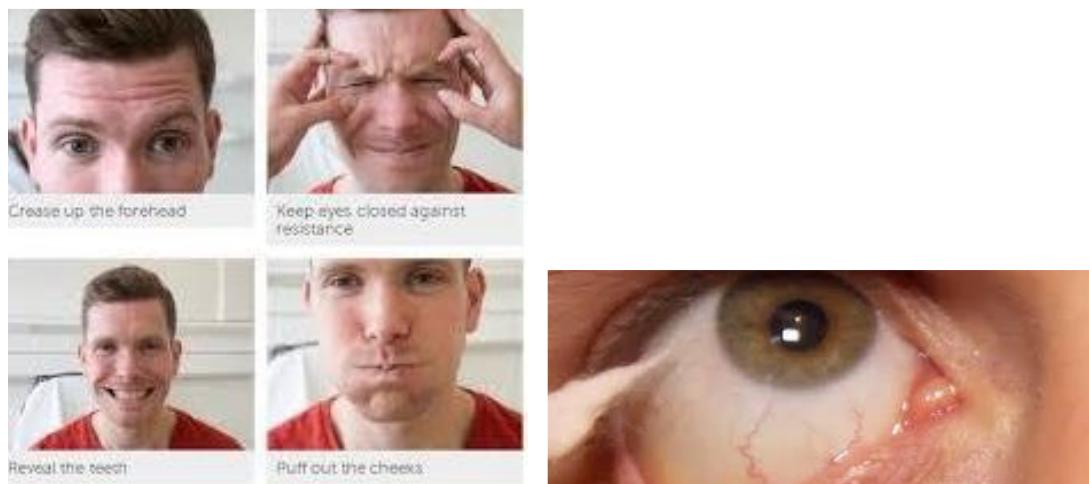
- Flattening of nasolabial fold
- Ask patient to wrinkle forehead, close eyes against resistance, smile, show teeth

Reflex

- Corneal reflex

Sensory function

- Test each sides of anterior 2/3 tongue separately
- Test for sweet, sour, salty sensation
- Give sip of water between tastes



Bell's palsy (peripheral facial nerve palsy)

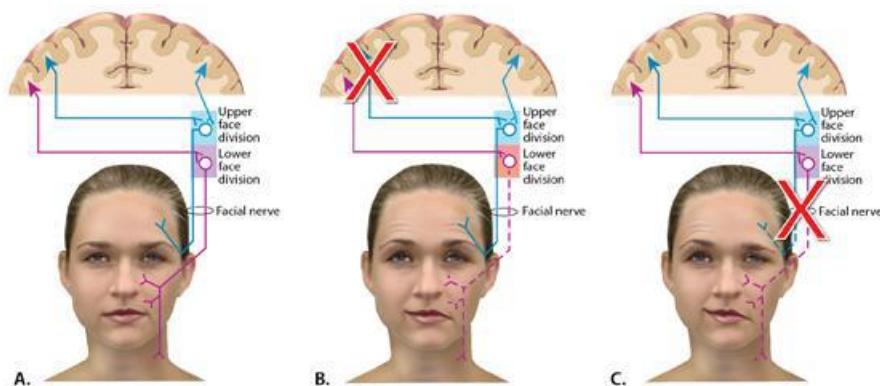
Loss of facial movement on affected side: inability to wrinkle or raise brow, close eye, drooping of the mouth corner

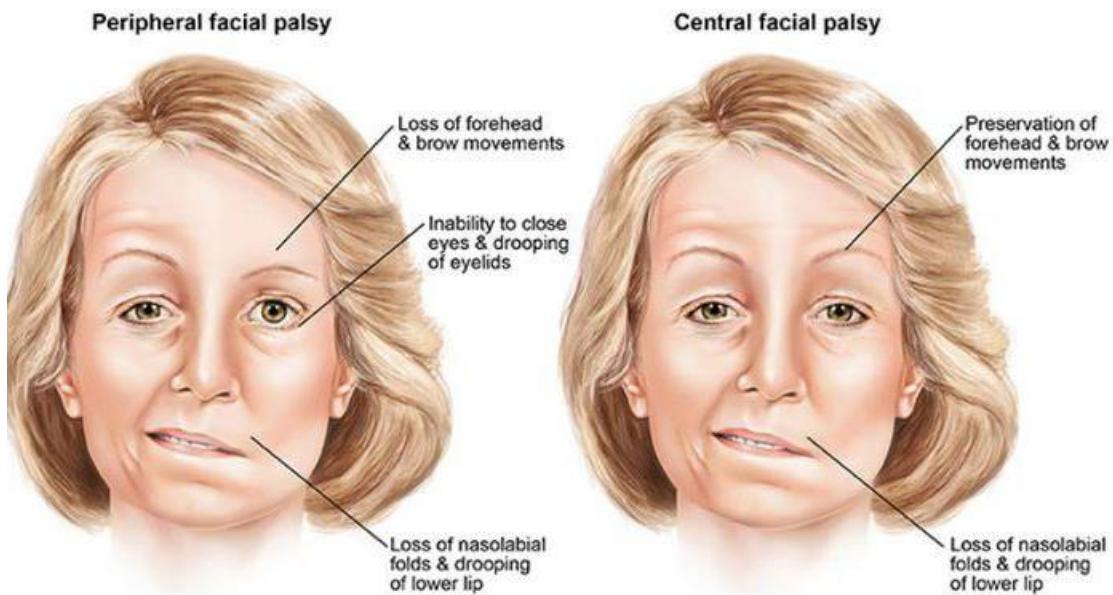
Additionally can be:

- Loss of taste on anterior 2/3 of tongue
- Hyposalivation (often is masked by functioning parotid glands)
- Dry eye
- Hyperacusis



Bell's palsy vs. central facial nerve palsy





14. VIII cranial nerve disorders.

VIII Vestibulo-cochlear n. (statoacoustic n., auditory n.)	sensory neurons in the cochlea (cochlear root) and in the semicircular canals, utricle, and saccule (vestibular root), peripheral afferent nerve trunk, brainstem nuclei, and projecting fibers to higher regions of the CNS	perception of sound and of bodily position, movement, and acceleration; regulation of balance
---	--	---

VIII	Vestibulocochlear Vestibular	Sensory (SSA)	From utricle and saccule and semicircular canals—position and movement of head	Internal acoustic meatus
	Cochlear	Sensory (SSA)	Organ of Corti—hearing	

Table 11-1 The Letter Symbols Commonly Used to Indicate the Functional Components of Each Cranial Nerve		
Component	Function	Letter Symbols
Afferent Fibers	Sensory	
General somatic afferent	General sensations	GSA
Special somatic afferent	Hearing, balance, vision	SSA
General visceral afferent	Viscera	GVA
Special visceral afferent	Smell, taste	SVA
Efferent Fibers		
General somatic efferent	Somatic striated muscles	GSE
General visceral efferent	Glands and smooth muscles (parasympathetic innervation)	GVE
Special visceral efferent	Branchial arch striated muscles	SVE

Vestibulocochlear nerve
Origin: pons and medulla

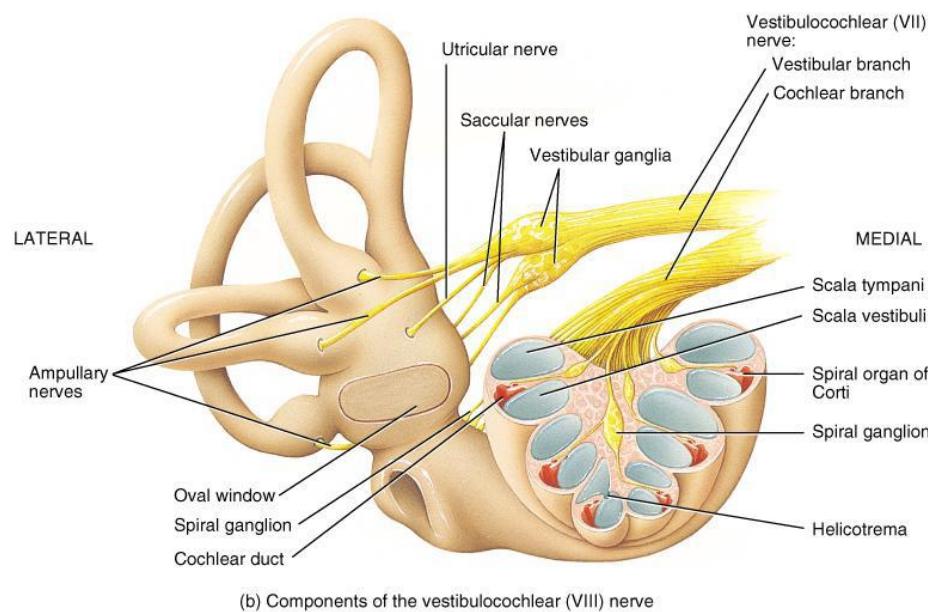
Innervation:

-Inner ear

Function: sensory

-Cochlear part –hearing

-Vestibular part –balance, maintenance of head and body position, proprioception



•Weber's test

•Rinne's test

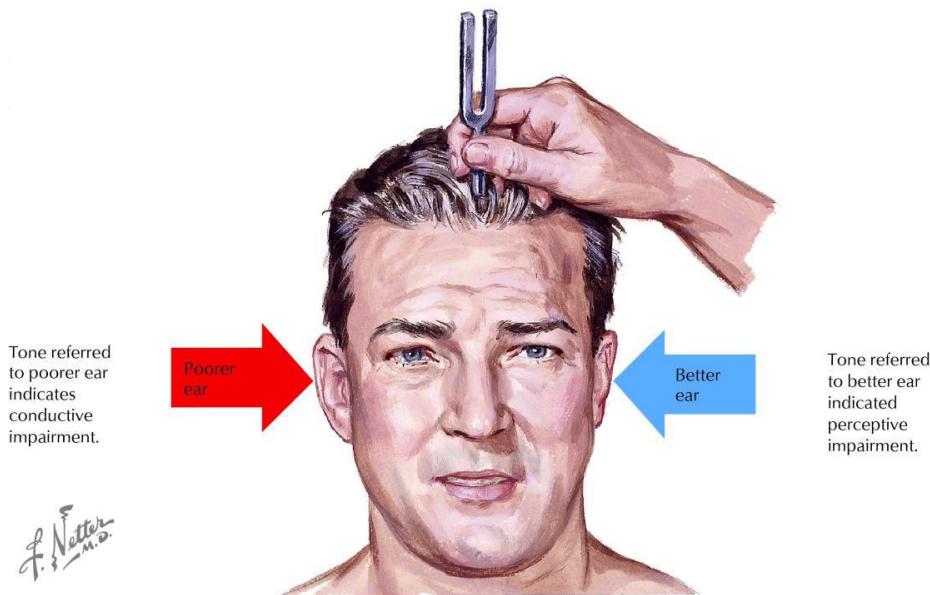
AC>BC is normal

BC>AC –middle ear disease

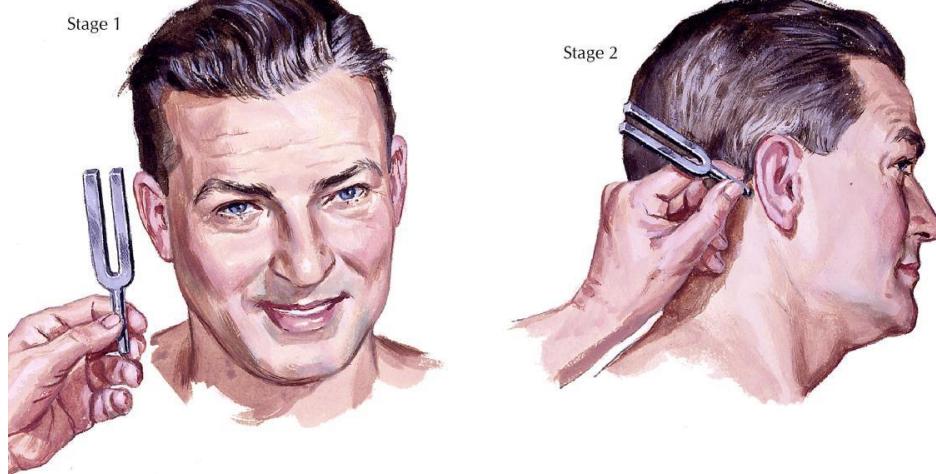
Both diminished indicative of nerve damage

Hearing Test: Weber and Rinne

Weber Test



Rinne Test



Tone heard longer by air conduction = Rinne positive: indicates perceptive loss
Tone heard longer by bone conduction = Rinne negative: indicates conductive loss

Vestibulocochlear nerve

Dysfunction

- Cochlear

- Unilateral deafness

- Loss of sound differentiation

- Tinnitus

•Vestibular

-Vertigo

-Balance disturbance



VERTIGO

is a sensation of spinning. If you have these dizzy spells, you might feel like you are spinning or that the world around you is spinning.

Vertigo is often triggered by a change in the position of your head.



Don't lower
your head
beneath
your
shoulders.



Don't
extend your
neck.

People with vertigo typically describe it as feeling like they are:

- Spinning
- Tilting
- Swaying
- Unbalanced
- Pulled to one direction

Other symptoms that may accompany vertigo include:

- Feeling nauseated
- Abnormal eye movements (nystagmus)
- Headache
- Ringing in the ears
- Sweating
- or hearing loss

Symptoms can last a few minutes to a few hours or more and may come and go.

15. IX, X and XI cranial nerves and their disorders.

IX Glossopharyngeal n.	nucleus ambiguus (medulla, motor fibers for the muscles of the soft palate and pharynx), nucleus of the tractus solitarius (gustatory fibers from the posterior third of the tongue, somatosensory fibers from the palatal and pharyngeal mucosa); inferior salivatory nucleus, otic ganglion (secretory fibers for the parotid gland); nerve	motor innervation of the palatal and pharyngeal muscles; somatosensory innervation of the palatal and pharyngeal mucosa; taste on the posterior third of the tongue; control of swallowing
X Vagus n.	nucleus ambiguus (medulla, motor fibers for the muscles of the soft palate and pharynx), dorsal nucleus of the vagus n., nucleus of the tractus solitarius (visceromotor and viscerosensory fibers for the thoracic and abdominal viscera), spinal nucleus of the trigeminal n. (sensory fibers from the pharynx, larynx, and external auditory canal); nerve trunk	innervation of the laryngeal musculature, speech, sensation in the external ear canal and the posterior cranial fossa, autonomic fibers to the thoracic and abdominal viscera
XI Accessory n.	nucleus ambiguus (medulla, cranial root) and spinal nucleus of the accessory n. (C1–C5, spinal root), nerve trunk, sternocleidomastoid m. and upper portion of the trapezius m.	turning the head to the opposite side, shrugging the shoulders

IX	Glossopharyngeal	Motor (SVE) Secretomotor (GVE) parasympathetic Sensory (GVA,SVA,GSA)	Stylopharyngeus muscle— assists swallowing Parotid salivary gland General sensation and taste from posterior one-third of tongue and pharynx; carotid sinus (baroreceptor); and carotid body (chemoreceptor)	Jugular foramen
X	Vagus	Motor (GVE,SVE) Sensory (GVA,SVA,GSA)	Heart and great thoracic blood vessels; larynx, trachea, bronchi, and lungs; alimentary tract from pharynx to splenic flexure of colon; liver, kidneys, and pancreas	Jugular foramen
XI	Accessory Cranial root Spinal root	Motor (SVE)	Muscles of soft palate (except tensor veli palatini), pharynx (except stylopharyngeus), and larynx (except cricothyroid) in branches of vagus Sternocleidomastoid and trapezius muscles	Jugular foramen

Table 11-1 The Letter Symbols Commonly Used to Indicate the Functional Components of Each Cranial Nerve

Component	Function	Letter Symbols
Afferent Fibers	Sensory	
General somatic afferent	General sensations	GSA
Special somatic afferent	Hearing, balance, vision	SSA
General visceral afferent	Viscera	GVA
Special visceral afferent	Smell, taste	SVA
Efferent Fibers		
General somatic efferent	Somatic striated muscles	GSE
General visceral efferent	Glands and smooth muscles (parasympathetic innervation)	GVE
Special visceral efferent	Branchial arch striated muscles	SVE

Glossopharyngeal nerve

Origin: medulla

Innervation: mucous membranes of tonsils, pharynx, posterior third of tongue, pharyngeal muscles, carotid sinus and carotid body

Function: sensory, parasympathetic and motor -touch, temperature, pain sensation of pharynx, taste sensation of posterior third of tongue, sensory part of gag and swallow reflex, secretion of parotid gland, movement of stylopharyngeus muscle

Dysfunction: decrease of gag reflex, swallowing problems, neuralgia, taste absence on posterior third of tongue

Vagus nerve

Origin: medulla

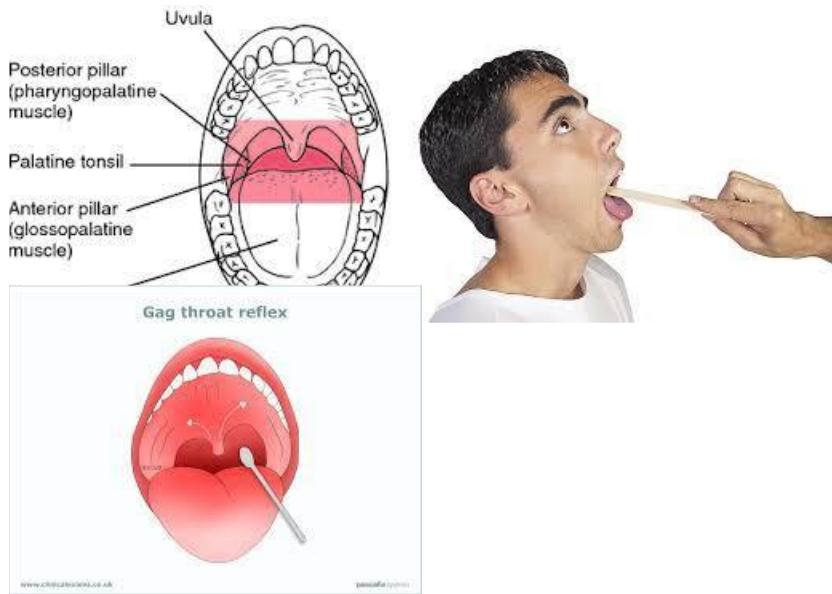
Innervation: muscles of pharynx, larynx and soft palate, parasympathetic innervation of thoracic and abdominal viscera

Function: parasympathetic and motor -motor part of gag and swallowing reflex, innervation of heart, lungs, digestive tract and spleen

Clinical evaluation

Glossopharyngeal and vagus nerve are always tested together

- evaluate voice quality (dysarthria, dysphonia, hoarseness)
- ask patient to open mouth, say “ah”, observe elevation of soft palate, midline position of uvula
- test gag reflex and soft palate reflex bilaterally
- swallowing
- taste sensation on posterior third of tongue (bitter)



- Negative findings

Loss of voice quality (dysarthria, dysphonia, hoarseness)

Deviation of uvula toward non-paralyzed side

Swallowing difficulty or nasal regurgitation



Spinal accessory nerve

Origin: spinal cord and medulla

Innervation: sternocleidomastoid and trapezius muscles

Function: motor -the sternocleidomastoid muscle tilts and rotates the head, the trapezius muscle has several actions on the scapula, including shoulder elevation and abduction of the arm.

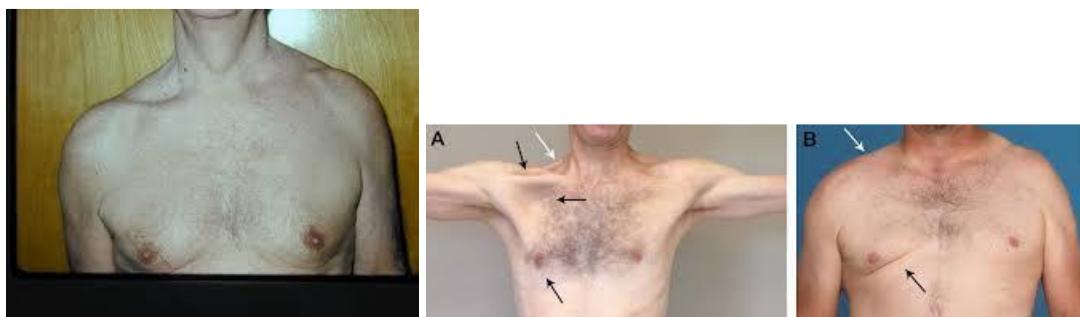
Dysfunction: muscles weakness

Clinical evaluation

- Observe muscles symmetry, scapula position and palpate muscles
- Ask patient to turn head one side and flex head against resistance, palpate sternocleidomastoid muscles
- Ask patient to lift shoulders against resistance



Spinal accessory nerve damage





Bulbar palsy

•impairment of function of the cranial nerves IX, X, and XII, which occurs due to a lower motor neuron lesion (nuclear or fascicular level in the medulla oblongata or from lesions of the cranial nerves outside the brainstem)

Main symptoms:

- Dysphagia (difficulty in swallowing)
- Dysphonia (defective use of the voice, inability to produce sound due to laryngeal weakness)
- Dysarthria (difficulty in articulating words)

Clinical signs

- Gag and soft palate reflexes are diminished or absent
- Atrophy of tongue
- Weakness of soft palate, nasal regurgitation can occur

Pseudobulbar palsy

- impairment of function of cranial nerves IX-XII due to upper motor neuron lesions of the corticobulbar tracts in the mid-pons. Such lesions must be bilateral as these cranial nerve nuclei receive bilateral innervation.

Main symptoms:

- Dysphagia (difficulty in swallowing)
- Dysphonia (defective use of the voice, inability to produce sound due to laryngeal weakness)
- Dysarthria (difficulty in articulating words)

Clinical signs:

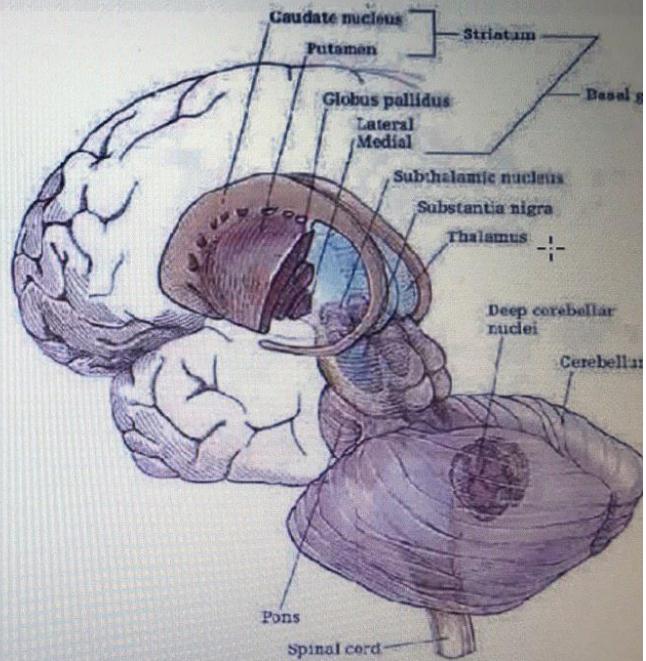
- Gag and soft palate reflexes are increased
- Atrophy is not present
- Pathological laughter or crying
- Brisk jaw jerk
- Frontal release signs are positive (palmomentalreflex, snout reflex, glabellarreflex a et.)

16. Disorders of motor coordination. Ataxia.

For this question should read points number 1 and 2 and number 3 for better understanding

1.

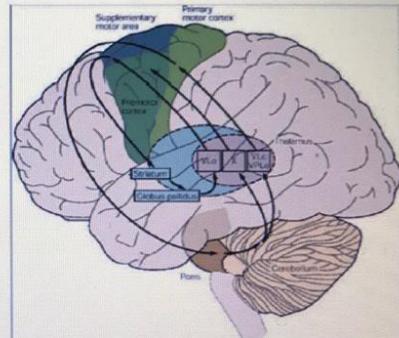
Cerebellum and basal ganglia dysfunction. Gait disorders



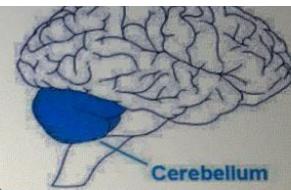
Motor system control

- **Pyramidal system**
provides voluntary movements by directly innervating motor neurons of the spinal cord or brainstem
- **Extrapyramidal system (basal ganglia)**
modulates motor activity, helps plan and control complex patterns of movements
- **Cerebellum**
monitors and makes corrective adjustment to motor plan, major role in timing of motor activities

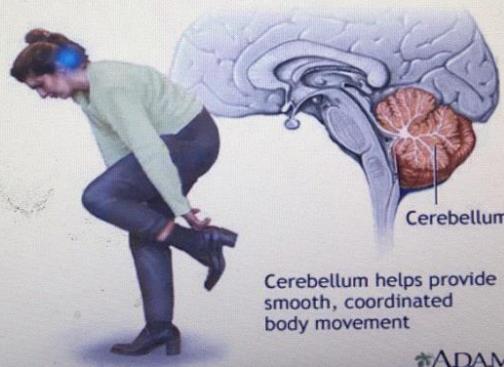
MOTOR CORTEX AFFERENT



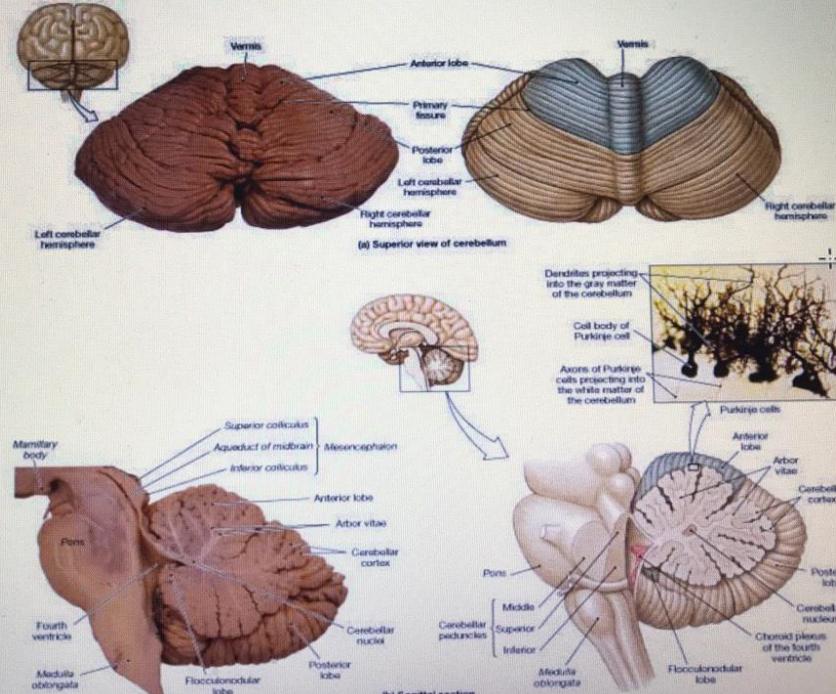
Cerebellum



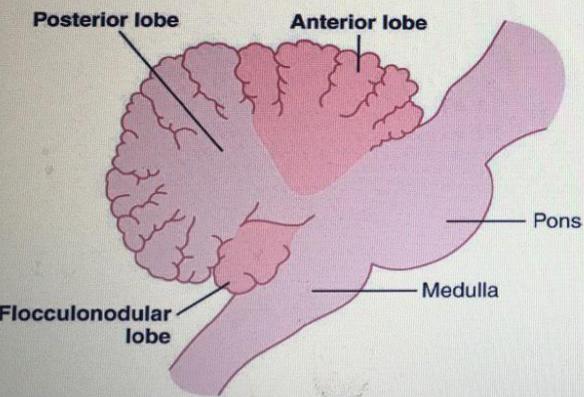
- plays an important role in motor control and motor memory
- does not initiate movement, but it contributes to coordination, precision, and accurate timing
- Cerebellar damage produces disorders in fine movement, equilibrium, posture, muscle tone and motor learning.



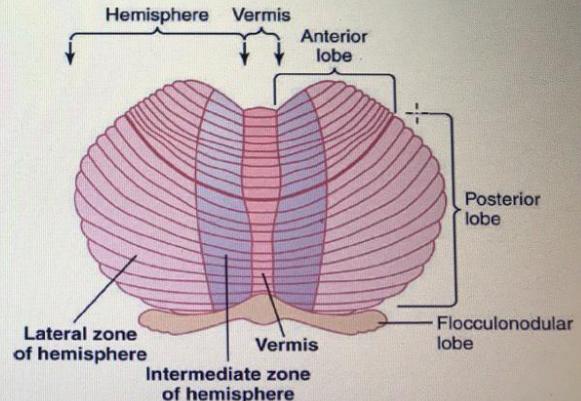
Cerebellum anatomy



Anatomical Functional Areas of the Cerebellum



Hall, Guyton and Hall Textbook of Medical Physiology, 12th Edition
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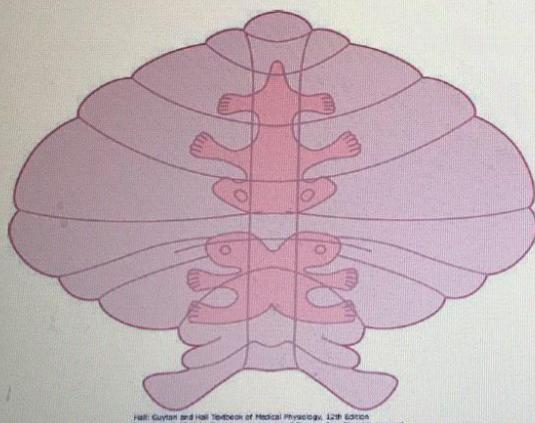


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Fig. 56.1 Anatomical lobes of the cerebellum

Fig. 56.2 Functional parts of the cerebellum

Anatomical Functional Areas of the Cerebellum



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3 broad classes of human movements controlled for by the cerebellum

Vestibulocerebellum - control postural movements, regulates balance and eye movements.

Spinocerebellum - feedback control of distal limb movement, prevention of overshooting of movements, control of ballistic movements

Cerebrocerebellum - Planning of sequential movements, timing function, extramotor predictive functions

Cerebellar tracts

- 3 highways leading in and out "peduncles"

- Superior (mixed) connects to midbrain

Cerebellothalamic tract

Cerebellorubral tract

Cerebelloreticular tract

Ventral spinocerebellar tract

- Middle – connects to pons (afferent)

Pontocerebellar tract

- Inferior – connects to medulla oblongata (afferent)

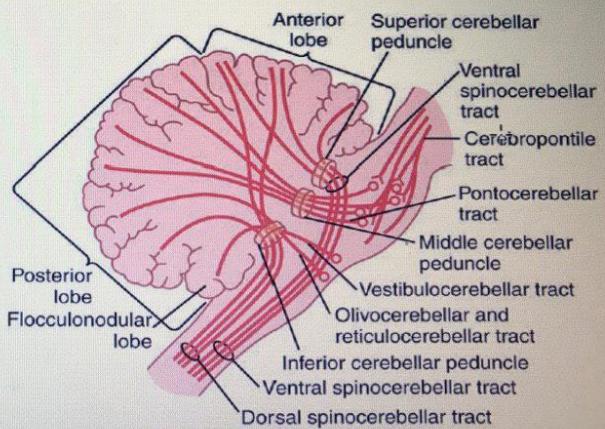
Dorsal spinocerebellar tract

Bulbo-cerebellar tract

Olivo-cerebellar tract

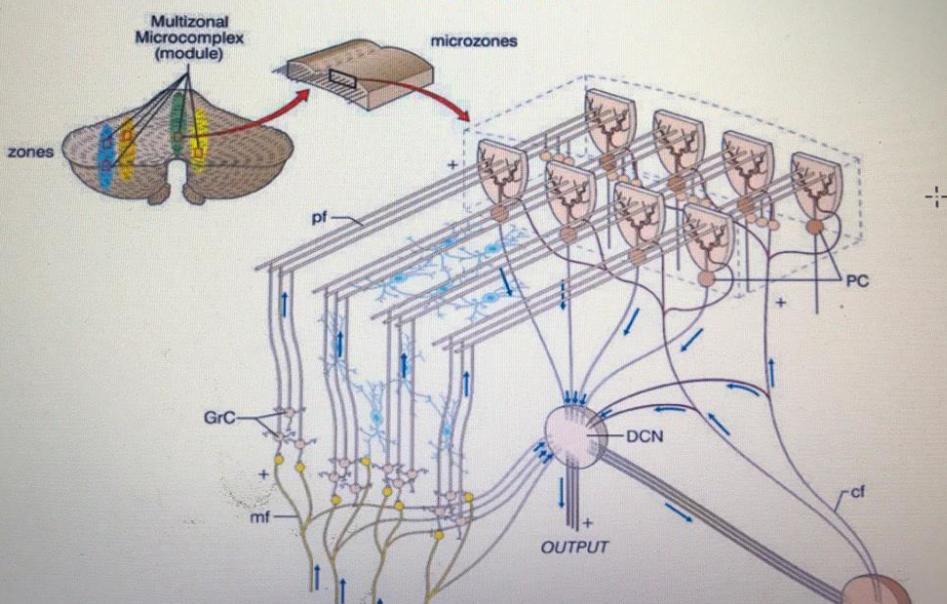
Vestibulo-cerebellar tract

Reticulo-cerebellar tract



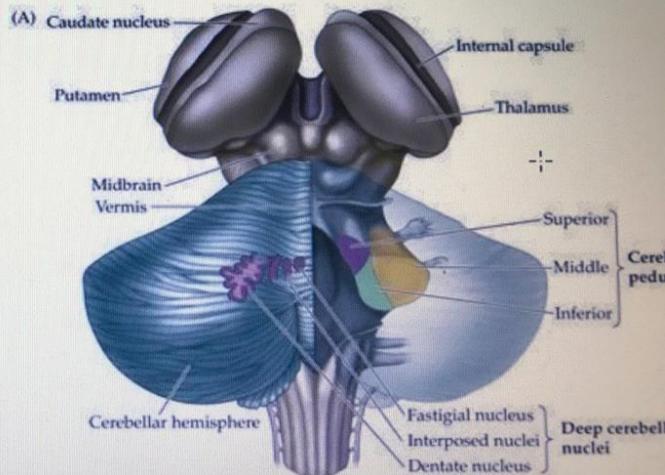
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High density of neurons in cerebellar cortex results in cerebellum accounting for 1/10 of total brain volume but contains more than 50% of CNS neurons

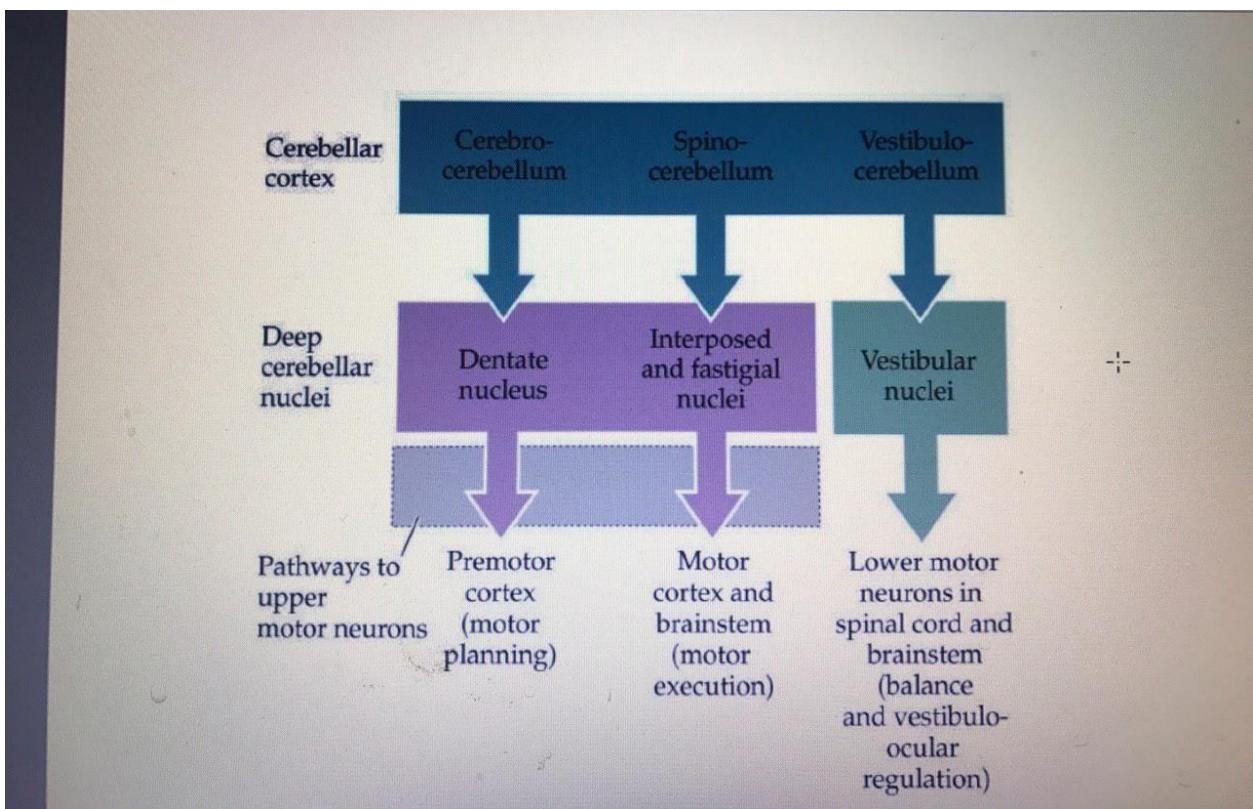


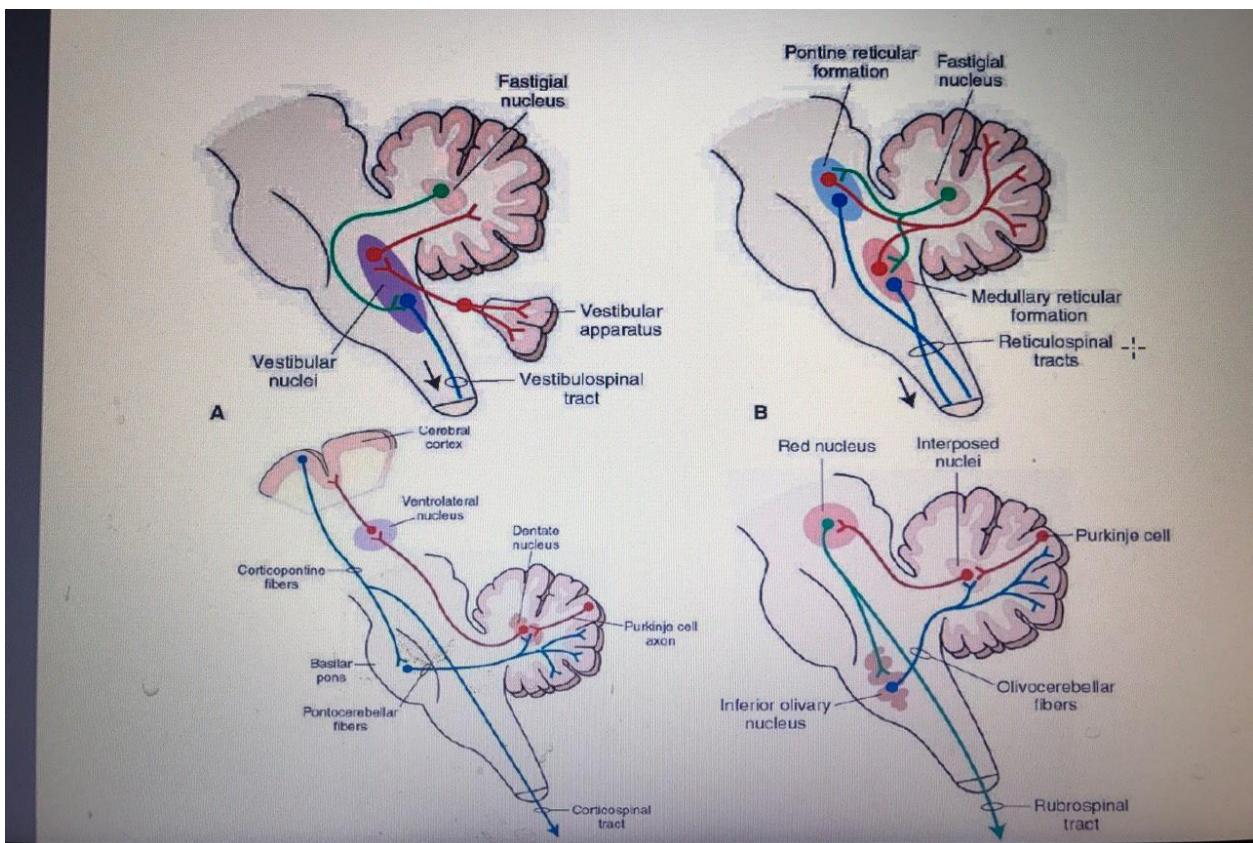
Deep cerebellar nuclei

- receive input from cerebellar cortex and send projection to thalamus
- Dentate nucleus
- Interposed nuclei (globose and emboliform)
- Fastigial nucleus



NEUROSCIENCE, Fourth Edition, Figure 19.1 (Part 1)





Motor Function- Cerebellar Function

□ Balance tests

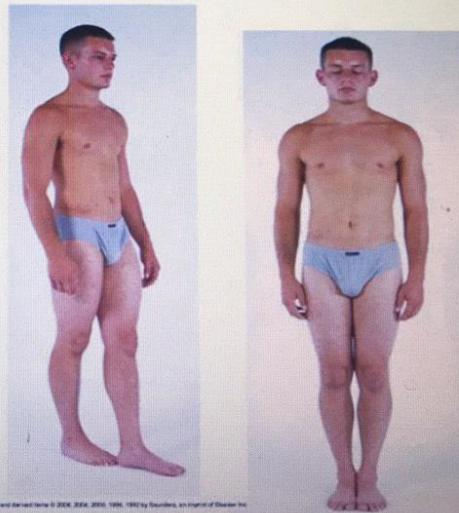
■ Gait

- Walk 10-20 feet, turn, and walk back the other way
 - *Gait smooth & coordinated*

- Tandem walk
 - *Able to tandem walk*

■ Romberg Test

- Feet together, arms at side, and eyes closed and hold 20 seconds
- No swaying = *negativ Romberg*



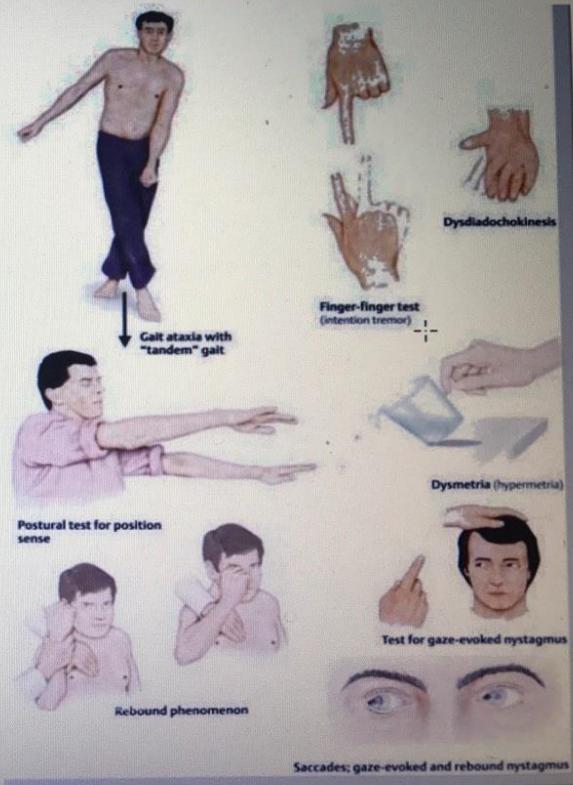
Limb examination

- finger-nose test, finger-finger test, finger-chase test
- heel-knee test
- rapid alternation movements
- rebound phenomenon

