ROLE OF THE DESCENDING PATHWAYS FROM THE BRAINSTEM IN MAINTAINING POSTURE AND MUSCLE TONE, SPINAL SHOCK

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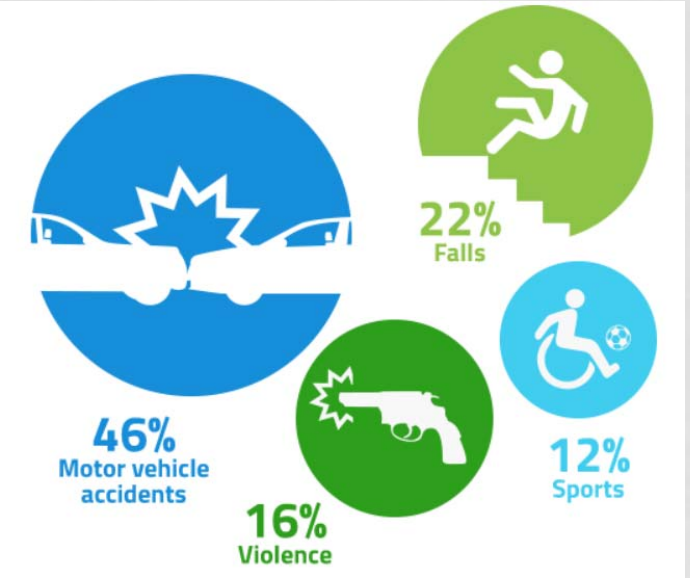
INCIDENCE AND HISTORY OF SPINAL CORD INJURY

- Trauma to the spinal cord causes dysfunction of the cord, with loss of sensory and motor function distal to the point of injury.
- In USA ≈ 400 000 patients with SCI
- incidence rate 1:1000 per year
- M : F = 4 : 1

Claire E. Hulseboch. Recent advances in pathophysiology and treatment of spinal cord injury. APS:238-255; 2002.

The leading causes of SCI:
Motor vehicle accidents (47%),
sports-related accidents:
diving injuries (24%), falls (12%),
Violence (16%):
gun shot, knife injuries (7%).



- **Three general groups**:
  - young individuals (16-25 years) – trauma
  - older individuals – cervical spinal stenosis caused by congenital narrowing or spondylosis
  - people with gunshot wounds

- 30-40 years ago: "*nothing can be done" and "wait and see what recovers"*
  - Urinary disfunction, trombosis, cardiac disfunction, hyper or hypotension

Life expectancy of SCI patients

- during World War II ≈ three months.
- In 1966, ≈ 20 years, with renal problems contributing to the majority of deaths.
- Currently, ≈ 25 to 30 years, the contributing cause of death is cardiac and respiratory dysfunction.

PATHOPHYSIOLOGY OF SPINAL CORD INJURY

Four general types of SCI:

- ***Maceration***, -the morphology of the cord is severely distorted;
- ***Laceration*** – gun shot or knife wounds
- ***Contusion injury*** -leads to a central hematomyelia that may evolve to syringomyelia
- ***Solid cord injury***, -in which there is no central focus of necrosis as in contusion injury.

THREE PHASES OF SCI RESPONSE THAT OCCUR AFTER INJURY

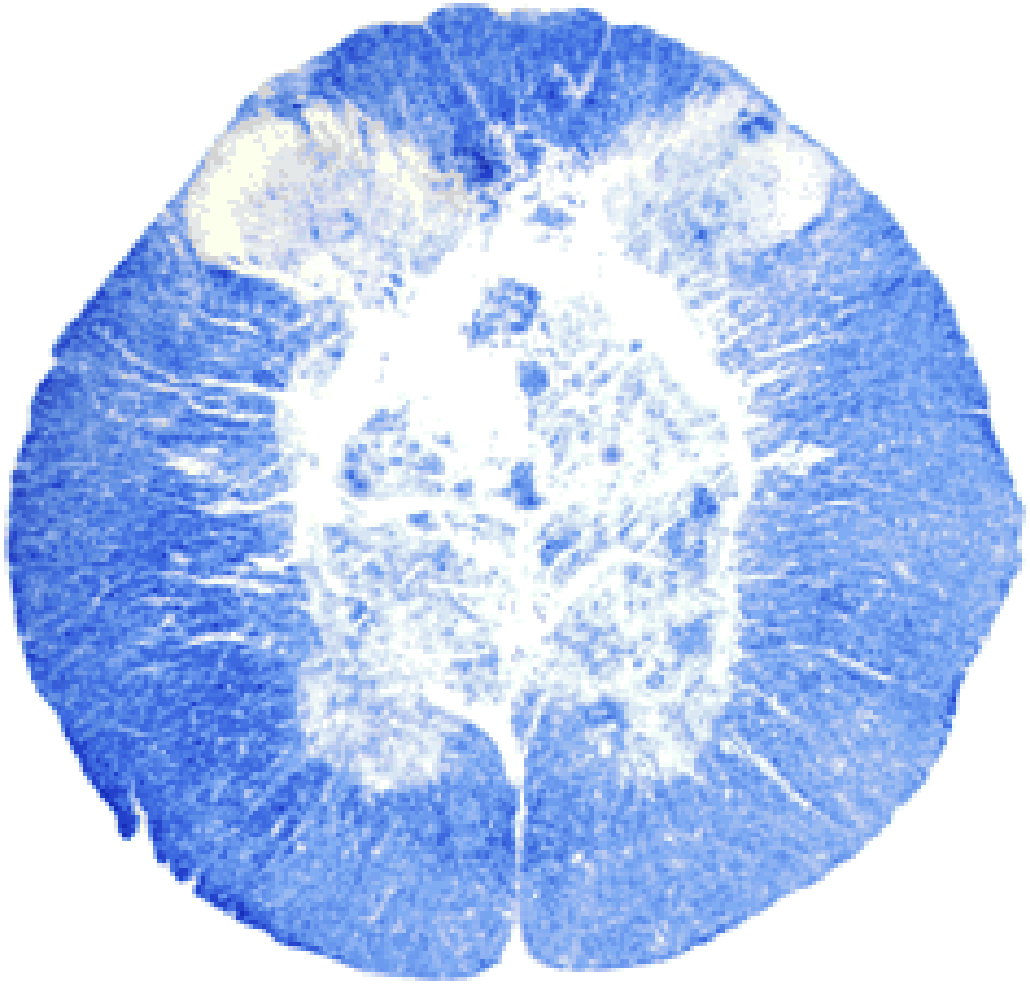
- Acute,
- Secondary,
- Chronic injury processes

- **Acute phase** (the moment of injury and extends for the first few days):
  - Immediate mechanical damage to neural tissue, necrosis, cell death
  - Injury-induced barrage of action potentials, electrolytic shifts -Na^{+}_{i} ↑, K^{+}_{e} ↑ Ca^{++}_{i} ↑
  - Spinal shock, hemorrhage, localized edema, thrombosis,
  - Compression of the spinal cord, neural injury↑

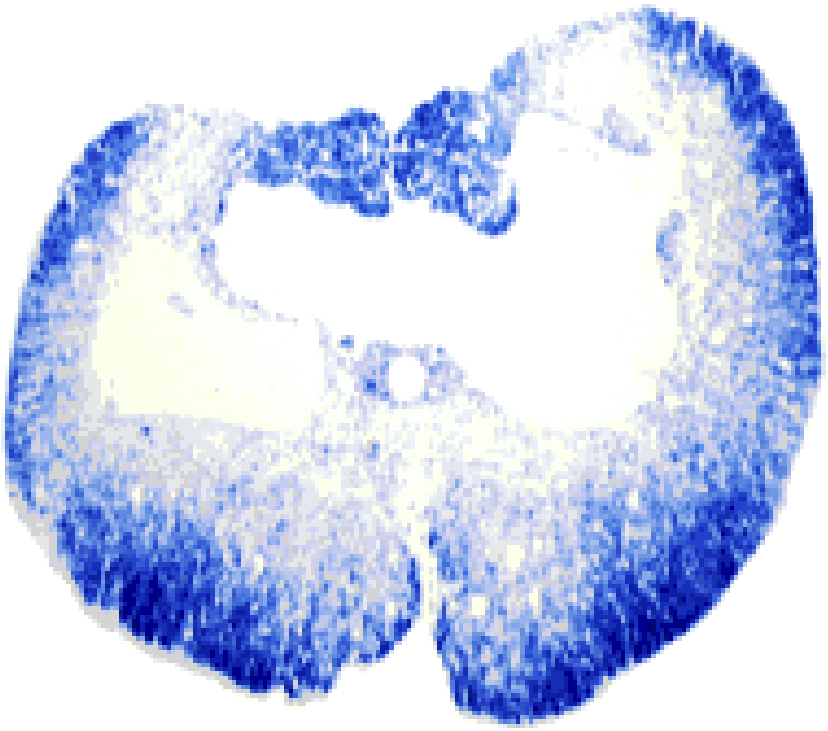
- **Secondary phase** (hours to weeks)
  - Continue from the acute phase
  - Glu_{e} cytotoxic concentrations (6-8x higher than normal)
  - Free-radical production, apoptosis – programmable cell death

- **Chronic phase**: (days to years)
  - Ortograde and retrograde apoptosis
  - Changes of receptors and ion channels
  - demyelination
  - Cyst form, syringomyelia
  - Lregenerative and sprouting response up to 1mm
  - Alterations in neural circuits due to changes in inhibitory and excitatory input
  - Permanent hyperexcitability – chronic pain syndromes!

Tissue Loss after Spinal Cord Injury

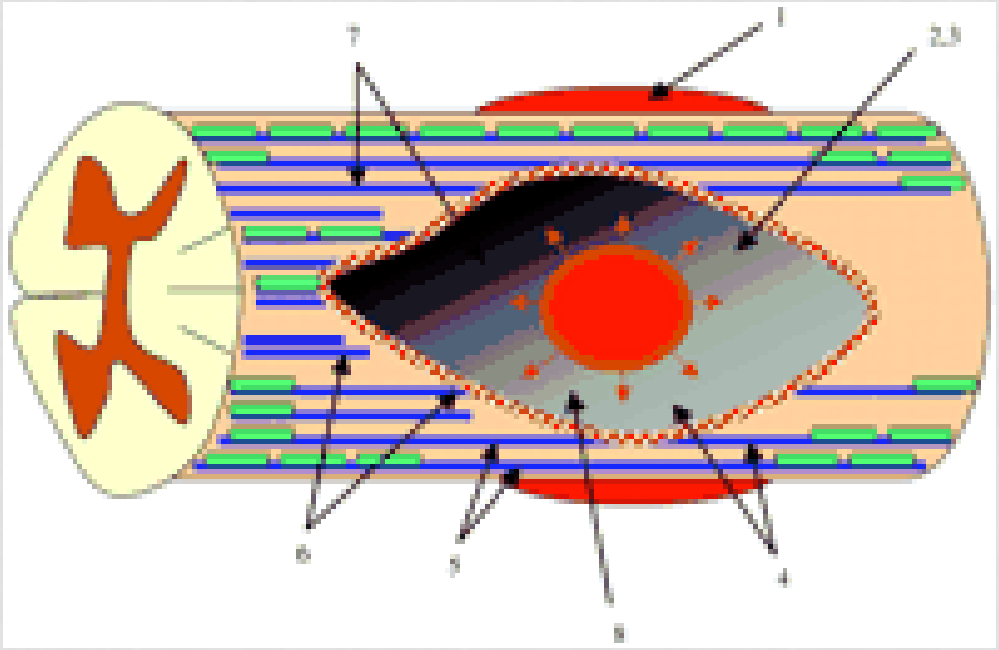


1 Hour Post Injury



60 Days Post Injury

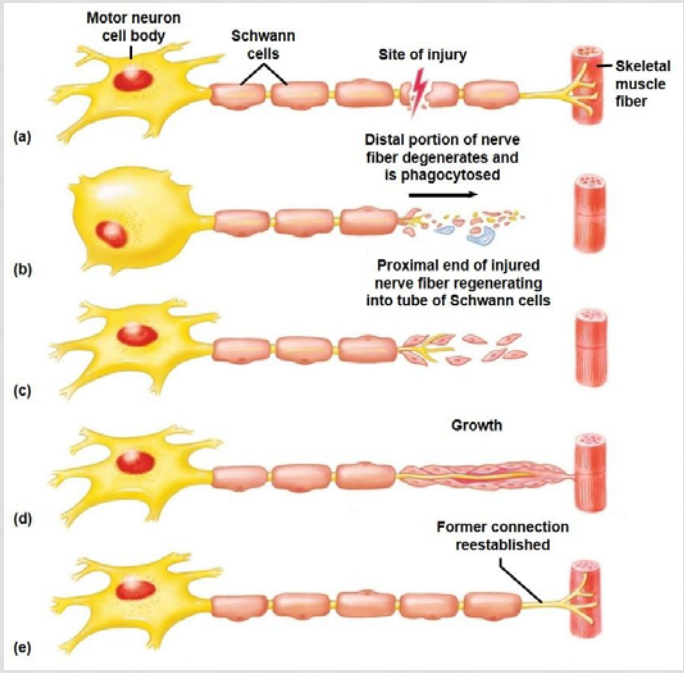
C.E. HULSEBOCH, ADVANCES IN PHYSIOLOGY EDUCATION, DECEMBER 2002.



C.E. HULSEBOCH, ADVANCES IN PHYSIOLOGY EDUCATION, DECEMBER 2002.

REGENERATION OF THE NEURONS

- Classically: neurons are incapable of regeneration!?
- 1958: Liu and Chambers: central projections of primary afferent fibers can sprout in the spinal cord.
- Proteins involved in neuronal regeneration:
  - GAP-43 (growth associated protein)
  - Protooncogene Bcl-2



ITERVENTION IN SCI

- hemostasis, decompression and stabilization of the vertebral column to prevent further trauma
- 1. Reduction of edema and free radical production
  - Methylprednisolone or lazaroid (for neuroprotection) within 8 h of injury
- 2. Inhibition of Glu Toxicity: Stopping the Excitotoxicity cascade and NO production
  - NMDA blocker: **MK-801,** AMPA antagonist:**NBQX, L-NAME**
- 3. Anti-Inflamatory Agents
- 4. Apoptosis-Rescue from Neural Cell Death

INTERVENTION IN SCI (CONT)

- 5. Demyelination and Conduction Deficits
  - Inhibiting the voltage-gated Na^+ channels: TTX
  - Inhibiting the voltage-gated K^+ channels: 4-AP phase III clinical trials (Acorda and Washington University)
- 6. Promoting Axonal Regeneration
- 7. Cell Replacement Strategies
  - Stem cells, Schwann cells
- 8. Transplant strategies
- *9. Aggressive Physical Therapy*
- *10. Electrical Stimulation*
- *11. treating chronic central pain: "Not all in their Head"*

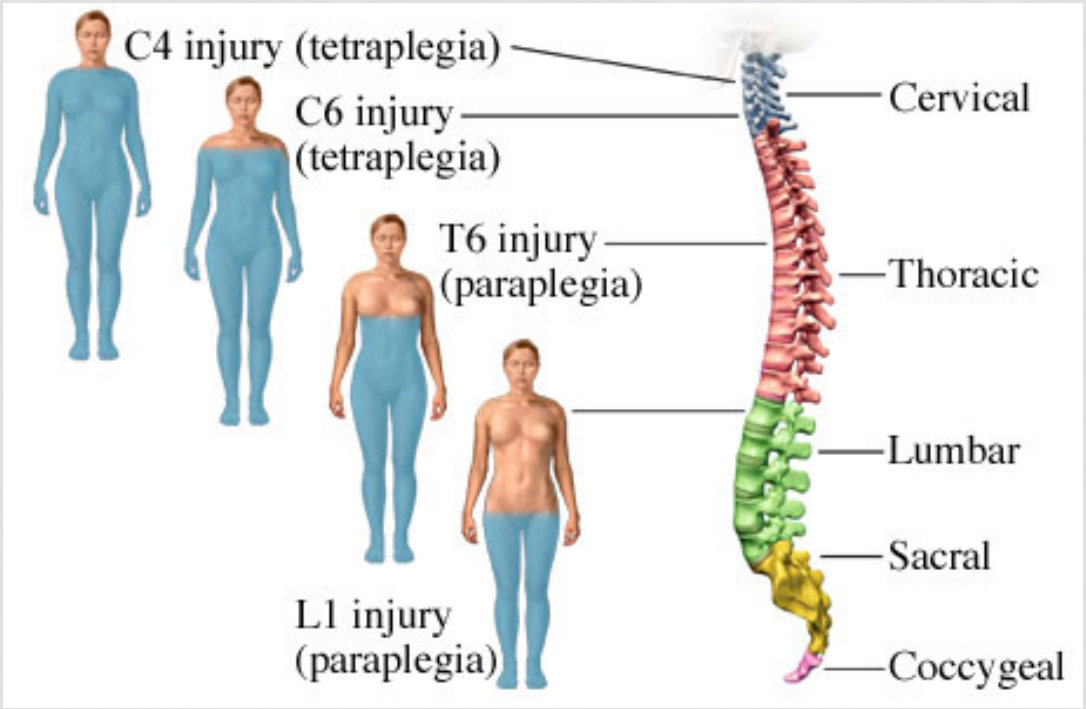
Kent Waldrep
National Paralysis Foundation

Christopher Reeves
National Paralysis Foundation

TERMINOLOGY

- Plegia = complete lesion
- Paresis = some muscle strength is preserved
- Tetraplegia (or quadriplegia)
  - Injury of the cervical spinal cord
  - Patient can usually still move his arms using the segments above the injury (e.g., in a C7 injury, the patient can still flex his forearms, using the C5 segment)
- Paraplegia
  - Injury of the thoracic or lumbo-sacral cord, or cauda equina
- Hemiplegia
  - Paralysis of one half of the body
  - Usually in brain injuries (e.g., stroke)

- MONOPLEGIA
- DIPLEGIA
- PARAPLEGIA
- HEMIPLEGIA
- TETRAPLEGIA (Quadriplegia)
  - HIGH (C1-C4)
  - LOW (C5-C8)



WHAT IS THE DIFFERENCE BETWEEN SPINAL SHOCK AND NEUROGENIC SHOCK?