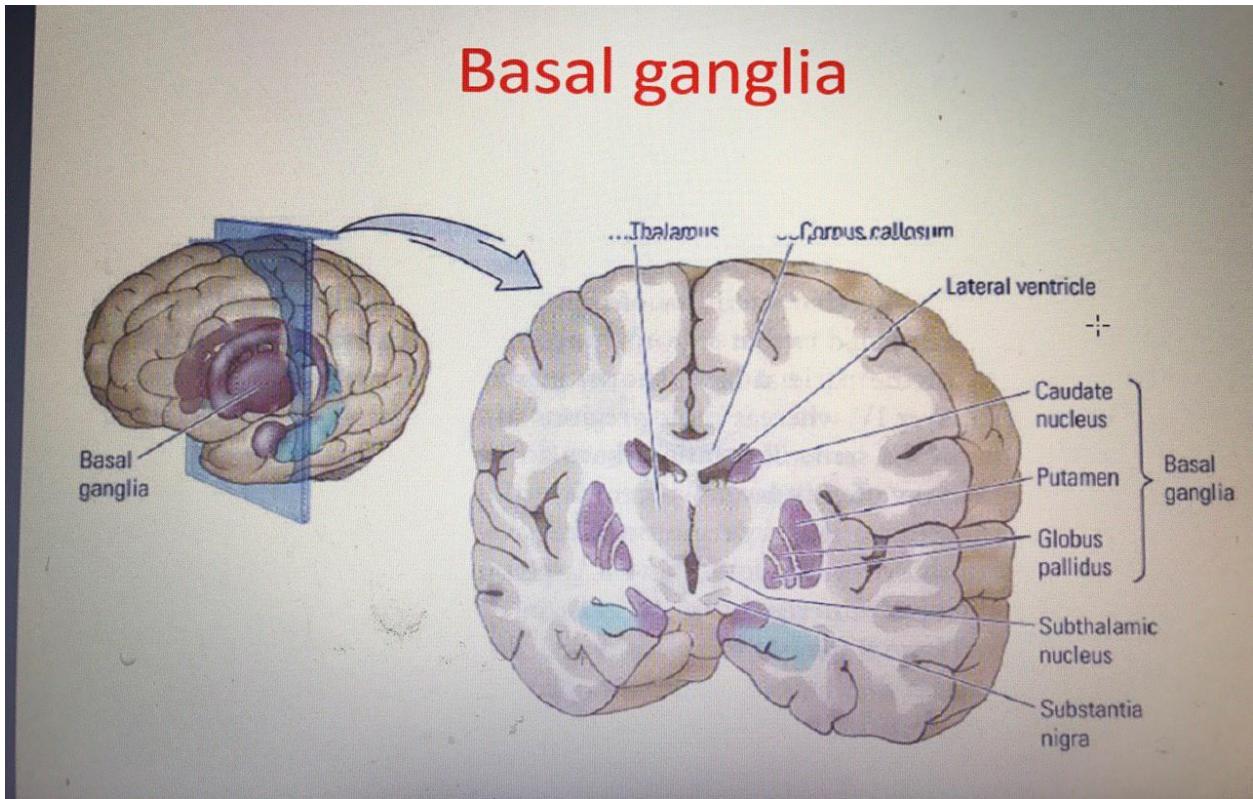


Cerebellar lesion

- decomposition of movement ie. complex movements performed as a series of successive simple movements (**dysynergia**),
- the inability to judge distance and when to stop (**dysmetria**),
- the inability to perform rapid alternating movements (**dysdiadochokinesia**),
- position and movement tremors (**postural and intention tremor**),
- staggering, wide based walking (**ataxic gait**),
- change of muscle tone (**hypotonia**),
- slurred speech (**ataxic dysarthria**)
- abnormal eye movements (**nystagmus**) or eyes have difficulty maintaining fixation; they drift from the target and then jump back with a corrective saccade

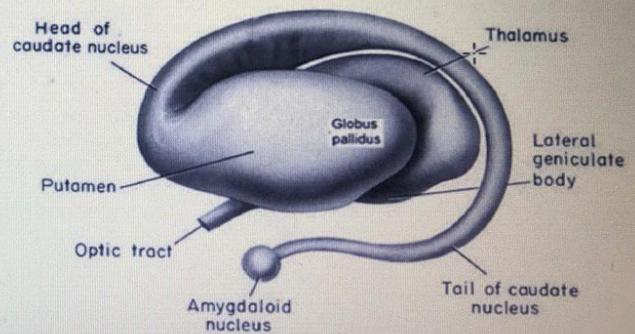


Basal ganglia



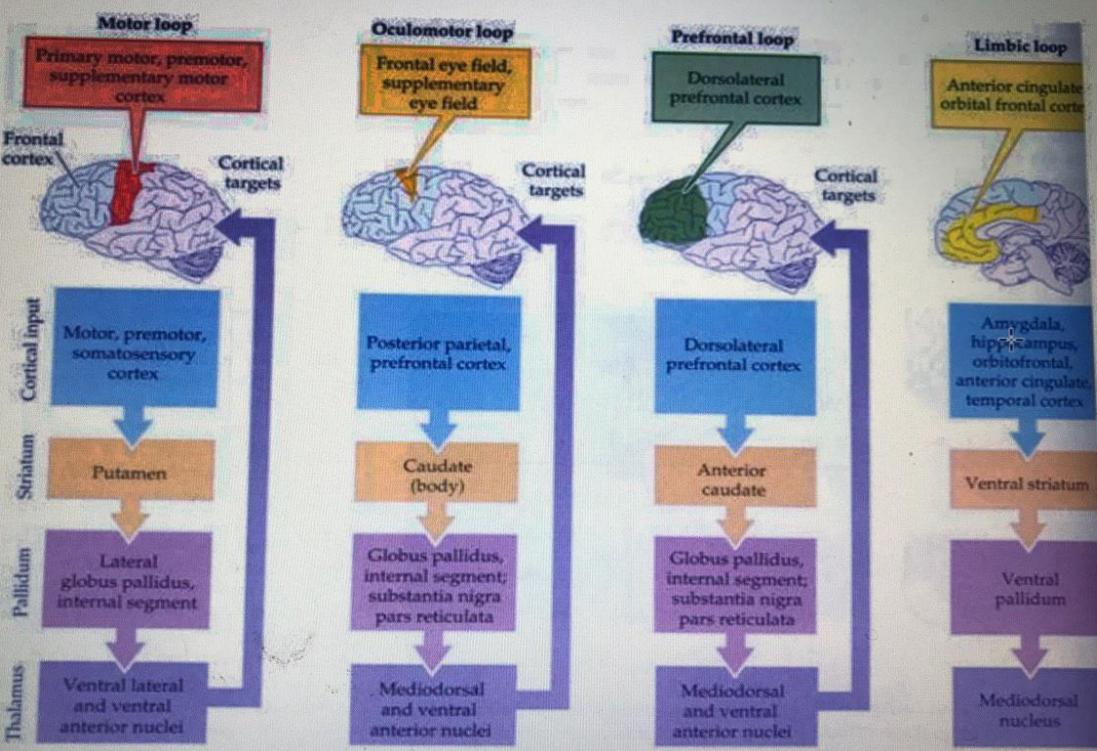
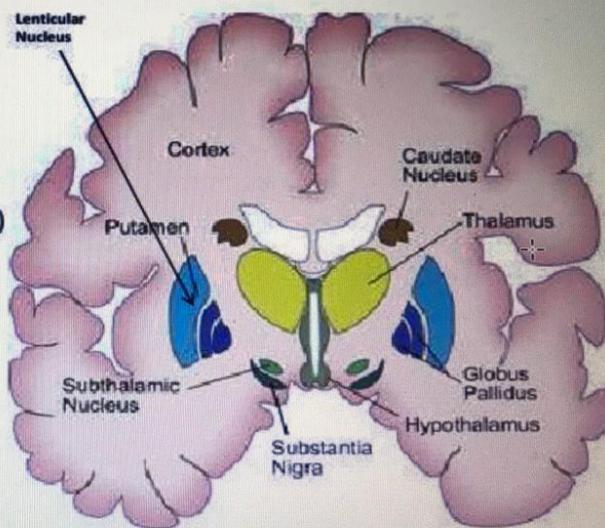
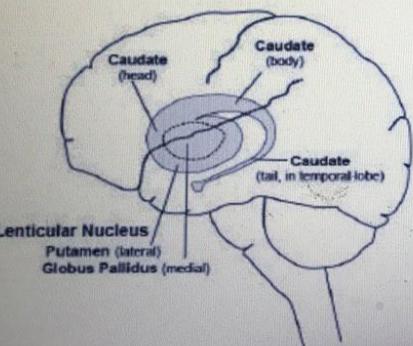
BG motor system influence

- Initiation of movement
- Programming and correcting movement while in progress
- Regulate muscle tone and force
- Change from one pattern to other
- Postural control
- Two types basal ganglia influence
 - Promotion of desired movements
 - Inhibition of unwanted movements
- Lesions of the basal ganglia result in disturbances of muscle tone and dyskinesias
 - Hyperkinesia
 - Hypokinesia



FUNCTIONAL DIVISIONS

- 1. Striatum**
 - a. caudate nucleus
 - b. putamen
- 2. Pallidum**
 - a. Globus Pallidus Interna (Gpi)
 - b. Globus Pallidus Externa (Gpe)
- 3. Thalamus**
- 4. Subthalamic Nucleus**
- 5. Substantia Nigra**



Basal Ganglia-Their Motor Functions

- **Function of the Basal Ganglia in Executing Patterns of Motor Activity—the Putamen Circuit**

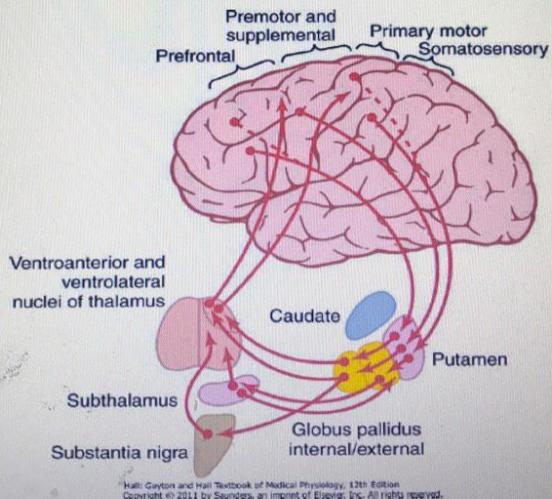
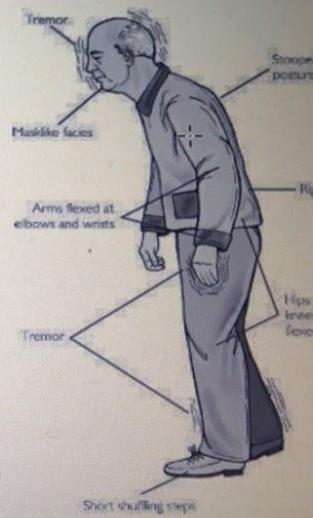


Fig. 56.11 Putamen circuit

Hall, Guyton and Hall Textbook of Medical Physiology, 12th Edition
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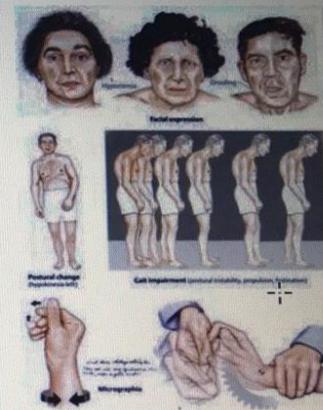
Parkinson's disease

- motor system disorder, which is the result of the loss of dopamine-producing brain cells.
- The four primary (motor) symptoms of PD are
 - resting tremor, or trembling in hands, arms, legs, jaw, and face;
 - rigidity, or stiffness of the limbs and trunk;
 - bradykinesia, or slowness of movement;
 - postural instability, or impaired balance and coordination.
- Non-motor symptoms may include depression and other emotional changes; difficulty in swallowing, chewing, and speaking; urinary problems or constipation; skin problems; and sleep disruptions.
- There are currently no blood or laboratory tests that have been proven to help in diagnosing sporadic PD.
- Therefore the diagnosis is based on medical history and a neurological examination



PD examination

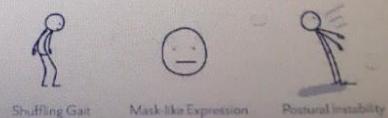
- observation (masked face, resting tremor)
- muscle tone (rigidity)
- rapid alternating movement (bradykinesia)
- postural instability
- gait



Parkinson's Disease



Other motor features:



Hyperkinesia

- **Hyperkinesia**, also known as **hyperkinesis**, refers to an increase in muscular activity that can result in excessive abnormal movements, excessive normal movements, or a combination of both.

Types of hyperkinesia

- chorea
- athetosis
- dystonia
- tremor
- hemiballismus
- myoclonus
- tics

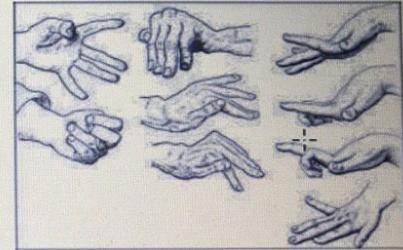
Chorea

- Chorea is an abnormal involuntary movement disorder characterized by brief, abrupt, irregular, unpredictable, non-stereotyped movements. In milder cases, they may appear purposeful; the patient often appears fidgety and clumsy. They can affect various body parts, and interfere with speech, swallowing, posture and gait.

Chorea may worsen with anxiety and voluntary movements, and subsides during sleep.

Most often causes:

Huntington's disease
Sydenham's chorea
Chorea gravidarum



Dystonia

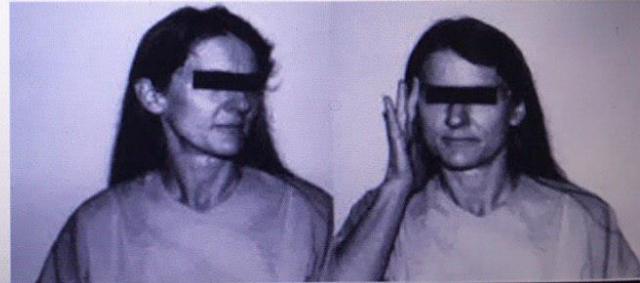
Involuntary movements and prolonged muscle contraction that result in twisting body motions, tremors, and abnormal posture. These movements may involve the entire body or only an isolated area.

- Generalized dystonia
- Focal dystonia
- Multifocal dystonia
- Segmental dystonia
- Hemidystonia



Dystonias can also be classified as syndromes based on their patterns:

- Blepharospasmus
- Cervical dystonia or torticollis
- Cranial dystonia
- Oromandibular dystonia
- Spasmodic dystonia
- Tardive dystonia
- Paroxysmal dystonia
- Torsion dystonia
- Writer's cramp



Medscape ®

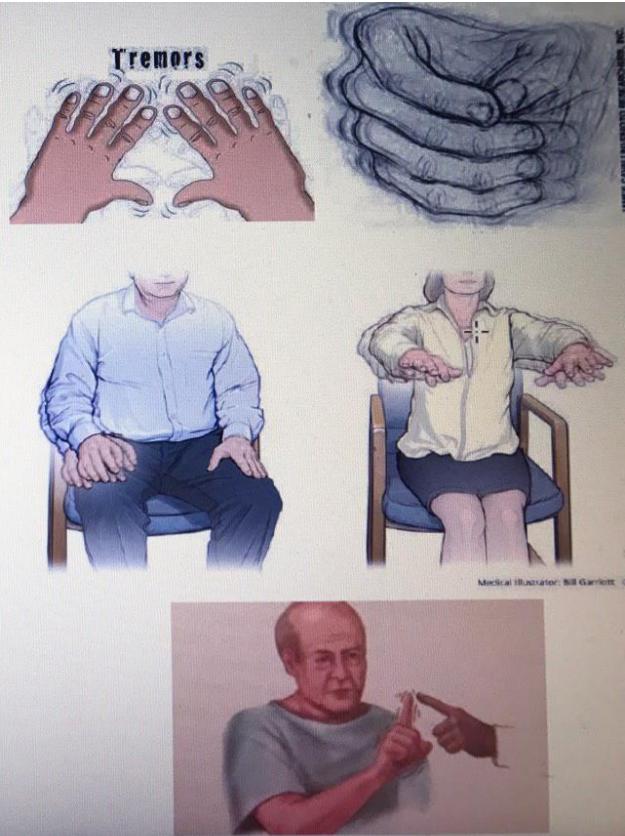
<http://www.medscape.com>

Tremor

- defined as a rhythmic, back and forth or oscillating involuntary movement about a joint axis.
- Tremors are symmetric about a midpoint within the movement, and both portions of the movement occur at the same speed.

Types of tremor

- resting tremor
- position (postural) tremor
- Intention tremor



- **Athetosis** is a *slow, continuous, involuntary writhing movement that prevents maintenance of a stable posture*.
- **Myoclonus** is a *sequence of repeated, often non-rhythmic, brief shock-like jerks due to sudden involuntary contraction or relaxation of one or more muscles*.
- **Hemiballismus** is *repetitive, but constantly varying, large amplitude involuntary movements of the proximal parts of the limbs observed on one side of the body*.
- **Tics** are *repeated, individually recognizable, intermittent movements or movement fragments that are almost always briefly suppressible and are usually associated with awareness of an urge to perform the movement*.

Comparison of motor system

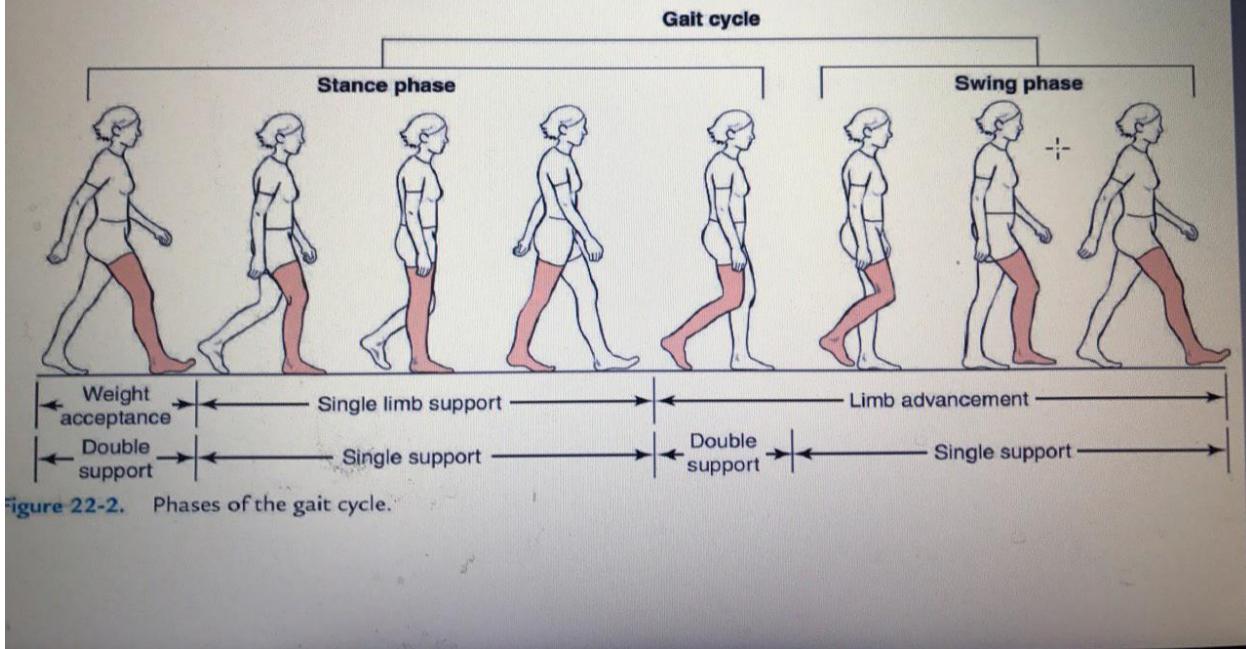
	Lower Motor Neuron Spinal Cord	Upper Motor Neuron Corticospinal Tract	Cerebellum	Basal Ganglia
Normal				
	Efferent part of monosynaptic reflex	Voluntary movement	Rapid coordinated alternating skilled movements that are learned	Facilitates intentional movements and inhibit extraneous movements
	Muscle tone by inhibiting antagonists	Muscle tone	Eye-head movements	Autopilot for motor activities
	Maintains muscle fibers (trophic factors)	Fine control, espec. finger flexors	Posture and Gait	
		Inhibitory to Lower motor neurons	Balance, equilibrium, orientation in space	Voluntary movements in an automatic manner.
	Weakness or paralysis	Weakness or paralysis	timing, duration, and amplitude	
Abnormal				
	Areflexia	Hyperreflexia Hyperactive deep tendon reflexes	Truncal ataxia, gait ataxia	Shuffling or festinating gait, small steps, hard to turn
	Fasciculation	Babinski- extensor plantar reflex	Nystagmus, Dizziness	Masked facies, few blinks
	Muscle Atrophy	S pasticity	Decomposition of movement	Difficulty turning or starting, hypokinetic = bradykinesia
	Flaccid paralysis		Dysmetria- ataxia of arms	Paucity of associated movements
			Dysynergia	
			Dysdiadochokinesia- inability to do rapid alternating movements	Chorea, athetosis, hyperkinetic
			Hypotonia- pendular reflexes	Rigidity (lead-pipe) (cogwheel).
			Intention tremor	Resting tremor
			Scanning speech	Soft speech

Gait

pattern of movement of the limbs of animals, including humans, during locomotion over a solid substrate.

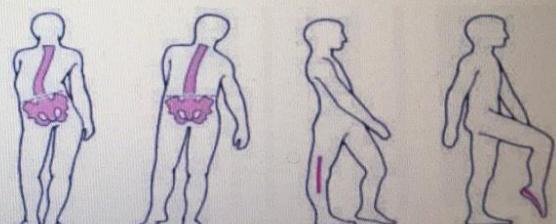
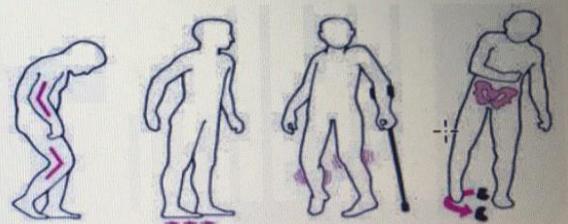
- The parameters taken into account for the gait analysis are as follows:
- *Step length*
- *Stride length*
- *Cadence*
- *Speed*
- *Dynamic Base*
- *Progression Line*
- *Foot Angle*
- *Hip Angle*
- **Pathological gait** may reflect compensations for underlying pathologies, or be responsible for causation of symptoms in itself.

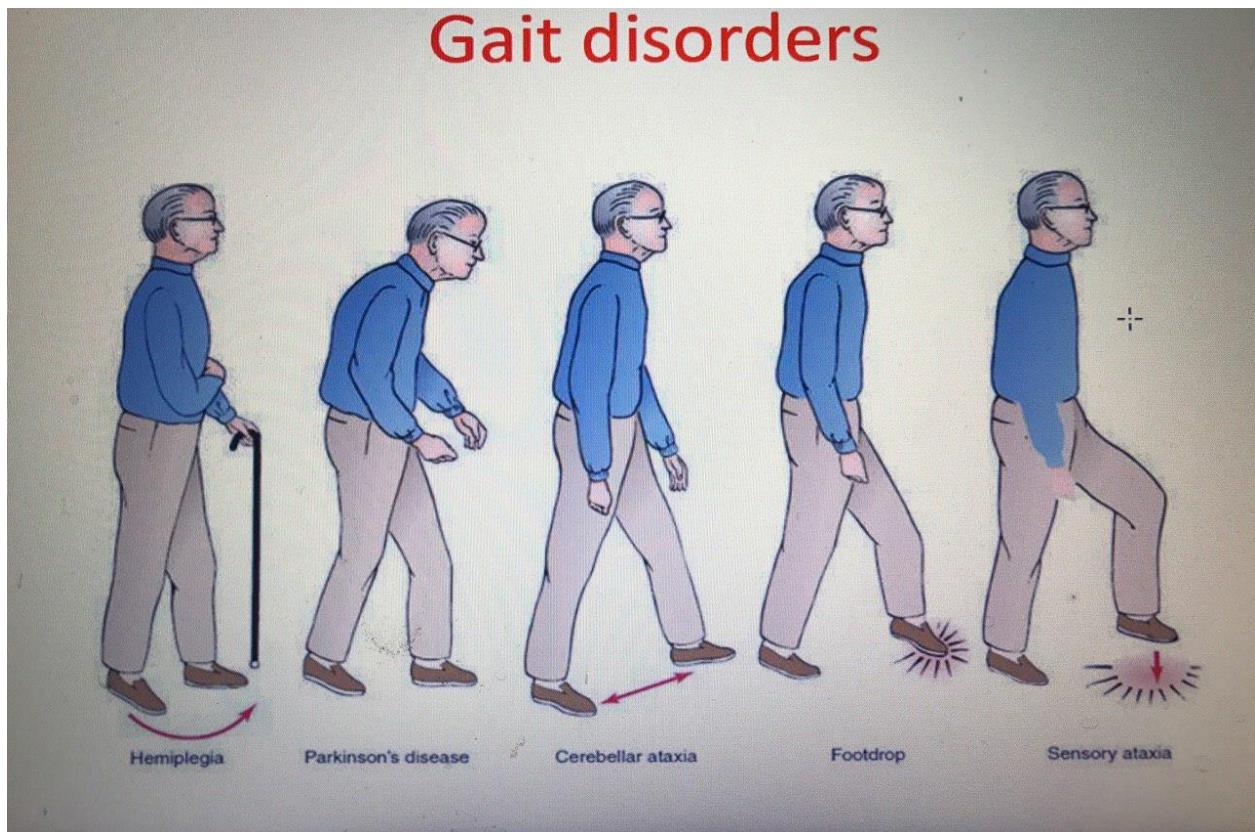
Phases of gait cycle



Types of gait abnormalities

- hemiplegic gait
- diplegic gait
- ataxic gait (cerebellar gait)
- sensory gait (stamping gait)
- myopathic gait (waddling gait)
- choreiform gait
- parkinsonian gait
- steppage gait
- antalgic gait
- gait apraxia





2. <https://www.slideshare.net/AmrHasanNeuro/cerebellum-ataxia>

(slides 18 to 39)

3. for better understanding

<https://www.slideshare.net/Physiotherapy2015/coordination-58532719>

17. Extra-pyramidal disorders.

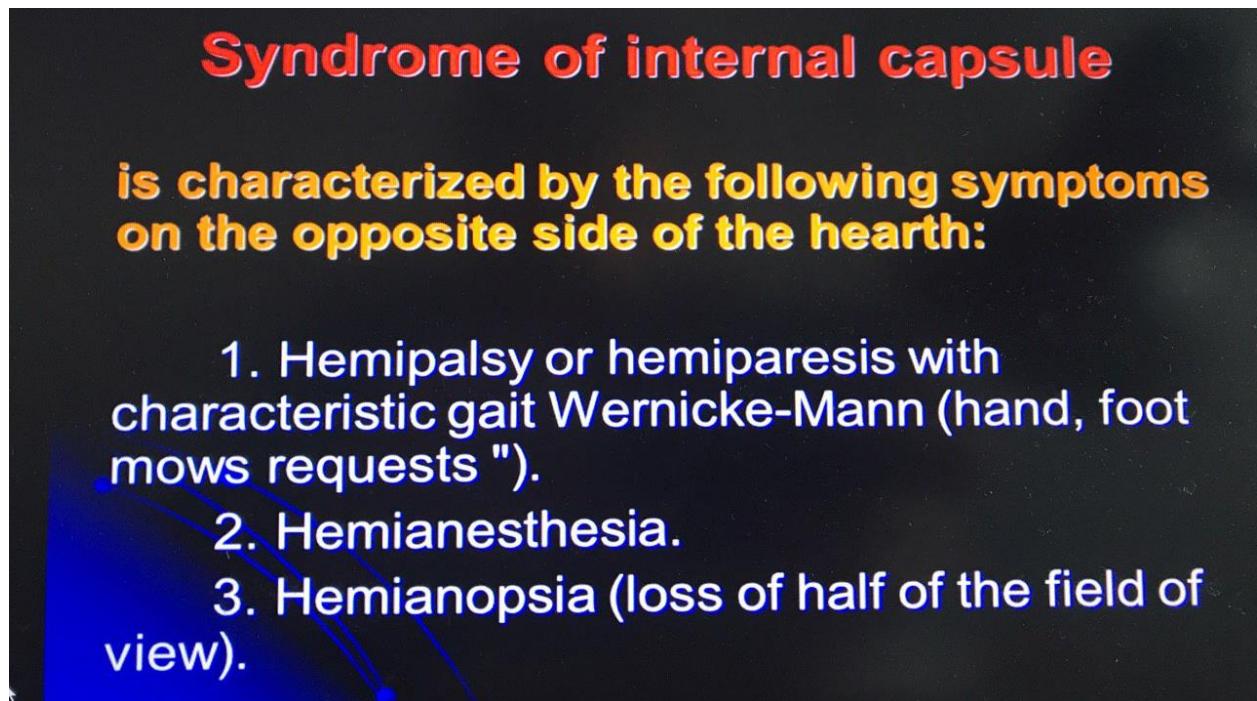
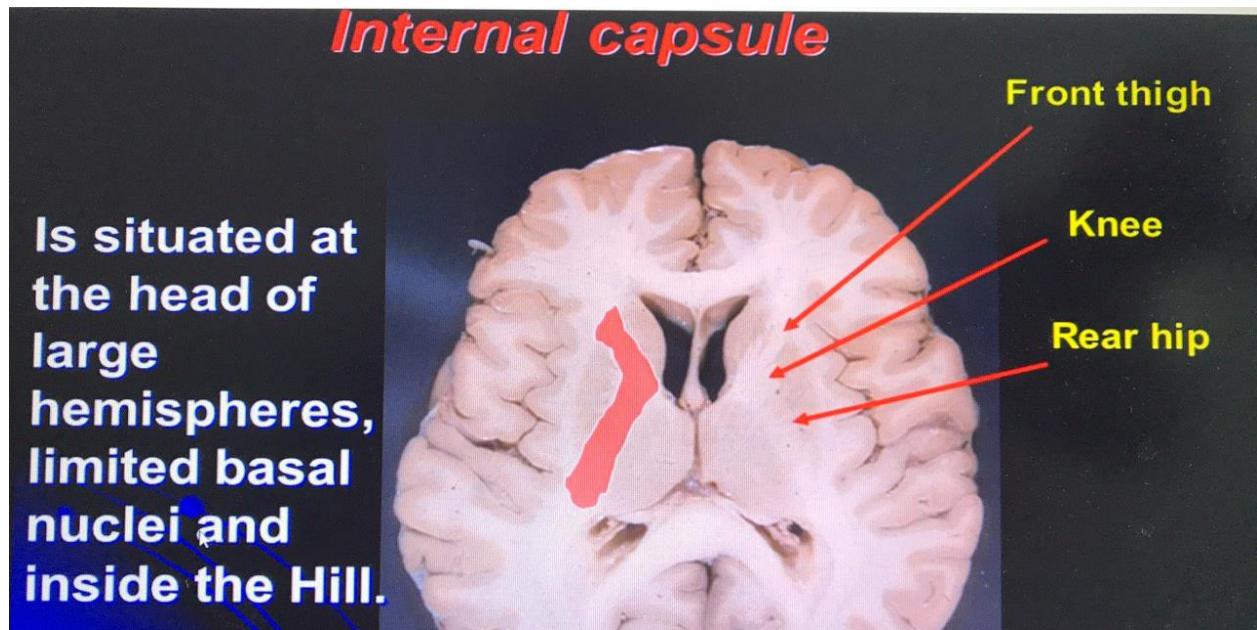
1. <http://etest.bsmu.by/mod/page/view.php?id=151387> (slide

no 12 to 32)

2. <https://www.slideshare.net/ASNasrullah/extrapyramidal-tract>

18. Internal capsule lesion syndromes.

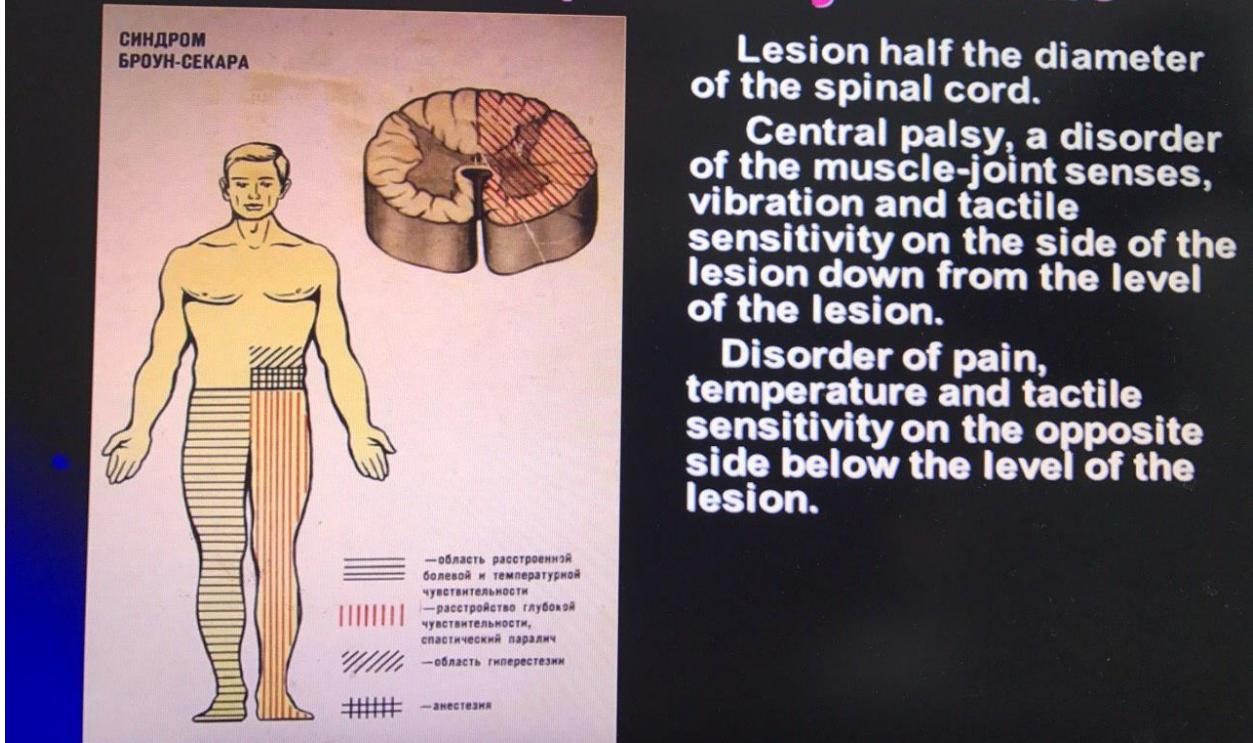
For anatomy part → <https://www.slideshare.net/ananthatiger/internal-capsule>



Syndrome of internal capsule



Brown-Séquard Syndrome



Lesion half the diameter of the spinal cord.

Central palsy, a disorder of the muscle-joint senses, vibration and tactile sensitivity on the side of the lesion down from the level of the lesion.

Disorder of pain, temperature and tactile sensitivity on the opposite side below the level of the lesion.

More information about internal capsular stroke

<https://stanfordmedicine25.stanford.edu/the25/ics.html>

19. Thalamic syndrome.

Thalamic Syndromes

Function. The thalamus is the *synaptic relay station for many somatosensory and special sensory pathways*; it transmits afferent impulses from peripheral extero- and proprioceptors, as well as from the higher sensory organs (eye, ear), to higher centers. In the thalamus, impulses pertaining to the body's various senses are *integrated, affectively colored, and then passed on to the cortex* (conscious perception appears to be possible only if the impulses reach the cortex). The thalamus also receives neural input from the extrapyramidal motor system and participates in the *regulation of attention and drive* as a component of the ascending reticular activating system (see below). Finally, certain components of the thalamus play a role in *memory*.

Deficits. Because the functions of the thalamus are as we have just described, lesions affecting it can produce **the following deficits:**

— **Somatosensory deficits:** these mainly consist of impaired proprioception on the side opposite the lesion. There may also be painful, burning sensations

that either arise spontaneously (*dysesthesia*) or are induced by, and outlast, a tactile stimulus delivered to the skin (*hyperpathia*).

— **Deficits of movement and coordination:** there may be contralateral hemiparesis (which is usually transient) or hemiataxia.

— **Contralateral hemianopsia** may be present.

Abnormal posture, particularly of the hands, may be present. In the “*thalamic hand*,” the metacarpophalangeal joints are flexed, while the interphalangeal

joints are hyperextended

for more information

1. <https://www.dovemed.com/diseases-conditions/thalamic-syndrome/>

20. Hypothalamic syndrome.

1. <https://www.slideshare.net/100002840600351/hypothalamus-71525756>
2. <https://www.slideshare.net/ATMHasibulHasan/disorder-of-hypothalamus>

21. Disorders of limbic-reticular system.

Anatomy and definition

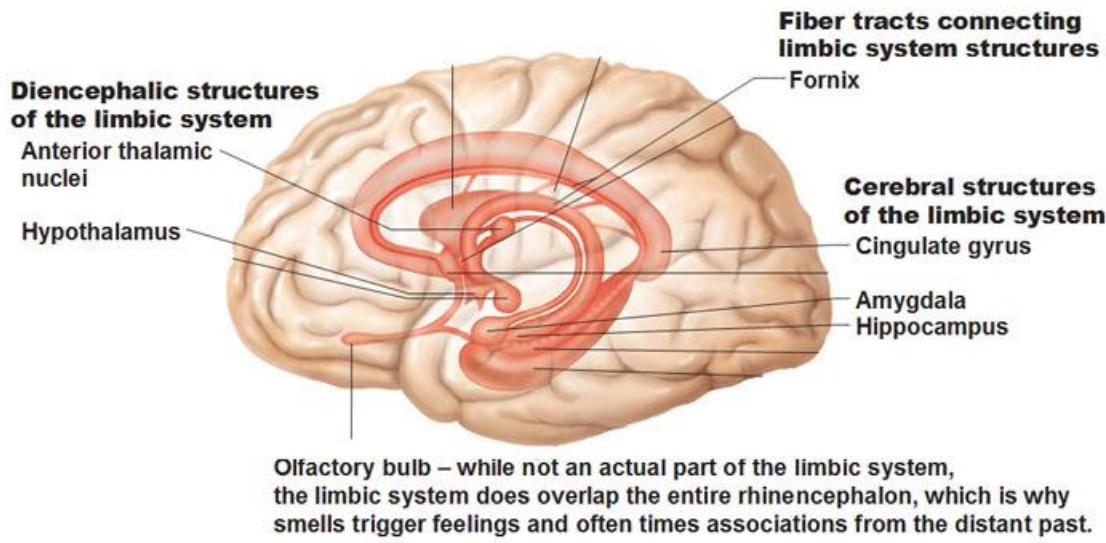
The Limbic System and the Reticular Formation

The limbic system and reticular formation are networks of neurons that function together even though they are widely separated.

The **limbic system** is the “emotional brain” made of deep gray matter structures linked together by the **fornix**. The fiber tracts have the appearance of oval fibers and looks like the corpus callosum but it’s not the same. The limbic system also includes certain structures of the diencephalon: the anterior thalamus and hypothalamus.

The hypothalamus in the middle of all this stuff has a direct between fearful stuff, memories and the autonomic nervous system. It’s a very strong anatomical connection.

The Limbic System (the basics)



Cingulate gyrus is involved with shifting thoughts, expressing emotions through gestures and resolving frustration.

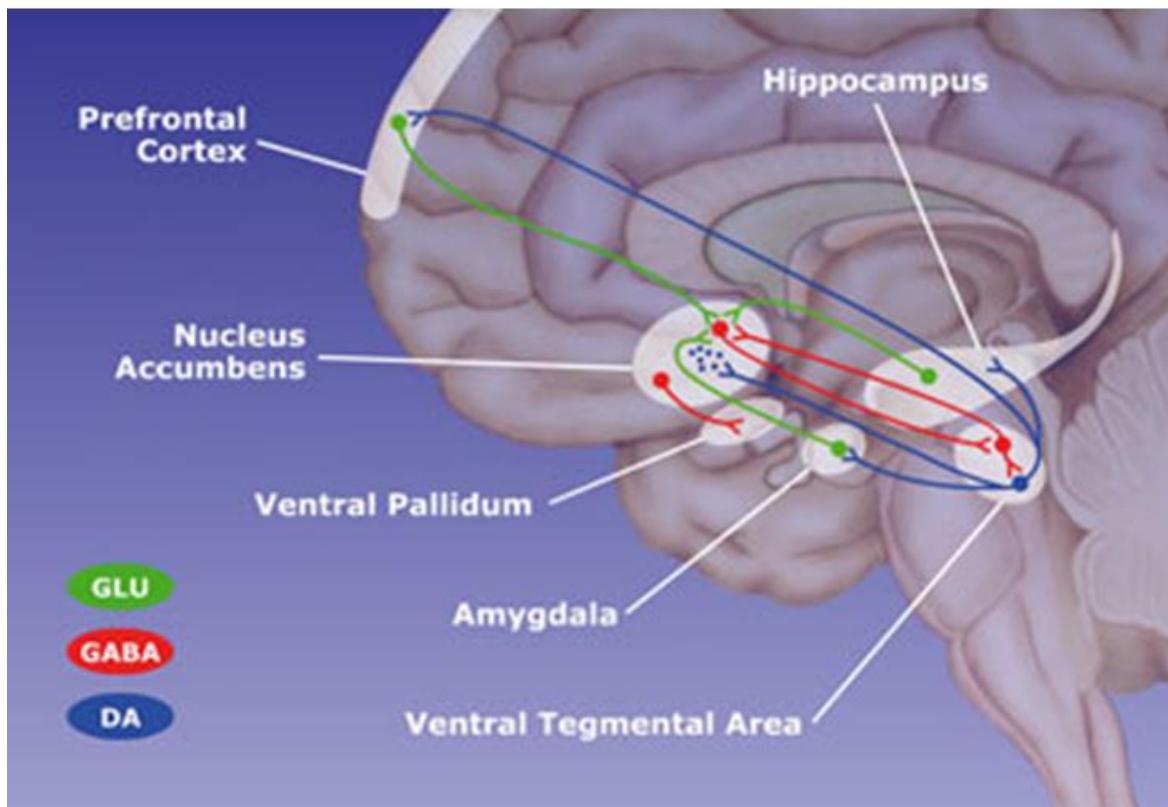
The **amygdala** (fear and its sympathetic response) and **hippocampus** (form and retrieve memories) reside very close together.

The Reward Circuit

This is called the reward circuit because when studies were done with rats and were provided with pleasure, they would actually neglect their babies to the point of death. We are hardwired for fear and pleasure.

The reward circuit includes the prefrontal cortex which includes judgments, habits, and behavior.