- **Spinal shock** is mainly a loss of motor and sensory activity accompanied by loss of reflexes (flaccid paralysis, areflexia) caudal to the site of injury
- **Neurogenic shock** is mainly hypotension and bradycardia due to loss of sympathetic tone

SPINAL SHOCK :

- Transient reflex depression of cord function below level of injury
- Initially hypertension due to release of catecholamines
- Followed by hypotension
- Flaccid paralysis
- Bowel and bladder involved
- Sometimes priaprism develops
- Symptoms last several hours to days, or even weeks

Phases

Phase	Time	Physical exam findings	Underlying physiological event
1	0-1 days	Areflexia	Loss of descending facilitation
2	1-3 days	Initial reflex return	Denervation supersensitivity
3	1-4 weeks	Hyperreflexia	Axon-supported synapse growth
4	1-12 months	Hyperreflexia, Spasticity	Soma-supported synapse growth

http://www.nature.com/sc/journal/v42/n7/full/3101603a.html

NEUROGENIC SHOCK:

- Triad of i) hypotension
  ii) bradycardia
  iii) hypothermia
- More commonly in injuries above T_6
- Secondary to disruption of sympathetic outflow from $T_1 – L_2$

NEUROGENIC SHOCK:

- Loss of vasomotor tone – pooling of blood
- Loss of cardiac sympathetic tone – bradycardia
- Blood pressure will not be restored by fluid infusion alone
- Massive fluid administration may lead to overload and pulmonary edema
- Vasopressors may be indicated
- Atropine used to treat bradycardia

NEUROGENIC SHOCK

- Seen in cervical injuries
- Due to interruption of the **sympathetic input** from hypothalamus to the cardiovascular centers
- Hallmark: ***hypotension*** (due to vasodilation, due to loss of sympathetic tonic input) is associated with ***bradycardia*** (not tachycardia, the usual response), due to inability to convey the information to the vasomotor centers in the spinal cord

SO WHAT DO YOU EXPECT WITH A CERVICAL LESION?

- Quadriplegia or quadriparesis
- Bowel/bladder retention (spastic)
- Various degrees of breathing difficulties
- Neurogenic and/or spinal shock

LOW CERVICAL INJURIES (C6-T1)

- Usually able to breathe, although occasionally cord swelling can lead to temporary C3-C5 involvement (need mechanical ventilation)
- The level can be determined by physical exam

THORACIC INJURIES (T2-L1)

- Paraparesis or paraplegia
- UMN (upper motor neuron) signs

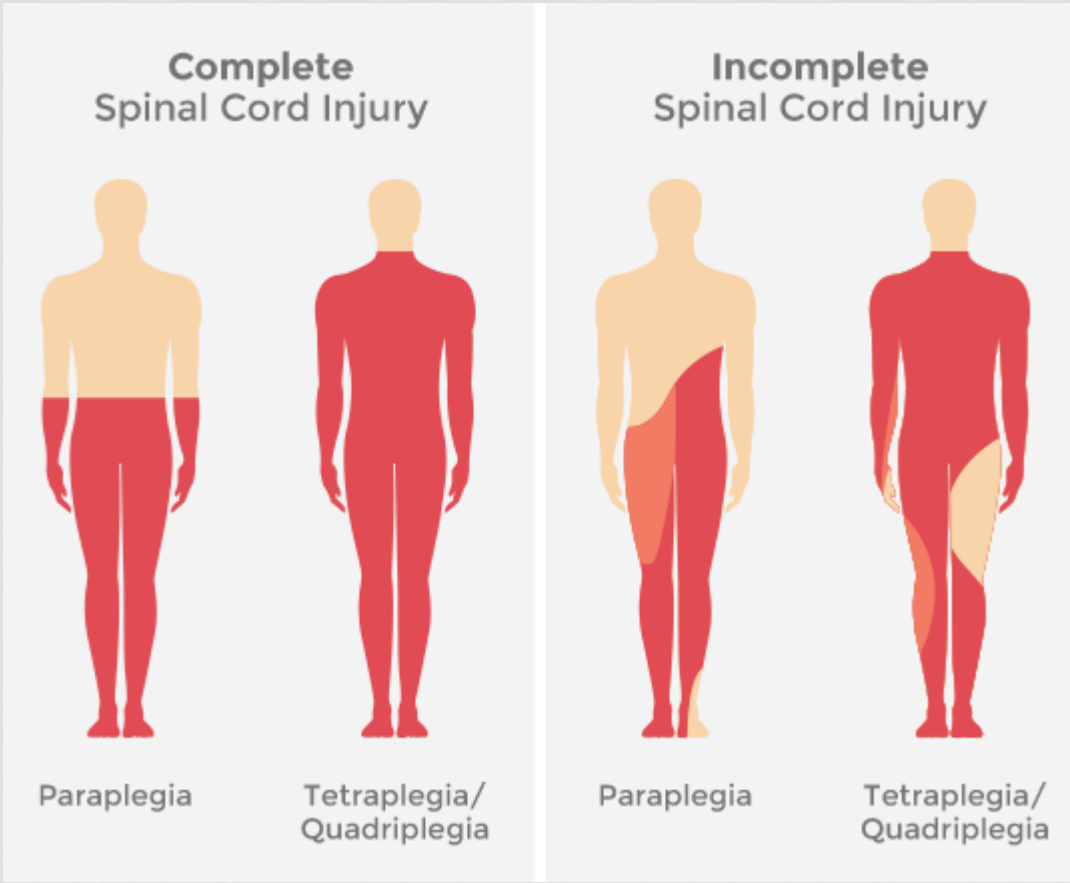
CAUDA EQUINA INJURIES (L2 OR BELOW)

- Paraparesis or paraplegia
- LMN (lower motor neuron) signs
- Thigh flexion is almost always preserved to some degree

WHAT IS AN *INCOMPLETE* LESION?

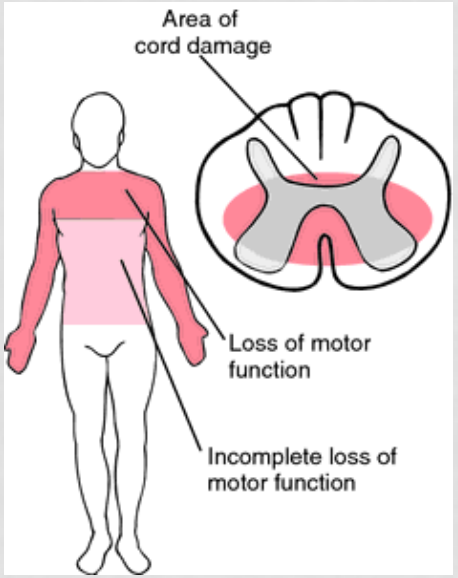
ASIA IMPAIRMENT SCALE

- **A = Complete:** No motor or sensory function is preserved in the sacral segments S4-S5.
- **B = Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.
- **C = Incomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.
- **D = Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.
- **E = Normal:** motor and sensory function are normal

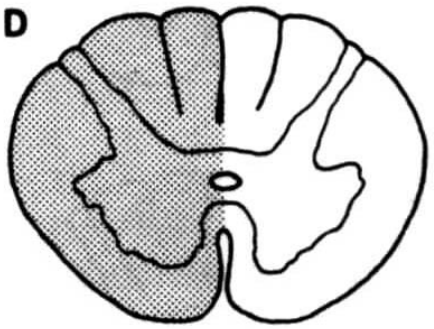


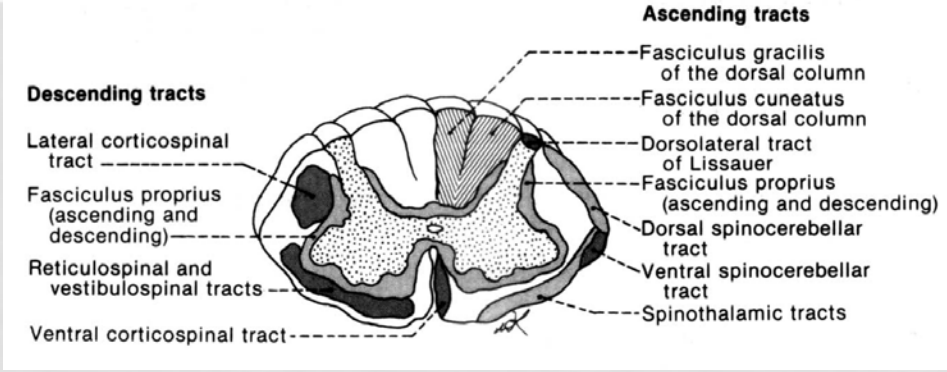
WHAT IS THE CENTRAL CORD SYNDROME?

- Cervical spinal cord involvement with arms more affected than legs
- May occur with trauma, tumors, infections, etc
- Traumatic lesions tend to improve in 1-2 weeks
- Surgical decompression may be indicated if there is spinal stenosis



BROWN-SEQUARD SYNDROME





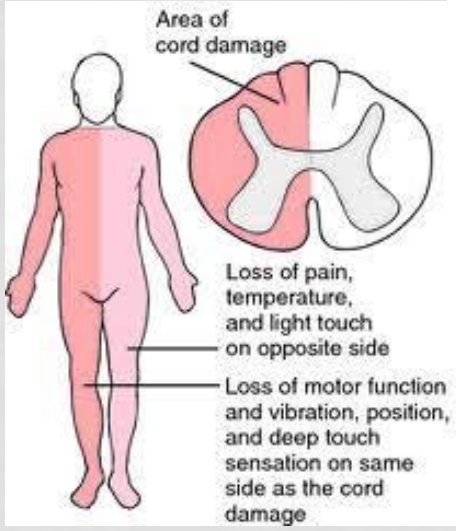


Table 7-4. Clinical Features of the Brown-Séquard Syndrome (Spinal Cord Hemisection)

Clinical Findings	Anatomical Basis
Contralateral effects	
Loss of pain and temperature sensation caudal to lesion	Interruption of spinothalamic tract
Ipsilateral effects	
Paralysis of voluntary movements caudal to level of lesion; hyperreflexia, spasticity, and extensor toe sign	Interruption of lateral corticospinal tract
Loss of vibration, position sense, form perception, and two-point discrimination	Interruption of dorsal columns
Segmental weakness and atrophy	Destruction of ventral motoneurons at level of lesion
Segmental anesthesia	Destruction of dorsal rootlets at level of lesion
Loss of sweating caudal to level of lesion, and, if lesion is cervical, ipsilateral Horner's syndrome (pupilloconstriction, ptosis, and hemifacial anhidrosis)	Interruption of descending autonomic fibers in ventral column

INITIAL MANAGEMENT IN SCI

- **Immobilization**
  - Rigid collar
  - Sandbags and straps
  - Spine board
  - Log-roll to turn
- **Prevent hypotension**
  - Pressors: Dopamine, not Neosynephrine
  - Fluids to replace losses; do not overhydrate
- **Maintain oxygenation**
  - O_2 per nasal canula
  - If intubation is needed, do NOT move the neck

MANAGEMENT IN THE HOSPITAL

- **NGT** (Naso-Gastric-Tube) to suction
  - Prevents aspiration
  - Decompresses the abdomen (paralytic ileus is common in the first days)
- **Foley catheter** (a thin, sterile tube inserted into the bladder to drain urine)
  - Urinary retention is common
- **Methylprednisolone** (Solu-Medrol)
  - Only if started within 8 hours of injury
  - Exclusion criteria
    - Cauda equina syndrome
    - GSW
    - Pregnancy
    - Age <13 years
    - Patient on maintenance steroids

LONG TERM CARE

- Rehab for maximizing motor function
- Bladder/bowel training
- Psychological and social support

MOTOR: HOW DO YOU TEST EACH SEGMENT?