The Reward Circuit



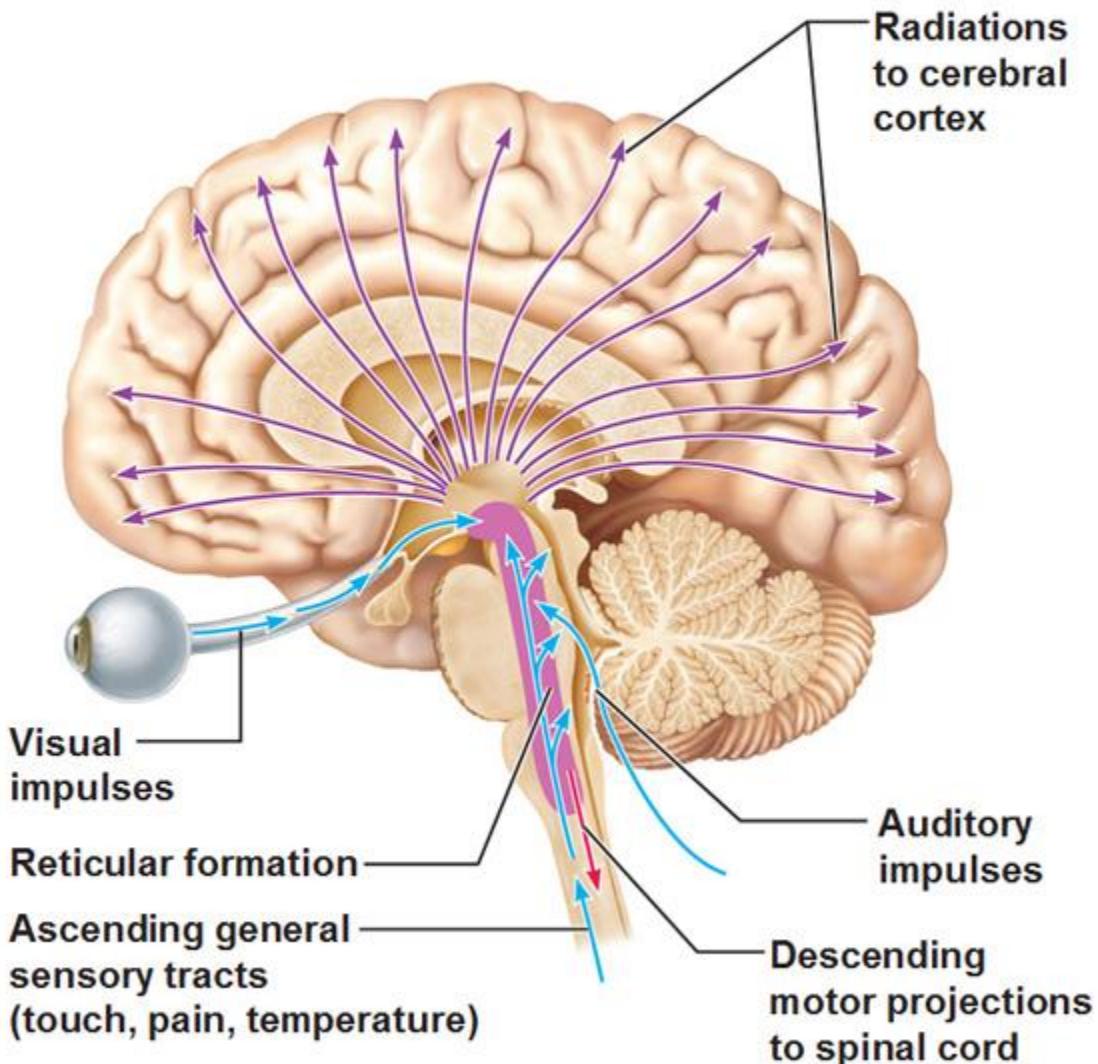
Nucleus accumbens (physically deep in the frontal lobe) and Ventral tegmental area (physically part of the midbrain) both release and mediate the neurotransmitter, dopamine, which specifically produces and mediates sensations of pleasure and relaxation.

Because the limbic system output passes through the hypothalamus and reticular formation, it in turn controls visceral responses. Therefore severe emotional stress can easily lead to visceral disorders.

The Reticular Formation

Runs through the central core of the brainstem with connections throughout cerebral hemispheres. It is made up of ascending and descending fibers. It plays a big role in filtering incoming stimuli to discriminate irrelevant background stimuli. The **Reticular Activating System (RAS)** is made up of the reticular formation and its widespread connections. It maintains consciousness and alertness and functions during sleep and arousal from sleep.

The Reticular Formation



Disorder:

1. <https://www.slideshare.net/ANANT1984/limbic-system-15625028> (slides 41 to 53)

22. Syndrome of hypothalamic-pituitary region lesions.

1. <https://slideplayer.com/slide/12904856/>

23. Syndrome of vegetative dystonia (somatoform disorders).

1. <https://www.slideshare.net/arunmadanan/somatoform-disorders-50168569>

24. Bladder and bowel dysfunctions of neurological origin.

Bladder, Bowel, and Sexual Function Anatomy.

The neural elements controlling bladder, bowel, and sexual function are the following:

— **Sympathetic fibers from spinal cord segments T12–L2 and parasympathetic fibers from spinal cord segments S2–S4** innervate the smooth muscle of the urinary bladder, rectum, and internal genitalia, **including the corpora cavernosa**. The sympathetic fibers travel to their target organs after a synaptic relay in the superior hypogastric plexus, while the parasympathetic fibers do so after a synaptic relay in the inferior hypogastric plexus. There are ganglion cells and synapses not just in the plexuses, but also within the walls of the target organs. Visceral sensory (afferent) fibers return to the spinal cord from the urinary bladder, genitalia, and rectum.

— The spinal center for micturition and defecation receives **supranuclear input from multiple higher cortical areas** (paracentral lobule – voluntary initiation of micturition and defecation) through a number of different pathways in the spinal cord, and it also conveys afferent information back upward to the brain (– conscious perception of bladder filling and of noxious and thermal stimuli). These mechanisms are

the basis of the voluntary control of micturition and defecation.

— The striated skeletal muscle of the pelvic floor and of the external sphincters of the bladder and rectum,

which are under voluntary control, is innervated by the **pudendal n.**, whose fibers are derived from spinal cord segments **S2–S4**. This nerve also conveys afferent impulses arising in the urethra, prostate gland, anal canal, and external genitalia.

Disturbances of bladder, bowel, and sexual function.

The clinical manifestations depend on the site of the lesion (peripheral/central, unilateral/bilateral):

— Spinal cord transection above the sacral level cuts off the bladder and bowel from the supraspinally derived (cortical) impulses subserving the voluntary control of micturition and defecation, but all of the afferent and efferent nerve pathways of the bladder remain intact, including the spinal reflex arc for bladder emptying. The result is a **spastic (automatic) neurogenic bladder**, which empties itself reflexively whenever it is filled to a certain volume .

Penile erection remains possible, though there may be retrograde ejaculation into the bladder.

Lesions of the conus medullaris, cauda equina, sacral plexus, and pelvic plexus.

Lesions of these structures

inactivate the sacral centers for micturition and defecation.

The result is **atony of the bladder and bowel musculature**, leading to severe impairment of emptying.

Bladder filling can no longer be perceived, either consciously or unconsciously. Tone is preserved in the sympathetically

elevated vesical sphincter; the bladder, therefore, continues to fill until the passive intravesical pressure overcomes the closing force of the sphincter. The continually overfilled bladder lets out small amounts of urine at short intervals (**overflow incontinence**). Defecation, meanwhile, occurs passively and in uncontrolled fashion through a *patulous anal sphincter*. In the male, lesions of these structures cause **erectile impotence**. Psychosexually mediated arousal remains possible in rare cases because of the preserved sympathetic efferent innervation through the hypogastric plexus. Thus, a small number of affected men are still able to have an emission of semen, but without ejaculation, and without rhythmic contraction of the pelvic floor muscles.

Lesions of the pudendal n. An isolated lesion of the pudendal n., which contains parasympathetic fibers from segments S2–S4, causes **erectile dysfunction**: the sacral erection center can no longer be activated because its somatosensory afferent input has been interrupted. Moreover, because the somatic efferent impulses to the bulbocavernosus and ischiocavernosus mm. no longer reach their targets, the maximal tumescence of the corpora cavernosa mediated by these muscles also fails to occur.

Impairment of the sympathetic innervation of the pelvic organs can be caused, for example, by tumor infiltration

or by surgical procedures. Bilateral lesions of the sympathetic chain and lesions of the superior hypogastric plexus abolish seminal emission into the proximal urethra; if ejaculation does occur, then the semen goes into the bladder, in retrograde fashion. As long as the

parasympathetic innervation of the genital organs by the pelvic plexus and their somatic sensory and motor innervation by the pudendal n. remain intact, the affected men are still able to have erections, and affected persons of both sexes can still experience pelvic floor contractions and orgasm. This constellation of symptoms (preserved ability to experience orgasm, in the absence of seminal emission) is seen in about half of all men who have undergone bilateral sympathectomy. It does not occur after unilateral lumbar sympathectomy.

From another source

Bladder and Bowel Dysfunction

Bladder dysfunction and bowel dysfunction refer to problems with urinating and passing stools. These may lead to the unwanted passage of urine or stool, called urinary or fecal incontinence.

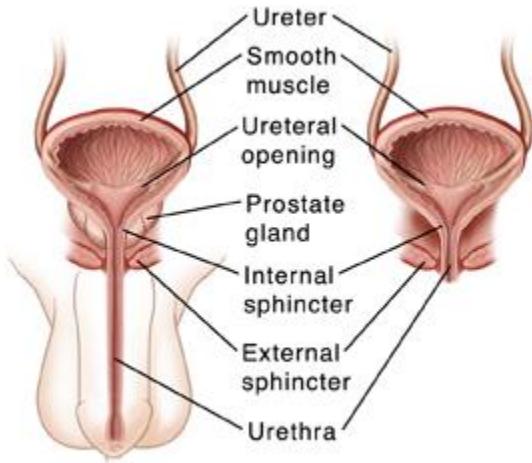
If you have these types of bladder and bowel problems, you may feel embarrassed at the thought of bringing them up with your doctor or other health care provider. The conditions can be physically and emotionally difficult to deal with, but you shouldn't feel uncomfortable about talking to your health care provider. Health care providers are used to dealing with these issues and can help you manage the problem.

Causes of bladder and bowel dysfunction

For the bladder and bowel to function correctly, certain nerves in your body need to control the right muscles, telling them when to contract and when to release in order to allow urine and feces to be eliminated when you want them to.

This happens when the nerves in the spinal cord send messages from the brain to the bladder and sphincter muscles to control the flow of urine. The muscles within the rectum and anus help control your bowels, and sphincter muscles control or release stool.

Urinary problems

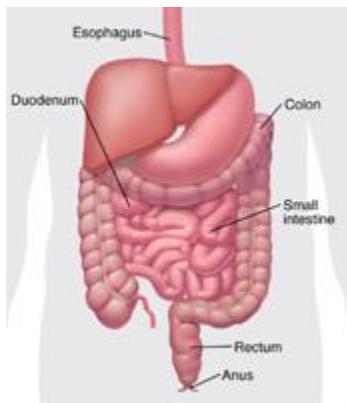


A number of conditions may affect the nerves and muscles that control the bladder and bowel, resulting in dysfunction and possible incontinence.

For the bladder, these conditions include:

- **Overactive bladder.** As the name suggests, you may have to go much more frequently than you would like. This can include an urgent need to urinate or having to urinate up to eight times or more a day and twice at night.
- **Difficulty controlling sphincter muscles.** If the nerves to these muscles have been damaged, they may not cooperate when you want to tighten or release them to pass urine.
- **Holding urine in too long (urine retention).** Sometimes nerve damage means that the bladder muscles don't get the chemical message that it's time for you to go. If the urine stays in the bladder too long, pressure may build up and lead to infection or damage of the bladder or kidneys.

Bowel problems



Fecal incontinence means you may not make it to the bathroom when you have a bowel movement, or you may "leak" a little when you pass gas.

Conditions that raise the risk for fecal incontinence include:

- Diarrhea
- Constipation
- Damage to the nervous system from disease or injury
- Poor health
- Vaginal childbirth
- Rectal prolapse (when the rectum protrudes into the anus)
- Rectocele (when the rectum pushes into the vagina)

Other conditions

These are other health issues that may contribute to bladder and/or bowel dysfunction:

- Medication side effects
 - Stress
 - Multiple sclerosis
 - Stroke
 - Alzheimer's disease
 - Diabetes
 - Infections, including spinal cord or brain infections
 - Hemorrhoids
 - Problems with the pelvic floor
 - Digestive problems, such as constipation or diarrhea
 - Abnormalities that affect the urinary or digestive tract
 - Problems affecting the nerves that control the urinary or digestive tract
- You could also have metal poisoning, congenital nerve-related problems, or injury or damage to the rectum caused by surgery or by conditions, such as Crohn's disease or ulcerative colitis.

Managing bladder and bowel dysfunction

Depending on the nature of your problem and your symptoms, your health care provider will work with you to create a plan of action. Here are some common treatments:

- **Changing your diet.** Gradually increasing your fiber intake can help manage the diarrhea and constipation that can lead to fecal incontinence. Drinking plenty of fluids can also ease

constipation, and restricting fluids at times can help manage overactive bladder or urinary incontinence.

- **Exercises.** Kegel exercises can strengthen the sphincter muscles and pelvic floor. This can provide better control and ease bladder and bowel dysfunction. Ask your doctor whether they might help in your case and, if so, how to do them.
- **Medications.** Some medications, including fiber supplements, can help control bowel dysfunction, and antidiarrheal medications can help manage diarrhea. Prescription medications are also available to help bladder muscles relax to promote better bladder control.
- **Training.** Programs that "train" the bowels and bladder can give you better control and manage dysfunction. This includes setting a regular schedule for using the toilet and attempting to urinate or have bowel movements at the same time each day.
- **Electrical stimulation.** This therapy can stimulate damaged nerves and promote better muscle control and control over urine and feces.
- **Surgery.** In rare cases, you may need surgery to repair damage to the muscles or nerves that are causing bladder or bowel dysfunction.

You don't have to suffer in silence. Finding the right therapy and management techniques can help you overcome bladder or bowel problems and avoid embarrassing incidents.

25. Cerebrospinal fluid abnormalities.

1.<https://slideplayer.com/slide/13206838/>+ a slide below

Test	Appearance	Pressure	WBC/ μ L	Protein mg/dL	Glucose mg/dL	Chloride
Normal CSF	Clear	90 – 180 mm	0-8 lymph.	15-45	50-80	115-130 mEq/L
Acute bacterial meningitis	Turbid	Increased	1000 -10000	100 – 500	< 40	Decreased
Viral meningitis	Clear	Normal to moderate increase	5-300, rarely >1000	Normal to mild increased	Normal	Normal
Tubercular meningitis	Slightly opaque cobweb formation	Increased/decreased, spinal block	100-600 mixed or lymph.	50-300 due to spinal block	Decreased	Decreased
Fungal meningitis	Clear	Increased	40-400 mixed	50-300	Decreased	Decreased
Acute syphilitic	Clear	Increased	About 500 lymph	Increased but <100	Normal	normal

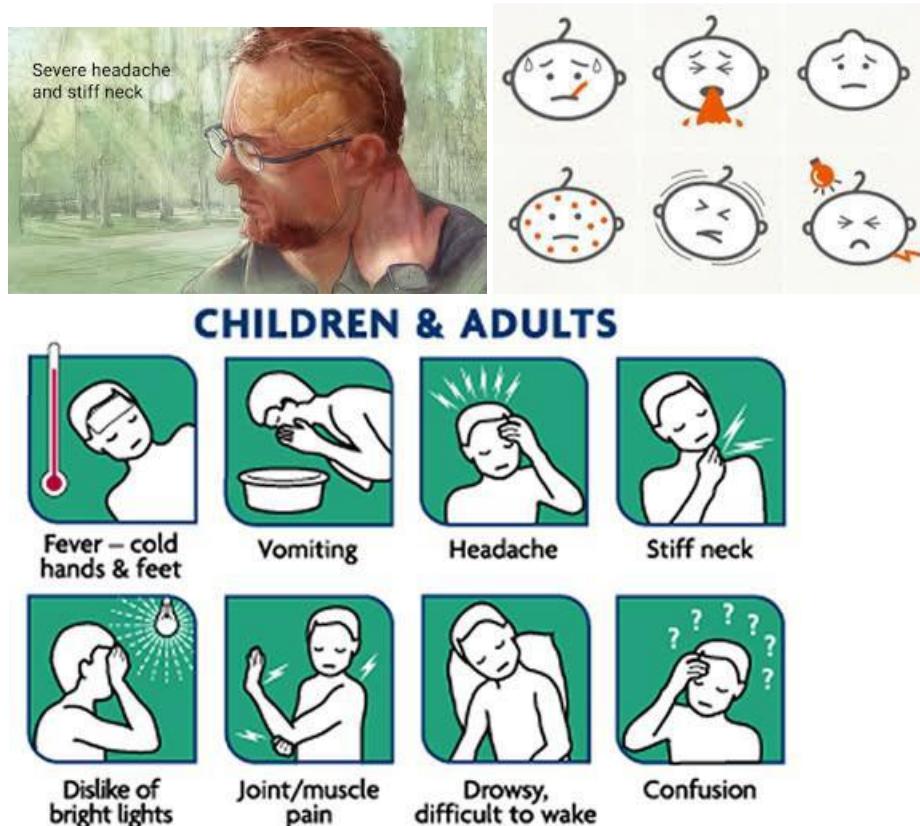
26. Syndrome of intracranial hypertension.

1. <https://slideplayer.com/slide/10327664/>
2. https://www.slideshare.net/medpk/headache-lecture-for-student?next_slideshow=1
3. <https://slideplayer.com/slide/5762836/> more information
4. Intracranial hypertension syndrome is characterized by an elevated intracranial pressure, papilledema, and headache with occasional abducens nerve paresis, absence of a space-occupying lesion or ventricular enlargement, and normal cerebrospinal fluid chemical and hematological constituents

27. Meningeal syndrome.

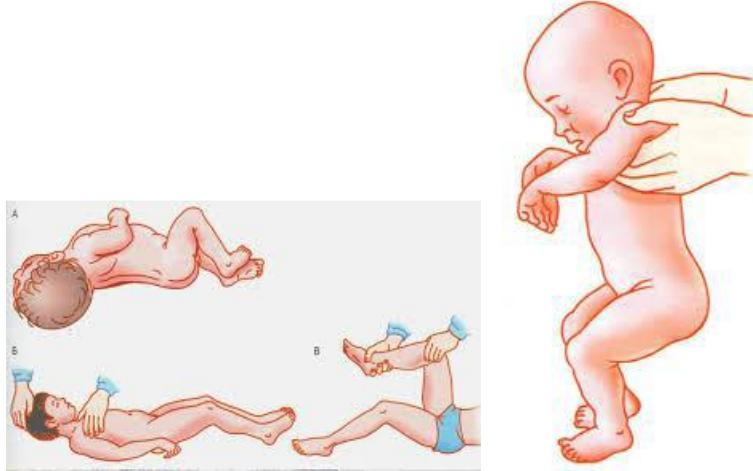
Meningeal syndrome

- Intracranial hypertension signs (headache, nausea, vomiting, consciousness disorders, seizures)
- Positive meningeal signs
- + Fever (if meningeal syndrome caused by infectious meningitis)



Major meningeal signs

- Meningeal pose
- Neck stiffness
- Kernig's sign
- Upper Brudzinski's symptom
- Middle Brudzinski's symptom
- Lower Brudzinski's symptom
- Lesage's sign



Brudzinski's Sign

Brudzinski's Sign

When the patient's neck is flexed (after ruling out cervical trauma or injury), flexion of the knees and hips is produced; when the lower extremity of one side is passively flexed, a similar movement is seen in the opposite extremity

Minor meningeal signs

- Zygomatic symptom of Bekhterev—tapping with finger or neurological hammer on malar arch increased headache and caused painful facial expression
- Mendel's symptom –tragus pressing caused or increased headache

NB!: symptoms of meningeal irritation may be absent in infants, elderly patients and patients in coma



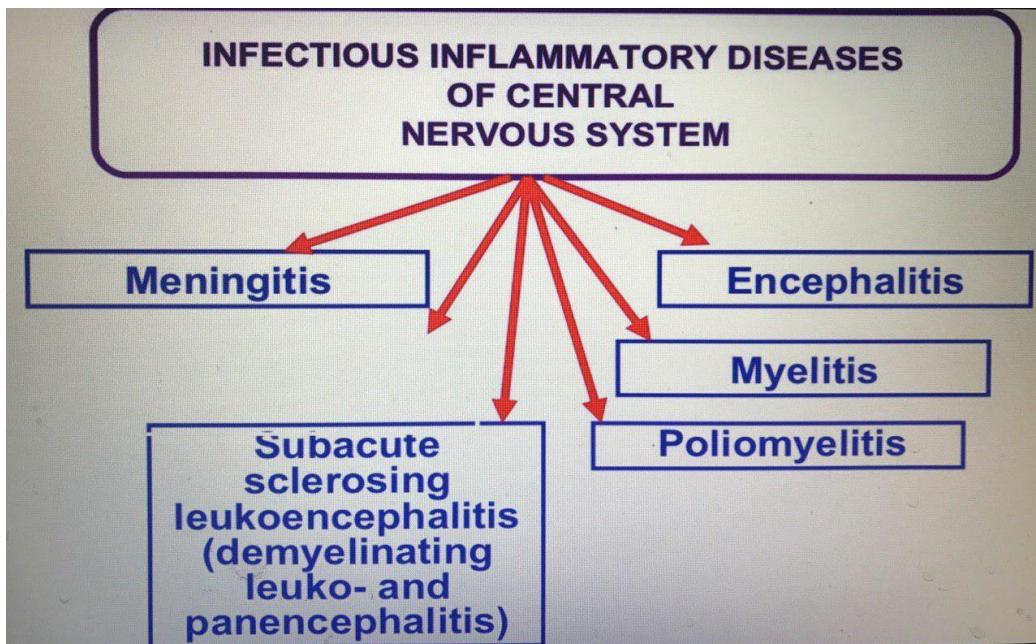
Investigations

Blood cultures -50-75 % positive

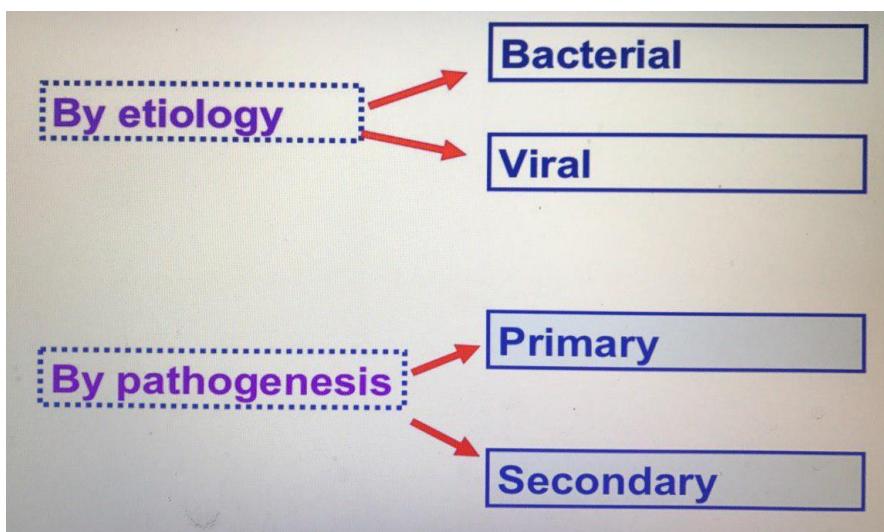
CT scan of the brain -especially if has a risk factor for mass lesion

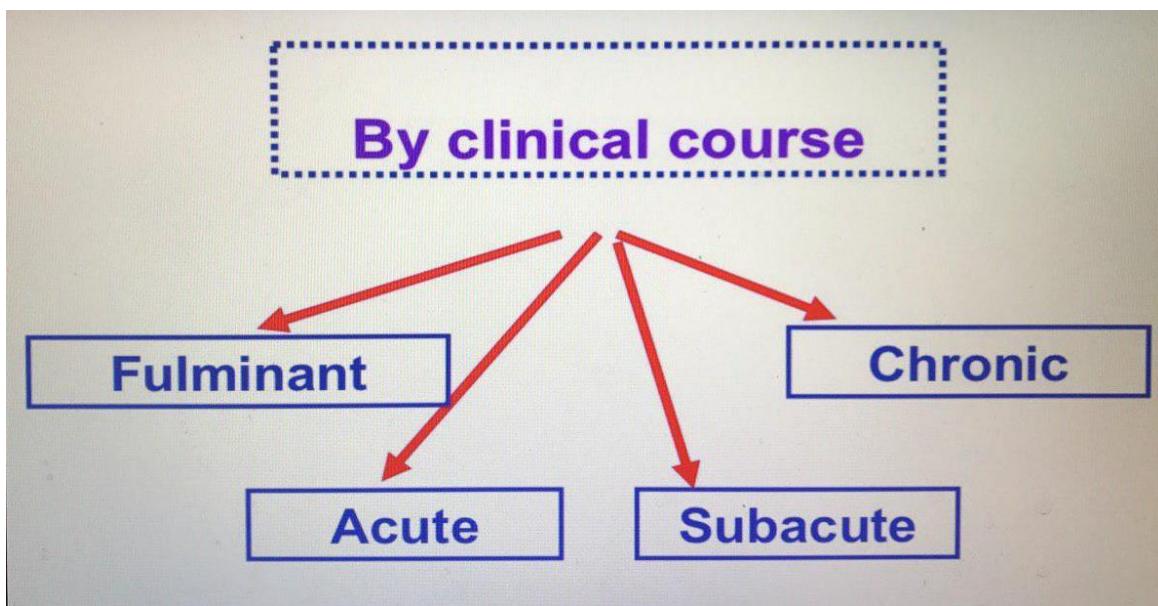
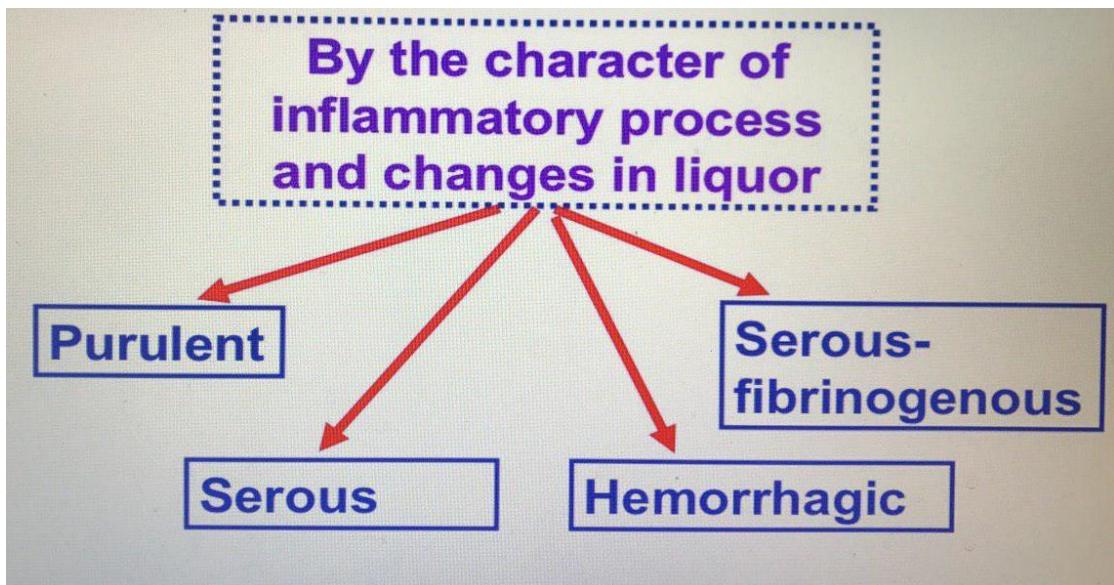
- Immunocompromised state (HIV, transplant, chemo therapy)
- History of CNS disease (mass lesion, stroke, focal infection)
- New onset seizures
- Papilledema
- Abnormal level of consciousness
- Focal neurologic deficit

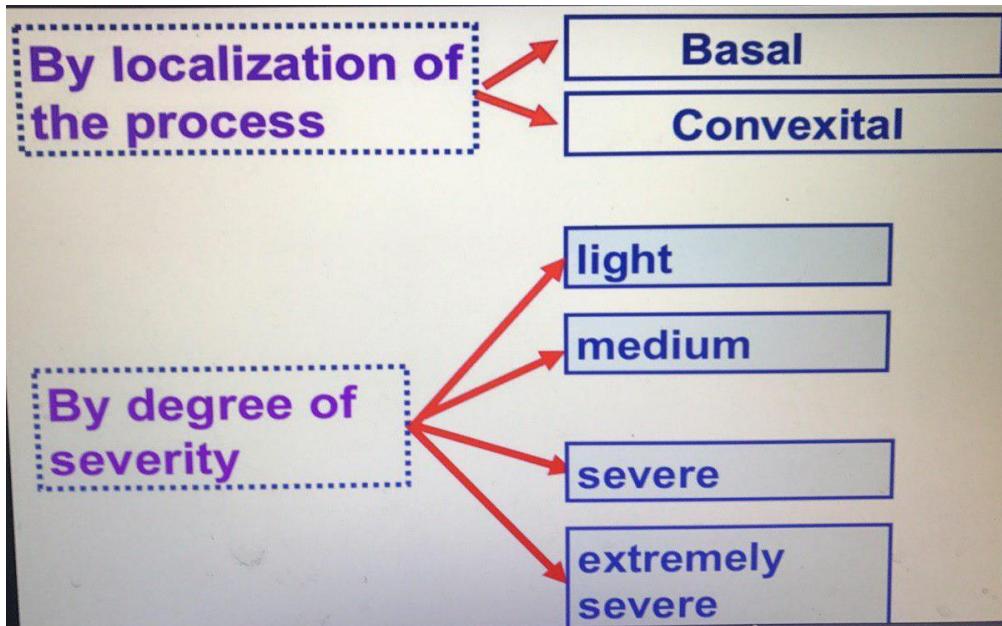
From another source:



CLASSIFICATION OF MENINGITIS







Clinical signs of meningitis

-
- Syndrome of infectious disease**
- fever
 - high body temperature
 - leucocytosis in blood with shift of the formula to the left,
 - erythrocyte sedimentation rate (EST)



Meningeal syndrome

Meningeal symptoms

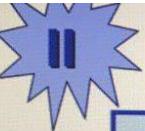
1. General hyperesthesia and hyperesthesia of organs of senses

2. Reactive pain phenomena:

- Bechterew's zygomatic symptom
- feeling of pain when you press on eyeballs, points of outlet of branches of trigeminal, occipital nerves

3. Muscular tonic tensions:

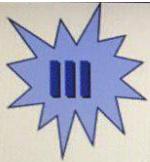
- rigidity of occipital muscles, long muscles of the back
- Kernig's symptoms, Brudzinski's upper, media, lower symptoms



Meningeal syndrome

General brain symptoms

- headache
- vomiting
- spasms
- psychomotor excitement
- impairment of consciousness



Syndrome of inflammatory changes in liquor

Purulent meningitis

Serous meningitis

Neutrophilic pleocytosis (thousands of cells per 1 mm³)

Lymphocytic pleocytosis (tens or hundreds of cells per 1 mm³)

Detection of pathogenic factor

Pathogenesis of meningitis

Ways of infection of membrane

Open craniocerebral trauma, which is combined with liquoria

Perineural or lymphogenous spread of pathogens in case of presence of purulent infection (sinusitis, otitis etc)

Hematogenous spread from primary sources of infection

Pathogenesis

Inflammation and edema of brain membranes (and adjacent brain tissue)

↓
Discirculation in brain and membranes vessels

↓
Hypersecretion of liquor and delay of its resorption

↓
High intracranial pressure and hydrocephalus

↓
Damage of membranes and roots of cranial and spinal nerves

Diagnostics

In the shortest term possible it's necessary to diagnose meningococcal infection only on the basis of clinical signs: acute beginning, fever, hemorrhagic rash.

For the patient's life hyperdiagnostics is much better than not timely diagnosis

- **Liquor.** Moderate pleocytosis (1-5 thousand or 10-12 thousand cells per 1 mm³) with cellular-proteinous dissociation, minor decrease of glucose level. The colour of liquor is like water with milk. When pleocytosis is more than 5-6 thousand per 1 mm³, liquor gets a yellowish shade.
- Express-diagnostics – bakterioscopy of thick drop of blood, blood smears and liquor. Colouring by gram already after 30 minutes allows to find out gram-negative diplococci.
- Inflammatory changes in peripheral blood.
- Clinical signs of meningitis

Treatment

- **meningococcal meningitis:** **cephalosporins of third generation, penicillin 300 000 units per 1 kilo of mass (18-24 mln units per 24 hours) ≈ 8 days.** To cancel when cytosis is less than 100 cells, when lymphocytes are less than 75 %
 - Ampicillin (200-400 mg/1 kilo of mass)
 - sulfonamides
 - dehydration
 - antipyretics (pirabutol, reoperin, ibuprofen)
 - seduxen (in case of psychomotor excitement)

28. Lumbar puncture and CSF examination. Diagnostic value. Indications, contraindications, complications.

Lumbar puncture

Opening pressure

- 350 mm H₂O (normal up to 200 mm H₂O)

CSF analysis

- Gram stain and culture
- Protein above 250 mg/dL (N-less than 50 mg/dL)
- Glucose below 45 mg/dL (N-greater than 45 mg/dL)
- White cell count above 1000/microliter (N-no cells)

Traumatic tap

- CSF clears between 1 to 3 tubes
- Blood pigments-present hemorrhage >12 hours, absent hemorrhage or traumatic tap <12 hours

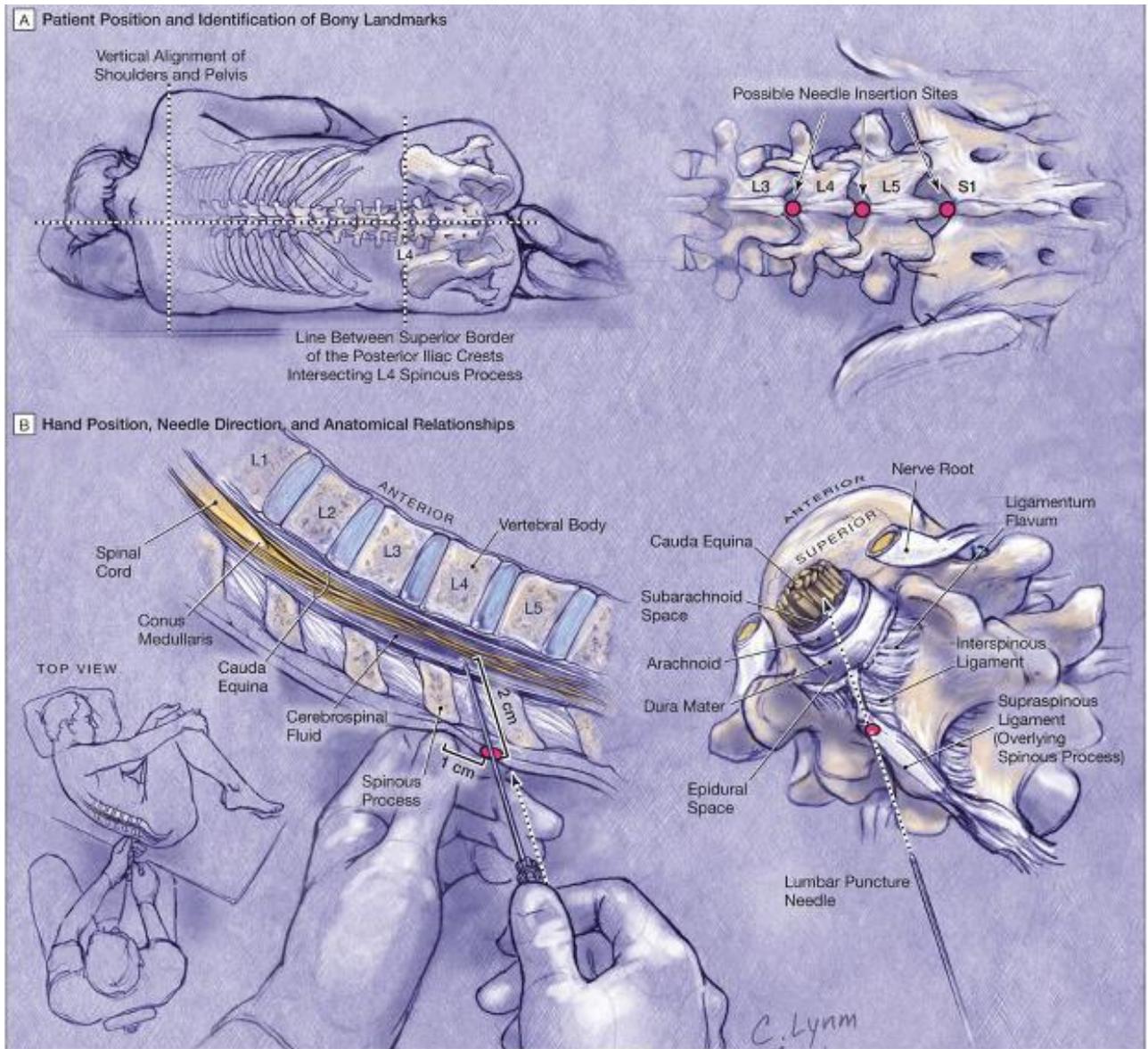
CSF cortisol level greater than 46.1 nmol/L

Latex agglutination test

- Detects antigens to common bacteria

Lumbar puncture

medical procedure in which a needle is inserted into the spinal canal to collect cerebrospinal fluid (CSF) for diagnostic testing or inject medication into CSF.



Lumbar puncture indications

- Suspicion of meningitis or other CNS infections
- Suspicion of subarachnoid hemorrhage (SAH)
- Suspicion of CNS disorders such as Guillain-Barre syndrome, carcinomatous meningitis or idiopathic intracranial hypertension .
- Intrathecal injection of medication

Lumbar puncture contraindications

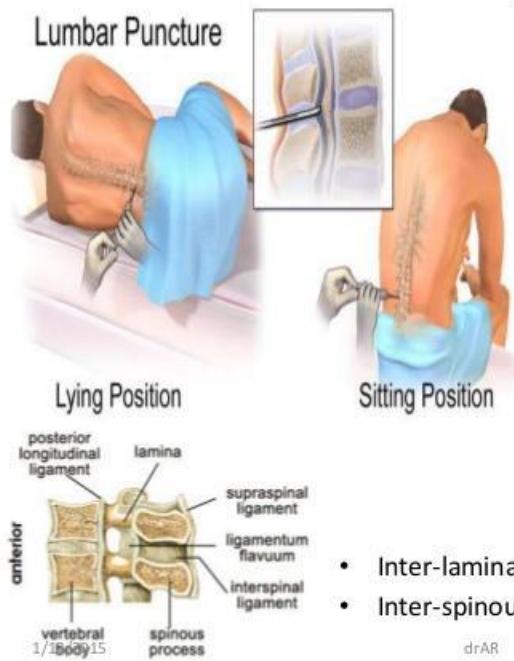
Absolute

- Raised intracranial pressure with brain herniation threat
- Infection of skin or soft tissue on the lumbar region or suspected spinal epidural abscess

Relative

- Severe condition as abnormal respiratory patterns, shock, bradycardia, decrease level of consciousness .
- Thrombocytopenia or other bleeding diathesis (including ongoing anticoagulant therapy)

Patient Preparation



- Local anesthesia is employed for lumbar puncture
- The patient is placed in the lateral recumbent position with the hips, knees, and chin flexed toward the chest so as to open the inter-laminar spaces or interspinous distance
- A pillow may be used to support the head
 - Inter-laminar space
 - Inter-spinous diameter

15

CSF test results

Test	Appearance	Pressure	WBC/ μ L	Protein mg/dL	Glucose mg/dL	Chloride
Normal CSF	Clear	90 – 180 mm	0-8 lymph.	15-45	50-80	115-130 mEq/L
Acute bacterial meningitis	Turbid	Increased	1000 -10000	100 – 500	< 40	Decreased
Viral meningitis	Clear	Normal to moderate increase	5-300, rarely >1000	Normal to mild increased	Normal	Normal
Tubercular meningitis	Slightly opaque cobweb formation	Increased/ decreased, spinal block	100-600 mixed or lymph.	50-300 due to spinal block	Decreased	Decreased
Fungal meningitis	Clear	Increased	40-400 mixed	50-300	Decreased	Decreased
Acute syphilitic	Clear	Increased	About 500 lymph	Increased but <100	Normal	normal

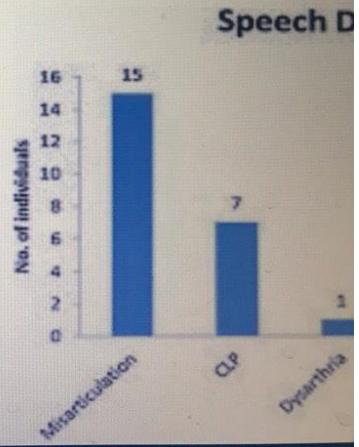
Lumbar puncture complications

- Post-lumbar puncture headache -starts several hours up to two days after the procedure and may be accompanied by nausea, vomiting and dizziness. The headaches are usually present when sitting or standing and resolve after lying down. Post-lumbar puncture headaches can last from a few hours to a week or more.
- Back discomfort or pain
- Bleeding
- Brain herniation

29. Speech disorders.

What is speech disorder ?

Speech disorders or speech impediments are a type of communication disorder where 'normal' speech is disrupted. This can mean stammer, stutter, lisps, etc. Someone who is unable to speak due to a speech disorder is considered mute. Such things may lead to low self-esteem but as development of depression.

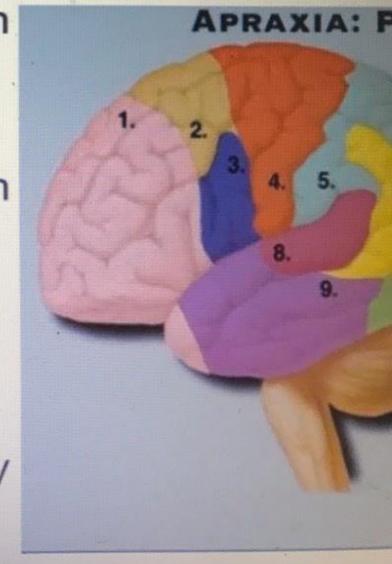


Some Common Speech Disorders

- 1) **Apraxia of Speech (AOS)**
- 2) **Stuttering – Stammering**
- 3) **Dysarthria**
- 4) **Lisping**
- 5) **Muteness – Selective Mutism**
- 6) **Aphasia**

Apraxia Of Speech (AOS)

Apraxia of Speech (AOS) happens when the neural pathway between the brain and a person's speech function (speech muscles) is lost or obscured. The person knows what they want to say – they can even write what they want to say on paper – however the brain is unable to send the correct messages so that speech muscles can articulate what they want to say, even though the speech muscles themselves work just fine.



Stuttering - Stammering

Stuttering, also referred to as stammering, is so common that everyone knows what it sounds like and can easily recognize it. Everyone has probably had moments of stuttering at least once in their life. The National Institute on Deafness and Other Communication Disorders estimates that three million Americans stutter, and reports that of the up-to-10-percent of children who do stutter, three-quarters of them will outgrow it. It should not be confused with cluttering.

H....h....h.

Dysarthria

Download

Dysarthria is a symptom of nerve or muscle damage. It manifests itself as slurred speech, slowed speech, limited tongue, jaw, or lip movement, abnormal rhythm and pitch when speaking.

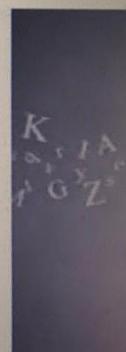
Speech-Language pathologists helps in understanding the speech of the affected person.

Characteristics of dysarthria subtypes	
Dysarthria subtype	Characteristics
Flaccid dysarthria	Speech may sound nasal or slurred
Spastic dysarthria	Speech may sound as if patient is squeezing out words from a pursed mouth
Ataxic dysarthria	Speech is uncoordinated; range, timing and direction may be inaccurate; rate is slow; may be explosive in quality
Hypokinetic dysarthria	Speech may sound monotonous, or slow-paced; rate may vary; rigidity may be present
Hyperkinetic dysarthria	Involuntary disruptions in sounds and/or movements

Lisp

A lay term, lisp can be recognized by anyone and is very common.

Speech language pathologists provide an extra level of expertise and can make sure that a lisp is not being confused with another type of disorder such as apraxia, aphasia, impaired development of expressive language, or a speech impediment caused by hearing loss.



Muteness - Selective Mutism

There are different kinds of mutism, and here we are talking about selective mutism. This used to be called elective mutism to emphasize its difference from disorders that caused mutism through damage to, or irregularities in, the speech process.

Selective mutism is when a person does not speak in some or most situations, however that person is physically capable of speaking. It most often occurs in children, and is commonly exemplified by a child speaking at home but not at school.

Selective Mutism is an Anxiety Disorder

- Children with Selective Mutism tend to speak in certain situations, such as in school despite being able to speak in other places, such as home.
- They will have difficulty speaking, laughing, reading aloud, singing in front of people outside of their "comfort zone".
- Parents, siblings and friends may make a habit of speaking for the child.
- These children often have symptoms of social phobia as well.

Aphasia

The National Institute on Neurological Disorders and Stroke estimates that one million Americans have some form of aphasia.

Aphasia is a communication disorder caused by damage to the brain's language capabilities. Aphasia differs from apraxia of speech and dysarthria in that it solely pertains to the brain's speech and language center.

Download

Aphasia

Treatment



Causes Of Speech Disorder

Download

Speech disorders affect the vocal cords, muscles, nerves, and other structures within the throat. Causes may include:

- a) Vocal cord damage
- b) Brain damage
- c) Muscle Weakness
- d) Respiratory Weakness
- e) Strokes
- f) Polyps or nodules on the vocal cords.

Symptoms of Speech Disorders

Download

Depending on the cause of the speech disorder, several symptom may be presented. Common symptoms experienced by people with speech disorders are :

- 1) Repeating sound, which is often seen in people who stutter.
- 2) Adding extra sound and words.
- 3) Taking frequent pauses when talking.
- 4) Visible frustration when trying to communicate.
- 5) Hoarseness, or speaking with a raspy or gravelly sounding voice.
- 6) Making jerking movements while talking, usually involving the head.

Treatment Of Speech Disorder

Download

Many of these types of disorders can be treated by [speech therapy](#), but others require medical attention by a doctor in phoniatrics. Other treatments include correction of organic conditions and psychotherapy.

A speech therapist will help you in muscle-strengthening exercises for the face as well as throat. You'll learn to control your breathing while speaking. Muscle-strengthening exercises along with controlled breathing help to improve the way words sound. This helps to learn the ways to practice smoother, and fluent speech. Muscle-strengthening exercises and controlled breathing help improve the way your words sound. You'll also learn ways to practice smoother, more fluent speech.

30. Higher mental functions disorders.

Lateralization of brain function

- Brain- two cerebral hemispheres, connected by corpus callosum
- 95% of R-handed & 80% of L-handed people have L hemisphere dominance for language
- Lateralization-
 - L hemisphere- exact calculation; language- grammar, vocabulary, literal
 - R hemisphere- approximate calculation; language- intonation/accents, prosody, pragmatic, contextual

Amnesia

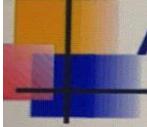
- Defect in memory
- Organic or functional
- Forms-
 - Anterograde-inability to memorize new things
 - Retrograde- inability to recall pre-existing memories
- Types-
 - Immediate- few seconds- in delirium
 - Recent- minutes-days- in dementia/delirium
 - Remote- months-years- usually intact, till late dementia
- Causes- delirium, post-traumatic, dementia

Types of amnesia

- Delirium- loss of all memory with loss of orientation to time, place, person
- Post-traumatic- transient or permanent, related to degree of injury
- Transient global amnesia- impaired recent memory, due to hippocampal dysfunction, complete recovery within a day
- Blackout- anterograde amnesia following binge drinking
- Korsakoff's syndrome- due to thiamine-B1 deficiency, severe memory loss with ataxia & confabulation
- Drug-induced- midazolam & propofol- medical use, recent memory loss of procedural events

Disorders of language

- **Language**- a system of signs-symbols or gestures- for encoding & decoding information
- Aphasia- defective processing of linguistic information- receptive and/or expressive
- **Speech**- a modality to convey language
- Dysarthria- defective articulation- pure motor, mainly consonants; due to neurological injury affecting V,VII,IX,X,XII cranial n.
- Dysphonia- impaired sound production- mainly vowels, due to defect in larynx/vocal cords



Aphasia

- **Causes-** stroke, trauma, tumor, degenerative
- **Manifestations-**
 - Inability to comprehend
 - Inability to speak spontaneously & fluently
 - Inability to form words or neologisms
 - Inability to read/write
 - Inability to name
 - Inability to repeat/persistent repetition



Testing for aphasia

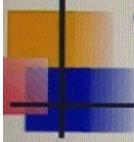
- **Spontaneous speech-** fluency, volume, initiation, pauses
- **Naming-** name objects
- **Repetition**
- **Comprehension-** point to objects named
- **Reading**
- **Writing**

Types of aphasia

- **Wernicke's- temporal lobe**
 - Defect in comprehension, fluent nonsensical speech
- **Broca's- frontal lobe**
 - Defect in expression- non-fluent, effortful, slow speech
 - Associated right face/arm hemiparesis
- **Conduction- arcuate fasciculus/auditory cortex**
 - Poor repetition & naming, good comprehension, fluent speech
- **Global- Wernicke's + Broca's**
- **Pure word deafness- impaired auditory comprehension**
- **Pure word blindness- impaired reading comprehension**

Apraxia- motor

Loss of ability to carry out learned purposeful movements,
despite having desire & physical ability to do so



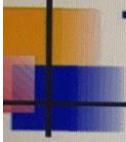
Types of apraxia

- Ideomotor- inability to carry out a motor command, limb or buccofacial
- Ideational- inability to create a plan for or an idea of a specific movement
- Constructional- inability to draw or construct simple configurations
- Speech- impaired ability to speak
- Gait- impaired coordination of leg movements



Agnosia- sensory

Loss of ability to recognize-
object/person/shape/sound/smell-
without any sensory or memory loss



Types of agnosia

- Alexia- inability to recognize text
- Amusia- inability to recognize/appreciate music
- Apperceptive- unable to distinguish visual shapes, can't copy shapes
- Integrative- can identify elements/parts, but not integrate them into whole
- Prosopagnosia- inability to recognize familiar faces
- Astereognosia- inability to recognize objects by touch, based on its texture, size or weight
- Visual- inability to recognize objects by seeing them



Delirium & Dementia

- Disorders of higher mental function
- Delirium-acute, Dementia-chronic

Delirium

- An acute confusional state, with fluctuating course
- A common disorder in hospitalised patients, specially elderly/in ICU
- Manifestation-
 - Attention deficits, abnormal behaviour, altered sensorium, incoherent speech
 - Cognitive defects- memory, orientation, planning
 - Changed sleep pattern, hallucination/delusion

Causes

- Majority outside brain
 - High-grade fever, infection in elderly
 - Hypoxia- ACS, CVA, pneumonia etc.
 - Hypoglycemia, acute renal/hepatic impairment
 - Electrolyte abnormality, specially Na, Ca
 - Alcohol/benzodiazepine withdrawl
 - Illicit drug abuse, poisoning, psychotropic drugs
 - Head injury
 - Brain disorders- stroke, bleed, tumor, raised ICP

Management

- Dx-
 - Clinical presentation
 - Ix- CBC, RBS, Cr, SGPT, Na, Ca, ABG, ECG, CxR
 - CT scan/MRI- rule in/out primary brain disease
- Rx-
 - Treat underlying cause
 - Supportive treatment- oxygenation, hydration, nutrition
 - Treat fever, pain, constipation
 - Optimize drugs

Dementia

- A chronic- >6 months- symptom complex
- Usually progressive & incurable
- Affects cognition- memory, attention, language, problem-solving
- Depression, anxiety, agitation are common
- Causes-
 - Fixed cognitive- one time insult- trauma, hypoxia, stroke
 - Slowly progressive- neurodegenerative- Alzheimer's, vascular, hypothyroidism, Wilson's, normal pressure hydrocephalus
 - Rapidly progressive- Prion diseases- Creutzfeldt-Jakob disease

Management

- Dx-
 - Cognitive testing- mini mental state examination- MMSE- orientation, registration, attention, calculation, recall, language
 - PET scan- for Alzheimer's disease
 - CT/MRI- to detect structural cause
 - CBC, Cr, SGPT, TSH- for a treatable cause
- Rx- mainly supportive
 - Drugs- anticholinesterase inhibitors- tacrine, donepezil, galantamine, rivastigmine or NMDA blocker- memantine
 - Antidepressants or anxiolytics- as required
- Px- progressive, incurable, life-span reduced

- 31. **Frontal lobe lesion syndromes.**
- 32. **Parietal lobe lesion syndromes.**
- 33. **Temporal lobe lesion syndromes.**
- 34. **Occipital lobe lesion syndromes.**



the answers of question 31 to 34 are on the site(2 lectures)

- 1. <http://etest.bsmu.by/mod/page/view.php?id=151544&forceview=1>
- 2. <http://etest.bsmu.by/mod/page/view.php?id=151545&forceview=1>

- 35. **Syndrome of cerebral pons lesions.**
- 36. **Syndromes of medulla oblongata lesions.**

- 1. <https://www.slideshare.net/kunalmahajan50/brainstem-stroke-syndromes-ppt> (**slides 24 to 43**)

for better understanding

- 1. <https://www.slideshare.net/docamruta24/pons-anatomy-and-syndromes>

{

37. Syndromes of brachial plexus lesions.
38. Syndromes of radial, ulnar and median nerves lesions.

1. <https://www.slideshare.net/zahoor111/brachial-plexus-injuries-12335691>
for better understanding
2. <https://slideplayer.com/slide/5742155/>

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39. Syndromes of lumbar and sacral plexus lesions.
40. Syndromes of feet nerves lesions.

Diseases of the Lumbosacral Plexus

It lies in a well-protected location in the **posterior wall of the pelvis**. Its cranial portion (the

lumbar plexus, L1–L4) gives off, as its main branches, the *ilioinguinal*, *iliohypogastric*, *femoral*, and *obturator nn.*

These nerves innervate most of the hip flexors and knee extensors. The caudal portion of the lumbosacral plexus (**the sacral plexus, L5–S3**) gives off the *superior* and *inferior gluteal nn.* for the gluteal muscles, as well as the *sciatic n.*, which supplies the knee flexors and all muscles of the lower leg and foot.

Typical deficits. The clinical manifestations of a lumbosacral plexus lesion depend on its location; in general, one finds a combination of the deficits seen in lesions of the individual **peripheral nerve trunks lying distal to the plexus lesion.**

Causes: Lumbosacral plexus palsy is usually due to a **local mass**, but it may also be due to prior *radiation therapy* or to an autoimmune disorder known as *chronic, progressive lumbosacral plexopathy*.

Diagnostic evaluation: Ancillary testing, primarily with CT or MRI, is generally needed to identify the etiology of a lumbosacral plexopathy. These imaging studies

can demonstrate the presence of a mass.

question number 39 for more information :

5. <https://www.sciencedirect.com/topics/neuroscience/lumbosacral-plexus>

there are 2 lectures on the site for question number 40 + answer below

1. <http://etest.bsmu.by/mod/page/view.php?id=151715&forceview=1>
2. <http://etest.bsmu.by/mod/page/view.php?id=151714&forceview=1>

Genitofemoral and Ilioinguinal Nn. (L1–L2)

Typical deficits: Lesions of these nerves cause local pain in the groin (*ilioinguinal nerve syndrome*), a sensory deficit in the corresponding zone(s) of cutaneous innervation, and sometimes, in men, loss of the cremaster reflex (because the afferent arm of the reflex loop is interrupted). The associated motor deficit only affects oblique muscles of the abdominal wall and is hardly noticeable.

Lateral Femoral Cutaneous N. (L2–L3)

Anatomy. This purely sensory nerve passes through the three layers of the abdominal wall and then penetrates the inguinal ligament, usually at a point three finger breadths medial to the anterior superior iliac spine, to emerge onto the anterior fascia of the thigh. It provides sensory innervation to a palm-sized area of skin on the *anterolateral surface of the thigh*

Typical deficits: The lateral femoral cutaneous n. is vulnerable to injury at the point where it penetrates the inguinal ligament. The resulting clinical disturbance is an entrapment neuropathy called **meralgia paresthetica**, characterized by *burning pain in the cutaneous distribution of the nerve*. The pain is better when the hip is flexed, e. g., when the patient raises the ipsilateral foot onto a lowstool; it is worse on hyperextension of the leg

(*reverse Lasègue sign*). The site where the nerve passes through the inguinal ligament is often tender to light pressure

. Most patients find the symptoms bearable and need only be reassured that the condition is benign. Surgery is only rarely necessary; the goal of the operation is to widen the aperture in the ligament through which the nerve passes, relieving compression. Causes. Meralgia paresthetica may be due to marked weight gain or pregnancy. It can also arise after prolonged, continuous extension of the hip joint (supine position). Some cases have no apparent cause.

Differential diagnosis: Meralgia paresthetica must be distinguished from an *L3 nerve root lesion*. L3 root lesions impair the quadriceps reflex; they also produce a more extensive sensory deficit, which, unlike that of meralgia paraesthetica, crosses over the midline of the thigh onto its anteromedial surface.

Femoral N. (L1–L4)

Anatomy. The femoral n. provides motor innervation to the *hip flexors* (iliacus and psoas major mm.) and the *knee extensors* (quadriceps femoris m.). It provides sensory innervation by way of *anterior cutaneous branches* to the anterior surface of the thigh, and, through its terminal branch, the *saphenous n.*, to the medial quadrant of the anterior surface of the lower leg.

Typical deficits: A lesion of the femoral n. impairs *hip flexion and knee extension*. The hip flexors are examined with the patient sitting up and the knee extensors are examined with the patient supine . In the standing patient, a *low-lying patella* is seen on the side

of the lesion. The *quadriceps reflex* (patellar tendon re-

flex) is absent. The patient cannot climb stairs with the affected leg and keeps it in a hyperextended position while walking. Sensation is diminished in the territory of the sensory terminal branches

Causes: Lesions of the femoral n. are commonly *traumatic* or *iatrogenic* (surgery). The nerve can also be involved by a pelvic *tumor* or, acutely, by a *hematoma in the psoas sheath*, e. g., in an anticoagulated patient.

Obturator N. (L3–L4)

Anatomy. The obturator n. supplies the *thigh adductors*. Its sensory innervation is to a small area of skin just above the medial aspect of the knee.

Typical deficits: A lesion of the obturator n. impairs *thigh adduction*.

The adductor reflex, elicited by a tap on the medial condyle of the femur, is diminished and there is a small area of hypesthesia on the medial aspect of the thigh, just above the knee. Sometimes, irritation of the obturator nerve trunk can produce pain in this area as the sole clinical manifestation. This is called the *Howship–Romberg phenomenon*.

Causes: Masses in the pelvis or the obturator foramen

are the usual causes; an obturator hernia is rarer.

Gluteal Nn. (L4–S2)

Anatomy. The gluteal nn. are purely motor. They innervate the hip abductors and extensors. The course of the two gluteal nn.

Lesions of the superior gluteal n. produce weakness of the hip abductors (gluteus medius and minimus mm. and tensor fasciae latae m.). This impairs the stability of the pelvis on the side of the stationary leg when the patient walks; the pelvis tilts to the side of the swinging leg (so-called *Trendelenburg gait*). In an incomplete superior

gluteal n. palsy, the patient barely manages to prevent tilting of the pelvis by inclining the trunk to the side of the stationary leg, thus displacing the body's center of gravity laterally .

Lesions of the inferior gluteal n. (L5–S2) produce weakness of the gluteus maximus m., impairing hip extension. This makes it difficult for the patient to climb stairs (for example). Atrophy of the gluteus maximus m. is usually difficult to see because of the overlying fatty tissue, but, when the gluteus maximus mm. on either side are simultaneously and actively contracted, the lack of muscle tone on the affected side is easily appreciated by palpation. The natal fold is lower on the affected side. Causes. The gluteal nn. are often injured by intramuscular injections with faulty technique.

Differential diagnosis. Trendelenburg gait can be observed in many diseases of the hip, e. g., in *congenital hip dislocation*. Weakness of the gluteus maximus m. can be

caused by an *S1 nerve root lesion* , while

weakness of the gluteus minimus and gluteus medius mm. can be caused by an *L5 nerve root lesion*. Bilateral weakness of the hip abductors is found, for example, in muscular dystrophy.

Sciatic N. (L4–S3)

Anatomy. The sciatic n. is the common trunk of the fibular (= peroneal or common peroneal) and tibial nn. It is the longest and thickest nerve in the human body.

The portions of the sciatic n. that are destined to become the fibular and tibial nn. are already clearly distinct from one another in the sciatic n. just distal to its exit from the pelvis, but they are usually ensheathed in a common epineurium nearly all the way down to the level of the popliteal fossa. The

sciatic nerve trunk, in its proximal portion, gives off cutaneous branches to the buttock and the posterior surface of the thigh (the *inferior cluneal nn.* and the *posterior femoral cutaneous n.*). Along its further course, it gives off *motor branches to the knee flexors*.

Typical deficits: The clinical manifestations of a sciatic nerve lesion depend on the level of the lesion and the extent to which it involves the fibular and tibial portions of the nerve. Proximal lesions (but not distal ones) produce **hypesthesia** on the buttock and the posterior surface of the thigh and impair knee flexion. The strength and reflexes of the knee flexors are best tested in the prone patient.

Causes: The sciatic nerve trunk can be injured by *fractures* of the pelvic ring or proximal portion of the femur, by *surgical procedures* in the region of the hip, or by faultily delivered *injections*. *Tumors* are a less common cause of sciatic nerve palsy.

Fibular N. (L4-S2)

Anatomy. The fibular (peroneal or common peroneal) n., after it separates from the tibial portion of the sciatic n., travels to the lateral margin of the popliteal fossa, winds around the fibular neck, and then enters into the body of the fibularis longus (peroneus longus) muscle, where it divides into the superficial and deep fibular (peroneal) nn. The **superficial fibular (peroneal) n.** provides motor innervation to the *fibular (peroneal) muscles* and sensory innervation to the *lateral surface of the lower leg* and the *dorsum of the foot*, with the exception of the space between the first and second toes (the first interosseous space). The latter is supplied by the **deep fibular (peroneal) n.**, which also innervates the *dorsiflexors of the foot and toes* and the *intrinsic muscles of the dorsum of the foot*.

Typical deficits: The clinical manifestations of a lesion of the deep fibular n. include *foot drop* and *steppage gait*. Sensation is impaired on the dorsum of the foot and completely abolished in the first interosseous space. A lesion of the superficial fibular n. causes *weakness of pronation of the foot* (i. e., inability to elevate the lateral edge of the foot); when the patient walks, the lateral edge of the foot hangs downward. Sensation is impaired in the lower leg and on the dorsum of the foot. If the trunk of the fibular n. (= the common peroneal n.) is affected, all of the above deficits are seen.

Causes: The trunk of the fibular n. can be injured by *penetrating or blunt trauma*, e. g., by knee fractures. *Injection palsies* of the sciatic n. usually affect its fibular portion. The most common cause of fibular nerve palsy, however, is compression of the nerve at the fibular neck by local, external *pressure* (faulty surgical positioning, a cast, etc.). This type of palsy is spontaneously reversible. The site of the lesion can be precisely localized with the aid of electroneurography .

Differential diagnosis: A foot drop combined with loss of sensation on the dorsum of the foot can be seen in *combined lesions of the L4 and L5 nerve roots*, but such lesions will additionally impair abduction of the hip and inversion of the foot. Bilateral foot drop caused either by *Steinert myotonic dystrophy* or by *peroneal muscle atrophy* in the setting of *HMSN type I* can mimic a bilateral fibular nerve palsy. An initially isolated, progressive, unilateral foot drop without any associated sensory deficit may be the first symptom of *spinal muscular atrophy* or *ALS*.

Tibialis anterior syndrome. This syndrome often causes difficulties in differential diagnosis. It is caused by *infarction*

(due to compression) of the muscles in the anterior compartment of the lower leg, because of overuse, trauma, or a hematoma. Steadily rising local pressure within the anterior compartment, which is tightly encased in fascia, leads first to intense local pain, and then to muscle swelling. The pain increases on passive extension of the muscles by plantar flexion of the foot. The muscles become necrotic in 12 to 24 hours and are later replaced by connective tissue. The resulting contracture prevents the appearance of the flaccid foot drop that is otherwise characteristic of fibular nerve palsy. In the acute phase of the tibialis anterior syndrome, the deep fibular n. can be damaged, because its course passes through the anterior compartment of the lower leg. The resulting sensory deficit may be diagnostically misleading, because it may be taken to imply a peripheral nerve

lesion as the primary causative event.

Tibial N. (L4-S3)

Anatomy. This nerve, derived from the medial portion of the sciatic n., innervates the *plantar flexors of the foot and toes* in the lower leg, as well as all of the *intrinsic muscles of the foot*, except those on the dorsum. It provides sensory innervation to the heel and sole

Typical deficits: Weakness of plantar flexion makes tiptoe walking impossible, while weakness of the intrinsic muscles of the foot makes the patient unable to fan the toes. The sensory deficit on the sole of the foot is particularly troublesome because of the important protective function of sensation in this area.

Tarsal tunnel syndrome is an entrapment neuropathy affecting the terminal branch of the tibial n. as it passes under the medial malleolus. It is seen almost exclusively after *fractures or sprains* of the upper ankle joint. Its

typical feature is local *pain* behind the medial malleolus or on the sole of the foot, which increases when the patient walks. The nerve trunk is tender to palpation behind the medial malleolus. Sensation is diminished on the sole of the foot and the plantar skin is abnormally smooth and dry. The patient can no longer fan the toes **Morton metatarsalgia**. A painful neuroma can develop on a digital nerve (a sensory terminal branch of the tibial n.) if the nerve is chronically injured by being compressed between two adjacent metatarsal heads. This condition, called Morton metatarsalgia, causes pain in the forefoot, which is initially felt only on walking, but later also at rest. The pain can be induced by the examiner by laterally compressing the anterior arch of the foot or by squeezing the metatarsal heads against each other. An injection of local anesthetic along the course of the nerve, applied from the dorsal surface of the foot proximal to the site of the neuroma, will bring complete, though transient, relief. Specially padded shoe inserts may be therapeutically useful. If the pain persists, the neuroma should be surgically excised through a plantar approach

41. Disorders of consciousness. Glasgow coma scale.

Variants of comatose states. Vegetative state.

Differential diagnosis of comatose states.

1. <https://www.slideshare.net/HenaJawaid/disorders-of-consciousness-50046093>
2. <https://slideplayer.com/slide/4303457/>
3. <https://www.slideshare.net/mariasalema/definitions-and-approach-to-coma>

Differential diagnosis

Locked in state Akinetic mutism

Persistent vegetative state Catatonic stupor

Pseudocoma Abulia

State	Stimulus needed for arousal
Drowsiness	Verbal and light touch
Obtundation	Deep touch
Stupor	Vigorous, painful, or noxious stimulation

42. Diagnostic value of skull radiography.

1. <https://www.slideshare.net/tarekhegazy/skull-x-ray-plain-evaluations> (slides 29 to 47)

43. Ultrasound dopplerography. Cerebral angiography. Diagnostic value.

1. <https://www.slideshare.net/samirelansary/brain-angiography>
2. <https://www.slideshare.net/NeurologyKota/neurosonology>
3. For more information : <https://www.slideshare.net/shaffar75/principles-of-doppler-ultrasound>

44. Diagnostic value of electroencephalography.

1. <https://www.slideshare.net/ashikh/electroencephalogrameeg>

45. Diagnostic value of electroneuromyography.

Electromyoneurography (EMNG) is the combined use of electromyography and electroneurography. This technique allows for the measurement of a peripheral nerve's conduction velocity upon stimulation (electroneurography) alongside electrical recording of muscular activity (electromyography). Their combined use proves to be clinically relevant by allowing for both the source and location of a particular neuromuscular disease to be known, and for more accurate diagnoses.

Characteristics

Electromyoneurography is a technique that uses surface electrical probes to obtain electrophysiological readings from nerve and muscle cells. The nerve activity is generally recorded using surface electrodes, stimulating the nerve at one site and recording from another with a minimum distance between the two. The time difference of the potential is a measure of the time taken for the potential to travel the distance across the two sites and is a measure of the conduction velocity along the nerve. The amplitude of the potential, measured baseline to peak, or peak to peak, is a measure of the number of fibers conducting the response. Abnormality in data obtained from nerve measurements, such as absent or low amplitude, indicates potential nerve damage.

This technique is used in many medical fields today. One example of its use is to detect neuropathy due to diseases like diabetes mellitus. It can also be used to detect muscle weakness or paralysis due to sepsis or multi-organ failure in comatose patients. This method remains a largely used medical technique due to its efficiency and relative simplicity. **It is especially attractive due to the lack of special precautions or preparation involved with**

this procedure. There is minimal pain and no significant risks except those associated with needle use.

I. Myopathy (disease or disturbance of striated muscle fibers or cell membrane)

Primary (muscle fiber): muscular dystrophy

Duchenne muscular dystrophy

Facioscapulohumeral muscular dystrophy

Limb-girdle muscular dystrophy

Cell membrane hyper-irritability (attributed to spindle cell hyperactivity)

Myotonic dystrophy

Myotonia congenita

Paramyotonia congenita

Myasthenia

Myasthenia gravis

Lambert-Eaton myasthenic syndrome

Hypokalemia

Glycogen storage disease type V

Cushing's syndrome



II. Neuropathy (disease or disorder of the lower motor neuron)
Myelopathy (lesion involving motor neuron in anterior horn of the spinal cord)
<i>Spinal muscular atrophy</i>
<i>Progressive muscular atrophy</i>
<i>Poliomyelitis</i>
<i>Amyotrophic lateral sclerosis</i>
<i>Charcot–Marie–Tooth disease</i>
Radiculopathy (lesion involving the nerve root)
<i>Spinal disc herniation</i>
<i>Spinal stenosis</i>
<i>Guillain–Barré syndrome</i>
Axonopathy (disease or damage to the axon or peripheral nerve)
<i>Carpal tunnel syndrome</i>
<i>Radial neuropathy</i>
<i>Meralgia paraesthesia</i>
<i>Hypothyroidism</i>

1.more information : <https://www.slideserve.com/kellan/electrophysiologic-evaluation-in-brachial-plexus-lesion>

46. Spine radiography. Myelography. Diagnostic value.

1. <https://www.slideshare.net/muhammadbinzulfiqar5/spine-radiography>
2. <https://www.slideshare.net/shatham/myelography>

47. Computer tomography and magnetic resonance imagination of the brain. Diagnostic value.

1. <https://www.slideshare.net/AjayNagisetti/ct-scan-vs-mri-scan>
2. <https://slideplayer.com/slide/7060562/>

48. Cerebral blood supply, symptoms of circulatory disorders in the carotid and vertebral-basilar systems.

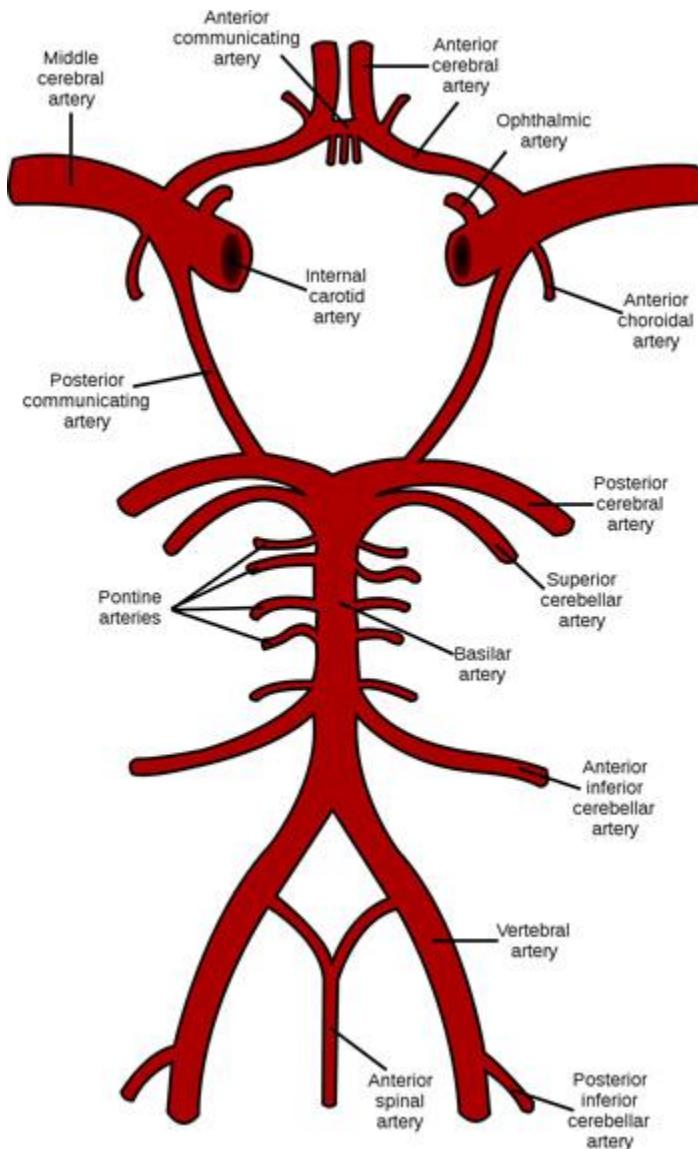
- 1.** <https://www.slideshare.net/meladbassim/the-blood-supply-of-the-brain-and-spinal-cord>
- 2.** <http://www.columbianeurology.org/neurology/staywell/document.php?id=35842>
- 3.** <https://www.medicalnewstoday.com/articles/322275.php>
- 4. For more information:**

Carotid and Vertebrobasilar Disorders

Blood flows from the heart to the brain via two large arterial systems: the **carotid** and the **vertebrobasilar** arterial systems. The vast majority of strokes—both ischemic and hemorrhagic—occur in the part of the brain supplied by the carotid circulation, which channels blood to most of the cerebral hemispheres.

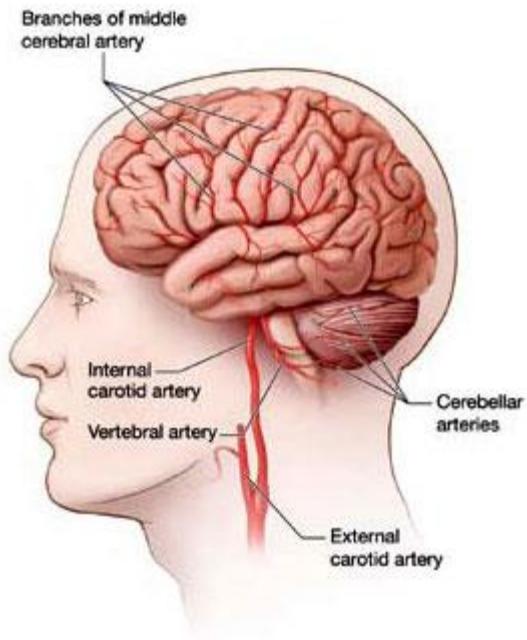
The middle cerebral artery, the anterior cerebral artery, and the ophthalmic artery are the three clinically important branches of the **carotid circulation**. The middle cerebral artery supplies blood to the lateral part of the cerebral cortex, to most of the basal ganglia, and parts of the internal capsule. At the base of the brain, the carotid and vertebrobasilar arteries form a circle of communicating arteries known as the **circle of Willis**.

The Circle of Willis



Schematic representation of the circle of Willis showing the arteries of the brain and brain stem. Source: Wikimedia Commons. Used with permission.

Carotid and Vertebral Arteries

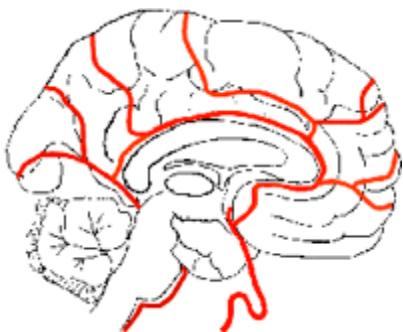


The carotid and vertebral arteries ascend through the neck and divide into branches that supply blood to different parts of the brain. Source: NINDS, Stroke Challenge Brochure.,

The **middle cerebral artery**, which supplies blood to the lateral surface of each hemisphere, is the largest of the cerebral arteries and the most common artery involved with stroke; embolism is the most common cause of blockage (Slater, 2014). Men are affected by middle cerebral artery stroke more often than women at a male-to-female ratio of 3 to 1 (Slater, 2014).

Because the middle cerebral artery is the area most commonly affected by ischemic stroke, its symptoms are the most familiar to healthcare providers: contralateral weakness and sensory loss in the face, neck, and arm (and to a lesser degree in the leg) and homonymous hemianopsia (loss of half of the visual fields of both eyes), as well as cognitive deficits that affect speech, language, and comprehension.

Anterior Cerebral Artery



Medial surface of the brain showing the areas perfused by the anterior cerebral artery. Source: Lauren Robertson. Used by permission.

The **anterior cerebral artery** supplies the medial surface of the brain, and the ophthalmic artery supplies blood to the eye and adjacent structures of the face. Deep branches from the carotid system also supply blood to the regions of the brain below the cerebral cortex—the basal ganglia and the thalamus, together sometimes referred to as the extrapyramidal system, as noted earlier.

Blood traveling through the two vertebral arteries joins at the level of the brainstem to form the **basilar artery**. The vertebrobasilar artery supplies blood to the posterior part of the cerebral hemispheres, including the occipital lobes and the posterior portions of the temporal lobes, the cerebellum, and the brainstem. This is referred to as the **vertebrobasilar or posterior circulation**.

Carotid (Anterior) Circulation Disorders

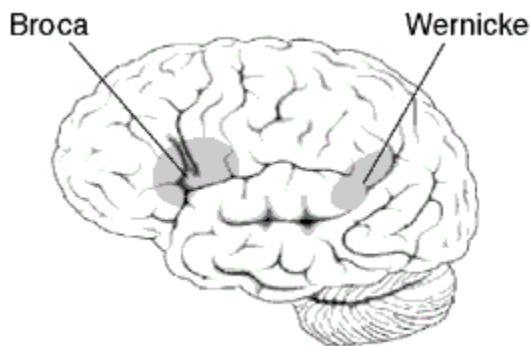
A stroke in any of the major arteries within the *carotid* circulation (middle cerebral, anterior cerebral, and ophthalmic artery) disrupts higher cognitive, motor, and sensory processing. The most common problems—aphasia, apraxia, agnosia, and hemi-neglect, and other cognitive losses—occur in the areas of the brain supplied by the *middle* cerebral artery. Similar problems can occur with occlusions of the *anterior* cerebral artery, in which case the lower extremities and proximal upper extremities are more affected.

A stroke occurring as a result of a blockage in the middle cerebral artery on the left side of the brain can lead to a type of language impairment called **aphasia**. There are different types of aphasia, which are typically defined by the region of the brain that has been damaged.

Wernicke's aphasia is caused by damage to the lateral surface of the left temporal lobe. It is sometimes referred to as receptive or fluent aphasia because

a patient's speech is fluent but the words carry no meaning. Sentences can be long and meandering—usually longer than seven words.

Areas Related to Broca's and Wernicke's Aphasia



Source: Wikimedia Commons.

Broca's aphasia is caused by damage to the lateral surface of the left frontal lobe. It is sometimes referred to as expressive or non-fluent aphasia because a patient is unable to communicate and sentences are short and choppy—usually less than seven words. **Global aphasia** is a combination of Wernicke's and Broca's aphasia in which a person is unable to understand the spoken word or communicate with speech. A severe stroke may begin with global deficits then slowly resolve to a lesser deficit.

If damage occurs on the right side of the brain, speech and comprehension are usually unaffected but other high-level cognitive deficits occur, including behavioral changes, general confusion and disinhibition, unintentional fabrication of information, memory deficits, attentional deficits, apraxia, and neglect.

Apraxia is another common cognitive problem caused by damage from a stroke in the carotid circulation. **Apraxia** is the loss of the ability to organize a movement or perform a purposeful act. It is a disorder of the execution of movement that cannot be attributed to weakness, incoordination, sensory loss, poor language comprehension, or attention deficit. Apraxia is a weakening of the top-down formulation of an action—the inability to sustain the intent to complete a movement. As a result, the nervous system is easily influenced by irrelevant input—a sort of pathologic absent-mindedness.

Apraxia affects all modalities including speech, writing, gesturing, dressing, and all activities of daily living (ADLs). It is difficult for caregivers to

understand and identify. Examples of apraxia are: picking up a telephone and beginning to talk without dialing, lighting a candle and trying to smoke it as if it were a cigarette, using a knife to brush one's hair, using a pencil to butter bread. In all these examples the brain commands the body to perform a movement but the command fades before the movement is completed. The patient tries to complete the movement but has already forgotten what the task was. Nevertheless, an attempt is made to complete the task—perhaps by guessing.

Agnosia is a sensory disorder in which a person is unable to recognize an object by sight, touch, or hearing in the absence of defects in the sensory apparatus of these systems. The person can touch, hear, and see but cannot recognize or identify the object. Agnosia is usually tested by asking a person to identify a series of objects that are placed out of sight in a bag or behind a partition. The person with agnosia will be unable to name an object by touch alone but will be able to identify the object using vision.

Anosognosia (hemi-neglect) is a sensory disorder caused by damage to the parietal lobe in which a person is unaware of the contralateral (opposite) side of the body including half of the visual field. It causes a disruption of a person's body schema and spatial orientation and affects balance and safety awareness. The person is often unaware that the second half of the body exists and will deny that anything is wrong. Those with hemi-neglect may ignore food on the left side of a plate, walk into objects in the left half of the visual field, and completely ignore the left extremities. They may even claim that the affected arm or leg belongs to another person.

A stroke in the ACA circulation affects the medial surface of the brain. It can cause contralateral weakness and sensory loss, primarily in the leg. There may be some weakness in the contralateral arm, especially proximally. It affects the lower extremities more than the upper extremities, leading to difficulties with balance, gait, and mobility. Behavioral disturbances and confusion may be present, and urinary incontinence is not uncommon.

A small clot (**microembolus**) in the ophthalmic artery, the first branch of the internal carotid artery, can cause partial or complete loss of vision in one eye lasting seconds to minutes; this is called temporary monocular blindness or *amaurosis fugax* (fleeting blindness). It is caused by temporary loss of blood flow to the retina and can be a sign of an impending stroke. It is often described as a gray or black shade that comes down over the eye or as blurring, fogging,

or dimming of vision. A clot lodged in the ophthalmic artery can also lead to a sudden and brief bilateral symmetric loss of vision in half of the visual fields that is called **homonymous hemianopsia**.

Loss of Visual Fields in Homonymous Hemianopsia



Paris as seen with right homonymous hemianopsia. The right visual field is missing in both eyes. Source: Wikimedia Commons.

Thalamic Disorders

After a stroke affecting the thalamus, a person may become hypersensitive to pain. This syndrome, called thalamic pain or “central pain syndrome,” is due to damage to the spinal tracts that carry pain and temperature sensation from the periphery to the thalamus. Damage to these tracts, called the **spinothalamic** or **trigeminothalamic** tracts result in severe, spontaneous pain in the parts of the body connected to the damaged tracts. Thalamic pain starts several weeks after the stroke and presents as an intense burning pain on the side of the body affected by the stroke; it is often worsened by cutaneous stimulation.

Pain is typically constant, may be moderate to severe in intensity, and is often made worse by touch, movement, emotions, and temperature changes, usually cold temperatures. One or more types of pain sensations may be present—the most prominent being burning. Mingled with the burning may be sensations of pins and needles; pressing, lacerating, or aching pain; and brief, intolerable bursts of sharp pain similar to the pain caused by a dental probe on an exposed nerve. Individuals may have numbness in the areas affected by the pain. The burning and loss of touch sensations are usually most severe on the distant parts of the body, such as the feet or hands.

Basal Ganglia Disorders

In addition to the lateral surface of the cerebral cortex, the middle cerebral artery also supplies blood to the basal ganglia. A stroke affecting the basal ganglia usually causes motor control problems rather than hemiparesis. Damage typically causes too much movement (hyperkinesia) or too little movement (hypokinesia).

Hyperkinesia

Hyperkinesia is too much movement, and although our understanding of its cause may be unclear, we have many words to describe such disorders. **Chorea** is a hyperkinetic movement disorder characterized by arrhythmic, rapid, involuntary movement that flows from one part of the body to another. The most common type of non-drug-related chorea is Huntington's chorea. **Dystonia** is a hyperkinetic movement disorder characterized by involuntary movement that is twisting, sustained, and repetitive. Over time, the affected body part may assume a fixed posture involving one joint (focal dystonia), two joints (segmental dystonia), or several joints (generalized dystonia).

Athetosis is a hyperkinetic movement disorder characterized by spontaneous writhing movements of the hand, arm, neck, or face. **Tardive dyskinesia** is a slow-onset, drug-induced hyperkinetic movement disorder characterized by rhythmic, unwanted movements of the face and extremities such as facial grimacing, tongue movements, and pill-rolling motions with the fingers. **Tourette syndrome** is characterized by excessive energy, tics, jerks, verbal noises, compulsive behavior, and grimaces. It is also associated with other behavioral disorders such as attention deficit disorder.

Hypokinesia

Hypokinesia is too little movement. Parkinson's disease (*paralysis agitans*) is one of the most common hypokinetic movement disorders and is characterized by resting tremor, rigidity, masked faces, bradykinesia, and festinating gait. Parkinson's disease is caused by widespread destruction of a portion of the brainstem (the substantia nigra), which is responsible for sending dopamine to the basal ganglia. Although Parkinson's disease is not caused by stroke it is mentioned here as an example of a hypokinetic movement disorder.

Vertebrobasilar (Posterior) Circulation Disorders

Recall that the vertebral artery supplies blood to the posterior part of the cerebral hemispheres, including the occipital lobes and the posterior portions of the temporal lobes, the cerebellum, and the brainstem. Posterior circulation ischemia causes a variety of symptoms that are distinctly different from those found with carotid artery strokes. If the damage is in the area of the brainstem there may be loss of brainstem function, cranial nerve abnormalities (with or without hemiparesis), or hemi-sensory deficits.

If damage is in the area of the cerebellum, you can expect to see ataxia, intention tremor, and hypotonia. **Ataxia** is motor incoordination due to irregularities in the timing, rate, and force of a muscular contraction. Ataxia causes unsteady, grossly uncoordinated, or “drunken” gait, loss of balance, and a tendency to fall. It also affects the ability to judge the distance or scale of a movement, typified by overshooting or undershooting an object (dysmetria). As a result, vertigo, nausea, vomiting, and nystagmus are common occurrences following a cerebellar stroke.

Intention or action tremor is another common type of abnormal movement associated with cerebellar damage. The tremor is not present at rest (as with Parkinson’s) but occurs as soon as a movement is initiated. For example, a person may reach for a glass of water but be unable to control the force and range of the movement, especially at the end of the movement. While reaching for the glass the tremor increases and the individual may overshoot the glass entirely, touch the glass with too much force, or lift it too rapidly.

Hypotonia is a decreased resistance to the passive stretch of a joint. Muscles feel soft to the touch and lack normal tone. Hypotonia can be tested by tapping the patellar tendon reflex with a reflex hammer. A tap on the patellar tendon will normally produce a quick extension of the lower leg, which will come to rest after one or two swings. If cerebellar damage is present, a tap on the patellar tendon will cause the lower leg to oscillate 6 or 7 times before coming to rest. This is called a **pendular swing** and is typical of cerebellar damage.

❖ Blood Supply

❖ **Anterior: Carotid Arteries** – middle & anterior cerebral arteries

- ❖ frontal, parietal, temporal lobes; basal ganglion; part of the diencephalon (thalamus & hypothalamus)

❖ **Posterior: Vertebral Arteries** – basilar artery

- ❖ Mid and lower temporary & occipital lobes, cerebellum, brainstem, & part of the diencephalon

❖ **Circle of Willis** – connects the anterior & posterior cerebral circulation

Clinical syndromes

Anterior cerebral artery occlusion:

The effect of occlusion depends on the relation with respect to the anterior communicating artery. Occlusion proximal to the anterior communicating artery is normally well-tolerated, because of adequate cross-flow, and thus few symptoms result.