Table 26-7 ASIA motor scoring system
(EXTREMITIES)

RIGHT grade	Segment	Muscle	Action to test	LEFT grade
0-5	C5	deltoid or biceps	shoulder abduction or elbow flexion	0-5
0-5	C6	wrist extensors	cock up wrist	0-5
0-5	C7	triceps	elbow extension	0-5
0-5	C8	flexor digitorum prof	squeeze hand	0-5
0-5	T1	hand intrinsics	abduct little finger	0-5
0-5	L2	iliopsoas	flex hip	0-5
0-5	L3	quadriceps	straighten knee	0-5
0-5	L4	tibialis anterior	dorsiflex foot	0-5
0-5	L5	EHL	dorsiflex big toe	0-5
0-5	S1	gastrocnemius	plantarflex foot	0-5
50	← TOTAL POSSIBLE POINTS →			50
GRAND TOTAL: 100				

American
Spinal
Injury
Association

MOTOR: HOW DO YOU GRADE THE STRENGTH?

https://www.youtube.com/watch?v=vZBpNsFPJVQ

Grade	Strength	
0	no contraction	
1	flicker or trace contraction	
2	movement with gravity eliminated	
3	movement against gravity	
4	movement against resistance	4- slight resistance 4 moderate resistance 4+ strong resistance
5	normal strength	

https://www.youtube.com/watch?v=tzsUE-uuxqQ

→ https://www.youtube.com/watch?v=suXg-eOM-ZY

→ https://www.youtube.com/watch?v=Axlo5lRQR5M

→ https://www.youtube.com/watch?v=-v30NYgd2ao

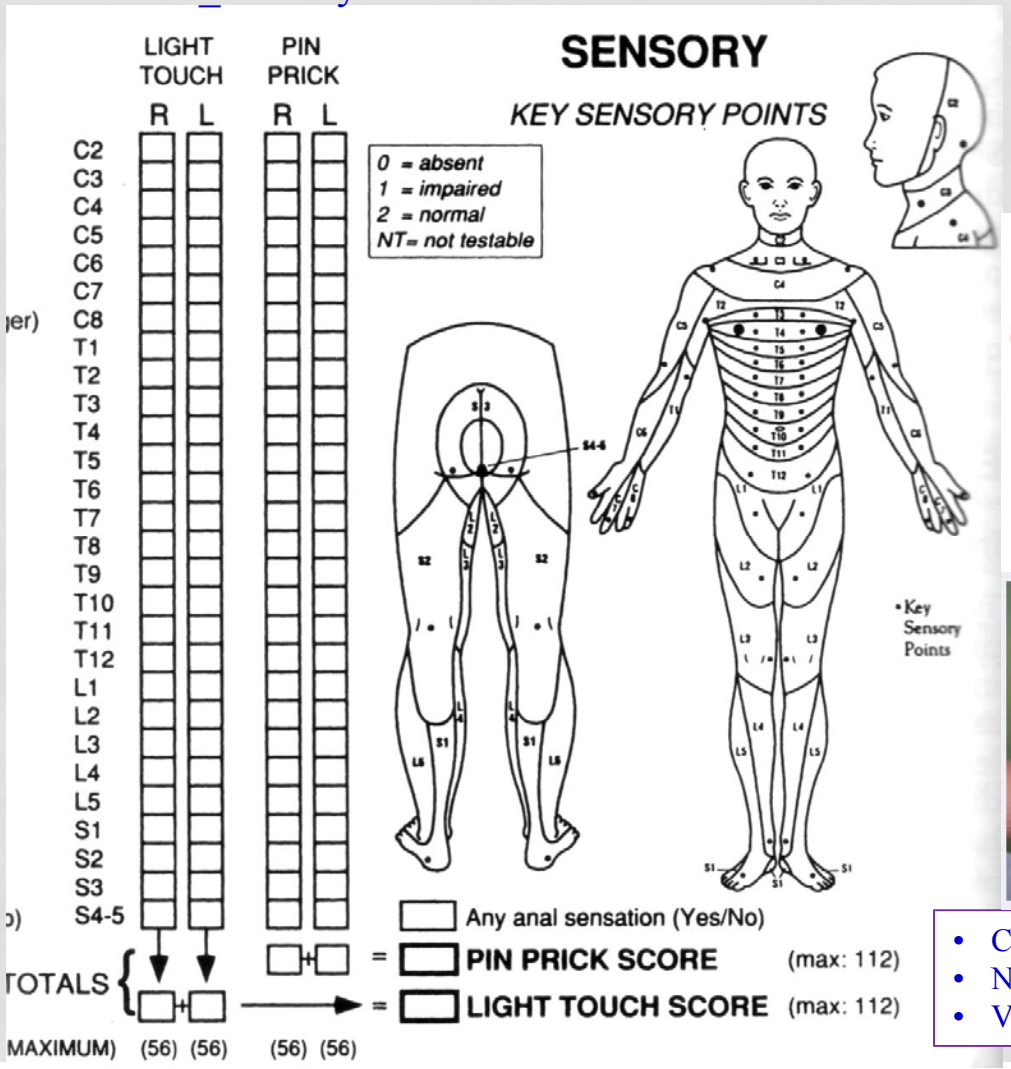
https://www.youtube.com/watch?v=eYXP54vKvCE

SENSORY: HOW DO YOU DETERMINE THE LEVEL?

https://www.youtube.com/watch?v=f2U_-K6dByw

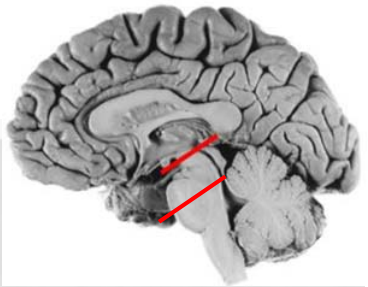
Table 26-6 Key sensory landmarks

Level	Dermatome
C4	shoulders
C6	thumb
C7	middle finger
C8	little finger
T4	nipples
T6	xiphoid
T10	umbilicus
L3	just above patella
L4	medial malleolus
L5	great toe
S1	lateral malleolus
S4-5	peri-anal

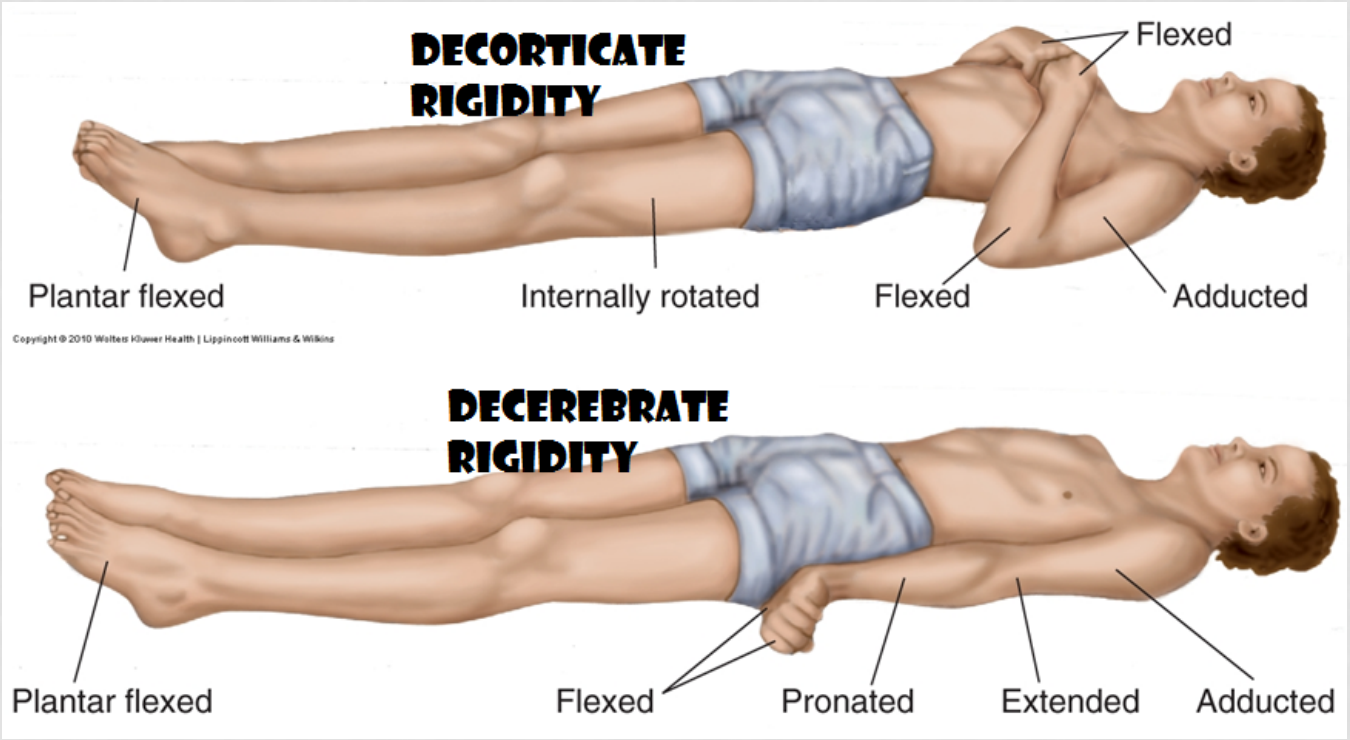


- Cotton ball, brush
- Neurologic pin
- Vibrating tuning fork

DECEREBRATE AND DECORTICATE RIGIDITY

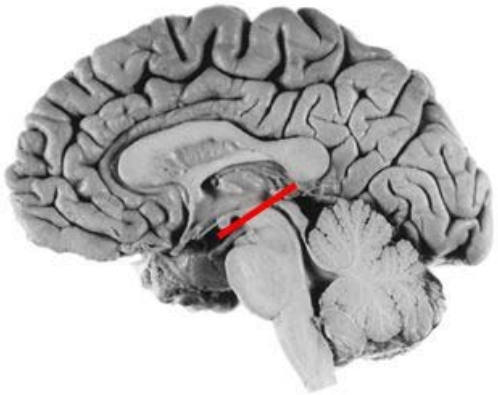


- Injury at the level of **pons** or **mesencephalon**

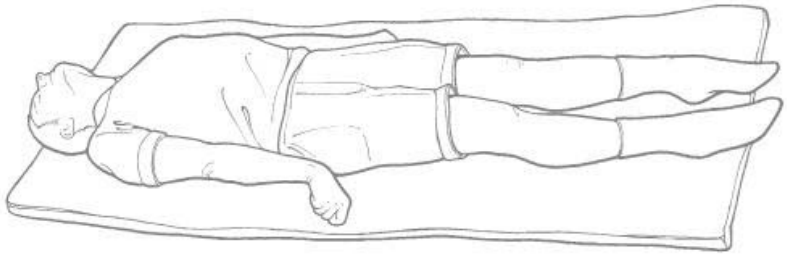


- Brainstem & its connections to spinal cord are responsible for these postures

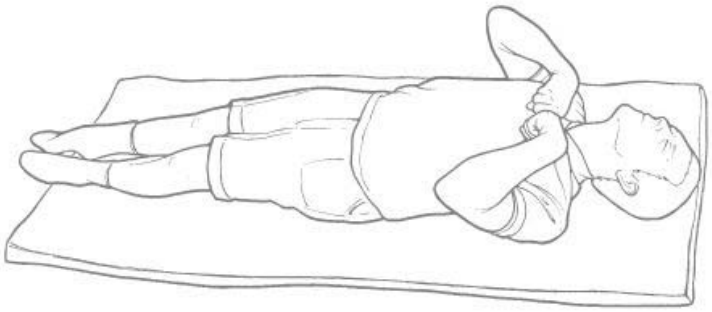
Forebrain connections removed



Lesions separate forebrain from brainstem



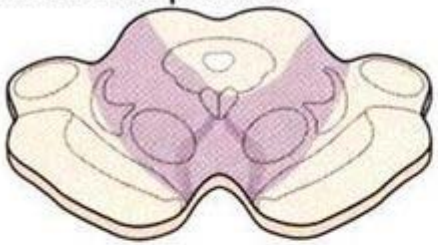
Decerebrate (all 4 limbs extended – extensor rigidity)



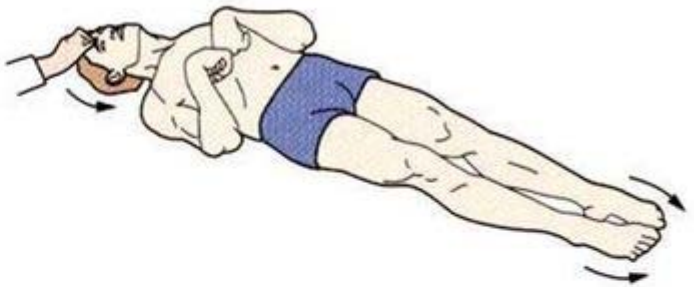
Decortitate (UE's flexed, Le's extended)

HT 24-13, 24-15

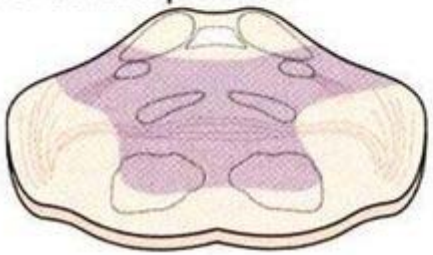
Upper midbrain damage
Decorticate posture

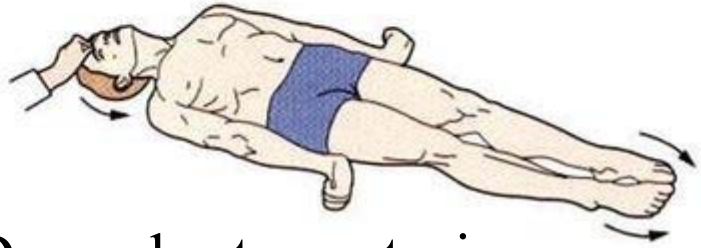


Decorticate posturing



Upper pontine damage
Decerebrate posture





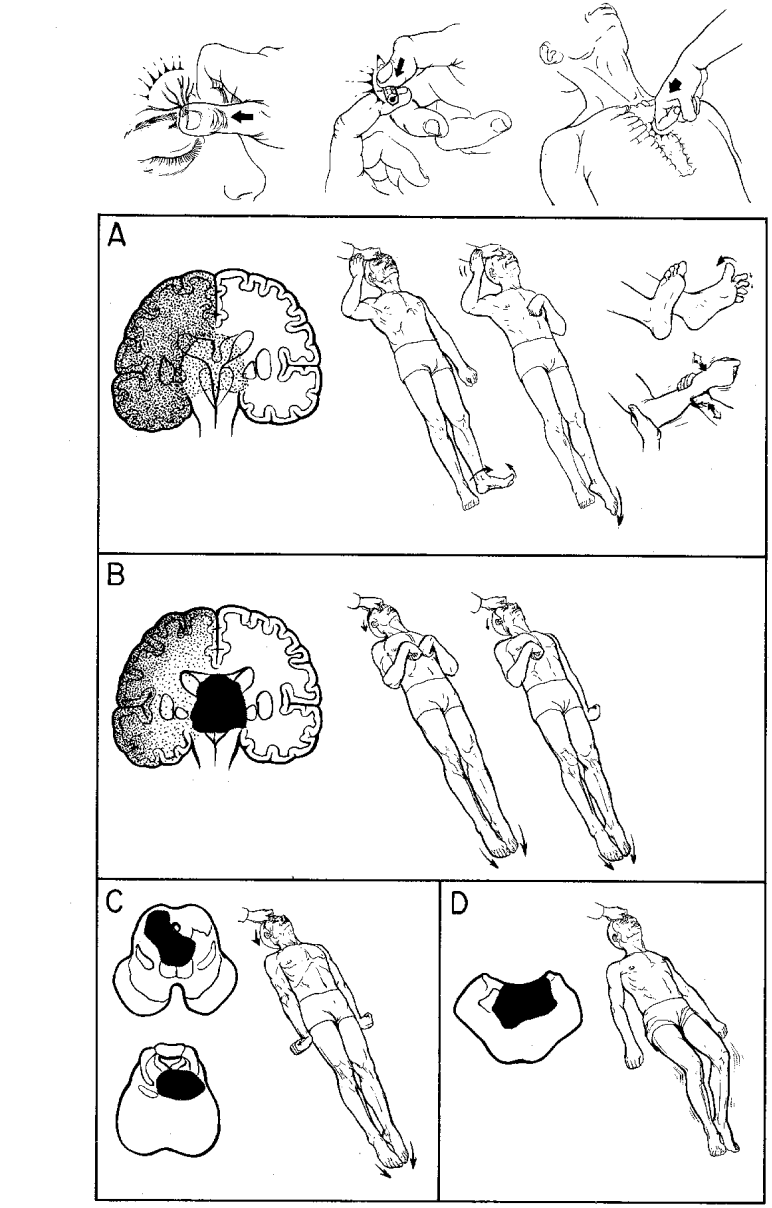
Decerebrate posturing

A. diffuse cortical-thalamic damage: patient exhibits **contralateral flexion of arms** and **extension of legs = DECORTICATE RIGIDITY**

B. focal cerebral lesion with central mass effect (pushes on deeper structures) **decorticate rigidity** = flexion/arms and extension/legs

C. Lesion lies between superior and inferior colliculi = **bilateral extension of upper and lower limbs = DECEREBRATE RIGIDITY**