- contralateral leg paresis; *contralateral leg hypoesthesia*;
- frontal ataxia and walking apraxia, astasia-abasia
- frontal lobe behavior;
- contralateral grasp and other primitive reflexes
- hyperkinesis in the face and arm
- incontinence

Clinical syndromes

Middle cerebral artery occlusion:

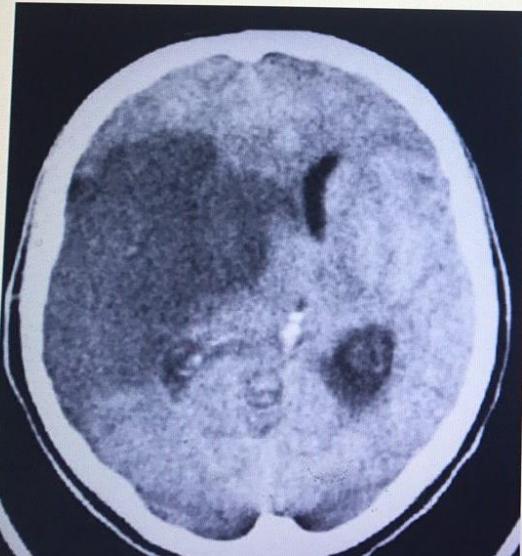
- Contralateral hemiparesis (including the lower part of the face and relative sparing of the leg);
- Contralateral cortical hemisensory loss;
- Contralateral homonymous hemianopia
- Dominant hemisphere: aphasia
- Non-dominant hemisphere: neglect of contralateral limb and dressing apraxia

Brain infarction in the left middle cerebral artery



- Speech disorders (aphasia)
- Right side hemiparesis
- Right side hypoesthesia
- Right side hemianopsia

Infarction of the brain in the right middle cerebral artery



- Left side hemiparesis
- Left side hypoesthesia
- Left side hemianopsia
- Ignoring an existing defect

Clinical syndromes

Vertebro-basilar occlusion

- dizziness;
- tinnitus;
- nausea, vomiting;
- muscular hypotonia, ataxia, adiadochokinesis (cerebellar syndrome);
- diplopia, nystagm, ptosis, strabismus, etc.(oculomotor disorders);
- cranial nerves lesion (ipsilateral);
- hemiparesis and hypoesthesia (contralateral);
- dysarthria, dysphagia, dysphonia (bulbar syndrome).

Clinical syndromes

Intracerebral hemorrhage:

The most common non-traumatic causes include chronic hypertension, aneurisms and vascular malformations

Clinical syndromes

The clinical signs depend on the location but are often associated with mass effect and therefore reduced consciousness level.

Clinical syndromes

Subarachnoid hemorrhage:

- Severe headache of instantaneous onset
- Transient or prolonged loss of consciousness or seizure may follow immediately
- Nausea and vomiting
- Drowsiness or coma may continue for hours or days
- Signs of meningism occur after 3-12 hours
- Focal signs from haematoma may be present
- Papilloedema may be present and accompanied by subhyaloid and vitreous haemorrhage

49. Blood supply of the spinal cord, symptoms of circulatory disorders.

1. <https://www.slideshare.net/meladbassim/the-blood-supply-of-the-brain-and-spinal-cord>

Circulatory Disorders of the Spinal Cord

Vascular lesions of the spinal cord, as of the brain, are of two main types: **hemorrhage** and **ischemia**. The latter is due to blockage of either the arterial blood supply (e. g., because of thrombosis or embolism) or the venous outflow.

Blood Supply of the Spinal Cord

The spinal cord receives arterial blood from three vessels: the unpaired **anterior spinal a.**, which runs down the anterior median fissure of the cord and supplies the anterior two-thirds of its cross-sectional area, and the paired **posterior spinal aa.** Each of these spinal arteries is made up of a series of individual segments that are linked with one another along the longitudinal axis and receive arterial blood from various sources .

At cervical levels, the anterior spinal a. receives blood mainly from the *vertebral a.* and the *costocervical and thyrocervical trunks*; further down the spinal cord, it is supplied by *segmental arteries* arising from the aorta (spinal branches and radicular arteries, each of which has an anterior and a posterior branch).

.
The largest of these, called the *great radicular a.* or the *artery of Adamkiewicz*, usually enters the spinal canal between T10 and L2, more commonly on the left side.

. Venous blood flows out of the spinal

cord through radicular veins and into the vena cava

Arterial Hypoperfusion

Global (arterial)myelomalacia. Infarction of the entire cross-section of the spinal cord at a particular level may be due to the **occlusion of a local spinal artery or of a**

radicular artery, or to extraspinal vascular pathology, such as an aortic aneurysm. The clinical presentation is usually an acute spinal cord transection syndrome (complete or partial), though, in some patients, symptoms develop subacutely over the course of a few days, or stepwise. Affected patients usually remain paraplegic, particularly if the ischemic lesion is very extensive.

Anterior spinal artery syndrome. Thrombotic or embolic occlusion of the anterior spinal a. damages the anterolateral aspect of the spinal cord over one or more segments

. An occlusion at a distal location along the course of the anterior spinal a., e. g., in a *sulcocommissural artery*, may cause a partial **Brown-Séquard syndrome**, with preservation of the sense of touch.

Central cord infarction. Infarction of the spinal cord, whether it involves the entire cross-section of the cord or only a part of it, is usually not restricted to a single cord segment in the vertical dimension, but rather tends to involve multiple segments. As part of this process, necrosis affects the motor neurons of the anterior horn, causing flaccid paresis and areflexia at the level of the lesion, in addition to the spastic paresis below the level of the lesion due to involvement of the corticospinal tracts. In a few weeks' time, the flaccid muscles become atrophic. The clinical picture is, therefore, that of a “*peripheral*” *paralysis at the level of the transection* and also

a short distance below it.

Intermittent spinal ischemia is very rare and causes a type of spinal intermittent claudication with fluctuating spastic paraparesis.

Chronically progressive vascular myelopathy can cause slowly progressive spastic paraparesis, as well as muscle atrophy owing to involvement of the anterior horns.

Impaired Venous Drainage

Spinal cord ischemia due to impaired venous drainage is a rare cause of infarction. It is usually due to a *spinal arteriovenous fistula* or *arteriovenous malformation*.

Spinal Arteriovenous Malformations and Fistulae

Arteriovenous malformations are usually found in the thoracolumbar region, while fistulae are usually found at lower lumbar levels. Both types of vascular anomaly are more common in men. They tend to present between the ages of 10 and 40, often with (*bandlike*) pain as the initial symptom. *Neurological deficits* referable to the spinal cord are often only intermittent at first and are (partially) reversible at this stage; later, they take a chronic, progressive course and become permanent. A dural arteriovenous fistula, for example, can cause chronically progressive spastic paraparesis. These vascular anomalies also occasionally present with spinal subarachnoid hemorrhage. *MRI* is the most important diagnostic study for the establishment of the diagnosis. *Spinal angiography* can provide useful additional anatomical detail.

Circulatory Disorders of the Spinal Cord

Mumenthaler /

Hemorrhage in or Adjacent to the Spinal Cord

The function of the spinal cord can be affected by an *intramedullary*, *subdural*, or *epidural hemorrhage*. These

types of hemorrhage can arise spontaneously in anticoagulated

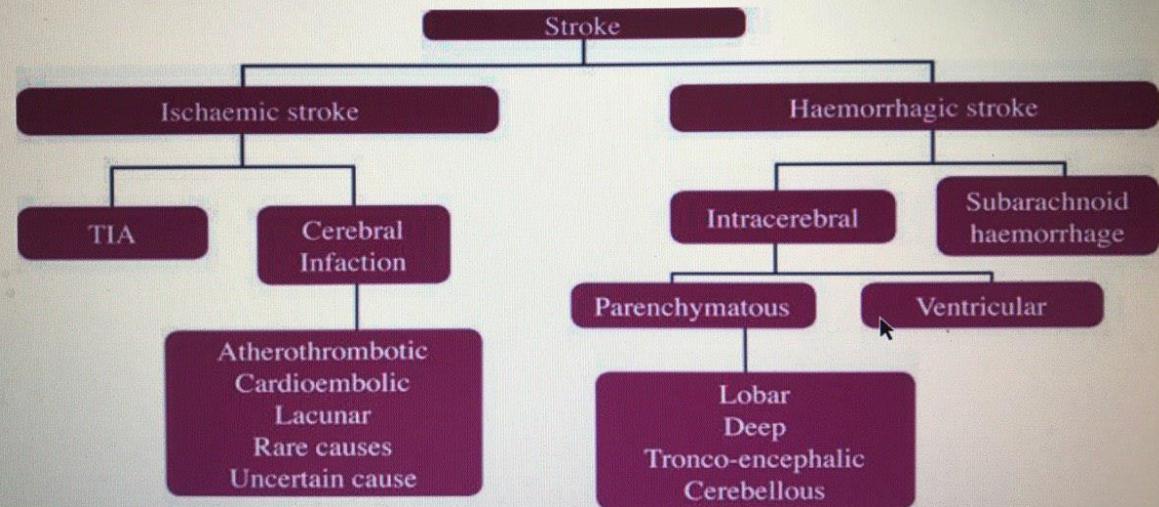
patients, or they can be caused by ruptured vascular malformations or trauma. They usually produce *intense pain* and more or less pronounced *neurological deficits*, depending on their site and extent. Hemorrhage in or adjacent to the spinal cord requires immediate diagnostic evaluation and, in some patients, emergency neurosurgical decompression.

50. Classification of cerebrovascular disorders. Transient ischemic attack.

Classification of cerebral circulation disorders (RESEARCH INSTITUTE of Neurology of AMS, USSR 1985)

1. Diseases and pathological conditions leading to cerebrovascular diseases
2. Cerebrovascular diseases:
 - A. initial manifestations of cerebrovascular insufficiency
 - B. Transient disorders of cerebral circulation:
 - transient ischemic attack
 - hypertensive crisis
 - Acute hypertensive encephalopathy
 - C. Stroke:
 - cerebral infarction
 - cerebral hemorrhage (non-traumatic)
 - subarachnoidal hemorrhage (non-traumatic)
 - extradural hematoma (non-traumatic)
 - subdural hematoma (non-traumatic)
 - D. Progressive cerebral circulation disorders:
 - Chronic subdural hematoma
 - Discirculatory encephalopathy

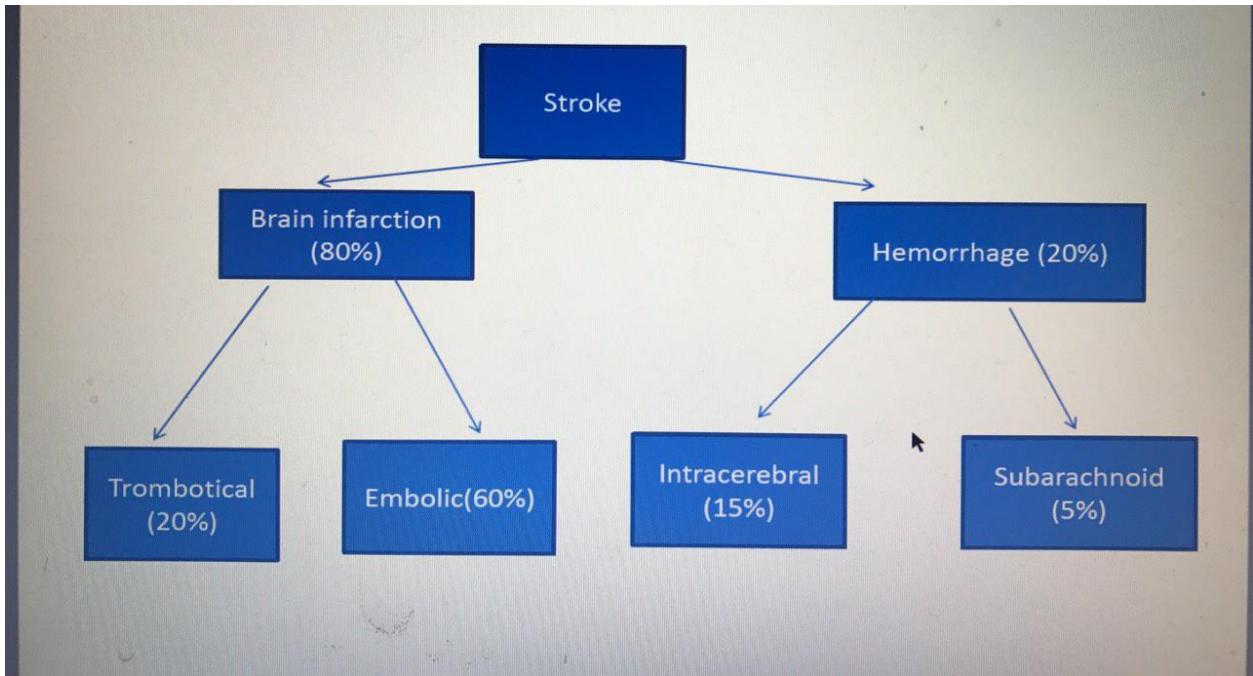
Classification



Stroke subtypes classification (TOAST)

Classification (haemorrhagic strokes)

- **1. Intra-axial haemorrhage**
- a) *Primary*
 - i. Haemorrhage
 - ii. Microhaemorrhage
- b) *Secondary*
 - i. Tumours
 - ii. Vascular malformations
 - iii. Aneurysms
 - iv. Haemorrhagiparous diseases. Coagulopathies
 - v. Anti-thrombotics
 - vi. Fibrinolytics
 - vii. Sympathomimetics
 - viii. Infections
 - ix. Vasculitis
 - x. Retarded posttraumatics
 - xi. Vein or sinus thrombosis
- **2. Subarachnoid haemorrhage**
- a. Aneurismatic
- b. Non-aneurismatic
- **3. Subdural haematoma**
- **4. Epidural haematoma**



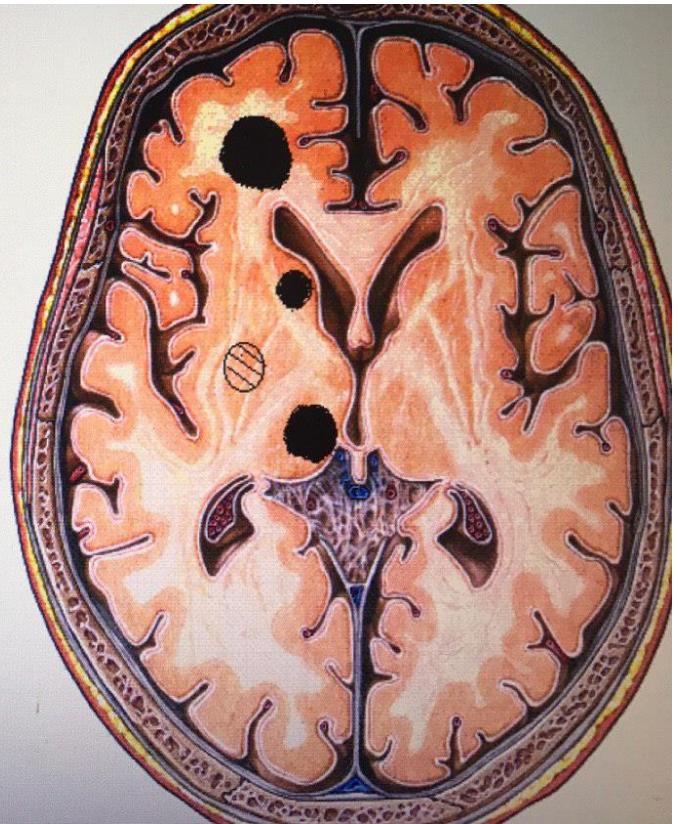
1. <https://www.slideshare.net/avinash0025/dont-ignore-transient-ischemic-attack>

51. Classification of cerebrovascular disorders. Acute hypertensive encephalopathy.

The answer of first part is the same as question 50

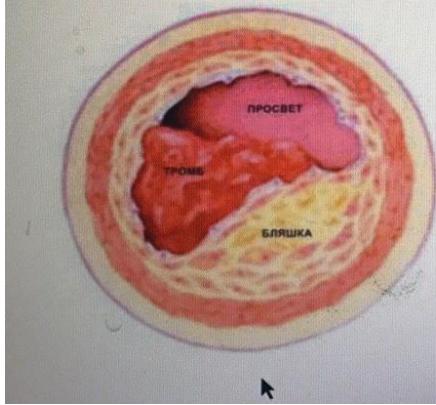
1. <https://www.slideshare.net/sazzad92/hypertensive-encephalopathy-and-emergencies>

**Encephalopathy
(chronic cerebral
circulation)**
- the most
frequent diagnosis
in neurology



The most frequent causes of
encephalopathy

Atherosclerosis



Arterial hypertension



Criteria for the diagnosis

- ❖ detected objectively neuropsychological or neurological symptoms;
- ❖ signs of cerebrovascular disease, including risk factors (arterial hypertension, Hyperlipidemia, diabetes mellitus, cardiac arrhythmia, etc.) and the data history
- ❖ evidence of a causal relationship between the aforementioned criteria: (a) compliance with the dynamics of neuropsychological and neurological deficit of cerebrovascular disease characteristics (the tendency to progression of alternating periods of rapid deterioration, partial regression and relative stability) and/or (b) correspondence identified in CT/MRI changes the substance of the brain vascular Genesis leading clinical manifestations;
- ❖ signs of cerebrovascular lesions and/or brain matter, MRI/CT SCAN of the brain;
- ❖ exclude other diseases that could explain the clinical picture.

Encephalopathy is divided into three degrees

1 grade (pseudoneurotic)

Dominated by subjective disorders: headache, dizziness, noise in the head, reducing the memory and attention, emotional lability.

2 grade (microsymptoms)

Pyramid and extrapiramidal disorders.

3 grade (psychoorganic)

Personality change, dementia, more objective neurological disorders.

The main clinical features of encephalopathy

Subject complaints

1. Memory loss and mental abilities
2. Increased fatigue and decreased performance.
3. Emotional lability.
4. Headache.
5. Dizziness.
6. Sleep disturbance.

Objective neurological findings

1. Extrapyramidal disorders.
2. Ataxia.
3. Pseudobulbar syndrome.
4. Pyramid violations.

Decline of cognitive functions is mandatory sign encephalopathy

- Light and mild cognitive impairment (memory loss and other cognitive functions without violations of professional and social activities)
- Dementia (decline in memory and other cognitive functions in violation of professional and/or social activities)

The most common types of brain changes when encephalopathy

- I. Bilateral white matter lesion (leukoencephalopathy).
- II. Clinical assessment of multiple lesions

Treatment of patients with encephalopathy on the basis of evidence-based medicine

- Prevention of progression of cerebrovascular disease.
- Training of cognitive functions.
- Treatment of cognitive, emotional and behavioral disorders and other neurological disorders.

52. Ischemic stroke.

1. <https://www.slideshare.net/DrSmashAMC/ischemic-stroke-46002893>

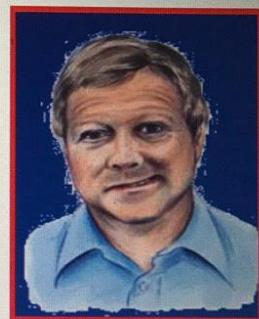
Basis for the diagnosis of "stroke"

- Acute development of neurological deficit
- The presence of risk factors
- Clinical symptoms
- Neuroimaging data

DIAGNOSIS OF STROKE

—Prehospital stroke scale

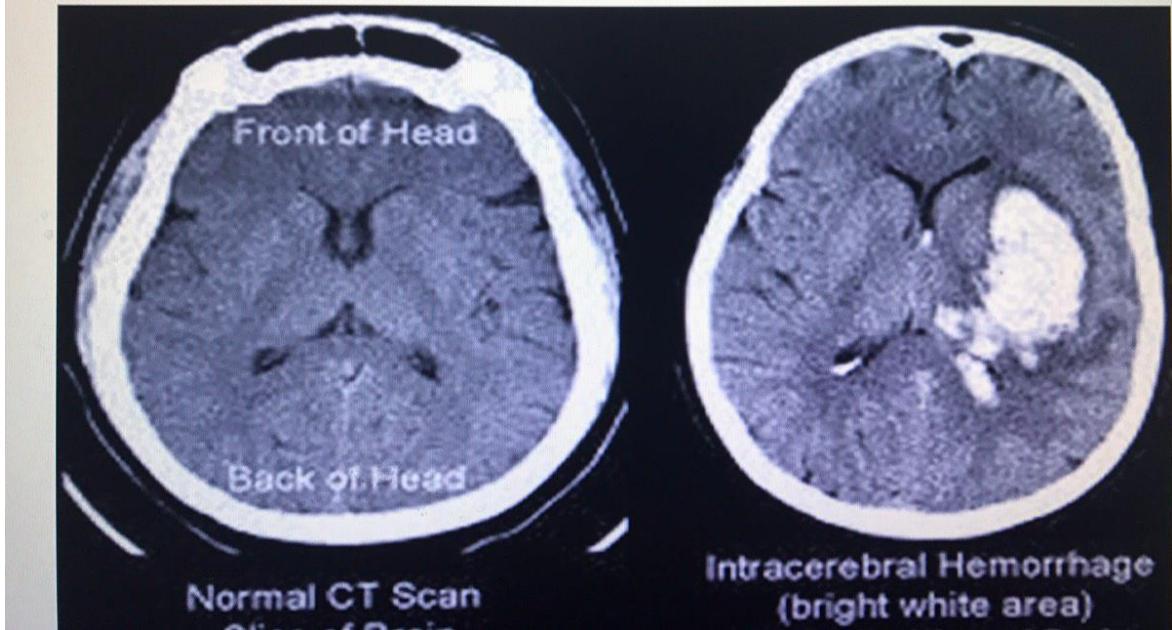
- Facial Droop
- Arm Drift
- Speech



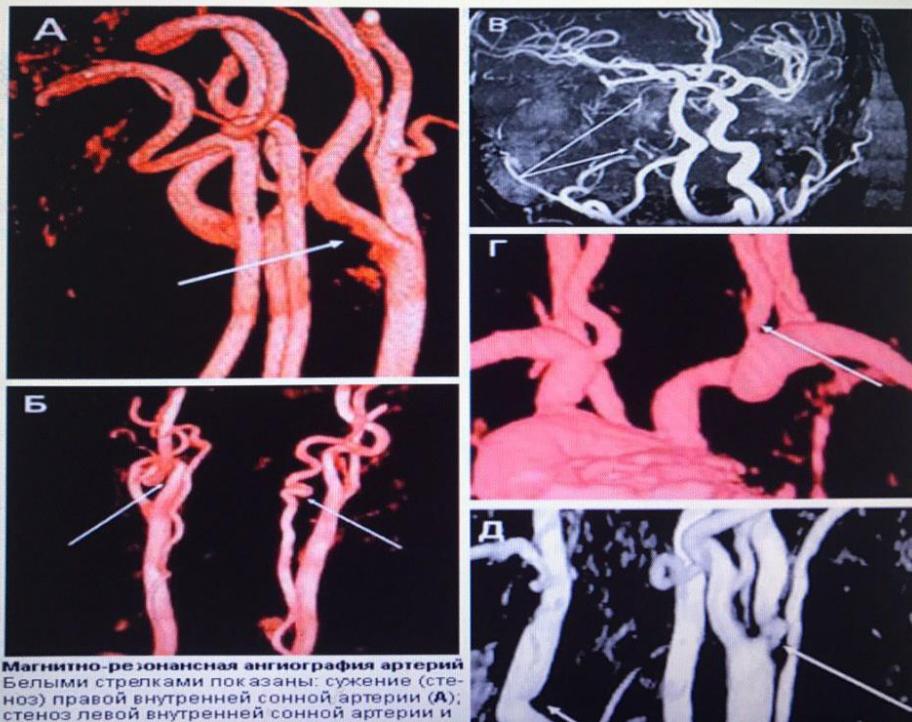
Examination of STROKE patients

- Neuroimaging: CT or MRI of the brain
- Lumbar puncture (inflammatory disease of the CNS or SAH suspected or no CT or MRI available)
- Ultrasound dopplerography of the brain arteries
- ECG, ECHO-Cardiography if needed.
- General and biochemical blood tests, blood coagulation tests
- Carotid or vertebral angiography (in case of suspected aneurisms)

Neuroimaging-should be carried out in all patients with suspected STROKE



MRI angio



Differential diagnosis of stroke

- Traumatic brain injury
- Meningitis, encephalitis
- Intracranial lesions
- Todd's palsy
- Migraine
- Metabolic disorders
 - Hyper-and hypoglycemia
 - Posthypoxic encephalopathy
 - Drugs overdose

TREATMENT

The success of aid in stroke depends on the 4 "chain"

- Rapid detection of possible signs of a stroke
- Immediate ambulance call
- Alert Hospital where the patient will be delivered, and easy transport
- Qualitative diagnosis and treatment

TREATMENT OF STROKE

Basic (undifferentiated) therapy

- is not dependent on the type of stroke

Special (differentiated) therapy

- is depending on the type of stroke after his verification

Basic therapy

- ❖ Ensuring adequate breathing.
- ❖ Maintaining blood circulation.
- ❖ Wrestling with swelling of the brain.
- ❖ Prevention and treatment of pneumonia and other complications.
- ❖ Neuroprotection.

The fight against brain edema

- 1) Osmodiuresis -mannitol. The drug is administered intravenously in the initial dose of 0.5 -1.5 g/kg body weight for 20 minutes, and then at a dose of 1/2 of the original.
- 2) Ventricles drainage

Neuroprotection

- There is now a wide range of neuroprotectors: presynaptic glutamate inhibitors (lubeluzol); calcium channel blockers (nimodipine); antioxidants (emokspin, mexidol, cytoflavin); Piracetam, actovegin, cerebrolysin, glycine etc.
- Proven their utility in experimental studies.
Efficacy of neuroprotective drugs in terms of evidence-based medicine is not approved.

Pre-hospital stage

- ensure sufficient ventilation and oxygenation;
- maintaining the stability of the systemic hemodynamics;

(blood pressure > 220/120 mm Hg do not decrease without a pressing need: asthma pulmonary edema, congestive heart failure, acute kidney damage);

- Anti-convulsive therapy (if needed);
- prevention of edema of the brain.

Special therapy:

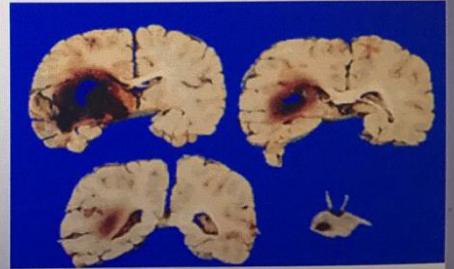
- **Trombolysis**
- **Anticoagulants**
- **Antiplatelet**
- **The most effective of the differentiated therapy of brain infarction is thrombolytic therapy**

Antiplatelet Agents

- **Aspirin-75-250 mg**
- **Aspirin+dipyridamol**
- **Plavix**
- **Plavix+aspirin**

Special therapy following a brain hemorrhage

- Specific medical treatments for a cerebral hemorrhage is now gone
- All patients with cerebral hemorrhage should be consulted by neurosurgeon
- surgical treatment:
 - Remove hematoma open way:
frontal, lateral hematoma;
 - Stereotactic surgery: medial hematoma.



Special therapy subarachnoid hemorrhage

- **Triple-H therapy:**
 - hipervolemia
 - hemodilution
 - arterial hypertension

Based on the fact that the cerebral blood flow depends on the cerebral perfusion pressure, blood viscosity and the diameter of the vessel. Impact on these parameters can improve cerebral blood flow and prevent cerebral vasoconstriction.

Nimodipine is currently the only calcium channel blocker with proven efficacy in the subarachnoid hemorrhage.

- **Methods of operative treatment:**
 - Clipping of aneurysm
 - Endovascular intervention
 - Stenting



53. Lacunar stroke.

1. <https://www.youtube.com/watch?v=7-QmrnjC4gE>

What is a lacunar stroke?

A stroke occurs when blood flow to the brain is **interrupted or blocked**. Strokes that are caused by blockages in blood vessels within the brain are called ischemic strokes. **Lacunar stroke is a type of ischemic stroke that occurs when blood flow to one of the small arteries deep within the brain becomes blocked.**

Lacunar strokes represent about one-fifth of all strokes. Any type of stroke is dangerous because brain cells are deprived of oxygen and begin to die within minutes.

What are the symptoms of lacunar stroke?

Signs of lacunar stroke can include:

- slurred speech
- inability to raise one arm
- drooping on one side of the face
- numbness, often on only one side of the body
- difficulty walking or moving your arms
- confusion
- memory problems
- difficulty speaking or understanding spoken language
- headache
- loss of consciousness or coma

What causes lacunar stroke?

Lacunar stroke is caused by lack of blood flow in smaller arteries that supply deep brain structures. The most important risk factor for the development of lacunar stroke is chronic high blood pressure. The condition can cause the arteries to narrow. This makes it easier for cholesterol plaques or blood clots to block blood flow to the deep brain tissues.

Who is at risk for lacunar stroke?

Risk of lacunar stroke increases **with age**. Those at risk include people with chronic **high blood pressure**, heart disorders, **or diabetes**. African-Americans, Hispanics, and people with a family history of stroke are also at a higher risk than other groups.

Additional factors that increase the likelihood of lacunar stroke include:

- smoking or exposure to secondhand smoke
- alcohol use
- drug abuse
- pregnancy
- use of birth control pills
- sedentary lifestyle
- poor diet
- **high cholesterol**
- **obstructive sleep apnea**

It's important to have annual physical examinations to screen for health issues that could raise your risk for stroke, including high cholesterol and obstructive sleep apnea.

How is lacunar stroke diagnosed?

Emergency treatment is necessary for any type of stroke, so it's imperative to seek diagnosis immediately. If your symptoms are consistent with stroke, immediate diagnostic testing will likely include a **CT scan or a MRI scan** to

take detailed images of your brain. A Doppler ultrasound may also be used. This will measure the amount of blood flowing through your arteries and veins.

Heart function tests, such as electrocardiogram and echocardiogram may be ordered. Kidney and liver function testing and various blood tests may also be administered.

What is the treatment for lacunar stroke?

. Once you arrive at the emergency room, you'll likely be given aspirin and other medications. This reduces your risk of having another stroke.

Supportive measures may be needed to assist your breathing and heart function. You may receive intravenous clot-busting drugs. In extreme circumstances a doctor can deliver medications directly into the brain.

Lacunar stroke can result in some brain damage. Depending how badly the underlying structures are damaged, you may not be able to care for yourself following a stroke. Recovery varies for each person and depends on the severity of the stroke.

Most people who experience a stroke require long-term treatment. This can include medication to treat high blood pressure, diabetes, or high cholesterol. After a lacunar stroke.

What is the long-term outlook?

Quality of life after lacunar stroke depends on many factors, including age and how quickly treatment began after symptoms started. For some patients, disabilities are permanent. These can include:

- paralysis
- numbness
- loss of muscle control on one side of the body
- tingling sensation in affected limb

54. Hemorrhagic stroke.

Classification (haemorrhagic strokes)

- **1. Intra-axial haemorrhage**
- *a) Primary*
 - i. Haemorrhage
 - ii. Microhaemorrhage
- *b) Secondary*
 - i. Tumours
 - ii. Vascular malformations
 - iii. Aneurysms
 - iv. Haemorrhagiparous diseases. Coagulopathies
 - v. Anti-thrombotics
 - vi. Fibrinolytics
 - vii. Sympaticomimetics
 - viii. Infections
 - ix. Vasculitis
 - x. Retarded posttraumatics
 - xi. Vein or sinus thrombosis
- **2. Subarachnoid haemorrhage**
- a. Aneurismatic
- b. Non-aneurismatic
- **3. Subdural haematoma**
- **4. Epidural haematoma**

1. <https://www.slideshare.net/raychow1/hemorrhagic-stroke-final-final>

55. Non-traumatic intracranial hemorrhages (cerebral arterial aneurisms).

1. <http://etest.bsmu.by/mod/page/view.php?id=151720> (**slides 1 to 32**)

56. Vascular dementia.

1. <https://www.slideshare.net/wef/vascular-dementia-and-mixed-dementia> (**slides 6 to 25**)

57. Cerebral palsy. Perinatal encephalopathy. Intracranial birth trauma.

1. <https://www.slideshare.net/bronzedchika/cerebral-palsy-1712524>
2. <https://www.slideshare.net/medicationdotnet/hypothermia-treatment-for-hypoxic-ischaemic-encephalopathy-in-newborn-infants>
3. <https://www.slideshare.net/SoumalyaKundu/birth-trauma-intracranial-haemorrhage>

58. Classification of meningitis.

for answers of questions 58 to 72 ,at first should read the lectures on the site

then the answer of each question separately

1. <http://etest.bsmu.by/mod/page/view.php?id=151550>
2. <http://etest.bsmu.by/mod/page/view.php?id=151551&forceview=1>



there are good slides in answer of question number 27 for the answer of this question

from another source

Meningitis

Meningitis is inflammation of the meninges : the pia and arachnoid maters and the cerebrospinal fluid(CSF)

Etiology

•Bacteria:

1. In the neonate, they include:

-Gram-negative bacilli (E. coli, Klebsiellaspp.)

2. In children:

-H. influenzae type B

-Streptococcus pneumoniae(pneumococcus)

-Neisseria meningitidis(meningococcus)

3. In adults:

-Streptococcus pneumoniae

-Neisseria meningitidis

4. In immunodeficiency, trauma, or neurosurgery:

-Staphylococcus aureus

-Listeria monocytogenes

-Proteus spp.

-Group A streptococci

•Acid-fast bacilli

-Mycobacterium tuberculosis –Tuberculous meningitis

•Spirochaetas

-Treponema pallidum(Syphilis)

-Borrelia burgdorferi(Lyme disease)

•Viruses

-Enteroviruses

- Mumps
- Herpes simplex type 2 (rarely type 1) and Epstein-Barr virus (EBV)
- HIV: due to the primary infection
- Fungi**

-Cryptococcus neoformans(common opportunistic organism in HIV-positive and other immunocompromised patients)

-Histoplasma capsulatum

Additional information:

Nosocomial meningitis

Meningitis that developed:

- more than 48 hours after hospitalization
- within one week of hospital discharge

Risk factors

- Neurosurgery
- Head trauma within the past month
- Neurosurgical device
- CSF leak

Causative agents

- Gram-negative bacilli
- Streptococcus
- Staphylococcus aureus
- Coagulase-negative staphylococci

Recurrent meningitis

a. Community-acquired meningitis

- Streptococcus pneumonia
- b. Nosocomial-acquired meningitis
- Gram-negative bacilli

Mechanism for developing meningitis

- Colonization of the nasopharynx
- Bloodstream invasion and subsequent CNS invasion
- Invasion of the CNS following bacteremia
- Localized source (endocarditis) or urinary tract infection

- Direct entry of organisms into the CNS
- From contiguous spread (sinuses, mastoid)
- Trauma
- Neurosurgery
- CSF leak
- Medical devices (shunts, ICP monitors, cochlear implants)

Causative organisms-site of entry

Neisseria meningitidis

- Nasopharynx

Streptococcus pneumonia

- Nasopharynx, direct extension across skull fracture

Listeria monocytogenes

- GI tract, placenta

Coagulase-negative staphylococcus

- Dermal or foreign body

Staphylococcus aureus

- Bacteremia, dermal, or foreign body

Gram negative rods

- Various

Haemophilus influenza

- Nasopharynx

Predisposing factors to meningitis

Host factors

- Asplenia
- Complement deficiency
- Corticosteroid excess
- HIV infection
- Recent infection (respiratory, otic)
- Recent exposure to someone with meningitis
- IV drug use
- Recent head trauma
- Otorrhea or rhinorrhea
- Travel to an endemic meningitis area (Africa-meningococcemia)

Mechanism of disease

Colonization and invasion

Evasion of the complement system

- Alternate pathway outside the CNS

Stimulation of the classic complement system inside the CNS

Inadequate humoral immunity in the CSF

- Rapid replication of the bacteria in the CNS

Cell wall components of the bacteria cause inflammation in CNS

- Leads to disruption of the blood-brain barrier
- Results in vasogenic brain edema, loss of cerebrovascular autoregulation, increased intracranial pressure
- Results in brain ischemia, cytotoxic injury and neuronal loss

Clinical features

Presenting manifestation

- Fever
- Nuchal rigidity
- Headache
- Photophobia
- Change in mental status

Other

- Seizures
- Cranial nerve palsies
- Papilledema
- Petechiae
- Palpable purpura
- Arthritis
- Otitis
- Sinusitis

59. Purulent meningitis, meningococcal meningitis.

Acute Meningitis

Acute bacterial meningitis

is caused by bacteria

that can reach the meninges by any of three routes:

hematogenous spread (e. g., from a focus of infection in the nasopharynx), **continuous extension**

(e. g., from the middle ear or paranasal sinuses), or **direct contamination** (through an open wound or CSF fistula).

The clinical onset of purulent meningitis is usually **acute or subacute** and patients very quickly become severely ill. The initiation of antibiotic therapy as rapidly as possible is essential for a good outcome

Etiology

The organisms that most commonly cause acute, purulent meningitis are:

— **in neonates**, *Escherichia coli*, group B streptococci, and *Listeria monocytogenes*;

— **in children**, *Hemophilus influenzae*, pneumococci, and meningococci (*Neisseria meningitidis*);

— **in adults**, pneumococci, meningococci, and, less commonly, staphylococci and gram-negative enterobacteria.

Clinical manifestations.

— myalgia, back pain;

— photophobia;

— if the infection is mainly located over the cerebral convexity, with irritation of the underlying brain parenchyma, epileptic seizures (40 %);

- cranial nerve deficits (10 to 20%, sometimes permanent deafness, particularly after pneumococcal infection);
- variably severe impairment of consciousness;
- in infection with *Neisseria meningitidis*, there may be petechial cutaneous hemorrhages and hemorrhagic necrosis of the adrenal cortex due to endotoxic shock (Waterhouse–Friderichsen syndrome).

Diagnostic evaluation.

The most important and most urgent component of the diagnostic evaluation is *lumbar puncture*. Whenever acute meningitis is suspected, a

lumbar puncture should be performed at once, **as soon as papilledema** (a sign of intracranial hypertension) has been ruled out by ophthalmoscopy. The CSF **is typically turbid**, with 1000 to several thousand cells/mm³ (mainly granulocytes), **the protein concentration markedly elevated** (positive Pandy test), **and the glucose concentration diminished**. CSF examination enables confirmation of the diagnosis of meningitis and, in two-thirds of patients, demonstration of bacteria by Gram stain and identification of the causative organism by CSF culture.

Treatment

begins with *antibiotic therapy*, with a single drug, or multiple drugs, chosen for their effectiveness against the most likely causative organisms in the given clinical setting. Once the organism has been identified by CSF culture and its antibiotic sensitivity spectrum has been determined, the antibiotic treatment can be tailored for maximum effectiveness against this organism.

! The antibiotic treatment of bacterial meningitis must be started immediately after the lumbar puncture, without waiting, e. g., for a CT or MRI to be performed (if these or other tests are planned). The elapsed time between the clinical presentation and the beginning of treatment is the most important prognostic factor!

Therapy

- ▶ **Antibiotic therapy**
- ▶ **Other therapeutic approach**

Antibiotic therapy

- ▶ **Choice for antibiotic**
- ▶ **Duration of antibiotic therapy**

Choice for initial antibiotic

The initial choice of therapy should be based on the antibiotic susceptibilities of *S. pneumoniae*, *N. meningococci*, and *H. influenzae*.

cefotaxime (200mg/kg.d) or

ceftriaxone (100mg/kg.d),

combined with **vancomycin** (60mg/kg.d).

Patients allergic to β -lactam should be treated with **chloramphenicol** (100mg/kg.d).

Choice When pathogen identified

- ▶ *S. pneumoniae*, Penicillin 200-400(10^3 U/kg.d)
- ▶ *N. meningococci*, Penicillin 200-400(10^3 U/kg.d)
- ▶ *H. influenzae* Ampicillin 200mg/kg.d

Duration of antibiotic therapy

- ▶ *N. meningococci* 7-10days
- ▶ *S.pneumoniae* and *H.influenzae* meningitis
10-14days
- ▶ *staphylococcus aureus* and *E. coli* >21days

Other therapeutic approach

(1) Intensive Care

(2) Treatment for high fever, seizure and infective shock.

► high fever: physical defervesce and antipyretic

► Seizure:

Luminal (phenobarbital) 8-10mg/kg.t im. and

Valium (diazepam) 0.3mg/kg.t iv.

phenytoin

(3) Treatment for increased ICP and preventing cerebral herniation: 20% mannitol 5-10ml/kg.t

(4) Corticosteroids Clinical data support the use of intravenous dexamethason, 0.6mg/kg.d, for 3-5 days.



Tip:

meningococcal infection

Early complications: Waterhouse–Friderichsen syndrome

Brain oedema with secondary brainstem syndrome

Meningococcal meningitis

Meningococcal meningitis, a bacterial form of meningitis, is a serious infection of the meninges that affects the brain membrane. It can cause severe brain damage and is fatal in 50% of cases if untreated.

Meningococcal meningitis, caused by *Neisseria meningitidis* bacteria, is of particular importance due to its potential to cause large epidemics. Twelve types of *N.*

meningitidis, called serogroups, have been identified, six of which (A, B, C, W, X and Y) can cause epidemics.

The disease can affect anyone of any age, but mainly affects babies, preschool children and young people.

The largest burden of meningococcal disease occurs in an **area of sub-Saharan Africa** known as the meningitis belt

Transmission

Neisseria meningitidis only infects humans; there is no animal reservoir. The bacteria are transmitted from person-to-person through droplets of respiratory or throat secretions from carriers. Smoking, close and prolonged contact – such as kissing, sneezing or coughing on someone, or living in close quarters with a carrier – facilitates the spread of the disease

The bacteria can be carried in the throat and sometimes overwhelms the body's defences allowing the bacteria to spread through the bloodstream to the brain.

- **Incubation period:** 1-5 days
- **Acute onset:** shivering, fever (39-40 C), increasing intensity headache with nausea and repetitive vomiting, delirium, agitation, seizures, impaired consciousness
- **Early meningeal signs (first hours)**
- **In severe cases:** CN involved (III,VI and rare –VII,VIII)
- **Early involvement of brain tissue -encephalitis**
(seizures, paresis, hyperkinesis, ataxia, nistagmus etc.)

Diagnostics:

-Clinical picture

-CSF analysis

Diff. Diagnosis:

-Other meningitis

-Subarachnoid hemorrhage

Treatment

Meningococcal disease is potentially fatal and should always be viewed as a medical emergency. Appropriate antibiotic treatment must be started as soon as possible, ideally after the lumbar puncture has been carried out if such a puncture can be performed immediately. If treatment is started prior to the lumbar puncture it may be difficult to grow the bacteria from the spinal fluid and confirm the diagnosis. However confirmation of the diagnosis should not delay treatment.

A range of antibiotics can treat the infection, including penicillin, ampicillin and ceftriaxone. Under epidemic conditions in Africa in areas with limited health infrastructure and resources, ceftriaxone is the drug of choice.

Prevention

1. Vaccination

There are three types of vaccines available:

- Polysaccharide vaccines are used during a response to outbreaks
- Conjugate vaccines are used in prevention (into routine immunization schedules and preventive campaigns) and outbreak response:

- Protein based vaccine, against *N. meningitidis B*. It has been introduced into the routine immunization schedule

2. Chemoprophylaxis

Antibiotic prophylaxis for close contacts, when given promptly, decreases the risk of transmission.

Ciprofloxacin antibiotic is the antibiotic of choice, and ceftriaxone an alternative.

Waterhouse–Friderichsen syndrome

adrenal gland failure due to bleeding into the adrenal glands.

The bacterial infection leads to massive hemorrhage into one or (usually) both adrenal glands. It is characterized by overwhelming bacterial infection meningococcemia leading to massive blood invasion, organ failure, coma, low blood pressure and shock, disseminated intravascular coagulation(DIC) with wide spread purpura, rapidly developing adrenocortical insufficiency and death.

Answer of Purulent meningitis from another source

Purulent Meningitis

Outline

- ▶ a common infectious disease in the central neural system (CNS)
- ▶ with a high rate of acute complication and risk of chronic neurological morbidity(1/3).