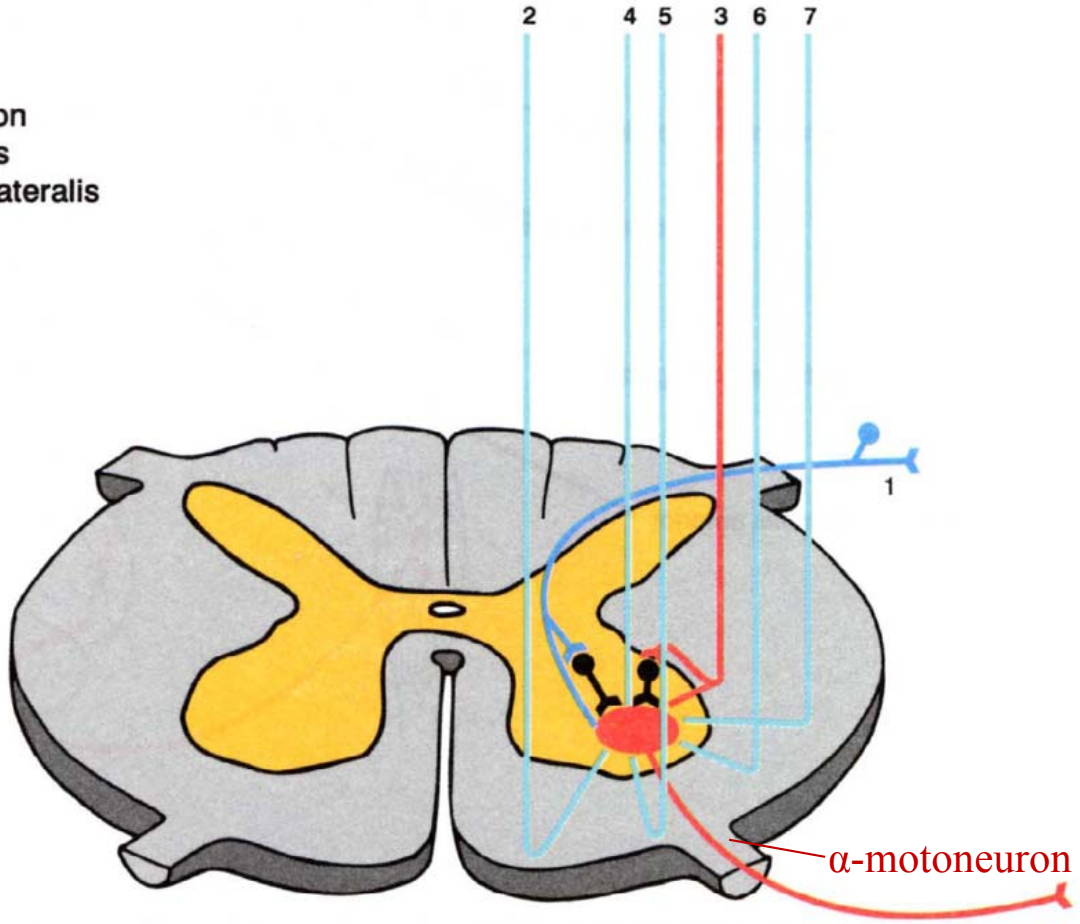
D. Lesion in medulla kind of **"wash"** motor-wise



Motor responses to noxious stimulation in patients with acute cerebral dysfunction. Noxious stimuli can be delivered with minimal trauma to the supraorbital ridge, the nail bed, or the sternum as illustrated at top. Levels of associated brain dysfunction are roughly indicated at left. The text provides details.

PROJECTIONS TO ALPHA-MOTONERON

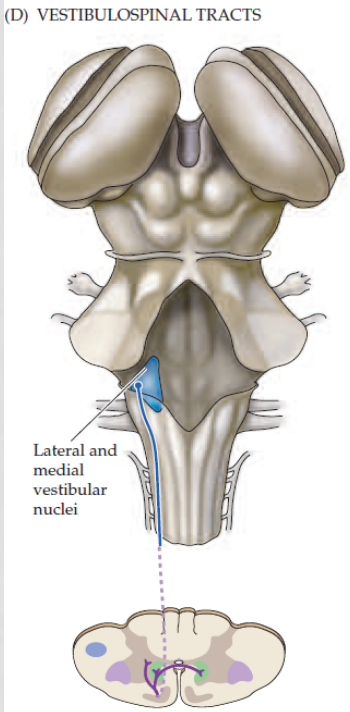
1 - primarni aferentni neuron
2 - tractus vestibulospinalis
3 - tractus corticospinalis lateralis
4 - tractus rubrospinalis
5 - tractus olivospinalis
6 - tractus tectospinalis
7 - tractus reticulospinalis

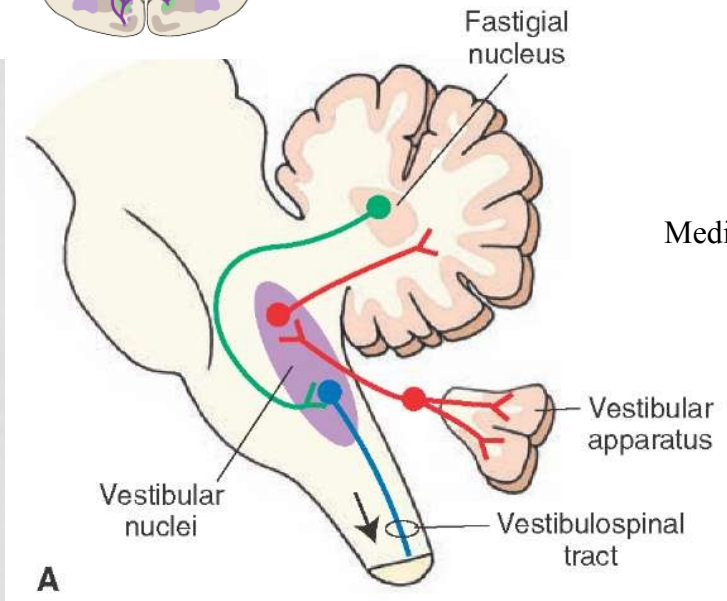


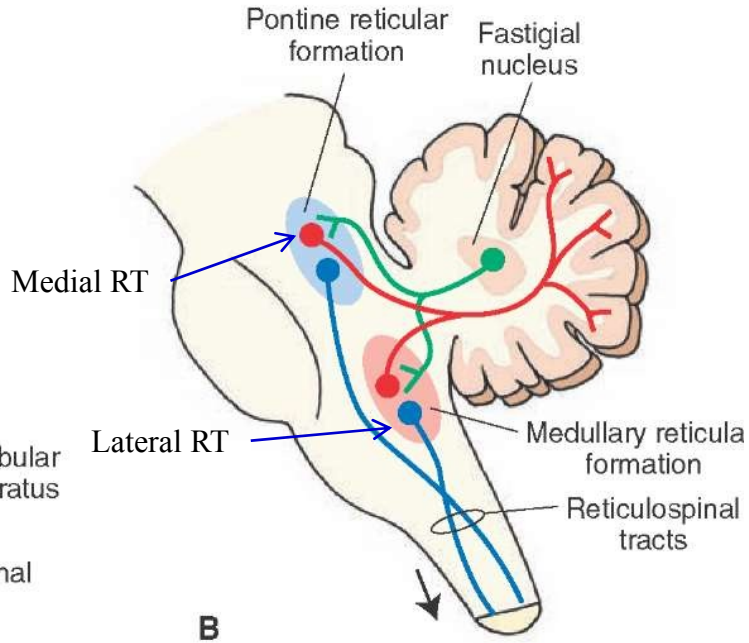
Slika 33-2. Na α-motoneuron djeluju četiri velike skupine sustava aferentnih aksona: a) primarna aferentna vlakna (1), b) izravni kortikospinalni put (3), c) silazni motorički putovi iz moždanog debla – npr. vestibulospinalni (2), retikulospinalni (7), rubrospinalni (4); d) silazni monoaminski (rafespinalni i ceruleospinalni) putovi (nisu prikazani na slici).

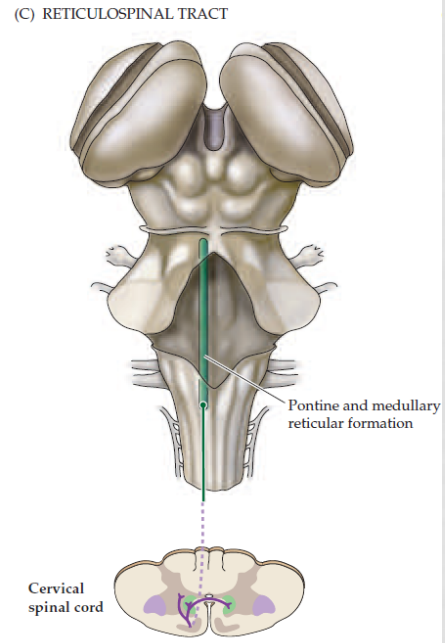
VESTIBULOSPINAL AND RETICULOSPINAL TRACTS

- Tractus vestibulospinalis lateralis (+E, lower extr.)
- Tractus vestibulospinalis medialis (-, neck, head)
- Tractus reticulospinalis medialis (pontine)(+E)
- Tractus reticulospinalis lateralis (medullary)(-E, +F)