Etiology (external cause)

The children in different age are susceptible to different bacteria

- ▶ The common bacteria(2/3) :meningococci, hemophilus influenzae, and pneumococci
- ▶ <2 months of age: enteric bacilli , staphylococci aureus
- ▶ 2 months to pre-adolescence: H. influenzae, meningococci, and pneumococci.
- ▶ Adolescence : meningococci and pneumococci

Etiology (internal cause)

The immunological and anatomical defect :

- ▶ The congenital immunological deficient;
- ▶ The acquired immunodepression : cortisone
→opportunistic organisms;
- ▶ Structural abnormalities: spinal meningocele;
- ▶ Therapeutic intervention: LP;

Clinical Manifestation

onset two patterns:

- ▶ sudden onset with rapidly progressive manifestations of shock, purpura, disseminated intravascular coagulation (DIC) and reduced levels of consciousness
- ▶ preceded by several days of upper respiratory tract or gastrointestinal symptoms, followed by nonspecific signs of CNS infection such as increasing **lethargy** and **irritability**.

Clinical Manifestation

Symptoms and signs

- ▶ **Nonspecific findings**
- ▶ **Acute Cerebral dysfunction**
- ▶ **Meningeal irritation**
- ▶ **Increased ICP (intracranial pressure)**

Nonspecific findings

- ▶ **fever** (present in 90-95%),
- ▶ **anorexia and poor feeding,**
- ▶ symptoms of upper respiratory tract infection, **headache**
- ▶ **myalgias, arthralgias,**
- ▶ **tachycardia, hypotension and shock**
- ▶ various **cutaneous signs**, such as **petechiae, purpura, macular rash.**

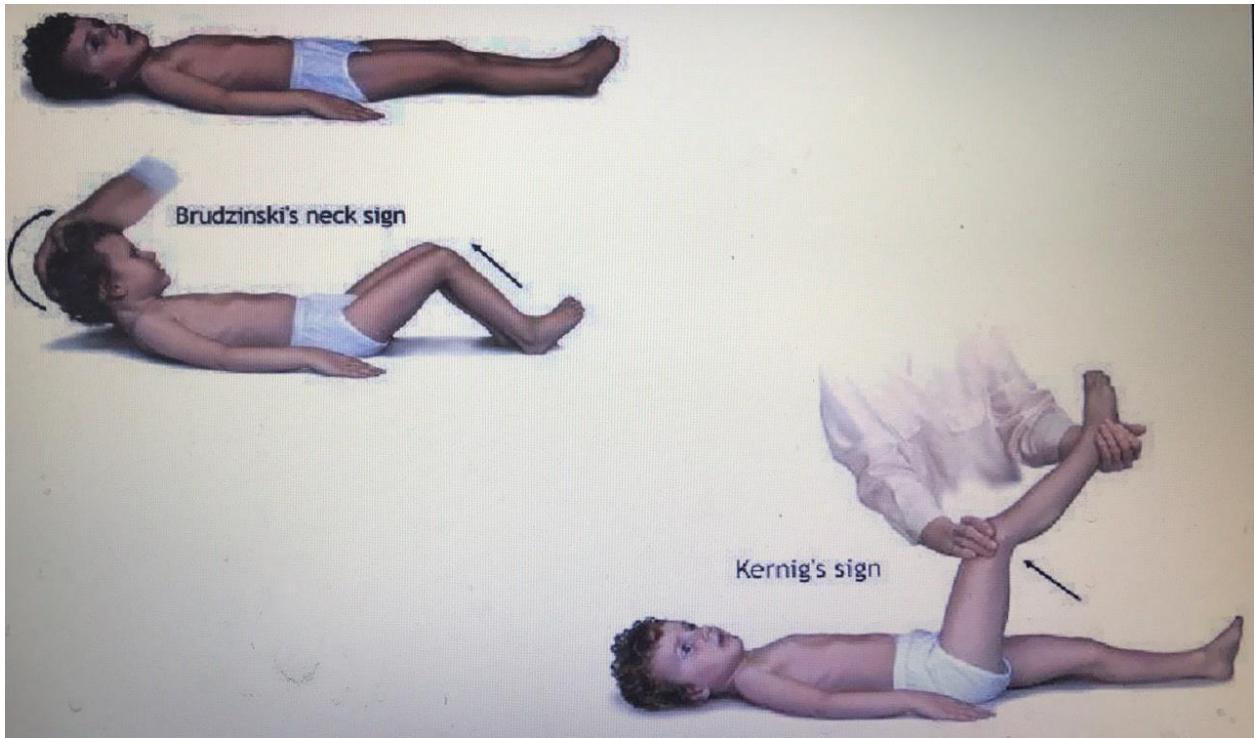
Acute Cerebral dysfunction

- ▶ **lethargy, irritability,**
- ▶ Alterations of mental status and a reduced level of consciousness include **stupor**, and **coma**.
- ▶ **Seizures** (20-30%) due to cerebritis, infarction, or electrolyte disturbances.
- ▶ **Focal neurological signs : limb paralysis.**
- ▶ **Cranial neuropathies** usually involve in cranial nerves of II, III, VI, VII, VIII.

Meningeal irritation

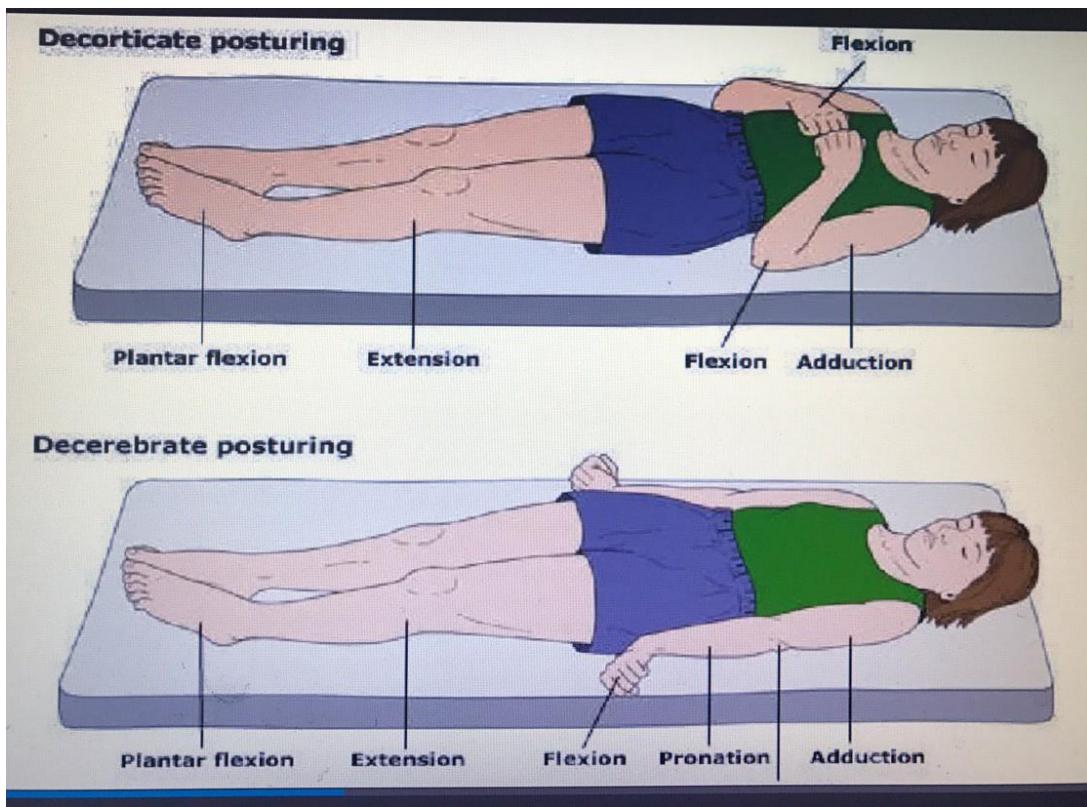
- ▶ **back pain,**
- ▶ **positive Kernig sign and Brudzinski sign.**
- ▶ **nuchal rigidity,**

In some children, particularly in those younger than 12-18 mo, Kernig and Brudzinski signs may not be evident with meningitis.



Increased ICP

- ▶ **headache, emesis,**
- ▶ **bulging fontanel or diastasis (widening) of the sutures,**
- ▶ **papilledema,**
- ▶ **hypertension with bradycardia,**
- ▶ **apnea or hyperventilation,**
- ▶ **decorticate or decerebrate posturing, coma, or signs of herniation.**



Laboratory Findings

- ▶ Peripheral blood check
- ▶ Lumbar Puncture
- ▶ CT or MRI

Laboratory Findings

► Peripheral blood check :

Leukocytosis of $20-40 \times 10^9/L$ usually occur in patients with meningitis, with the increase primarily of neutrophil, reaching above 80%.

Laboratory Findings

Lumbar Puncture:

- The CSF pressure usually be high,
- The CSF looks like turbidity.
- The CSF leukocyte count greater than $200-400/mm^3$ and reveals a neutrophilic predominance(75-95%).
- Elevated protein, and reduced glucose and chloride concentrations.
- The Gram stain is positive (70-90%).
- CSF cultures are the evidences for the pathogens of meningitis.

Laboratory Findings

CT or MRI :

- ▶ **High signal along subdural and piamater**
- ▶ **ventriculitis**
- ▶ **empyema**

Complication

- ▶ **Subdural effusion**
- ▶ **SIADH** (syndrome of inappropriate secretion of anti-diuretic hormone)
→hydrocephalus , hyponatremia →seizures
- ▶ **Ventriculitis**
- ▶ **Hydrocephalus**
- ▶ **Other: seizures, increased ICP, cranial nerve palsies, stroke, herniation.**

Diagnosis

- ▶ Nonspecific findings, acute cerebric dysfunction, and meningeal irritation
- ▶ Lumbar puncture should always be considered in any infant who has a fever with neurological signs and symptoms.
- ▶ confirmed by analysis of the CSF, which reveals microorganisms on Gram stain and culture, a neutrophilic pleocytosis, elevated protein, and reduced glucose concentrations.
- ▶ Determining the specific microorganisms

Contraindications for an immediate LP :

- (1) evidence of increased ICP;
- (2) severe cardiopulmonary compromise, such as shock;
- (3) infection of the skin overlying the site of the LP.

Differential Diagnosis

- ▶ CNS or non-CNS
 - Severe pneumonia, electrolytic disturbance**
- ▶ Infectious disease or noninfectious
 - Multiple sclerosis
- ▶ Different pathogen
 - viral vs bacterial, various bacteria

60. Serous meningitis. Enteroviral meningitis, mumps meningitis.

- Viral meningitis is inflammation of the leptomeninges as a manifestation of CNS infection. Viral meningitis syn. aseptic meningitis.
- In uncomplicated viral meningitis, the clinical course is usually self-limited, with complete recovery in 7-10 days. However, when the viral pathogen causes a more involved meningoencephalitis or meningomyelitis, the course can be significantly more protracted.
- Partially untreated bacterial meningitis in particular can present similarly to viral meningitis-devastating outcomes if misdiagnosed.

Causes

- Enteroviruses account for more than 85% of all cases of viral meningitis.
- Herpes family viruses: HSV-1, HSV-2, VZV, EBV, CMV, and human herpesvirus 6 collectively cause approximately 4% of cases of viral meningitis, with HSV-2 being the most common offender.
- Lymphocytic choriomeningitis virus
- Adenovirus
- Measles
- Mumps

Pathophysiology

- Viral pathogens may gain access to the CNS via 2 main routes: hematogenous or neural.
- Multiple host defenses (local and systemic immune responses, skin and mucosal barriers, and the blood-brain barrier) prevent viral inoculum from causing clinically significant infection.

Mortality/Morbidity

- Excluding the neonatal period, the mortality rate associated with viral meningitis is less than 1%; the morbidity rate is also low.

Viral Meningitis-Clinical Manifestations

- The classically taught triad of meningitis consists of fever, nuchal rigidity, and altered mental status.
- Upon presentation, most patients report fever, headache, irritability, nausea, vomiting, stiff neck, rash, or fatigue within the past 18-36 hours.
- Nuchal rigidity or other signs of meningeal irritation (Brudzinski or Kernig sign) may be seen in more than half of patients but is generally less severe than in bacterial meningitis. Pediatric patients, especially neonates, tend not to exhibit nuchal rigidity on examination.
- The neonate may exhibit hypotonia, irritability, and poor feeding.
- Some viruses cause rapid onset of the above symptoms, while others manifest as nonspecific viral prodromes, such as malaise, myalgia, and upper respiratory symptoms. In many cases, symptoms have a biphasic pattern; the nonspecific flu-like symptoms and low-grade fever precede neurologic symptoms by approximately 48 hours. With the onset of neck stiffness and headache, the fever usually returns.

- Headache is common and is characteristically severe.
- Photophobia is relatively common but may be mild. Phonophobia may also be present.
- Seizures occur occasionally and are usually from the fever, although the involvement of brain parenchyma (encephalitis) should be considered.
- Other signs of specific viral infection can aid in diagnosis:
- Pharyngitis and pleurodynia in enteroviral infections
- Skin manifestations, such as zoster eruption in VZV, maculopapular rash from measles and enteroviruses, vesicular eruption by herpes simplex, and herpangina in coxsackievirus A infections.
- Pharyngitis, lymphadenopathy, and splenomegaly suggest Epstein-Barr virus infection.
- Immunodeficiency and pneumonia should suggest adenovirus, cytomegalovirus , or HIV as the causative agent.
- Parotitis and orchitis can occur with mumps
- Gastroenteritis and rash occur with most enteroviral infections.

Viral Meningitis-Diagnosis

- CT scan usually is performed prior to LP to rule out intracranial hematoma, mass effect, or obstructive hydrocephalus.
- PCR testing for viral DNA.
- The following are some CSF characteristics used to support the diagnosis of viral meningitis:
- Cells: Pleocytosis with WBC counts in the range of 50 to $>1000 \times 10^9/L$ of blood has been reported in viral meningitis. Mononuclear cell predominance is the rule, but PMNs may comprise the majority of cells in the first 12-24 hours; the cell count usually is then dominated by lymphocytes in the classic CSF pattern of viral meningitis. This helps to distinguish viral from bacterial meningitis, which has a much higher cell count and a predominance of PMNs in the cell differential; this is by no means an absolute rule, however.

- Protein: CSF protein level usually is only slightly elevated, but can range from being normal to as high as 200 mg/dL.
- Glucose: Normal in most cases.
- Culture, Gram stain, and acid-fast stain

Viral Meningitis-Treatment

- Mostly supportive-Rest, hydration, antipyretics, and pain or anti-inflammatory medications. The most important decision is whether to initiate antimicrobial therapy empirically for bacterial meningitis while waiting for the cause to be identified. Patients with signs and symptoms of meningoencephalitis should receive acyclovir early to possibly curtail HSV encephalitis.
- Enteroviruses and HSV are both capable of causing viral septic shock in newborns and infants. In these young patients, broad-spectrum antibacterial coverage and acyclovir should be instituted as soon as the diagnosis is suspected.

- Seizures should be treated immediately with IV anticonvulsants such as lorazepam, phenytoin, midazolam, or a barbiturate.

Serous meningitis

Aseptic meningitis

Aseptic meningitis is the inflammation of the meninges, a membrane covering the brain and spinal cord in patients whose **cerebral spinal fluid test result is negative with routine bacterial cultures**. The testing for both meningitis and aseptic meningitis is mostly **the same**. A cerebrospinal fluid sample is taken by lumbar puncture and is tested **for leukocyte levels** to determine if there is an infection .The symptoms are the same for both meningitis and aseptic meningitis but the severity of the symptoms and the treatment can depend on the certain cause.

The most common cause of aseptic meningitis is by viral infection. Other causes may include side-effects from drugs and connective tissue disorders.

Signs and symptoms

Aseptic meningitis is a disease that can depend on the patient's age. A variety of patients notice a change in body temperatures (higher than normal temperatures 38-40°C), marked with the possibility of vomiting, headaches, firm neck pain, and even anorexia.

In younger patients, like babies,

possibility of hepatic necrosis and myocarditis. (In serious cases, a multiple organ failure can also signal aseptic meningitis and oftentimes) in babies, seizures and focal neurological deficits can be early symptoms of aseptic meningitis. In fact, in newborns, the mortality rate is 70%. The next set of age group, like children, have similar but varying symptoms of sore throat, rashes, and diarrhea. In adults, symptoms and the harshness of them tend to be less in duration. Additionally, the probability of developing aseptic meningitis increases when patients have a case of mumps or herpes.

Symptoms of meningitis caused by an acute viral infection last between one and two weeks. When aseptic meningitis is caused by cytomegalovirus 20 percent of individuals face mortality or morbidity. If left untreated it can affect an individual's hearing and learning abilities.

Causes

The most common cause of aseptic meningitis is a viral infection, specifically by enteroviruses. Other viruses that may cause aseptic meningitis are varicella zoster virus, herpes, and mumps. Other causes may include mycobacteria, fungi, spirochetes, and complications from HIV. Side effects of certain drugs such as nonsteroidal anti-inflammatory drugs (NSAIDs), antibiotics (e.g., trimethoprim-sulfamethoxazole or amoxicillin), and antiepileptic drugs can also cause aseptic meningitis.

- Viral meningitis

- Enterovirus (EV) caused meningitis.
- Human immunodeficiency virus (HIV)
- Mumps meningoencephalitis
- Mosquito carried viruses of the flavivirus family. Saint Louis encephalitis (SLE) and West Nile virus (WNV) are the most typical.
- Specific types of Herpes can result in aseptic meningitis. These are (HSV)-1, (HSV)-2, varicella-zoster virus, and (HHV6).
- **Bacteria**
 - Lyme disease
 - Syphilis
 - Leptospirosis
- **Fungi**
 - Cryptococcal infection
 - Coccidioidal infection
- **Drug-induced aseptic meningitis (DIAM)**
 - Irritation of the meninges from drugs administered directly to the spinal canal or subarachnoid space. The hypersensitivity to the drug results in an immune response.
- **Autoimmune diseases**
 - Systemic lupus erythematosus.
- **Cancer-caused aseptic meningitis such as neoplastic meningitis**
 - This affects about 5% of all cancer cases, with a predominance in leukemias.
- **Neurosarcoidosis**

Diagnosis

the medical history of the individual and family, physical examination, and laboratory test

One common medical test used when diagnosing aseptic meningitis is **lumbar puncture**. Is analyzed by microscope

examination or by culture to distinguish between bacterial and aseptic meningitis. Samples of CSF undergo cell count, Gram stains, and viral cultures, and polymerase chain reaction (PCR). Polymerase chain reaction has increased the ability of clinicians to detect viruses such as enterovirus, cytomegalovirus, and herpes virus in the CSF. Other laboratory tests include blood, urine, and stool collection. also computed tomographic (CT) scan or magnetic resonance imaging (MRI), these tests help observe calcifications or abscesses.

Treatment

If CSF levels are irregular among individuals, they will undergo hospitalization where they receive antiviral therapy. If aseptic meningitis was caused by herpes simplex virus (HSV), the individual will receive acyclovir, an antiviral drug. If infants are diagnosed, medical professionals will order regular check-ins for hearing and learning disabilities.

From another source:

Viral Meningitis

Usually acute or subacuteonset, self-limiting and lasts for 4-10 days

Causes

- 85% enterovirus,HIV,HSV2,EBV,varicella zoster virus,mumps,lymphocytic choriomeningitis(LCV)

Presents

- Intense headache,fever,malaise,myalgia,photophobia

Clinically

- Nuchal rigidity, look for focal signs, less likely to have altered mental status

Lumbar puncture

- Negative gram stain, WBC 10-1000/mm³, normal glucose, protein up to 150mg/dL
- Send for nested PCR (two loci primers)
- Can send for direct viral cultures (only 6% return)

Blood

- HIV test in 2-3 months

Treatment

- supportive

Etiotropictherapy:

- Tiloron(RNA & DNA viruses)
- Interferons
- Intravenous immunoglobulins

In case of Bacterial complications –antibiotics

Desintoxication and symptomatic therapy

Intracranial hypertension –dehydration

Neuroprotection, metabolic therapy, vitamins.

61. Tuberculous meningitis.

Brain and spine Tuberculosis

Tuberculous meningitis: tuberculosis infection of the meninges. It is the most common form of CNS tuberculosis.

- Causative agent: *Mycobacterium tuberculosis* is an aerobic gram-positive rod.
- CNS tuberculosis most commonly occurs in those infected with HIV and those from South East Asia where TB is still endemic

Frequency

The World Health Organization (WHO) estimates that one third of the world's population is infected by *M. tuberculosis*.

Clinical Manifestations

Tuberculous meningitis progresses rapidly with headache, fever, tremor, and cranial nerve deficits (esp CN-IV palsy). Focal neurological deficits may include monoplegia, hemiplegia, aphasia, and tetraparesis. Vasculitis with resultant thrombosis and hemorrhagic infarction may develop in vessels. Visual findings-Papilledema is the most common visual effect of TBM.

- The clinical picture in primary spinal meningitis is often characterized by myelopathy, with radicular pain and progressive paraplegia or tetraplegia.

Brain and spine Tuberculosis-Pathophysiology

Diagnosis

- Diagnosis of TB meningitis is made by analysing cerebrospinal fluid collected by lumbar puncture. A spider-web clot in the collected CSF is characteristic of TB meningitis, but is a rare finding.
- Culture for *M. tuberculosis* takes 2 weeks. More than half of cases of TB meningitis cannot be confirmed microbiologically, and these patients are treated on the basis of clinical suspicion only before the diagnosis is confirmed.
- PCR

Brain and spine Tuberculosis

Treatment

- The treatment of TB meningitis is isoniazid, rifampicin, pyrazinamide and ethambutol for two months, followed by isoniazid and rifampicin alone for a further ten months. Corticosteroids are always used in the first six weeks of treatment when cerebral edema, subarachnoid block, or both occur.
- **Treatment must be started as soon as there is a reasonable suspicion of the diagnosis. Treatment must not be delayed while waiting for confirmation of the diagnosis.**

Mortality/Morbidity

- Death follows within weeks in untreated CNS tuberculosis. Mortality is greatest at the extremes of age 20% at <5 years of age and 60% at >50 years or if illness has been present more than 2 months (80%)

Tuberculous meningitis -Clinical Manifestations

- Tuberculous meningitis progresses rapidly with headache, fever, meningismus, and cranial nerve deficits (esp CN-IV palsy). Focal cerebral or cerebellar deficits are followed by altered sensorium and coma.

Visual findings

- Papilledema is the most common visual effect of TBM. In children, papilledema may progress to primary optic atrophy and blindness resulting from direct involvement of the optic nerves and chiasma by basal exudates (ie, opticochiasmatic arachnoiditis).
- In adults, papilledema may progress more commonly to secondary optic atrophy, provided the patient survives long enough.
- Apart from papilledema, fundus examination occasionally reveals a retinal tuberculoma or a small grayish-white choroidal nodule, highly suggestive of TB. These lesions are believed to be more common in miliary TB than in other forms of TB.

Tuberculous meningitis-Clinical Manifestations

Neurologic findings

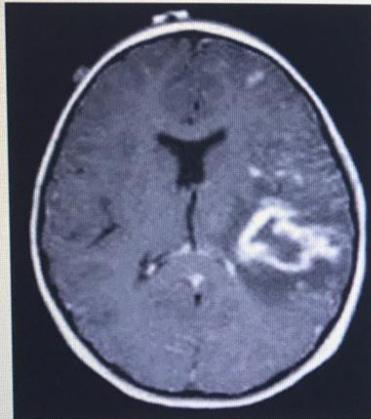
- Cranial neuropathies, most often involving CN VI, may be noted. CNs III, IV, VII, and, less commonly, CNs II, VIII, X, XI, and XII, also may be affected.
- Focal neurological deficits may include monoplegia, hemiplegia, aphasia, and tetraparesis.
- Tremor is the most common movement disorder seen in the course of TBM. In a smaller percentage of patients, abnormal movements, including choreoathetosis and hemiballismus, have been observed, more so in children than adults. In addition, myoclonus and cerebellar dysfunction have been observed. Deep vascular lesions are more common among patients with movement disorders.
- Vasculitis with resultant thrombosis and hemorrhagic infarction may develop in vessels that traverse the basilar or spinal exudate or lie within the brain substance. Eventually, fibrinoid degeneration within small arteries and veins produces aneurysms, multiple thrombi, and focal hemorrhages, alone or in combination.

Tuberculous meningitis-Pathophysiology

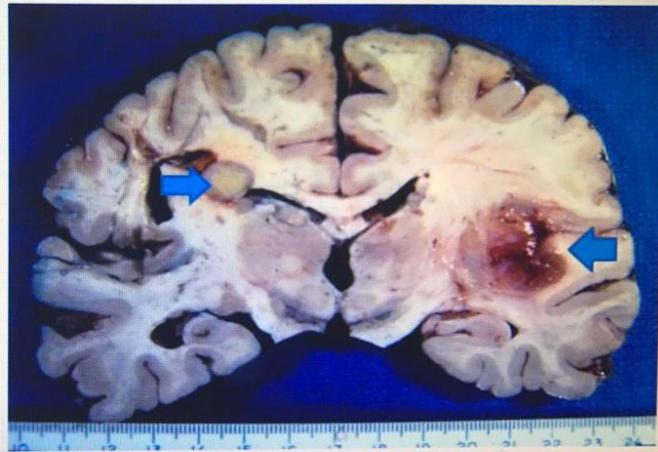
- Many of the symptoms, signs, and sequelae of tuberculous meningitis (TBM) are the result of an immunologically directed inflammatory reaction to the infection. The development of TBM is a 2-step process. *M. tuberculosis* bacilli enter the host by droplet inhalation, the initial point of infection being the alveolar macrophages. Localized infection escalates within the lungs, with dissemination to the regional lymph nodes to produce the primary complex. During this stage, a short but significant bacteremia is present that can seed tubercle bacilli to other organs in the body.
- In persons who develop TBM, bacilli seed to the meninges or brain parenchyma, resulting in the formation of small subpial or subependymal foci of metastatic caseous lesions. These are termed Rich foci, after the original pathologic studies of Rich and McCordick.

- The second step in the development of TBM is an increase in size of a Rich focus until it ruptures into the subarachnoid space. The location of the expanding tubercle (ie, Rich focus) determines the type of CNS involvement. Tubercles rupturing into the subarachnoid space cause meningitis. Those deeper in the brain or spinal cord parenchyma cause tuberculomas or abscesses. While an abscess or hematoma can rupture into the ventricle, a Rich focus does not.
- A thick gelatinous exudate infiltrates the cortical or meningeal blood vessels, producing inflammation, obstruction, or infarction. Basal meningitis accounts for the frequent dysfunction of cranial nerves (CNs) III, VI, and VII, eventually leading to obstructive hydrocephalus from obstruction of basilar cisterns. Subsequent neurological pathology is produced by 3 general processes: adhesion formation, obliterative vasculitis, and encephalitis or myelitis.

Tuberculomas are conglomerate caseous foci within the substance of the brain. Under conditions of poor host resistance, this process may result in focal areas of cerebritis or frank abscess formation, but the usual course is coalescence of caseous foci and fibrous encapsulation (ie, tuberculoma).



T1 w MRI IMAGE



Tuberculoma is the round gray mass in the left corpus callosum. The red meninges on the right are consistent with irritation and probable meningeal reaction to tuberculosis.

Tuberculous spinal meningitis-Clinical manifestations

- Tuberculous spinal meningitis may manifest as an acute, subacute, or chronic form.
- The clinical picture in primary spinal meningitis is often characterized by myelopathy, with progressive ascending paralysis, eventually resulting in basal meningitis and associated sequelae.
- In some cases with acute onset, in addition to variable constitutional symptoms, patients develop acute paraplegia with sensory deficits and urinary retention. The clinical picture often mimics transverse myelitis or Guillain-Barré syndrome.
- The subacute form is often dominated by myeloradiculopathy, with radicular pain and progressive paraplegia or tetraplegia.
- A less virulent chronic form might mimic a very slowly progressive spinal cord compression or a nonspecific arachnoiditis.
- The dorsal cord seems to be affected most commonly, followed by the lumbar and the cervical regions.

Tuberculous spinal meningitis -Pathophysiology

- In the tuberculous process, the spinal meninges may be involved, owing to the spread of infection from intracranial meningitis, primary spinal meningitis in isolation as a result of a tuberculous focus on the surface of the cord rupturing into the subarachnoid space, or transdural extension of infection from caries of the spine.
- Pathologically, a gross granulomatous exudate fills the subarachnoid space and extends over several segments. Vasculitis involving arteries and veins occurs, sometimes resulting in ischemic spinal cord infarction.

- The earliest lesion in the vertebra is invariably due to hematogenous spread, often involving the body of the vertebra near an intervertebral disk. The intervertebral disk is almost always involved with the spread of the disease to the adjacent vertebra and eventually along the anterior or posterior longitudinal ligaments or through the end plate. Soon, a cold abscess develops, either as a paraspinal abscess in the dorsal and lumbar regions or as a retropharyngeal abscess in the cervical region. As the disease progresses, increasing decalcification and erosion result in progressive collapse of the bone and destruction of intervertebral disks, involving as many as 3-10 vertebrae in one lesion, resulting in kyphosis. The abscess may rupture intraspinally, resulting in primary spinal meningitis, hyperplastic peripachymeningitis, intraspinal abscess, or tuberculoma.

Tuberculous spondylitis-Clinical manifestations

- Tuberculous spondylitis is also known as Pott disease or spinal caries.
- In regions where the disease is endemic, such as Asia and Africa, this condition still accounts for 30-50% of all cases of compressive myelopathy resulting in paraplegia. Spinal TB also accounts for approximately 50% of all bone and joint TB cases.
- In the lumbar region, tuberculous spondylitis may result in a psoas abscess that often calcifies.
- It usually runs a subacute or a chronic course, with back pain and fever and variable neurological deficits.
- Spondylitis can also result in various symptoms, including local and radicular pain, limb motor and sensory loss, and sphincter disturbances.
- Eventually, complete spinal cord compression with paraplegia, the most dreaded complication, may supervene.

Diagnosis of TB meningitis is made by analyzing cerebrospinal fluid collected by lumbar puncture. The CSF usually has a high protein, low glucose and a raised number of lymphocytes. Acid-fast bacilli are sometimes seen on a CSF smear, but more commonly, M. tuberculosis is grown in culture. A spider-web clot in the collected CSF is characteristic of TB meningitis, but is a rare finding.

- Culture for M tuberculosis; takes 2 weeks (50-80% of known cases of TBM yield positive results). More than half of cases of TB meningitis cannot be confirmed microbiologically, and these patients are treated on the basis of clinical suspicion only before the diagnosis is confirmed..
- Polymerase chain reaction (PCR): Results imply that PCR can provide a rapid and reliable diagnosis of TBM, although false-negative results potentially occur in samples containing very few organisms.
- Differential diagnosis: Cryptococcal antigen and herpes antigen testing; Syphilis serology

Brain and spine Tuberculosis-Treatment

- The treatment of TB meningitis is isoniazid, rifampicin, pyrazinamide and ethambutol for two months, followed by isoniazid and rifampicin alone for a further ten months. Corticosteroids are always used in the first six weeks of treatment when cerebral edema, subarachnoid block, or both occur.
- Treatment must be started as soon as there is a reasonable suspicion of the diagnosis. Treatment must not be delayed while waiting for confirmation of the diagnosis.

Surgical Care

- Hydrocephalus occurs as a complication in about a third of patients with TB meningitis and will require placement of a ventriculoperitoneal shunt.
- Mortality/Morbidity
- Death follows within weeks in untreated CNS tuberculosis. Mortality is greatest at the extremes of age 20% at <5 years of age and 60% at >50 years or if illness has been present more than 2 months (80%)

From another source:

Tuberculous meningitis (TBM)

- Typically a chronic meningitis
- May occur years after primary infection, and in many cases there is no history of prior TB
- Meningeal signs take several weeks to develop
- Associated with non-specific symptoms: vague headache, malaise, anorexia, sleep disturbances, fatigue, sweating etc.
- TB often affects base of the brain (damage to the brainstem, arteries to the brain, block of the CSF flow –causing hydrocephalus)

Diagnostics:

- Anamnesis (contacts with TB patients, previous TB history)
- Clinical picture
- CSF analysis

Treatment:

- First 2 month: 4 out of 5 –rifampin, isoniazid, pyrazinamide, ethambutol, streptomycin.
- Correction of the scheme after determination of sensitivity to antibiotics
- After 2-3 months 2 medications are left (usually Rifampin and Isoniaside)
- Minimal treatment duration: 6-12 months

Control of toxic effects:

- VIII CN (Etambutol)
- Hepatotoxicity
- Peripheral neuropathies–pyridoxine, polyvitamins, tioctic acid

For prevention of adhesive pachymeningitis and hydrocephalus – oral corticosteroids

62. Secondary purulent meningitis. Otogenic meningitis. Complications.

Classification of meningitis and meningoencephalitis							
I. A. Primary B. Secondary							
II. According to etiology							
1 bacterial	3 rickettsial	5 viral	7 protozoal				
2 fungal	4 helminth	6 spirochetal	8 mixed				
III. By the nature of the CSF							
1- purulent		2- serous					
IV. According to the degree of gravity							
1- light	2- middle	3- heavy					
V. Кечиши буйича							
A By the character:							
1. smooth							
2. not smooth	a) with complications						
	b) with secondary infection						
	c) with the aggravation of chronic diseases						
B. According to duration:							
1. Acute	2. Prolonged	3. chronic					

The main likvorological signs in meningitis

Signs	Normal likvor	Serous meningitis	Purulent meningitis
Color and clarity	Colorless, clear	Colorless, clear	Whitish or green, muddy
Pressure(in mm of water .up.)	130-180	200-250	increased
The speed of leakage from the needle	40-60	60-90	Jet
Cytosis(number of cells in 1mm)	2-8	20-800	1000-1500 and more
Cytogram: Lymphocytes % Neutrophils%	80-85 3-5	80-100 0-20	0-60 40-100
Protein	0,25- 0,33	0,33-1,0	0,66- 16,0
Reaction "Pandi"	«neg»	+ (++)	+++(+++)

Secondary purulent meningitis and meningoencephalitis are distinguished:

Otogenic
Septic
Streptococcal
Esherihiozik
Etc

In otogenic meningitis bacteria^{Slide}
penetrate the meninges by

- By contact
- By vascular way

Distinguishes:

Labirintogenic meningitis

Timpanogenic meningitis

In the otogenic purulent meningitis
affects

- For the first the membranes of the base brain
- Then the posterior fossa
- Rigidity of the muscles of the back of the head
- Early and sudden expressed than symptoms Kernig and Brudzinsk

Likvorologikal changes in otogenic meningitis

SlidePia

- SF is turbid
- Cytosis is neutrophil
- Protein is increased

OTOGENIC MENINGITIS

- Inflammation of all brain shells otologic origin .
- Can be serous or purulent

OTOGENIC MENINGITIS

Meningeal symptoms

- Appears due to tension of spinal nerve roots to the dura mater. They are:
- **Rigidity of neck muscles** - impossibility to get by a patient's chin to physician's thumb located on jugular fossa;
- **Kerning** - impossibility to unbend a leg in a knee joint after simultaneous bending of knee and coccyx joints;
- **Brudzinsky upper** – when patient try to get by the chin to jugular fossa the patient's knees are reduced to a stomach;
- **Brudzinsky middle** – during pressing by physician's fists on patient's symphysis the knees are reduced to the stomach;
- **Brudzinsky lower** - during checking Kerning symptom - other leg are reduced to the stomach.

OTOGENIC MENINGITIS

II. Brain symptoms

- Appears due to raising of spinal liquor pressure:
- Deterioration of consciousness (sopor, stupor or coma).
- Severe headache,
- Nausea, vomiting,
- hyper aesthesia (light phobia, noise phobia, raising of tactile and temperature sensitivity).
- Forced position of the patient: „ Raising cock” pose;

OTOGENIC MENINGITIS

The common – intoxication symptoms

- Fever, temperature curve - constant;
- Tachycardia;
- Tachypnoe;
- Blood hypertension
- Hypo or anuria.

Additional methods of inspection

- **Blood analysis** : neutrophilic leucocytosis, accelerated SOE is expressed;
- **Spinal puncture** - liquid implies under the increased pressure, it is muddy, and also contains too much cells elements, protein, sometimes - bacteria, contents of sugar and chlorides are reduced.
- **Bacteriological research of ear pus and spinal canal liquid** shows the agent and its antibiotics resistance (to choose the adequate antibiotic therapy).
- **X-ray of temporal bone**.
- **Computer tomography and magnet -nuclear resonance research** - detection of cavities, filled with exudates in temporal bone.

Treatment

- Urgent hospitalization in ENT -department, where urgent surgical procedure will performed (**extended radical mastoidectomy**). From usual mastoidectomy it differs by maximum wide disclosure of a dura mater of middle and posterior cranial fosses, and available mastoid cells. Postauricular wound is not closed to give possibility of cleaning of operational cavity and introduction medicines in operational wound.
- The intensive conservative therapy includes: treatment by antibiotics in maximum dozes, that penetrate through blood-brain barrier (including in spinal canal), massive des intoxication, de hydratation, hormones.

63. Para-infectious encephalitis (measles, chickenpox, rubella, mumps).

The answer of this question is written in the lecture (viral part)

1. <http://etest.bsmu.by/mod/page/view.php?id=151550&forceview=1>

64. Encephalitis, classification.

Encephalitis

Inflammation of the brain parenchyma, usually caused by **viruses**. Direct–infective organism directly causes the encephalitis at the time of infection

Immune-mediated: causing an allergic or post infectious encephalomyelitis

Encephalitis -Etiology

The causative organisms are often not identified and viral aetiology is presumed.

The most common:

- Echovirus
- Coxsackie virus
- Mumps virus

-Herpes Simplex (causes severe viral encephalitis and is often associated with hemorrhagic changes within the temporal lobes and progressive personality changes and memory loss)

More rare:

- Adenovirus
- Varicella-Zoster
- Measles
- Influenza

In the Far East, the most common cause is **Japanese B arbovirus**, which causes epidemic encephalitis with high mortality

Clinical picture

Most of agents will cause a mild self-limiting disease with **headache and drowsiness**, but some will cause severe illness. Herpes simplex type 1 accounts for most of the severe cases.

Non-specific features: headache, pyrexia, myalgia, malaise

Meningism: headache, photophobia, neck stiffness, and lymphocytic pleocytosis in the CSF

Parenchymal involvement (depends whether the inflammation is diffuse or local): confusion, dysphasia, hemiparesis, seizures, ataxia, cranial nerve palsies, autonomic dysfunction.

Virus specific features: e.g. parotid swelling in mumps.

Encephalitis -Diagnostics

Definitive diagnosis is often difficult in viral encephalitis due to the number of viral agents.

Investigations:

CT-scan(shows non-specific cerebral oedema)

MRI(subtle inflammatory changes are much more apparent and appearance of herpes simplex encephalitis in one or both temporal lobes are very distinctive)

EEG(non-specific slow wave changes and/or periodic complexes, findings in temporofrontal regions suggests herpes simplex aetiology)

Viral serology/PCR of blood and CSF

Lumbar puncture

Brain biopsy (seldom performed)

Treatment

Herpes simplex –Acyclovir IV

- Supportive treatment for comatose patients
- Anticonvulsants for seizures
- Control of cerebral oedema
- Corticosteroids in the first week (discussed)

65. Tick borne encephalitis.

Tick-borne encephalitis (TBE) is a viral infectious disease involving the **central nervous system**. The disease most often manifests as meningitis, encephalitis, or meningoencephalitis. TBE is posing a concerning health challenge to Europe.

The tick-borne encephalitis virus is known to infect a range of hosts including **ruminants, birds, rodents, carnivores, horses, and humans**. The disease can also be spread from animals to humans, with ruminants and dogs providing the principal source of infection for humans.

Signs and symptoms

the median incubation period is 8 days (range, 4–28 days) after tick bite. **Non specific symptoms of mild fever, malaise, headache, nausea, vomiting and myalgias may be present as first manifestation of the disease and spontaneously resolve within 1 week.** After another week the patient may develop neurological symptoms. The virus can result in long neurological symptoms, **infecting the brain (encephalitis), the meninges (meningitis) or both (meningoencephalitis)**.

Cause

TBE is caused by **tick-borne encephalitis virus**, Russia and Europe report about **5,000–7,000 human cases annually**.

Transmission

It is transmitted by the bite of several species of infected woodland ticks, including *Ixodes scapularis*, *I. ricinus* and *I. persulcatus*, or (rarely) through the non-pasteurized milk of infected cows.

Diagnosis

Detection of specific IgM and IgG antibodies in patients sera combined with typical clinical signs, is the principal method for diagnosis. In more complicated situations, e.g. after vaccination, testing for presence of antibodies in cerebrospinal fluid may be necessary .

PCR (Polymerase Chain Reaction) method is rarely used, since TBE virus RNA is most often not present in patient sera or cerebrospinal fluid at the time of clinical symptoms.

Prevention includes non-specific (tick-bite prevention, tick checks) and **specific prophylaxis in the form of a vaccination**.

Treatment

there is no specific drug therapy for TBE. Symptomatic brain damage requires hospitalization and supportive care based on syndrome severity. Anti-inflammatory drugs, such as corticosteroids, may be considered under specific circumstances for symptomatic relief. Tracheal intubation and respiratory support may be necessary.

Epidemiology

The disease is most common in Central and Eastern Europe, and Northern Asia.

66. HIV, neurological complications.

Human immunodeficiency virus (HIV)

Infection with the retrovirus HIV can cause neurological involvement either directly or via opportunistic infections.

Neurological involvement develops in 80% of patients.

Both the central and peripheral nervous system can be affected

HIV (central nervous system)

Primary HIV infection:

- HIV encephalopathy (AIDS Dementia): subacute or chronic onset
- HIV myelopathy – could be reversible (during seroconversion) or irreversible (later form)
- Acute atypical meningitis: self-limiting, occurs during seroconversion, with cranial neuropathies and pyramidal signs

Opportunistic Infection:

- CNS toxoplasmosis
- Cryptococcal meningitis
- Progressive multifocal leucoencephalopathy(PML)

- Cytomegalovirus (CMV): retinitis, myelitis, sacral radiculitis, encephalitis
- Herpes simplex type 2 myelitis
- Varicella-Zoster: radiculitis and encephalitis
- Other: Candida, Aspergillus, Coccidioides.

Neoplasia:

- Primary CNS lymphoma: presents as mass lesions
- Other malignancies: spread from systemic non-Hodgkin's lymphoma, metastasis from Kaposi's sarcoma etc.

HIV (peripheral nervous system)

Peripheral neuropathy

- Distal symmetrical polyneuropathy
- Chronic inflammatory demyelinating polyradiculoneuropathy
- Guillain-Barre syndrome
- Multifocal neuropathy

Myopathies:

- Polymyosites
- Type-2 fibremuscle atrophy
- Drug-induced myopathy (zidovudine)



For better understanding use this link

1. <https://slideplayer.com/slide/6659377/>

67. Herpetic encephalitis

Viral Encephalitis

Herpes Simplex Encephalitis

Herpes simplex encephalitis is a serious infectious condition caused by the **herpes simplex virus, type I**.

Pathogenesis.

This viral disease is characterized by **hemorrhagic–necrotic inflammation of the basal portions of the frontal and temporal lobes**, combined with **severe cerebral edema**. The inflammatory foci are found in both hemispheres, but one is usually more strongly affected than the other.

Clinical manifestations.

After a *nonspecific prodromal phase* with **fever, headache, and other general symptoms**, the disease presents with **progressive impairment of consciousness, epileptic seizures** (usually of complex partial type, with or without secondary generalization, because of the temporal localization of the disease), and **focal neurological and neuropsychological deficits**, particularly **impairment of memory and orientation**. **Aphasia** and **hemiplegia** may ensue.

Diagnostic evaluation.

CSF examination reveals up to 500 cells/mm³, mainly lymphocytes but also granulocytes; the CSF is sometimes bloody or xanthochromic.

Viral DNA can be identified in the CSF by the polymerase chain reaction (**PCR**) in the first few days of illness and, two weeks later, **IgG specific for herpes simplex virus can be identified in the CSF as well**. The **EEG**, in addition

to nonspecific changes, may reveal characteristic focal

findings over one or both temporal lobes. The **CT scan** is usually normal at first but, within a few days, reveals temporal or frontal hypodense areas, which may contain foci of hemorrhage . **MRI** may reveal corresponding signal changes even earlier.

Treatment.

Acyclovir is given intravenously. *Corticosteroids* are given to combat cerebral edema and *antiepileptic drugs* to prevent seizures.