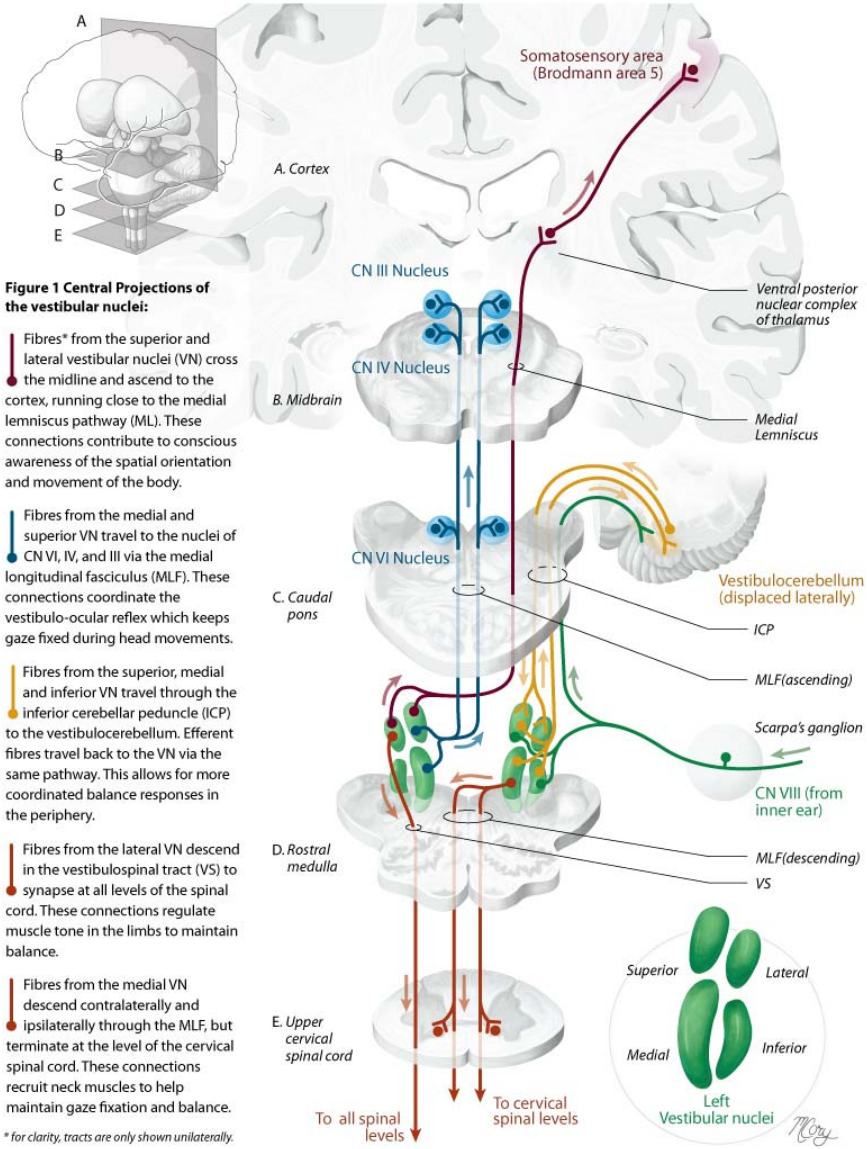


Figure 1 Central Projections of the vestibular nuclei:

Fibres* from the superior and lateral vestibular nuclei (VN) cross the midline and ascend to the cortex, running close to the medial lemniscus pathway (ML). These connections contribute to conscious awareness of the spatial orientation and movement of the body.

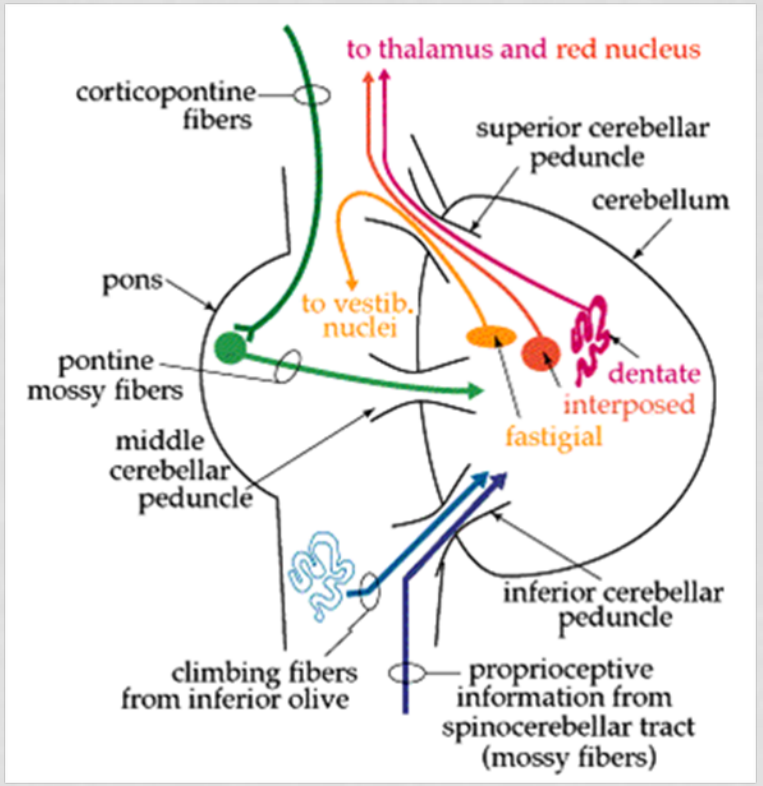
Fibres from the medial and superior VN travel to the nuclei of CN VI, IV, and III via the medial longitudinal fasciculus (MLF). These connections coordinate the vestibulo-ocular reflex which keeps gaze fixed during head movements.

Fibres from the superior, medial and inferior VN travel through the inferior cerebellar peduncle (ICP) to the vestibulocerebellum. Efferent fibres travel back to the VN via the same pathway. This allows for more coordinated balance responses in the periphery.

Fibres from the lateral VN descend in the vestibulospinal tract (VS) to synapse at all levels of the spinal cord. These connections regulate muscle tone in the limbs to maintain balance.

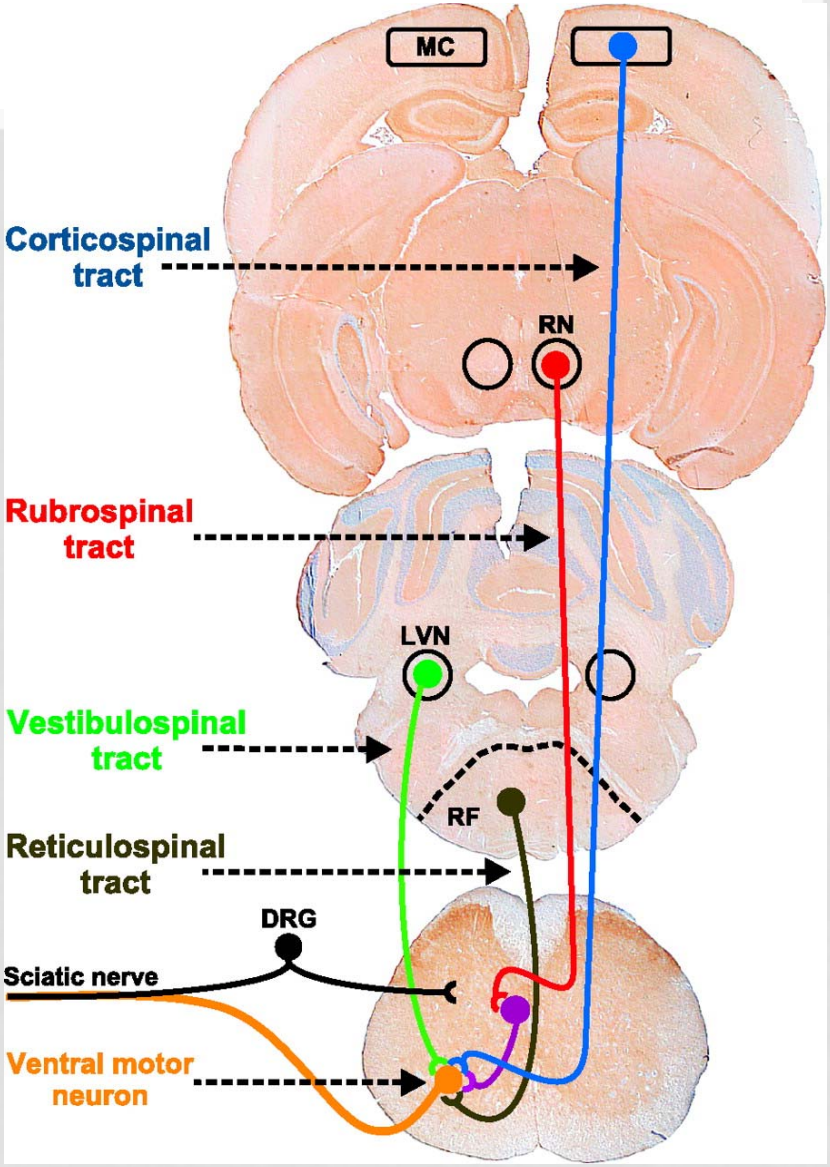
Fibres from the medial VN descend contralaterally and ipsilaterally through the MLF, but terminate at the level of the cervical spinal cord. These connections recruit neck muscles to help maintain gaze fixation and balance.

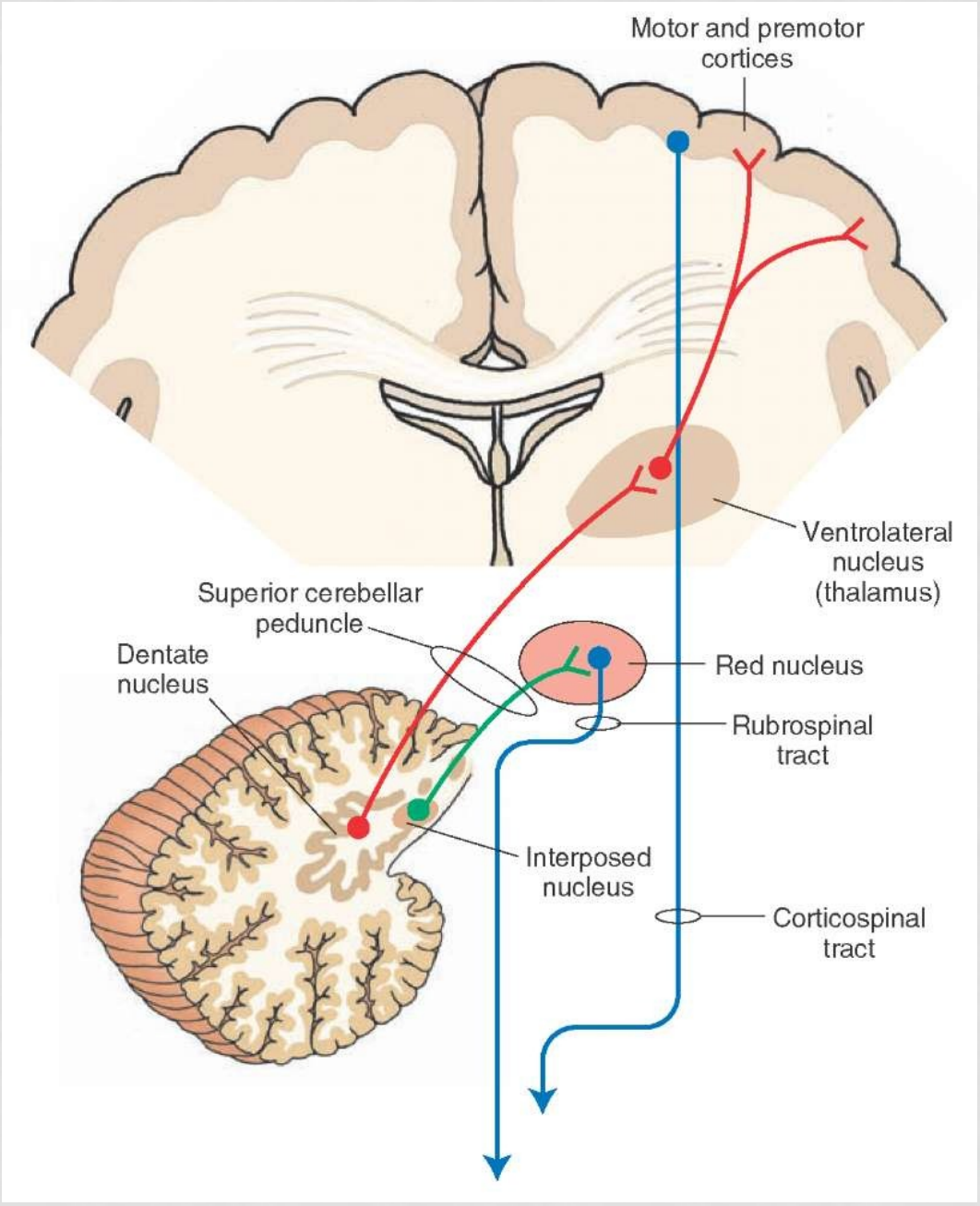
** for clarity, tracts are only shown unilaterally.*



RUBROSPINAL AND CORTICOSPINAL TRACTS

- Field MI and MII
- Cerebelum
- Nucleus ruber



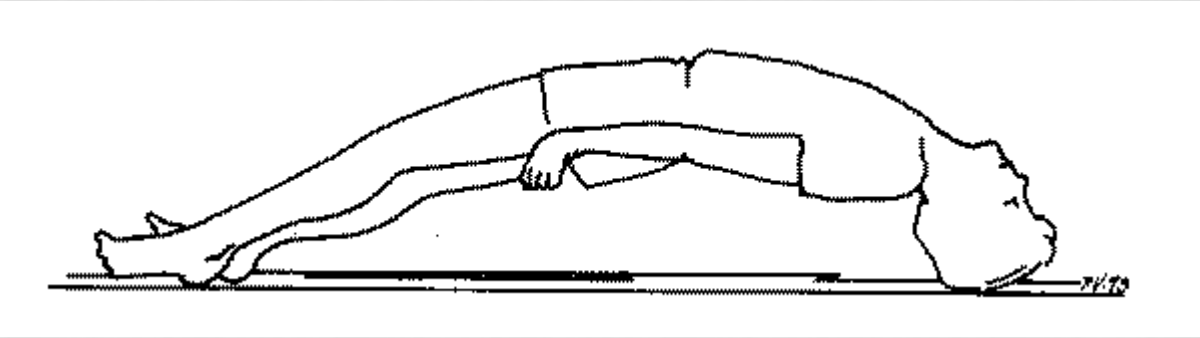

Motor and premotor cortices
Ventrolateral nucleus (thalamus)
Superior cerebellar peduncle
Dentate nucleus
Red nucleus
Rubrospinal tract
Interposed nucleus
Corticospinal tract

SUMMARY: WHERE IS THE LEVEL OF INJURY?

- In decerebrate rigidity?
- In decorticate rigidity?

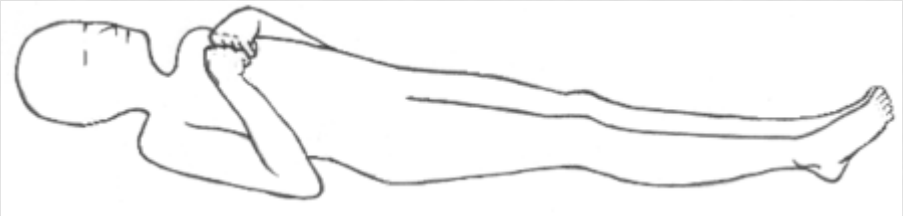
DECEREBRATE RIGIDITY

- Injury is at the level of **rostral pons**
- Corticospinal and rubrospinal projections are interrupted
- Hyperextension, opisthotonus (spasm of the muscles causing backward arching of the head, neck, and spine, as in severe tetanus, some kinds of meningitis, strychnine poisoning).



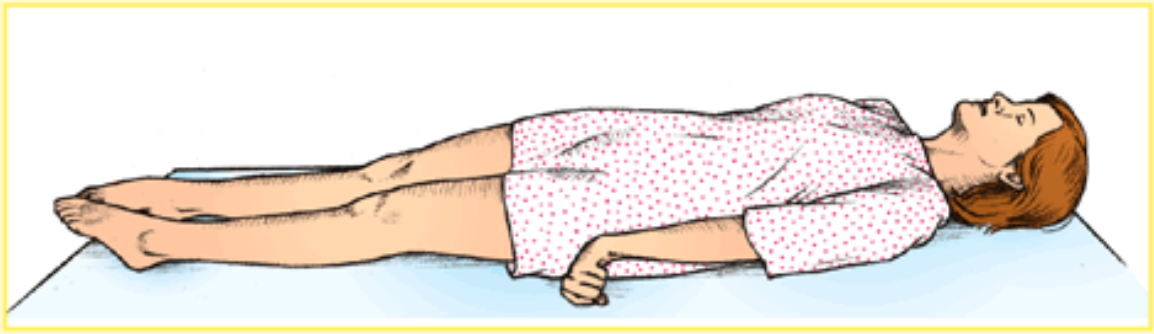
DECORTICATE RIGIDITY

- Injury is at the level of **rostral mesencephalon**
- Corticospinal projections are interrupted, rubrospinal are preserved
- Hyperextension of the lower extremities, flexion of upper extremities

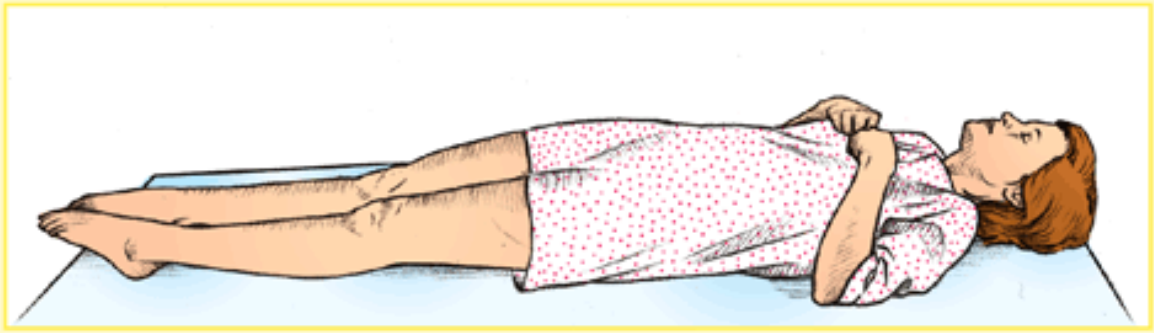


Comparing decerebrate and decorticate postures

Decerebrate posture results from damage to the upper brain stem. In this posture, the arms are adducted and extended, with the wrists pronated and the fingers flexed. The legs are stiffly extended, with plantar flexion of the feet.



Decorticate posture results from damage to one or both corticospinal tracts. In this posture, the arms are adducted and flexed, with the wrists and fingers flexed on the chest. The legs are stiffly extended and internally rotated, with plantar flexion of the feet.



USEFUL VIDEOS

- https://www.youtube.com/watch?v=RmLJ_LRKAGE
- https://www.youtube.com/watch?v=yZUE2Dvf1Q4
- http://neuroscience.uth.tmc.edu/s3/chapter06.html
  (Neuroscience, Chapter 6: Disorders of the Motor System)

THE END