If there is good reason to suspect herpes simplex encephalitis (progressive impairment of consciousness, aphasia, epileptic seizures [particularly of the complex partial type], an inflammatory CSF profile, focal EEG abnormalities), intravenous acyclovir therapy must be started immediately.

Herpes zoster encephalitis is accompanied by a segmental vesicular rash in the territory of a peripheral nerve (cranial nerve). CSF examination reveals lymphocytic pleocytosis up to 200 cells/mm³. The disease may appear in particularly severe form after a generalized herpes zoster infection.

68. Brain abscess.

Brain abscesses are produced by **focal infection of the brain parenchyma** leading to tissue destruction and pus formation. They can be solitary or multiple. A special form is **focal encephalitis**, in which systemic sepsis or the embolization of infectious material into the central nervous system gives rise

to **multilocular, disseminated microabscesses**

Brain Abscess

Etiology.

Brain abscesses are caused by one or more pathogens, mainly streptococci and staphylococci and, less commonly, *Pseudomonas*, *Actinomyces*, and fungi. Like the organisms that cause bacterial meningitis, these pathogens can reach the brain through **local extension**

of infection (especially mastoiditis, sinusitis, and otitis), **hematogenous dissemination** from a distant infectious focus (usually pulmonary infections or endocarditis), or **direct contamination** (open brain injury).

Immunocompromised patients are at increased risk.

Clinical manifestations.

A large **brain abscess** exerts

mass effect and typically causes **fever**, leukocytosis, and **rapidly progressive intracranial hypertension**. Marked perifocal edema generally adds to the mass effect. Alternatively, there may be a **subdural empyema** between the dura mater and the arachnoid, or an **epidural abscess** between the dura mater and the inner table of the skull. These processes usually arise as a **complication of sinusitis or otitis**, less commonly after trauma. Fever, headache, and meningism, accompanied by neurological deficits, are their **clinical hallmarks**. The course of subdural empyema is often fulminant and lifethreatening, that of epidural abscess usually more protracted.

Diagnostic evaluation.

The diagnosis is suspected on the basis of the **typical clinical findings** (intracranial hypertension with papilledema, impaired consciousness, sometimes hemiparesis or other focal neurological deficits), **accompanying signs of infection** (fever, elevated laboratory parameters of inflammation), and **relevant aspects of the past medical history** (such as traumatic brain injuries, known lung or heart disease, and immune suppression or diseases of the immune system).

CSF examination may reveal inflammatory changes (predominantly

granulocytic pleocytosis, elevation of total protein), and the *CT or MRI scan* shows a ring-shaped area of contrast enhancement (abscess wall) surrounding the hypodense interior of the abscess.

Treatment.

Operative removal of the abscess is the preferred form of treatment in most patients, accompanied by *antibiotic therapy*, which is initiated before surgery

and continued thereafter for *at least six weeks*.

69. Myelitis.

Myelitis -Classification

Onset:

- Acute
- Subacute
- Chronic

Prevalence:

- limited*.The spinal cord is localized only one pathological focus;
- diffuse*.Inflammation covers the whole spinal cord;
- multipolar*.In certain areas the body is several centers (more than two);
- transverse myelitis*

Mechanism of development:

- primary myelitis
- secondary myelitis

Etiology:

- radiation;
- bacterial;
- post-vaccination;
- virul;

- traumatic;
- idiopathic;
- toxic.

Myelitis –Clinical picture

Intoxication(fewer, nausea, myalgia)

Cerebral symptoms (due to increased intracranial pressure)

Focal symptoms (motor, sensory, autonomous dysfunction)

Diagnostics

- History of the disease
 - Clinical picture
 - CSF analysis
 - Immunology/Bacteriological test
 - CT/MRI
 - Electroneuromyography
- Treatment
- Etiological
 - Anti-inflammatory
 - Symptomatic
 - Prevention of complications

From another source

What is Myelitis?

Myelitis involves the **infection or the inflammation** of the white matter or gray matter of the spinal cord which is a part of the central nervous system that acts as a bridge between the brain and the rest of the body.

- During an inflammatory response in the spinal cord, the myelin and axon may get damage which can cause symptoms such as **paralysis and sensory loss**.
- Myelitis can be divided into certain types depended on the area of the cause of inflammation.
- Myelitis mainly occurs in narrow region that can go and spread to other broad regions.

Symptoms:

- **Pain in your lower back**
- Weakness or paralysis in your legs or arms
- Sensitivity to touch to the point where slight fingertip pressure causes pain
- Numbness or a pins-and-needles feeling in your toes, feet, or legs
- Problems controlling your bladder or bowels
- Muscle spasms
- **Fever**
- Loss of appetite
- From age of 10 to 19 and 30 to 39 the risk is higher for **multiple sclerosis**, myelitis is common in women than men.

Types of myelitis:

- Poliomyelitis-Disease caused by infection in gray matter which shows symptoms of muscle paralysis and weakness.
- Transverse myelitis-It occurs when both side of one section of the **spinal cord** gets damaged. Many times the covering of the nerve cells, the myelin gets damaged.
- Meningococcal Myelitis (or meningomyelitis): lesions occurring in the region of meninges and the spinal cord
- Though there are three types of myelitis the most common myelitis is the transverse myelitis and doctors, and people often refer to any inflammatory

Causes of myelitis:

- Virus or other infection-Recent infection in **respiratory tract or gastrointestinal tract** can cause myelitis. Mostly myelitis occurs after infection is over.
- Viruses that can infect the spinal cord directly are herpes viruses, including the one that causes shingles and chickenpox (zoster), enteroviruses, and West Nile virus.
- Other viruses may trigger an autoimmune reaction without directly infecting the spinal cord.
- Parasites may infect the spinal cord in a rare condition, and some bacteria such as that of Lyme disease can cause a painful inflammation in the nerve roots of the spinal cord.
- Multiple sclerosis-It is a disease when the body's immune system affects the spinal cord cells. Transverse myelitis can be the first sign of multiple sclerosis.

continue: Causes of Myelitis-

- **Neuromyelitis optica** (Devic's disease) -It is a condition that causes inflammation and myelin loss around the spinal cord and the nerve in the eye.
- Transverse myelitis can be associated with neuromyelitis optica, which can affect both side of the body and can lead to eye problems even temporary vision loss.
- However people may not have any symptoms of Neuromyelitis optica, only can show **symptoms of Myelitis**.
- Autoimmune disease-It can cause myelitis in some people. As, antibody affection the spinal cord can sometime lead to transverse myelitis.
- Vaccinations -using vaccines for infectious diseases including **hepatitis B**, measles-mumps-rubella and diphtheria-tetanus vaccines have occasionally been associated as a possible trigger.

Treatments:

- Plasma exchange therapy: Patient not responding to steroids may be given therapy of plasma exchange. Here the plasma in **bone marrow** is replaced with another fluid.
- Antiviral medication. Patients having myelitis from viral infection can be treated with medicines against virus.
- Therapy against complications:
- Pain medication. Chronic pain is a common complication of transverse myelitis. Medications that may lessen muscle pain include common pain relievers, such as acetaminophen (Tylenol, others), ibuprofen (Advil, Motrin IB, others) and naproxen sodium (Aleve.)
- Nerve pain may be treated with antidepressant drugs, such as sertraline (Zoloft), and anticonvulsant drugs, such as gabapentin (Neurontin, Gralise) or pregabalin

Diagnosis of myelitis:

- **Magnetic resonance imaging (MRI)** -It uses a magnetic field and radio waves to create 3-D images of soft tissues. An MRI can show inflammation of the spinal cord, and other potential causes of the symptoms, including abnormalities affecting the spinal cord or blood vessels.
- Lumbar puncture (spinal tap) -In this technique a needle is used to draw small amount of cerebrospinal fluid (CSF), the protective fluid surrounding the spinal cord and brain. People with transverse myelitis, show abnormally high level of **white blood cells** in CSF or immune system proteins that indicate inflammation.
- Blood tests-Antibody blood tests can be done which checks for antibodies associated with neuro-myelitis optica.

Complications of Myelitis-

- **Magnetic resonance imaging (MRI)** -It uses a magnetic field and radio waves to create 3-D images of soft tissues. An MRI can show inflammation of the spinal cord, and other potential causes of the symptoms, including abnormalities affecting the spinal cord or blood vessels.
- Lumbar puncture (spinal tap) -In this technique a needle is used to draw small amount of cerebrospinal fluid (CSF), the protective fluid surrounding the spinal cord and brain. People with transverse myelitis, show abnormally high level of **white blood cells** in CSF or immune system proteins that indicate inflammation.
- Blood tests-Antibody blood tests can be done which checks for antibodies associated with neuromyelitis optica.

70. Poliomyelitis.

Poliomyelitis

Caused by **polio virus** (picornavirus from the group of enteroviruses).

Incidence of primary infection is extremely low since the immunisation start in 1957.

Remains endemic in the tropics, occurring especially in **late summer and autumn**

Poliomyelitis is spread by faeco-oral route and then enters the bloodstream, causing a viraemia.

Neurological involvement occurs only in some patients and targets the anterior horn cells of the spinal cord and the motor nuclei of the brain stem.

Incubation period 10-14 days.

Poliomyelitis – Clinical features

Asymptomatic (95%): with resultant immunity.

Abortive poliomyelitis (4-5%): a self-limiting illness with gastrointestinal and mild upper respiratory symptoms and pyrexia.

Non-paralytic poliomyelitis (0,5%): features of abortive poliomyelitis with meningism. Recovery is complete.

Paralytic poliomyelitis (0,1%)

Paralytic poliomyelitis

Initially features of abortive poliomyelitis, which subside and recur with meningism and myalgia.

There is subsequent asymmetrical paralysis with no sensory involvement
Respiratory failure due to respiratory muscles involvement.

The lower limb or limbs are most commonly affected, especially in children

Bulbar symptoms can occur with cranial nerves involvement

Diagnostics

• History of the disease

• Epidemiological data

• Neurological assessment

• CSF

• Paired serology (to show rising titre)

Treatment

Isolation and immunization of the contacts

- Careful nursing, preventing pressure ulcers
- Physiotherapy
- Fluid and electrolyte replacement
- In case of respiratory failure –artificial ventilation

71. Spinal epiduritis, abscess.

Spinal Abscesses

Spinal abscesses are most often *epidural*, less often subdural, and only rarely *intramedullary*. The most common pathogen is *Staphylococcus aureus*, which reaches the spinal canal from a site of primary infection outside it by way of the bloodstream (hematogenous spread). The typical clinical features are *general signs of infection* (fever, elevated erythrocyte sedimentation rate, leukocytosis, chills in some cases), *pain*, and *neurological deficits* referable to the spinal nerve roots or spinal cord, depending on the specific anatomic situation. Spinal abscesses usually require prompt *surgical treatment*,

followed by weeks of high-dose antibiotics

vertical extent; they usually arise in the patient's second or third decade.

Diagnostic evaluation.

Syringomyelia can be diagnosed from its typical symptoms and physical findings; the characteristic picture is of a *dissociated sensory deficit* combined with *trophic disturbances*. The diagnosis must then be confirmed with *neuroimaging*, specifically *MRI*.

Clinical course.

Syringomyelia is usually slowly progressive.

Treatment.

Neurosurgical methods are occasionally successful. The options include the *Puusepp operation* (opening the posterior aspect of a large syrinx into the subarachnoid space), drainage of the syrinx with a shunt, or operation of an accompanying Arnold–Chiari

malformation at the craniocervical junction.

From another source:

1. <https://www.slideshare.net/AdeWijaya5/spinal-epidural-abscess>
2. <https://www.slideshare.net/yimsmart90/epidural-abscess>

72. Neurosyphilis.

Syphilis

Caused by *T. pallidum*, transmission is almost invariably through sexual contact.

The natural history of untreated infection is divided into three stages. Neurological involvement occurs on the 3rd stage, which is typically many years after initial infection. Neurosyphilis occurs in less than 10% of all untreated cases

Syphilis -Diagnostics

Serology:

-Venereal disease reference laboratory

-*T. pallidum* haemagglutination assay

-Fluorescent treponema Antibodies absorbed

Test could be negative on the serum, but positive on CSF in neurosyphilis.

Syphilis -Treatment

Parenteral Penicillin for 2-3 weeks.

Established neurological disease can be arrested but may not be reversed

Jarisch-Herxheimer reactions may occur following treatment and high-dose steroid cover is often given with the penicillin

Neurosyphilis syndromes

- Asymptomatic Neurosyphilis
- Meningitis
- Tabes dorsalis
- General paralysis of the insane

- 
- 73. Acute disseminating encephalomyelitis.
 - 74. Multiple sclerosis.
 - 75. Acute inflammatory demyelinating polyradiculoneuropathies Guillain-Barre.



The answers of question 73 to 75 are on the site

1. <http://etest.bsmu.by/mod/page/view.php?id=151705>
2. <http://etest.bsmu.by/mod/page/view.php?id=151706&forceview=1>

- 76. Amyotrophic lateral sclerosis.

Amyotrophic Lateral Sclerosis

Motor Neuron Disease Terminology

Lower motor neuron ← → Upper motor neuron

Progressive
Muscular
Atrophy

Amyotrophic
Lateral
Sclerosis

Primary
Lateral
Sclerosis

Amyotrophic Lateral Sclerosis Pathology

- ✖ Degeneration and death of motor nerves
 - Upper Motor Neuron
 - within brain/spinal cord
 - Lower Motor Neurons
 - leaves brain (stem)/spinal cord
- ✖ Relatively spared
 - Eye movements and bowel/bladder function

Amyotrophic Lateral Sclerosis

Epidemiology

- ✖ Etiology – unknown
- ✖ Average age of onset mid-50's
- ✖ Mode of transmission
 - Sporadic – 90-95%
 - Familial – 5-10% (autosomal dominant)

Amyotrophic Lateral Sclerosis

Epidemiology

- ✖ Male : Female – 3:2
- ✖ U.S. Prevalence: 30,000
- ✖ Incidence 1-2.5 / 100,000
- ✖ Isolated areas of increased incidence
 - Kii peninsula of Japan
 - Chamorro natives of Guam

Amyotrophic Lateral Sclerosis

Clinical Presentation

- ✖ Lower motor neuron signs
 - Weakness, muscle wasting, hyporeflexia, muscle cramps, fasciculations
- ✖ Upper motor neuron signs
 - Spasticity, hyperreflexia, weakness

Amyotrophic Lateral Sclerosis

Clinical Presentation

- ✖ Asymmetric Weakness – most common
- ✖ Onset single limb or bulbar
- ✖ Local spread then regional spread
 - Bulbar, cervical, thoracic, lumbosacral
- ✖ Fasciculations

Amyotrophic Lateral Sclerosis Diagnosis

- ✖ Prominent upper and lower neuron signs with a progressive course without significant sensory or sphincter abnormalities
- ✖ Laboratory investigation to search for a more treatable condition

Amyotrophic Lateral Sclerosis Clinical Signs and Symptoms

- | | |
|-------------------|-----------------------|
| ✖ Weakness | ✖ Spasticity |
| ✖ Hyporeflexia | ✖ Hyperreflexia |
| ✖ Pain and cramps | ✖ Babinski's sign |
| ✖ Fasciculations | ✖ Emotional Liability |
| ✖ Wasting | |

Amyotrophic Lateral Sclerosis

Atypical Features

- ✗ Dementia - < 5 %
- ✗ Sensory loss – atypical
- ✗ 25% complain of paresthesias
- ✗ Oculomotor dysfunction
- ✗ Bowel or bladder dysfunction

Amyotrophic Lateral Sclerosis

Diagnosis

- ✗ Two experienced Neurologists

Laboratory Studies

- No study to prove or disprove
- Look for an alternate diagnosis

Amyotrophic Lateral Sclerosis Laboratory Studies

- ✖ Nerve conduction studies
 - assess for demyelinating vs. axonal involvement
- ✖ Electromyography
 - confirm ALS
 - myopathy

Amyotrophic Lateral Sclerosis Laboratory Studies

- ✖ MRI cervical spine
 - Cervical Spondylosis with cord compression
 - Herniated disc
 - Syrinx

Amyotrophic Lateral Sclerosis Laboratory Studies

- ✗ ESR – inflammatory/malignancy
- ✗ SPEP – monoclonal gammopathy
- ✗ TSH – hyperthyroidism
- ✗ B₁₂ – combined systems degeneration
- ✗ Calcium/PTH - hyperparathyroidism

Amyotrophic Lateral Sclerosis Prognosis

- ✗ Variable – difficult to predict in an individual patient
- ✗ 50% live 3-4 or more years
- ✗ 20% live 5 or more years
- ✗ 10% live 10 or more years
- ✗ Occasional patients live 20 years

Amyotrophic Lateral Sclerosis Treatment

- ✖ Rilutek
- ✖ 2 large clinical trials
 - Bulbar onset
 - Entire population
- ✖ Endpoint
 - Death
 - Ventilator dependence

Amyotrophic Lateral Sclerosis Treatment

- ✖ Bulbar onset
 - Prolonged survival
 - Improved muscle strength
- ✖ Entire population
 - Prolonged survival
 - No effect on decline in muscle strength
- ✖ Prolonged survival an average of 2-3 months

Amyotrophic Lateral Sclerosis

Rilutek 50 mg po bid

- ✖ Hepatotoxicity
 - Serum transaminase levels
 - Check every month x 3
 - Then every 3 months x 3 for the first year
- ✖ Adverse effects
 - Neutropenia
 - Nausea/vomiting

Amyotrophic Lateral Sclerosis

Rilutek 50 mg po bid

- ✖ Reasons for not taking the drug
 - Expense
 - Minimal benefit
 - Unwillingness to take a medication that would prolong life

Amyotrophic Lateral Sclerosis Management

- ✗ weakness
- ✗ fatigue
- ✗ nutrition
- ✗ dysphagia
- ✗ feeding tube
- ✗ dysarthria
- ✗ communication
- ✗ spasticity
- ✗ cramps
- ✗ pain
- ✗ depression
- ✗ anxiety
- ✗ breathing
- ✗ end-of -life

Amyotrophic Lateral Sclerosis Multidisciplinary Approach to Care

- ✗ Neurologist
- ✗ Clinical/research nurse
- ✗ Dietician
- ✗ Speech/swallowing therapist
- ✗ Family/caregivers
- ✗ Psychologists
- ✗ Physical therapist
- ✗ Occupational therapist
- ✗ Social worker
- ✗ GI physician
- ✗ Support organizations
- ✗ Homehealth/hospice
- ✗ Pulmonologist

77. Syringomyelia.

Syringomyelia, a condition coming under the general heading of **spinal dysraphism**, is sometimes seen in combination with other congenital defects such as the **Arnold–Chiari syndrome or spina bifida**. It has several different causes; it can

be classified into **primary syringomyelia** and **symptomatic forms due to** (for example) hemorrhage, infection, or a tumor.

Syringomyelia is defined by the **pathological finding** of a tubelike or cleftlike **cavity (syrinx)** within the spinal **cord**, often lined by ependyma, and usually extending over several spinal segments. The cavity may reach all the way up to the medulla, or even the midbrain (*syringobulbia, syringomesencephaly*). Mere widening of the central canal of the spinal cord is called *hydromyelia*. Clinical manifestations of syringomyelia depend on the

location of the syrinx within the spinal cord and on its regulation (with negative polio titers). Others use it for a syndrome with *progressive worsening of residual weakness* occurring decades after the acute illness. Before this problem can be ascribed to the earlier **polio infection**, other possible causes of weakness must be ruled out, e. g., compression of the spinal cord or spinal nerve roots because of secondary degenerative disease of the spine.

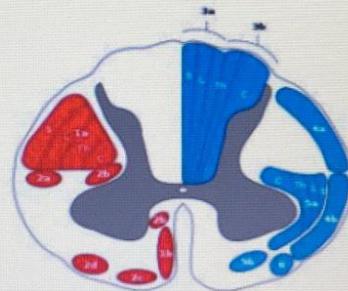
From another source:

Definition

- **Syringomyelia:** presence of fluid-filled cavity within the spinal cord. The cavity lies outside of the central canal and not lined by ependymal cells.
- **Communicating syringomyelia:** have direct connection with the fourth ventricle.
- **Noncommunicating syringomyelia:** no connection to the fourth ventricle.
- **Hydromyelia:** dilatation of the central canal of the spinal cord (cavity lined by ependymal cells)

Download

Cervical Spinal Cord Anatomy



Motor and descending (efferent) pathways (left, red)	Sensory and ascending (afferent) pathways (right, blue)
1. Pyramidal Tracts	3. Dorsal Column Medial Lemniscus System
1a. Lateral corticospinal tract	3a. Gracile fasciculus
1b. Anterior corticospinal tract	3b. Cuneate fasciculus
2. Extrapyramidal Tracts	4. Spinocerebellar Tracts
2a. Rubrospinal tract	4a. Posterior spinocerebellar tract
2b. Reticulospinal tract	4b. Anterior spinocerebellar tract
2b. Vestibulospinal tract	5. Anterolateral System
2d. Olivospinal tract	5a. Lateral spinothalamic tract
	5b. Anterior spinothalamic tract
3. Sensory Abbreviations:	6. Spino-olivary fibers
Si. Sacral; Li. Lumbar	
Tb. Thoracic; Ci. Cervical	



Clinical Presentation

- Gradual onset of symptoms usually between 25-40 years of age.
- Pain, numbness of the hands, stiffness of the legs, vertigo, neurogenic arthropathy.
- Horner syndrome is common.

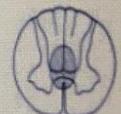
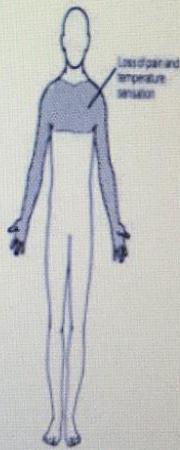


Figure 5-17. Syringomyelia involving the cervicothoracic portion of the spinal cord.

Clinical Presentation

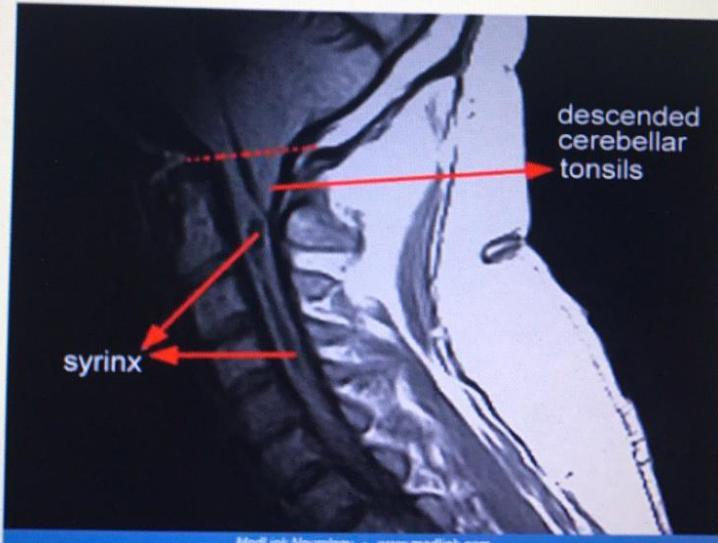


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Causes

- Familial often associated with Chiari type 1 malformation (basilar impression)
- Secondary to basal/spinal arachnoiditis after meningitis, SAH, TB, trauma, repetitive deceleration in skydivers, CSF leak after brachial plexus avulsion, reaction to contrast, spinal anesthesia.
- Secondary to intra or extramedullary spinal tumors.
- Secondary to cervical spondylosis.
- Secondary to injury to cord from trauma, radiation, infarction, hemorrhage, infection, transverse myelitis.
- Idiopathic

Type 1 Chiari malformation and syringomyelia, T1 MRI





Proposed Pathophysiology

- Multiple proposed mechanisms however not fully understood.
- Initial syrinx formation may be due to infarction of tissue with subsequent cyst formation.
- Hydrodynamic theory-cyst formation due to fluid flowing into the central canal from the forth ventricle secondary respiratory and arterial pressure waves.
- Cyst formation secondary to extracellular fluid edema after spinal cord injury.
- Changes in subarachnoid space compliance, CSF fluid or pressure dynamics from arachnoid adhesions, spinal stenosis, or cord compression could lead to syrinx enlargement.



Diagnosis

- MRI is the imaging modality of choice.
- Often demonstrates that syringes are asymmetrical, loculated, or multiple, and this information can aid operative planning.
- Phase-contrast cine MRI can localize subarachnoid space obstruction and demonstrate normalization of CSF flow following surgery.

Management

- No clear standard approach and area of controversy.
- 17-50% of patients have no progression of syrinx or symptoms over a 10 year follow-up period.
- Spontaneous resolution has been observed in children and adults.
- Surgery reported as effective in Chiari-related syringomyelia with 90% chance of long-term stabilization or improvement.
- Postoperative relief of headaches and neck pain observed in 83% of children with Chiari I malformation.

Management Continued

- Persistent dysesthetic pain can occur despite improvement or collapse of syrinx on post-op MRI.
- Aim of surgery is to reconstruct normal CSF pathways via bony decompression and duraplasty in cases associated with Chiari malformation.
- Shunting procedures can be used in cases not associated with Chiari malformation although the best type of shunting procedure is unknown (syringoperitoneal, syringopleural, syringosubarachnoid).



Management Continued

- With shunt insertion 12-53% of pts will improve, 10-56% will be unchanged, and 12-32% will get worse.
- Shunt failure rate is about 50% in most series.
- Operative complication rates range from 6-18%.
- In cases secondary to trauma or arachnoiditis post-op recurrence of adhesions is the rule.
- Fetal spinal cord stem cells have been transplanted in patients without deterioration however without evidence of growth into surrounding spinal cord.



78. Epilepsy. Differential diagnosis of seizures.

Management of single seizure.

79. Epilepsy: conservative and surgical treatment.

80. Epileptic status. Management of epileptic status.



The answers of question 78 to 80 are on the site (2

Lectures)

1. <http://etest.bsmu.by/mod/page/view.php?id=151707>

2. <http://etest.bsmu.by/mod/page/view.php?id=151708>



- 81. Migraine.**
- 82. Tension type headache.**
- 83. Trigeminal neuralgia. Conservative and surgical treatment.**
- 84. Facial nerve neuropathy.**



The answers of question 81 to 84 are on the site (3 Lectures)

- 1. <http://etest.bsmu.by/mod/page/view.php?id=151710>**
- 2. <http://etest.bsmu.by/mod/page/view.php?id=151711&forceview=1>**
- 3. <http://etest.bsmu.by/mod/page/view.php?id=151712&forceview=1>**

85. Classification of peripheral nervous system disorders. Compression-ischemic (entrapment) neuropathies.

- 1. <https://slideplayer.com/slide/7060554/>**

There are many types of peripheral neuropathy, often brought on by diabetes; genetic predispositions (hereditary causes); exposure to toxic chemicals, alcoholism, malnutrition, inflammation (infectious or autoimmune), injury, and nerve compression; and by taking certain medications such as those used to treat cancer and HIV/AIDS. Mayo Clinic researchers are working toward earlier and better diagnosis and treatment, and ultimately prevention of these debilitating nerve diseases. **The following are the major types of peripheral neuropathy:**

- **Neuropathy** is the disease of the nervous system in which there is a disturbance in the function of a nerve or particular group of nerves. The three major forms of nerve damage are: **peripheral neuropathy, autonomic neuropathy, and mononeuropathy**. The most common form is peripheral neuropathy, which mainly affects the feet and legs.

- **Sciatica** is pain, tingling, or numbness produced by an irritation of the sciatic nerve. Sciatica is a pain in the leg due to irritation of the sciatic nerve. Sciatica most commonly occurs when a branch of the sciatic nerve is compressed at the base of the spine.
- **Carpal tunnel syndrome** occurs when tendons in the wrist become inflamed after being aggravated. Tendons can become aggravated when the carpal (a tunnel of bones) and the ligaments in the wrist narrow, pinching nerves that reach the fingers and the muscle at the base of the thumb.
- **Polyneuropathy** is any illness that attacks numerous nerves in the body, sometimes causing weakness and/or pain. It tends to be a systemic problem that affects more than one nerve group at a time. Polyneuropathies are relatively symmetric, often affecting sensory, motor, and vasomotor fibers simultaneously.
- **Diabetic neuropathies** are neuropathic disorders that are associated with diabetes mellitus. These conditions usually result from diabetic microvascular injury involving small blood vessels that supply nerves (vasa nervorum).
- **Autonomic neuropathy** is a group of symptoms caused by damage to nerves supplying the internal body structures that regulate functions such as blood pressure, heart rate, bowel and bladder emptying, and digestion.
- **Postherpetic neuralgia** is pain that persists after an episode of shingles (herpes zoster) has resolved, resulting from damaged nerve fibers from the shingles.
- **Thoracic outlet syndrome** is a condition in which the nerves or vessels behind the collar bone (clavicle) become compressed or stretched, causing pain, weakness, or numbness in the arm on the same side. The thoracic outlet is an area at the top of the rib cage, between the neck and the chest. Several anatomical structures pass through this area, including the esophagus, trachea, and nerves and blood vessels that lead to the arm and neck region.

2. <https://www.slideshare.net/bikashnanda180/entrapment-neuropathies>

86. Classification of vertebrogenic disorders of the nervous system. Vertebrogenic chest pain?

87. Vertebrogenic cervical pain syndromes and radiculopathy.?

88. Lumbago, sciatica.

1. https://www.slideshare.net/drkmliau/low-back-pain-1683348?next_slideshow=1

2. <https://www.slideshare.net/Farshidmokhberi/sciatica-44352691>

89. Discogenic lumbo-sacral radiculopathy.

1. <https://www.slideshare.net/naveenrathor2/discogenic-lower-backache-by-dr-naveen-rathor>

90. Intercostal neuropathy. Herpes zoster.

What is intercostal neuralgia?

Intercostal neuralgia is neuropathic pain involving the intercostal nerves. These are the nerves that arise from the spinal cord, below the ribs.

Intercostal neuralgia tends to cause thoracic pain, which affects your chest wall and upper trunk.

What are the symptoms?

The main symptom of intercostal neuralgia is burning, sharp, or shooting pain. This pain may be felt:

- around the ribs
- in the upper chest
- in the upper back

Additional symptoms in these areas include:

- a squeezing pressure sensation that wraps around the chest from front to back
- tingling
- numbness

The pain might feel worse even when doing gentle physical activities, such as deep breathing or stretching. It might also intensify when you laugh, cough, or sneeze. Some people also notice referred pain in their shoulder blade or lower pelvis. Referred pain is pain that you feel in an area other than the affected one.

Intercostal neuralgia caused by the shingles virus ([postherpetic neuralgia](#)) can also make your skin itchy and extremely sensitive, even to clothing.

Symptoms of more severe cases of intercostal neuralgia include:

- involuntary muscle twitching
- loss of appetite
- paralysis
- muscle atrophy
- pain that feels like a lightning bolt

What causes it?

Intercostal neuralgia is caused by irritation, inflammation, or compression of your intercostal nerves, which are just below your ribs.

A number of things can cause this, including:

- trauma to your chest

- viral infections, such as [shingles](#)
- nerve entrapment or pressure
- injury from a surgical procedure that involved opening your chest to access your throat, lungs, heart, or diaphragm ([thoracotomy](#))

Sometimes, intercostal neuralgia doesn't have a clear cause. In this case, it's called idiopathic intercostal neuralgia.

How is it diagnosed?

Before diagnosing your intercostal neuralgia, your doctor will want to rule out any other causes of your pain. During a physical exam, they'll likely press the area between your ribs or ask you to take a deep breath. If either of these cause pain, you may have intercostal neuralgia.

Depending on your symptoms, you might also need a neurological exam to check for any problems with your nervous system. Your doctor might also use an [X-ray](#), [ultrasound](#), [CT scan](#), or MRI scan to look for any signs of injury.

How is it treated?

There are several options for relieving intercostal neuralgia, and many people find that a combination of treatments works best.

Over-the-counter options

Some over-the-counter topical treatments can provide temporary pain relief. These include:

- capsaicin creams or skin patches
- lidocaine gels or skin patches

Medication

Antidepressants are sometimes used to treat nerve-related pain. Common ones include:

- amitriptyline
 - desipramine (Norpramin)
 - duloxetine (Cymbalta)
 - imipramine (Tofranil)
 - nortriptyline (Aventyl, Pamelor)
 - venlafaxine
1. https://www.powershow.com/viewfl/44ad9e-ZjQ1N/Neurologic_Complications_of_Varicella-Zoster_Virus_Infection_powerpoint_ppt_presentation
 2. <https://www.slideshare.net/mohamedfazly31/herpes-zoster-50752503>

91. Traumatic lesions of peripheral nerves.

1. <https://emedicine.medscape.com/article/1172408-overview>

92. Polyneuropathy classification.

1. https://www.slideshare.net/rashim100/polyneuropathy?next_slideshow=1
2. <https://slideplayer.com/slide/4330711/>

93. Diabetic polyneuropathy.

1. <https://slideplayer.com/slide/4100216/>

94. Alcoholic polyneuropathy.

What is Alcoholic Neuropathy? (Definition/Background Information)

- Neuropathy is defined as a condition in which the **nerves that relay information between the body's extremities and brain become damaged**. Neuropathy associated with excessive alcohol use is termed Alcoholic Neuropathy
- Alcoholic Neuropathy, as the name suggests, affects those who consume excessive amounts of alcohol. Though any individual consuming excess alcohol is at risk, the majority of Alcoholic Neuropathy affected individuals **are men**
- Alcoholism and associated inadequate dietary nutrition are some known risk factors for the condition. Alcoholic Neuropathy can be caused through direct damage to the nerves by excessive and repeated alcohol use, nutritional inadequacy, and deficiency in thiamine
- Nerve damage in Alcoholic Neuropathy is felt as **numbness, pain, or tingling sensation in the arms and legs, as the brain struggles to communicate with the extremities**. Severe and irreversible damage can be observed in some cases, which could lead to complication such as pain in the affected areas, trouble urinating, impotence in men, etc.

Who gets Alcoholic Neuropathy? (Age and Sex Distribution)

- Alcoholic Neuropathy is a condition that is caused by **chronic alcohol abuse**; it is typically observed only in individuals who are alcoholic. The majority of these individuals (around 70%) are reported to be men

- Alcoholic Neuropathy could affect alcoholics of any age. As many as 50% of long-term alcohol users are likely to develop this condition
- All racial and ethnic groups are prone to this condition; this condition is observed worldwide

What are the Risk Factors for Alcoholic Neuropathy? (Predisposing Factors)

The risk factors for Alcoholic Neuropathy include:

- Excessive consumption of alcohol
- Inadequate nutrition, which is a common habit or feature associated with alcoholism

What are the Causes of Alcoholic Neuropathy? (Etiology)

The exact cause of Alcoholic Neuropathy is unclear.

- However, the direct toxicity of alcohol to the human nerves, as well as the poor nutritional habits prevalent among alcoholics is thought to contribute to the nerve damage
- Studies have shown that neither excessive alcohol use and normal nutrient levels or nutrient deficiency alone can cause Alcoholic Neuropathy
- The nutrient, thiamine that is acquired from one's diet, has been determined to play a critical role in proper nerve function

What are the Signs and Symptoms of Alcoholic Neuropathy?

The signs and symptoms of Alcoholic Neuropathy are:

- Numbness and tingling of the arms and legs
- Pain in the muscles, such as cramps or aches (muscle pain is usually more common in the legs than the arms)
- Difficulties with urination such as leaking urine and incomplete emptying of the bladder

- Constipation, diarrhea, even nausea and vomiting

How is Alcoholic Neuropathy Diagnosed?

Alcoholic Neuropathy is diagnosed using the following methods:

- Physical examination with study of (family) medical history including history of alcohol consumption
- Thorough neurological examination
- Nerve conduction studies
- Liver function test

The physician may also run tests to determine if minerals and nutrients in the blood are within adequate levels. Minerals and nutrients may not be absorbed by the body from one's diet, due to excessive consumption of alcohol.

What are the possible Complications of Alcoholic Neuropathy?

Complications due to Alcoholic Neuropathy could include:

- Disability in the affected areas
- Long-term pain and discomfort
- Injury to the extremities
- Impotence in men
- Trouble urinating

How is Alcoholic Neuropathy Treated?

The treatment measures for Alcoholic Neuropathy include:

- Complete cessation of alcohol consumption
- Administration of vitamin supplements
- Maintaining a healthy diet routine

How can Alcoholic Neuropathy be Prevented?

Alcoholic Neuropathy can be easily prevented by stopping alcohol consumption (or drinking very moderately) and maintaining a healthy diet.

What is the Prognosis of Alcoholic Neuropathy? (Outcomes/Resolutions)

- Depending on the patient's history with alcohol use and the extent to which the nerves are damaged, the existing nerve damage in Alcoholic Neuropathy can be permanent
- While this nerve damage is not life-threatening, it can seriously diminish one's quality of life

Alcoholic Neuropathy

- Chronic alcohol abuse leads to polyneuropathy
 - Calf pain is common
- Deficiency in thiamine due to alcoholism also causes neuropathy
 - Can lead to Wernicke-Korsakoff syndrome
 - Common presentation
 - Eye signs
 - Ataxia
 - Cognitive change
 - Delirium tremens
 - Hypothermia and hypotension

The diagram illustrates Thiamine Deficiency (Beriberi) with two main types: DRY BERIBERI and WET BERIBERI.

DRY BERIBERI: Common early manifestations include loss of reflexes in knees & feet, peristalsis, numbness of feet, emaciation, and foot drop. Aphasia may appear (Poor prognosis, vagus nerve involved).

WET BERIBERI: Symptoms include dyspnea, orthopnea, great weakness, edema, and Wernicke's Syndrome. Wernicke's Syndrome leads to ophthalmoplegia, confusion, coma, and death. Dilatation of right heart; heart failure is also shown.

95. Parkinson's disease.

1. <https://www.slideshare.net/Jijoallsaints/parkinsons-disease-31065515>

96. Essential tremor. Torsion dystonia.

- 1.** <https://www.slideshare.net/NeurologyKota/tremors-54024775>
- 2.** <https://www.epainassist.com/movement-disorders/dystonia>
- 3.** For more information <https://www.slideshare.net/drpsdeb/tremor-25107960>

97. Myodystrophy Duchenne, Dejerine-Landuzi, Erb-Roth.

(Dejerine-Landuzi, Erb-Roth have other names)

- 1.** <http://etest.bsmu.by/mod/page/view.php?id=151391&forceview=1> (slides 1 to 34 and 52 to 53)
- 2.** <http://etest.bsmu.by/mod/page/view.php?id=151392&forceview=1> (slides 1 to 8)

98. Myasthenia.

- 1.** <https://www.slideshare.net/manalihsolanki/myasthenia-gravis-17274167>
- 2.** <https://www.slideshare.net/smcmedicinedept/myasthenia-gravis-pathophysiology-cl-features-dd>

99. Myotonia. Paroxizmal myoplegia.

- 1.** <http://etest.bsmu.by/mod/page/view.php?id=151392&forceview=1> (slides 24 to 50)

Periodic paralysis (also known as **myoplegia paroxysmalis familiaris**) is a group of rare genetic diseases that lead to weakness or paralysis from common triggers such as cold, heat, high carbohydrate meals, not eating, stress or excitement and physical activity of any kind. The underlying mechanism of these diseases are malfunctions in the ion channels in skeletal muscle cell membranes that allow electrically charged ions to leak in or out of the muscle cell, causing the cell to depolarize and become unable to move.

The symptoms of periodic paralysis can also be caused by **hyperthyroidism**, and are then **labeled thyrotoxic periodic paralysis**; however, if this is the underlying condition there are likely to be other characteristic manifestations, enabling a correct diagnosis.

Periodic paralysis is an **autosomal dominant myopathy** with considerable variation in penetrance, leading to a spectrum of familial phenotypes (only one parent needs to carry the gene mutation to affect the children, but not all family members who share the gene are affected to the same degree). **Specific diseases include:**

Hypokalemic periodic paralysis where potassium leaks into the muscle cells from the bloodstream.

Hyperkalemic periodic paralysis, where potassium leaks out of the cells into the bloodstream.

Paramyotonia congenita, a form which often accompanies hyperkalemic periodic paralysis, but may present alone. The primary symptom of paramyotonia congenita **is muscle contracture which develops during exercise or activity**.

Paramyotonia congenita attacks may also be triggered by **a low level of potassium in the bloodstream**. This means people with both hyperkalemic periodic paralysis and paramyotonia congenita can **have attacks with fluctuations of potassium up or down**.

Andersen-Tawil syndrome, a form of periodic paralysis that includes significant heart **rhythm problems**, fainting and risk of sudden death. Potassium levels may be low, high, or normal during attacks of ATS. Patients with ATS may also have skeletal abnormalities like scoliosis (curvature of the spine), webbing between the second and third toes or fingers (syndactyly), crooked fingers (clinodactyly), a small jaw (micrognathia) and

low-set ears. Patients need to have another form of periodic paralysis to have the Andersen-Tawil.

Cause

One of the most common descriptions of periodic paralysis are episodic attacks of muscle weakness, which are commonly associated with serum potassium levels. Physical activity and diet content (carbohydrates) have been identified as PP triggers. Unlike non-dystrophic myotonias, the periodic paralysis phenotype is triggered after resting following exercise. Voltage-gated sodium channel mutations are among the key causes behind periodic paralysis.

Hyper-kalemic PP (hyperPP) is identified with high extracellular potassium levels which are typically greater than 5 mM during attacks; however, HyperPP attacks can also take place without rise in potassium concentrations. HyperPP has a prevalence rate of 1/100,000. Patients become symptomatic around the age of 10. The weakness attacks in hyperPP are relatively short lasting, and range from minutes to hours. The attacks can happen upwards of ten times per month.

Hypo-kalemic PP (hypoPP) is associated with low potassium levels. The onset of hypoPP occurs between the ages of 15 and 35. The prevalence of hypoPP is estimated to 1/100,000. HypoPP can be triggered by many external factors such as stress, high-sugar diet, and rest after exercise. During hypoPP attacks, the serum potassium concentrations can drop to less than 3 mM. Furthermore, hypoPP attacks are considerably longer lasting than hyperPP. As exercise is a trigger for periodic paralysis attacks, recently there is more research going into the physiological changes that accompany exercise including changes in blood pH.

Diagnosis

This disease is unusually difficult to diagnose. Patients often report years of wrong diagnosis and treatments that made them worse instead of better. Part of this may be that migraines are present in up to 50% of patients and can cause a confusing array of symptoms including headaches, speech difficulties and visual, auditory or sensory auras. DNA testing is available for only a half dozen common gene mutations, while dozens of known mutations are possible but are not routinely tested. Electromyography (EMG) findings are not specific but the McManis Protocol, also called the Compound Muscle Amplitude Potential test (CMAP) can be used by a skilled neurologist capable of utilizing the EMG, which can give assistance in diagnosing several of these PP disorders. The old glucose/insulin provocative testing can cause life-threatening symptoms and should not be used.

Also of note is that potassium levels do *not* have to range outside of normal limits to cause serious, even life-threatening paralysis. These diseases are *not* the same as having a very low level of potassium (hypokalemia) or high potassium (hyperkalemia) and must not be treated as such. The total body store of potassium is usually normal; it is just in the wrong place.

Treatment

Treatment of the periodic paralyses may include carbonic anhydrase inhibitors (such as acetazolamide, methazolamide or dichlorphenamide), taking supplemental oral potassium chloride and a potassium-sparing diuretic (for hypos) or avoiding potassium (for hypers), thiazide diuretics to increase the amount of potassium excreted by the kidneys (for hypers), and significant lifestyle changes including tightly controlled levels of exercise or activity. Treatment of periodic paralysis in Andersen-Tawil syndrome is similar to that for other types. However, pacemaker insertion or an implantable cardioverter-defibrillator may be required to control cardiac symptoms.

100. Spinal and neural amyotrophy Werdnig-Hoffman, Kugelberg-Welander, Charcot-Marie.

1. <https://www.slideshare.net/KannanChinnasamy2/spinal-muscular-atrophy-76056904>
2. <https://www.slideshare.net/ShadyMahmoud4/spinal-muscle-atrophy-59689629>
3. <https://www.slideshare.net/ArunK29/charcot-mari tooth-disease>
4. More information <https://www.slideserve.com/ianthe/spinal-muscular-atrophy>

101. Familial spastic paraplegia. Spinal-cerebellar degenerations.

1. <https://www.slideshare.net/ysasi/hereditary-spastic-paraplegia>
2. <https://www.slideshare.net/marina761/spinocerebellar-ataxia-type-1>

102. Hepatolenticular degeneration. Huntington's chorea.

1. <https://www.slideshare.net/sunethweerarathna/wilsons-disease-34852809>
2. <http://etest.bsmu.by/mod/page/view.php?id=151395&forceview=1> (**57 to 60**)
3. <http://etest.bsmu.by/mod/page/view.php?id=151396&forceview=1> (**1 to 31**)
4. <https://www.slideshare.net/quelz/huntingtons-disease-30289859>

103. Radiation lesions of the brain.?

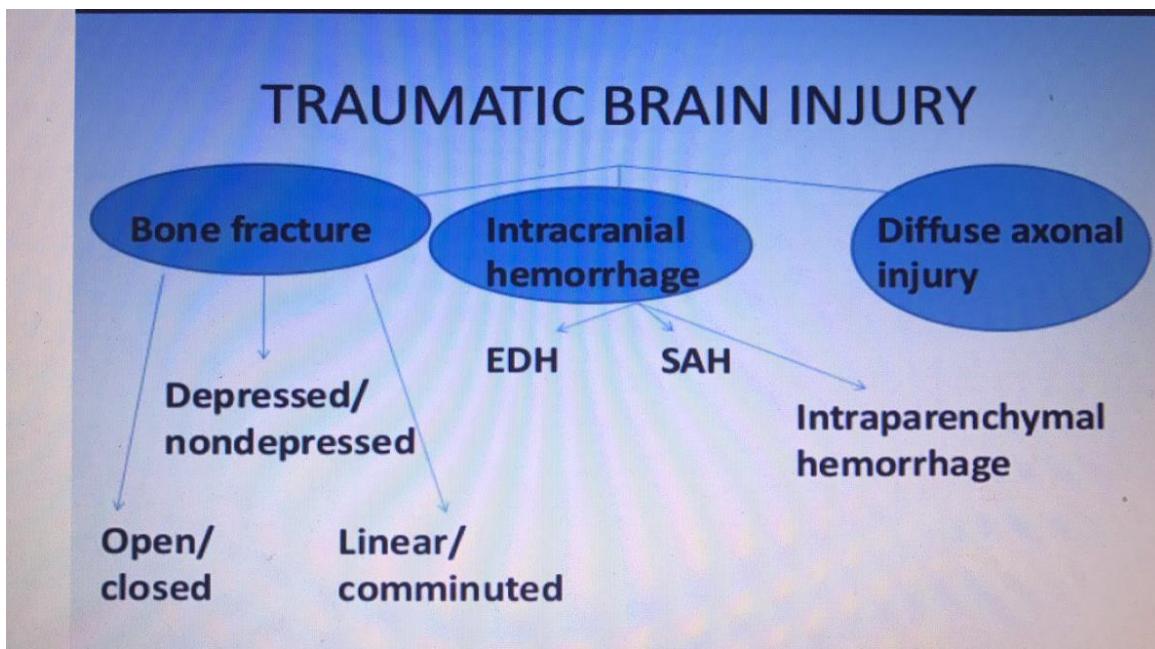
104. Brain dislocation.?

- 105. Traumatic brain injury classification.**
- 106. Concussion of the brain.**
- 107. Diffuse axonal brain injury.**
- 108. Depressed skull fractures.**
- 109. Traumatic subdural hematomas.**
- 110. Traumatic epidural hematoma.**
- 111. Traumatic brain injury in elderly patients and patients with concomitant alcohol intoxication.**
- 112. Examination of patients with traumatic brain injury.**
- 113. Severe traumatic brain injuries.**
- 114. Traumatic brain injury complications and consequences.**



There are 3 lectures on the site + answers below for questions 105 to 114

- 1.** <http://etest.bsmu.by/mod/page/view.php?id=151721&forceview=1>
- 2.** <http://etest.bsmu.by/mod/page/view.php?id=151722&forceview=1>
- 3.** <https://slideplayer.com/slide/5740059/>



- Symptoms of a skull fracture include
- swelling around area of impact,
- facial bruising, and
- bleeding from the nostrils or ears.

- A skull fracture is any break in the cranial bone, also known as the skull.
 - There are many types of skull fractures, but only one major cause: an impact or a blow to the head that's strong enough to break the bone.
- symptoms that can indicate a fracture include:
- ✓ swelling and tenderness around the area of impact
 - ✓ facial bruising
 - ✓ bleeding from the nostrils or ears

Depressed fracture

- This refers to a fracture that causes the skull to indent or extend into the brain cavity.



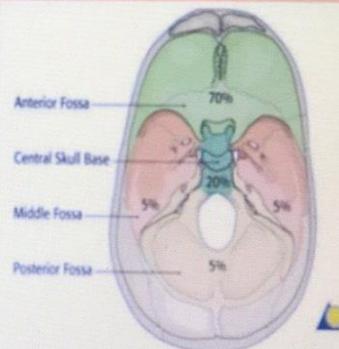
Basal fracture

- A basal fracture occurs in the floor of the skull: the areas around the eyes, ears, nose, or back, near the spine.

In addition to the above types, fractures can also classify as:

- linear (in a straight line)
- greenstick (incomplete)
- comminuted (broken into three or more sections)

SKULL BASE FRACTURES



70% of the skull base fractures occur in the anterior fossa.
20% in the middle central skull base
5% in the middle and posterior fossa.

Basilar Skull Fracture

Battle's sign



Raccoon eyes



- A skull fracture occurs when a force that is strong enough to break the bone hits the skull.
- Any type of impact to the head can cause a skull fracture, including
 - ✓ being hit with an object,
 - ✓ falling and hitting the ground,
 - ✓ injuring the head in a car accident, or
 - ✓ any other type of trauma.
- In some cases, as in an open or depressed fracture, it may be easy to see that the skull is broken. Sometimes, though, the fracture isn't obvious.

Symptoms of skull fractures

Serious symptoms of a skull fracture include:

- bleeding from the wound caused by the trauma, near the location of the trauma, or around the eyes, ears, and nose.
- bruising around the trauma site, under the eyes, or behind the ears
- severe pain at the trauma site
- swelling at the trauma site
- redness or warmth at the trauma site

• **Less severe symptoms**, or those that may not necessarily appear to be related to a skull fracture, may include:

- headache
- nausea
- vomiting
- blurred vision
- restlessness
- irritability
- loss of balance
- stiff neck
- pupils not reacting to light
- confusion
- excessive drowsiness
- fainting

- A doctor may be able to diagnose a fracture by simply performing a **physical examination** of the head.
- However, it's useful to diagnose the extent and exact nature of the damage, which requires more exact diagnostic tools.
- X-rays and **magnetic resonance imaging (MRIs)** are typical methods for imaging the body and can help to diagnose skull fractures.
- An **X-ray** penetrates soft tissue and provides an image of the bone. An MRI produces an image of the bone and soft tissue, allowing a doctor to see both the skull fracture and the brain.
- The **most common tool** used is a **computerized tomography scan (CT or CAT scan)**. This technique usually provides the clearest picture of the fracture and any damage to the brain because it produces a 3-D

Treatment of skull fractures

- Treatment for a skull fracture depends on several factors.
- Following points to be taken into take into consideration
 - the person's age,
 - health, and
 - medical history,
 - type of fracture,
 - its severity, and
 - any resulting brain injuries.

- In some cases, such as in basal skull fractures, medication to control pain may be all the patient needs.
- The skull will heal itself in a majority of these instances.
- However, a basal fracture may require surgery if it results in excessive leakage of cerebrospinal fluid from the nose and ears.
- Surgery is more often a required course of treatment for depressed skull fractures.
- If the depression is severe enough, surgery may be necessary to correct it.
- Surgery may also be necessary if the depression puts pressure on the brain or if there is cerebrospinal fluid leakage.

EVALUATION OF A PATIENT WITH HEAD INJURY

Initial assessment

- The initial management is in accordance to ATLS guidelines.
 - A - airway
 - B - breathing
 - C - circulation

Airway

- Manual manoeuvres (chin lift, jaw thrust, recovery position, etc.)
- Insertion of oral or nasal airway
- Use of suction
- Assisted ventilation using bag–valve–mask
- Endotracheal intubation
- Cricothyroidotomy (with or without tracheostomy)

Airway

- Goals

- *Maintain SPO₂ > 90%*
- *Maintain PaO₂ > 60mmHg*

Indication for intubation

Unable to maintain airway

GCS ≤ 8

Loss of protective laryngeal reflexes

Unstable facial bone #

Bleeding into mouth

Seizures

Ventilatory insufficiency

Spontaneous hyperventilation

Irregular respiration

Breathing

- Assessment of respiratory distress and adequacy of ventilation
- Administration of oxygen
- Needle thoracostomy
- Chest tube insertion

Circulation

Goals

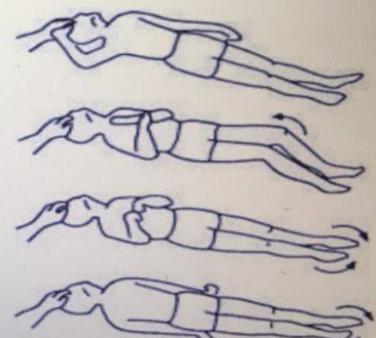
- Maintain SBP > 90mm of Hg
- Prevention of secondary brain injury

Circulation

- IV crystalloid
- Hypotensive resuscitation
- Colloid
- Blood
- Component transfusion

- Transport to equipped center
- Prognosis depends on initiation of primary care
- Enroute management

Examination: Glasgow Coma Scale		
<i>Best Eye Opening</i>		<i>Best Verbal Response</i>
4 Spontaneously		5 Converses, oriented
3 Voice		4 Converses, disoriented
2 To pain		3 Inappropriate words
1 Not at all		2 Incomprehensible sounds
		1 No verbalization
<i>Best Motor Response</i>		
6 Obeys	Follows motor commands	
5 Localizes	Clearly pushes painful stimuli away	
4 Withdraws <i>(flexion-withdrawal)</i>	Only withdraws arm or leg to painful stimuli	
3 Abnormal flexion <i>(decorticate)</i>	Flexion of arms with extension of legs to stimuli	
2 Abnormal extension <i>(decerebrate)</i>	Extension of all extremities to painful stimuli	
1 Flaccid	No response to painful stimuli	



Pupil

Pupil size:

- The normal diameter of the pupil is between 2 and 5 mm, and although both pupils should be equal in size,
 - a 1-mm difference is considered a normal variant.
 - Abnormal size is noted by anisocoria: >1 mm difference between pupils

Pupil

Pupil symmetry:

- Normal pupils are round, but can be irregular due to ophthalmological surgeries.
- Abnormal symmetry may result from compression of CNIII can cause a pupil to initially become oval before becoming dilated and fixed.

Pupil

Direct light reflex:

- Normal pupils constrict briskly in response to light, but may be poorly responsive due to ophthalmological medications.
- Abnormal light reflex may be seen in sluggish pupillary responses are associated with increased ICP
- A non-reactive, fixed pupil has <1 mm response to bright light and is associated with severely increased ICP.

Physical examination

Head and neck

- inspection for cranial nerve deficits, periorbital or postauricular ecchymoses, CSF rhinorrhoea or otorrhoea, haemotympanum (**signs of base of skull fracture**)
- fundoscopic examination for retinal haemorrhage (sign of abuse)[90] and papilloedema (**sign of increased ICP**)
- palpation of the scalp for haematoma, crepitance, laceration, and bony deformity (**markers of skull fractures**)

Physical examination

- auscultation for carotid bruits (**sign of carotid dissection**)
- evaluation for cervical spine tenderness, paraesthesia, incontinence, extremity weakness, priapism (**signs of spinal cord injury**)
- Extremities should receive motor and sensory examination (**for signs of spinal cord injury**)

Baseline laboratory investigations should include:

- CBC including platelets
- serum electrolytes and urea
- serum glucose
- coagulation status: PT, INR, activated PTT
- blood alcohol level and toxicology screening if indicated

Indications for CT scan

- eye opening only to pain or not conversing (GCS 12/15 or less)
- confusion or drowsiness (GCS 13/15 or 14/15) followed by failure to improve within
- at most one hour of clinical observation or within two hours of injury (whether or not intoxication from drugs or alcohol is a possible contributory factor)
- base of skull or depressed skull fracture and/or suspected penetrating injuries

Indications for CT scan

- **a deteriorating level of consciousness or new focal neurological signs**
- **full consciousness (GCS 15/15) with no fracture but other features, eg**
 - severe and persistent headache
 - two distinct episodes of vomiting
- **a history of coagulopathy (eg warfarin use) and loss of consciousness, amnesia or any neurological feature.**

Traumatic Brain Injury (TBI): Common Manifestations/Complications

- Increased ICP symptoms general and specific
- Restlessness- R/O respiratory; waking up
- Systemic effects of acute brain injury- hypermetabolism, brainstorming, SIADH
- Brainstorming- hypothalamic stimulation-ANS
- CSF leak- rhinorrhea/otorrhea- basal skull Fx
- Post concussion Syndrome
- Associated cervical spinal cord injury

Complications

- **Posttraumatic seizures;** frequently occur after moderate or severe TBI, they are usually general or partial.

Immediate seizures occur in the first 24 hours.

Early seizures occur in the first 2-7 days.

Late seizures occur after 7 days.

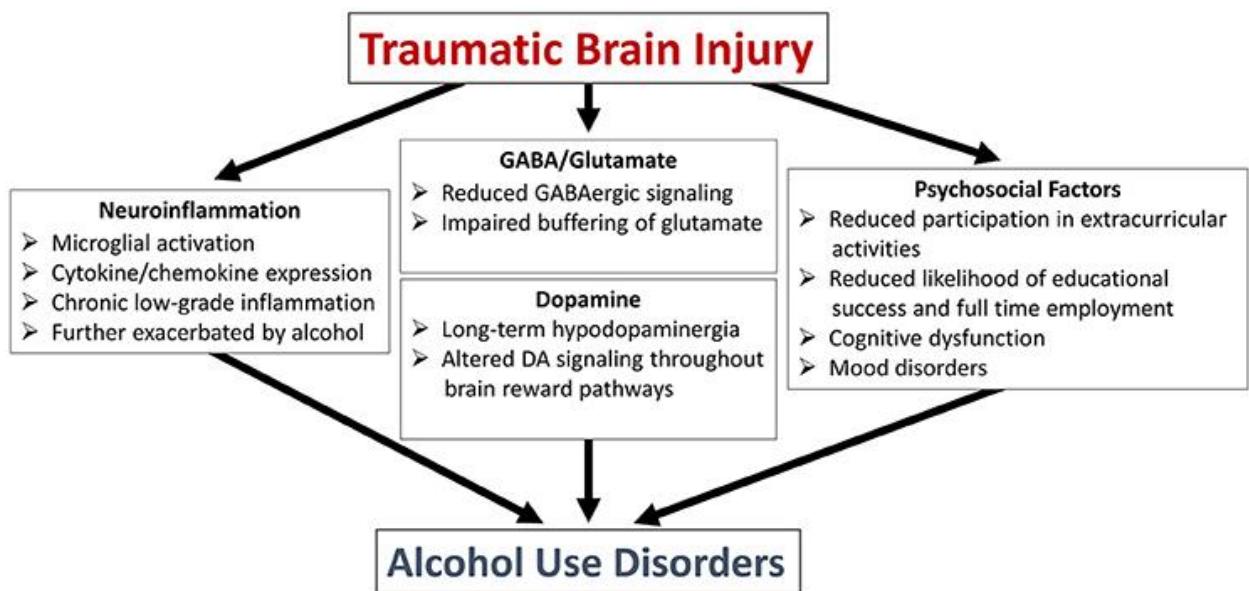
Temkin showed that prophylactic use of phenytoin is effective during the first week after TBI.

He recommended discontinuation after 1 week if no seizures develop because of its lack of effect in preventing late seizures.

Consequences of Traumatic Brain Injury

Physical Consequences related to the injury and secondary complications of central dysregulations and impaired mobility:

- Musculoskeletal system – Mobility - Motor control impairment
- Spasticity
- Swallowing and speech control impairment
- Sensory impairment
- Seizures
- Late intracranial complications
- Endocrine and Autonomous System dysregulation
- Respiratory system complications related to aspiration
- Incontinence
- Headache
- Neuropsychological consequences



115. Traumatic lesions of brachial plexus.

<https://www.slideshare.net/adityachakri/brachial-plexus-injuries-93887511>

116. Classification of traumatic spinal cord lesions. Spinal cord contusions.

117. Spinal cord injury (concussion and contusion).

118. Traumatic spinal cord compression.

119. Examination of patients with traumatic spinal cord lesions.

Anatomy Review

- 33 Vertebrae
- Spine supported by pelvis
- key ligaments and muscles connect head to pelvis
 - anterior longitudinal ligament
 - anterior portion of the vertebral body
 - major source of stability
 - protects against hyperextension
 - posterior longitudinal ligament
 - posterior vertebral body within the vertebral canal
 - prevents hyperflexion

Anatomy Review

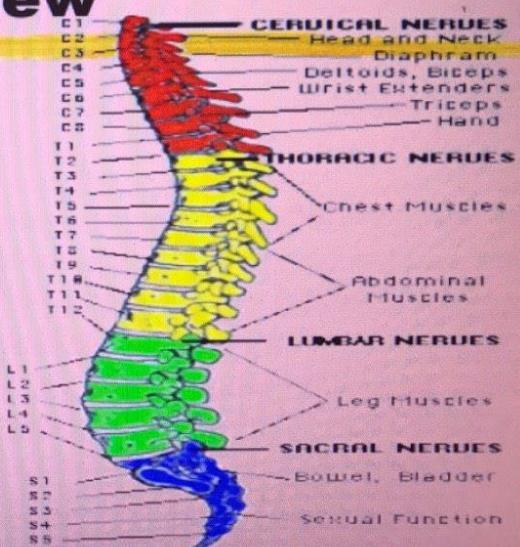
□ Bone Structure of the Spine

□ Cervical

□ Lumbar

□ Thoracic

□ Sacral/Coccyx



Anatomy Review

□ Cervical Spine

- 7 vertebrae
- very flexible
- C1: also known as the atlas
- C2: also known as the axis

□ Thoracic Spine

- 12 vertebrae
- ribs connected to spine
- provides rigid framework of thorax

Anatomy Review

□ Lumbar Spine

- 5 vertebrae
- largest vertebral bodies
- carries most of the body's weight

□ Sacrum

- 5 fused vertebrae
- common to spine and pelvis

□ Coccyx

- 4 fused vertebrae
- "tailbone"

Anatomy Review

Vertebral body

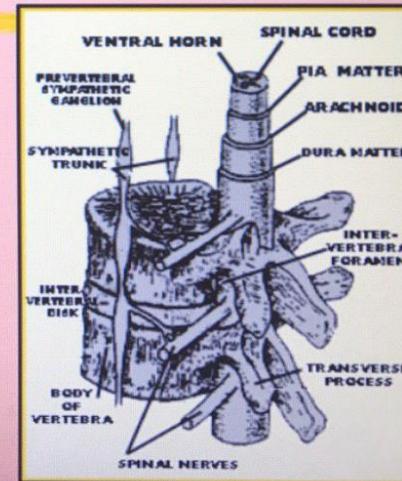
- posterior portion forms part of vertebral foramen
- increases in size from cervical to sacral
- spinous process
- transverse process

Vertebral foramen

- opening for spinal cord

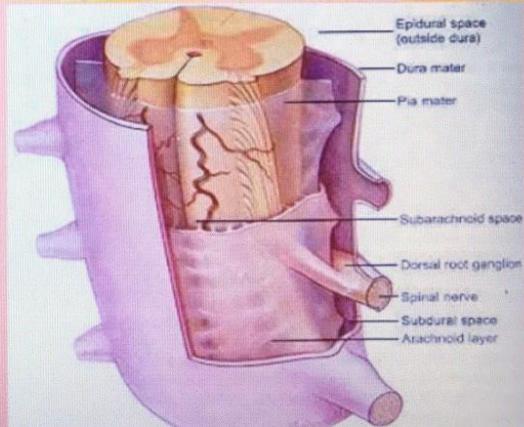
Intervertebral disk

- shock absorber (fibrocartilage)



Anatomy Review

- Ends at ~ L-2
 - cauda equina
- Blood supplied by vertebral and spinal arteries
- Gray matter: core pattern resembling butterfly
- White matter: longitudinal bundles of myelinated nerve fibers



Anatomy Review

□ Spinal Cord

- Thoracic and lumbar levels supply sympathetic nervous system fibers
- Cervical and sacral levels supply parasympathetic nervous system fibers

Spinal Cord Pathways

□ Ascending Nerve Tracts (sensory input)

- carry impulses from body structures and sensory information to the brain

□ Posterior column (dorsal)

- conveys nerve impulses for proprioception, discriminative touch, pressure, vibration, & two-point discrimination
- cross over at the medulla from one side to the other
 - e.g. impulses from left side of body ascend to the right side of the brain

Spinal Cord Pathways

- Spinothalamic Tracts (anterolateral)
 - Convey nerve impulse for sensing pain, temperature & light touch
 - Impulses cross over in the spinal cord not the brain
 - Lateral tracts
 - conduct impulses of pain and temperature to the brain
 - Anterior tracts
 - carry impulses of light touch and pressure

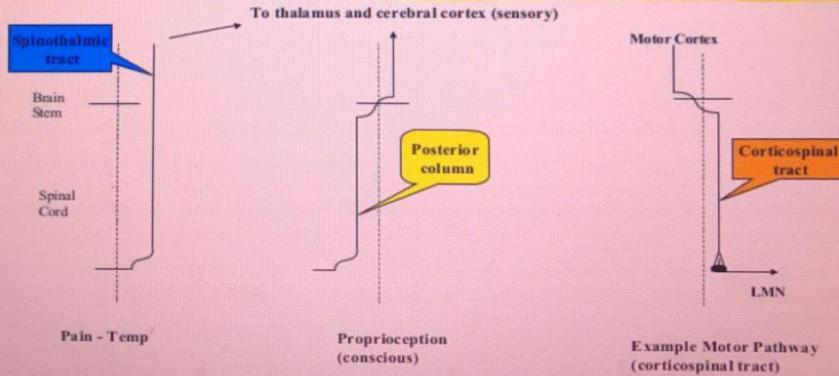
Spinal Cord Pathways

- Descending Motor Tracts (motor output)
 - conveys motor impulses from brain to the body
 - Pyramidal tracts: Corticospinal & Corticobulbar
 - Corticospinal tracts
 - destined to cause precise voluntary movement and skeletal muscle activity
 - lateral tract crosses over at medulla

Spinal Cord Pathways

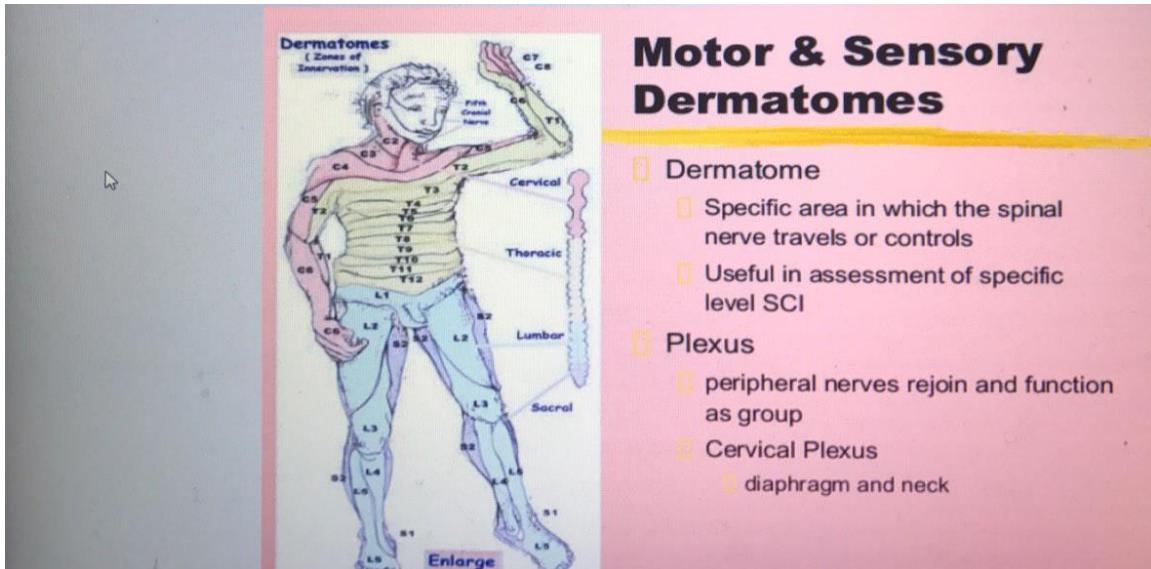
- Descending Motor Tracts (motor output)
- Extrapyramidal tracts
 - rubrospinal, pontine reticulospinal, medullary reticulospinal, lateral vestibulospinal and tectospinal
 - Pontine reticular and lateral vestibular have powerful excitatory effects on extensor muscles
 - brain stem lesions above these two areas but below midbrain cause dramatic increase in extensor tone
 - called decerebrate rigidity or posturing
 - Reticulospinal: impulses to control muscle tone & sweat gland activity
 - Rubrospinal: impulses to control muscle coordination & control of posture

Example Motor and Sensory Pathways



Spinal Nerves

- 31 pairs originate from the spinal cord
- Carry both sensation and motor function
- Named according to level of spine from where they arise
 - Cervical 1-8
 - Thoracic 1-12
 - Lumbar 1-5
 - Sacral 1-5
 - Coccygeal 1



Motor & Sensory Dermatomes

Dermatome

- Specific area in which the spinal nerve travels or controls
- Useful in assessment of specific level SCI

Plexus

- peripheral nerves rejoin and function as group
- Cervical Plexus
 - diaphragm and neck

Dermatomes

- | | |
|--|--|
| <ul style="list-style-type: none"> □ C3,4 <ul style="list-style-type: none"> □ motor: shoulder shrug □ sensory: top of shoulder □ C3, 4, 5 <ul style="list-style-type: none"> □ motor: diaphragm □ sensory: top of shoulder □ C5, 6 <ul style="list-style-type: none"> □ motor: elbow flexion □ sensory: thumb | <ul style="list-style-type: none"> □ C7 <ul style="list-style-type: none"> □ motor: elbow, wrist, finger extension □ sensory: middle finger □ C8, T1 <ul style="list-style-type: none"> □ motor: finger abduction & adduction □ sensory: little finger □ T4 <ul style="list-style-type: none"> □ motor: level of nipple □ T10 <ul style="list-style-type: none"> □ motor: level of umbilicus |
|--|--|

Dermatomes

- | | |
|---|---|
| <ul style="list-style-type: none"> □ L1, 2 <ul style="list-style-type: none"> □ motor: hip flexion □ sensory: inguinal crease □ L3,4 <ul style="list-style-type: none"> □ motor: quadriceps □ sensory: medial thigh, calf □ L5 <ul style="list-style-type: none"> □ motor: great toe, foot dorsiflexion □ sensory: lateral calf | <ul style="list-style-type: none"> □ S1 <ul style="list-style-type: none"> □ motor: knee flexion □ sensory: lateral foot □ S1, 2 <ul style="list-style-type: none"> □ motor: foot plantar flexion □ S2,3,4 <ul style="list-style-type: none"> □ motor: anal sphincter tone □ sensory: perianal |
|---|---|

Assessment of Spinal Injury

□ Mechanism of Injury -

- No longer consider all MOIs lead to SCI**
- Severe mechanism of injury is consistent with SCI**
- Other MOIs don't correlate to the risk of SCI**
- ED & Field Clearance protocols now commonly used**
- Exam and History findings help identify the potential SCI**
- Do No Harm!**

SCI General Assessment

□ Consider Mechanism of Injury & Kinematics

- Positive MOI ⇒ Should Require SMR**
 - high speed motor vehicle collision**
 - fall greater than 3 times the patient's height**
 - violent situations occurring near the spine**
 - stabbing
 - gun shot
 - sports injury (with force or velocity)**
 - confounding factors such as osteoporosis, extreme age**
 - other high impact, high force or high velocity conditions involving the head, spine or trunk**

SCI General Assessment

□ Consider Mechanism of Injury & Kinematics

- Negative MOI ⇒ Probably Do Not Require SMR**
 - force or impact does not suggest a potential spinal injury**
 - dropped a rock on foot
 - twisted ankle while running
 - isolated musculoskeletal injury
 - simple fall from standing position
 - low speed motor vehicle collision

SCI General Assessment

□ ABCs

- Airway and/or Breathing impairment**
 - Inability to maintain airway**
 - Apnea**
 - Diaphragmatic breathing**

- Cardiovascular impairment**
 - Neurogenic Shock**
 - Hypoperfusion**

SCI General Assessment

□ Neurologic Status:

- Level of Consciousness**
 - Brain injury also?**
 - Cooperative**
 - No impairment (drugs, alcohol)**
 - Understands & Recalls events surrounding injury**
 - No Distracting injuries**
 - No difficulty in communication**

SCI General Assessment

□ Assess Function & Sensation

- Palpate over each spinous process**
- Motor function**
 - Shrug shoulders**
 - Spread fingers of both hands and keep apart with force**
 - "Hitchhike" {T1}**
 - Foot plantar flexors (gas pedal) {S1,2}**
- Sensation (Position and Pain)**
 - weakness, numbness, paresthesia**
 - pain (pinprick), sharp vs dull, symmetry**
- Priapism**

Spinal Cord Injuries

Forces

- Direct traumatic injury
 - stab or gunshot directly to the spine
- Excessive Movement
 - acceleration
 - deceleration
 - deformation

Directional Forces

- flexion, hyperflexion
- extension, hyperextension
- rotational
- lateral bending
- vertical compression
- distraction

Spinal Cord Injuries

Primary Injury

- occurs at the time of injury
- may result in
 - cord compression
 - direct cord injury
 - interruption in cord blood supply

Secondary Injury

- occurs after initial injury
- may result from
 - swelling/inflammation
 - ischemia
 - movement of body fragments

Spinal Cord Injuries

Cord concussion & Cord contusion

- temporary loss of cord-mediated function

Cord compression

- decompression required to minimize permanent injury

Laceration

- permanent injury dependent on degree of damage

Hemorrhage

- may result in local ischemia

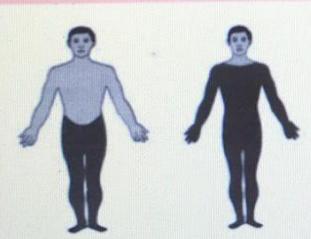
Spinal Cord Injuries

□ Cord transection

□ Complete

- all tracts disrupted
- cord mediated functions below transection are permanently lost
- determined ~ 24 hours post injury
- possible results
 - quadriplegia
 - paraplegia

Terminology



The dark shading shows the areas of the body affected by a T-11 level injury to the lower spinal cord. This person has paraplegia.

The dark shading shows the areas of the body affected by a C-3 level injury to the neck. This person has tetraplegia.

□ Paraplegia

- loss of motor and/or sensory function in thoracic, lumbar or sacral segments of SC (arm function is spared)

□ Quadriplegia

- loss of motor and/or sensory function in the cervical segments of SC

Spinal Cord Injuries

□ Cord transection

□ Incomplete

- some tracts and cord mediated functions remain intact
- potential for recovery of function
- Possible syndromes
 - Brown-Séquard Syndrome
 - Anterior Cord Syndrome
 - Central Cord Syndrome

Brown Sequard Syndrome

- Incomplete Cord Injury
 - Injury to one side of the cord (Hemisection)
 - Often due to penetrating injury or vertebral dislocation
 - Complete damage to all spinal tracts on affected side
 - Good prognosis for recovery

Brown Sequard Syndrome

- Exam Findings
 - Ipsilateral loss of motor function motion, position, vibration, and light touch
 - Contralateral loss of sensation to pain and temperature
 - Bladder and bowel dysfunction (usually short term)

Anterior Cord Syndrome

- Anterior Spinal Artery Syndrome
 - Supplies the anterior 2/3 of the spinal cord to the upper thoracic region
 - caused by bony fragments or pressure on spinal arteries

Anterior Cord Syndrome

- Exam Findings
 - Variable loss of motor function and sensitivity to pinprick and temperature
 - loss of motor function and sensation to pain, temperature and light touch
 - Proprioception (position sense) and vibration are preserved

Central Cord Syndrome

- Usually occurs with a hyperextension of the cervical region
- Exam Findings
 - weakness or paresthesias in upper extremities but normal strength in lower extremities
 - varying degree of bladder dysfunction

Cauda Equina Syndrome

- Injury to nerves within the spinal cord as they exit the lumbar and sacral regions
 - Usually fractures below L2
 - Specific dysfunction depends on level of injury
- Exam Findings
 - Flaccid-type paralysis of lower body
 - Bladder and bowel impairment

Neurogenic Shock

- Temporary loss of autonomic function of the cord at the level of injury
 - Usually results from cervical or high thoracic injury
- Does not always involve permanent primary injury
- Effects may be temporary and resolve in hours to weeks
- Goal is to avoid secondary injury

Neurogenic Shock

□ Presentation

- Flaccid paralysis distal to injury site
- Loss of autonomic function
 - hypotension or relative hypotension
 - vasodilation
 - loss of bladder and bowel control
 - priapism
 - loss of thermoregulation
 - warm, pink, dry below injury site
 - relative bradycardia
 - may have classic SNS response presentation above injury

Autonomic Hyperreflexia Syndrome

- Associated with SCI patients (usually T-6 or above) some time after initial injury
 - Vasculature has adapted to loss of sympathetic tone
 - Blood pressure normalized
 - No vasodilation response to increased BP
- ANA reflexively responds with arteriolar spasm
 - increased BP
 - stimulates PNS
 - results in bradycardia
 - peripheral and visceral vessels unable to dilate

Autonomic Hyperreflexia Syndrome

□ Presentation

- Paroxysmal hypertension, possible extreme
- headache
- blurred vision
- sweating and flushed skin above level of injury
- increased nasal congestion
- nausea
- bradycardia
- distended bladder or rectum

Non-Traumatic Conditions

□ Low Back Pain (LBP)

- 60-90% of population experience some form of LBP
- Very small number due to sciatica (lumbar nerve root)
- Most causes can not be specifically diagnosed
- Risk Factors
 - repetitious lifting or straining
 - chronic exposure to vibration (e.g. vehicle)
 - osteoporosis
 - age

Non-Traumatic Conditions

□ Low Back Pain (LBP)

- Causes
 - tumor
 - prolapsed disk
 - bursitis
 - degenerative joint disease
 - problems with spinal mobility
 - inflammation caused by infection
 - fractures
 - ligament strains

Non-Traumatic Conditions

- Low Back Pain (LBP)
 - Degenerative Disk disease
 - common over 50 years of age
 - narrowing of the disk
 - biochemical alterations of intervertebral disk
 - Herniated intervertebral Disk
 - tear in the posterior rim of capsule enclosing the gelatinous center of the disk
 - trauma, degenerative disk disease, improper lifting
 - commonly affects L-5, S-1 and L-4, L-5 disks

Management of SCI

- Primary Goal
 - Prevent secondary injury
- Stabilization of the spine begins in the initial assessment
 - Treat the spine as a long bone
 - Secure joint above and below
 - Caution with "partial" spine splinting
 - Dr. Robert's Rule: All or None
- Immobilization vs Motion Restriction

Management of SCI

- Neutral positioning of head and neck if at all possible
 - allows for the most space for cord
 - most stable position for spinal column
 - don't force it

Management of SCI

Cervical Motion Restriction

- Manual method
- Rigid collar comes later
- Interim device (KED)
- Move to long board or full body vacuum splint
- Manual continues until trunk and head secured
- "CID"
 - Don't use sand bags or IV fluid bags as head blocks
 - Tape works wonders!
 - Improvise with blanket rolls

Management of SCI

Don't forget the Padding

- Maintains anatomical position
- Limits movement on board
 - especially during transport on board or in vehicle
- fill all the voids
 - curvature of the lower back is normal - fill it
 - pillows, blankets, towels
- Tape along (even duct tape) is not enough

Management of SCI

Securing to the Board

- Straps, Tape, Cravats, whatever
- Torso first
 - then legs and feet and head
- Even patients extricated with a KED are secured to the board

Management of SCI

Pediatric Patient Considerations

- Elevate the entire torso if large occiput
 - Pad underneath
 - Short board underneath
 - Vacuum mattress
- Lots of voids to fill
- Difficult to find a correctly sized rigid collar
 - Improvise with
 - horse collar
 - blanket or towel rolls

Management of SCI

Helmeted Patients

- Removal should be limited to emergent need for access to airway and ventilation
- Leave in place if
 - good fit with little or no head movement within
 - no impending airway or breathing problems
 - can perform spinal motion restriction with helmet on
 - no interference in airway assessment or management
 - no cardiac arrest

Management of SCI

Helmeted Patients

- Types of Helmets
 - Sports (football, hockey)
 - Shoulder pads and helmet go together
 - Racing (motorcycle, car racer)
 - Recreational (motorcycle, bicycle)
- Various helmets create different problems for patient and for removal

Management of SCI

General

- Manual Spinal Motion Restriction
- ABCs
 - Increase FiO₂
 - Assist ventilations prn
 - IV Access & fluids titrated to BP ~ 90-100 mm Hg
- Consider High Dose methylprednisolone [SoluMedrol]: 30 mg/kg bolus over 15 mins then infusion after 1st hour
- Look for other injuries: "Life over Limb"
- Transport to appropriate SCI center

- 120. Brain tumors classification. Tumors of brain hemispheres.**
- 121. Brain tumors classification. Subtentorial brain tumors.**
- 122. Brain tumors classification. Supratentorial brain tumors.**
- 123. Frontal lobe tumors.**
- 124. Temporal lobe tumors.**
- 125. Tumors of pituitary region.**
- 126. Cerebellar tumors.**
- 127. Metastatic lesions of the CNS.**
- 128. Examination of patient with brain tumor.**
- 129. Spinal cord tumors. Intra-medullar tumors.**
- 130. Spinal cord tumors. Extra-medullar tumors.**



130

There are 4 lectures on the site for questions 120 to

- 1.** <http://etest.bsmu.by/mod/page/view.php?id=151718>
- 2.** <http://etest.bsmu.by/mod/page/view.php?id=151719&forceview=1>
- 3.** <https://www.slideshare.net/jvyom001/brain-tumor-68206369>
- 4.** <https://www.slideshare.net/AnilKumarGowda/spinal-cord-tumors-78943987>



- 131. Cerebral arterial-venous malformations.**
- 132. Carotid-cavernous fistula.**
- 133. Non-traumatic intracranial hemorrhage. Surgical approach.**



There are 2 lectures on the site for questions 131 to 133 + answers below

- 1.** <http://etest.bsmu.by/mod/page/view.php?id=151720>
- 2.** <https://www.slideshare.net/sureshBishokarma/caroticocavernous-fistula-ccf>

Nontraumatic intracranial hemorrhage is defined as a spontaneous hemorrhage into the brain parenchyma (**intracerebral hemorrhage**) or the cerebrospinal fluid space (**subarachnoid hemorrhage**).

Intracerebral hemorrhages cause acute signs and symptoms resembling those of cerebral ischemia and account for about 10% of strokes. One of the more common forms of intracerebral hemorrhage is **hypertensive hemorrhage**. The main symptom of subarachnoid hemorrhage is **headache**; its most common source is a ruptured aneurysm

General manifestations of intracranial hemorrhage.

These include:

- acute *headache*, often accompanied by *vomiting*;
- rapidly or very rapidly *progressive neurological deficits* (whose type depends on the site of hemorrhage);
- progressive *impairment of consciousness*, perhaps leading to coma;
- in many patients, *epileptic seizures*.

If these manifestations are present, an intracranial hemorrhage is the probable cause. The definitive diagnosis,

however, can only be made with neuroradiological methods.

Intracerebral Hemorrhage

Etiology

Most cases of intracerebral hemorrhage are due to the *rupture of vascular lesions of hypertensive origin* ("rhexis hemorrhages" of pseudoaneurysms of lipohyalinotic arterioles), *aneurysms, or arteriovenous malformations*. Intracerebral hemorrhage may also be a complication of therapeutic (over-) anticoagulation.

Causes of nontraumatic cerebral hemorrhage

Chronic arterial hypertension

Aneurysm rupture

Hemorrhage into a preexisting lesion (infarct, tumor)

Vascular malformation (cavernoma, arteriovenous malformation)

Vascular fragility due to vasculopathy, e. g., cranial arteritis, amyloid angiopathy

Bleeding diathesis due to hematologic disease or therapeutic anticoagulation

Cerebral venous thrombosis and venous sinus thrombosis

Rarely, in the setting of a hypertensive crisis or drug abuse (e. g., cocaine)

Clinical manifestations.

Chronic arterial hypertension and advanced age (typically 60–70) make a rhesis hemorrhage more likely. These hemorrhages are ultimately caused by hypertension and are usually very large. Common sites are the pallidum, the putamen, and the internal capsule, with the corresponding clinical manifestations: *contralateral, usually dense, hemiparesis or hemiplegia, horizontal gaze palsy, and initially, in many cases, déviation conjuguée and deviation of the head to the side of the lesion.* Less common sites are the subcortical white matter, brainstem, thalamus, and cerebellum. Very large hemorrhages, particularly if located in the posterior fossa, can rapidly elevate the intracranial pressure, causing brainstem compression and, in turn, impairment of consciousness and coma.

Acute worsening of more or less severe, preexisting

signs and symptoms, perhaps accompanied by additional

impairment of consciousness, suggests hemorrhage into an infarct or tumor.

Acute onset of focal or generalized epileptic seizures preceding the onset of the acute event point toward a tumor, vascular

malformation, or other structural lesion of the brain as the likely cause of hemorrhage

Diagnostic evaluation.

The diagnosis of intracranial hemorrhage is suggested by the characteristic *clinical findings and then definitively confirmed by the demonstration of blood on CT or MRI.* When performed in the acute phase, these studies may fail to reveal an underlying vascular malformation, if present, which may be obscured by the hemorrhage; *angiography* may be necessary to complete the diagnostic work-up. The obtaining of a complete *coagulation profile* is indicated

in some patients.

Treatment and prognosis.

Patients suffering from an acute intracerebral hemorrhage require *close clinical observation*; in particular, signs of intracranial hypertension (vomiting, progressive impairment of consciousness, and sometimes anisocoria and papilledema) must be vigilantly watched for. *Intracranial hypertension may be due to recurrent hemorrhage or to progressive brain swelling*; in either case, it must be promptly detected and treated (*for treatment measures, Treatment of intracranial hypertension includes the:*

- elevation of the head of the patient to 30°;
- hyperventilation (if the patient is intubated);
- osmotic diuretics, such as mannitol, given intravenously, in fractionated daily doses; rapid infusion is important for the generation of an effective osmotic gradient; saluretics, too, can transiently lower the intracranial pressure (caution: excessive use of diuretics can lead to dehydration and impairment of cerebral perfusion);
- corticosteroids (e. g., dexamethasone, given intravenously) are used to counteract cerebral edema, particularly of the vasogenic type; they are mainly effective against peritumoral and inflammatory brain edema, less so against ischemic and traumatic brain edema, which are predominantly of the cytotoxic type.). In addition,

stabilization of vital functions and the treatment of epileptic seizures, if present, are essential. In each case, the possible indication for *neurosurgical removal of the hematoma* should be carefully considered, in light of the neurological manifestations, site of the hemorrhage, and age and general condition of the patient. Cerebellar hemorrhage with mass effect generally confers a risk of impending brainstem compression and death and is often an indication for life-saving emergency surgery.

Although about one-third of all patients with an intracerebral hemorrhage will die of it, while others go on

to enjoy a more or less complete spontaneous recovery

134. Classification of spinal cord tumor. Extra medullar tumors.

the question was repeated