SPINAL CORD INJURY

Incidence

- 10 - 15 per million
- 18 - 35 years
- Male - 3:1
- RTA 51% - cars
- Domestic 16%
- Industrial 11%
- Sports 16% - diving incidents
- Self harm 5%

Types

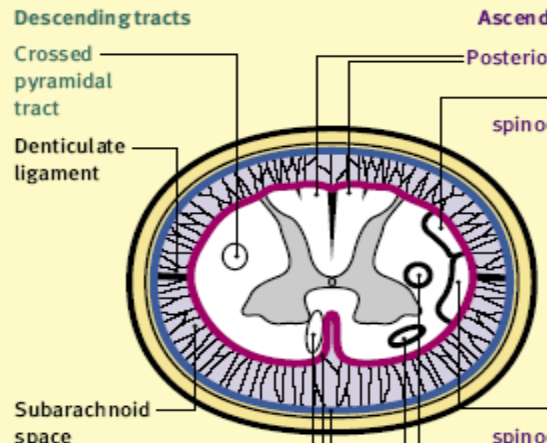
- Cervical 40%
- Thoracic 10%
- Lumbar 3%
- Dorso lumbar 35%
- Any 14%
- Injury – osseus/ligamentousus
- SCIWORA Syndrome
  (Spinal Cord Injury w/o Radiologic Abnormality)

Anatomy

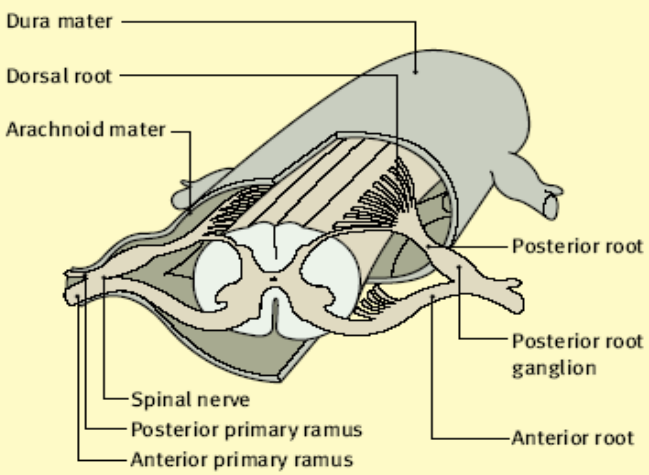
- Spinal cord ends below lower border of L1
- Cauda equina is below L1
- Mid dorsal spinal cord & neural canal space are of same diameter hence prone for complete lesion

- Mechanical injury - early ischaemia, cord edema - cord necrosis

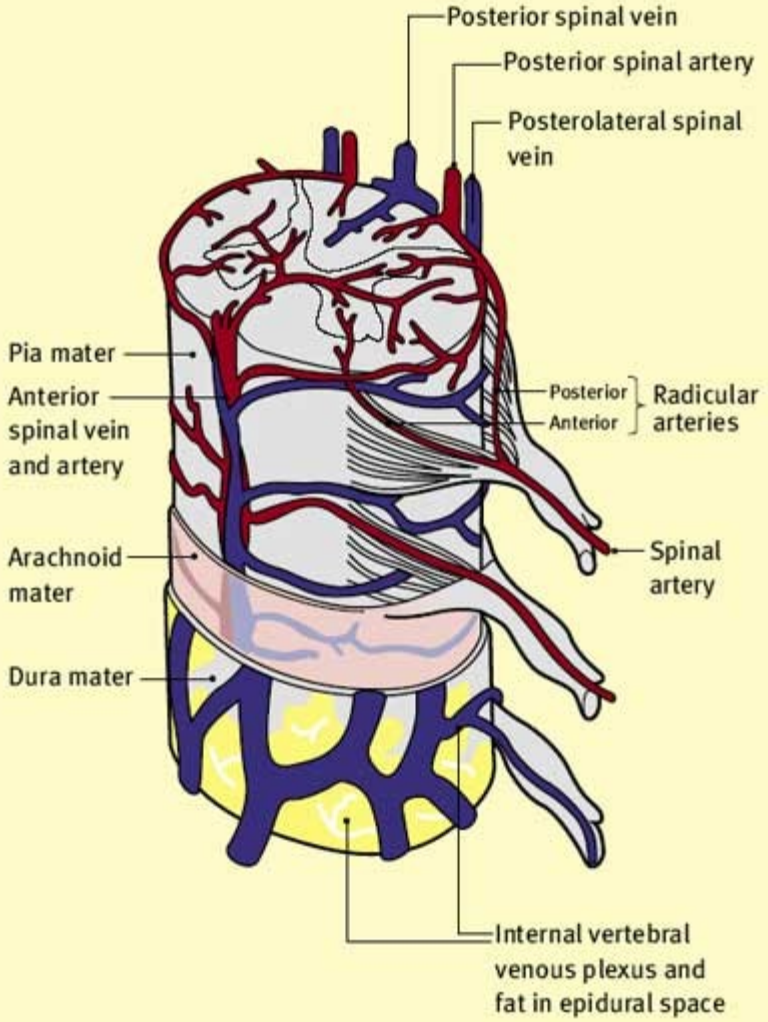
Cross-section of spinal cord showing meninges and denticulate ligaments and ascending and descending tracts



Spinal cord



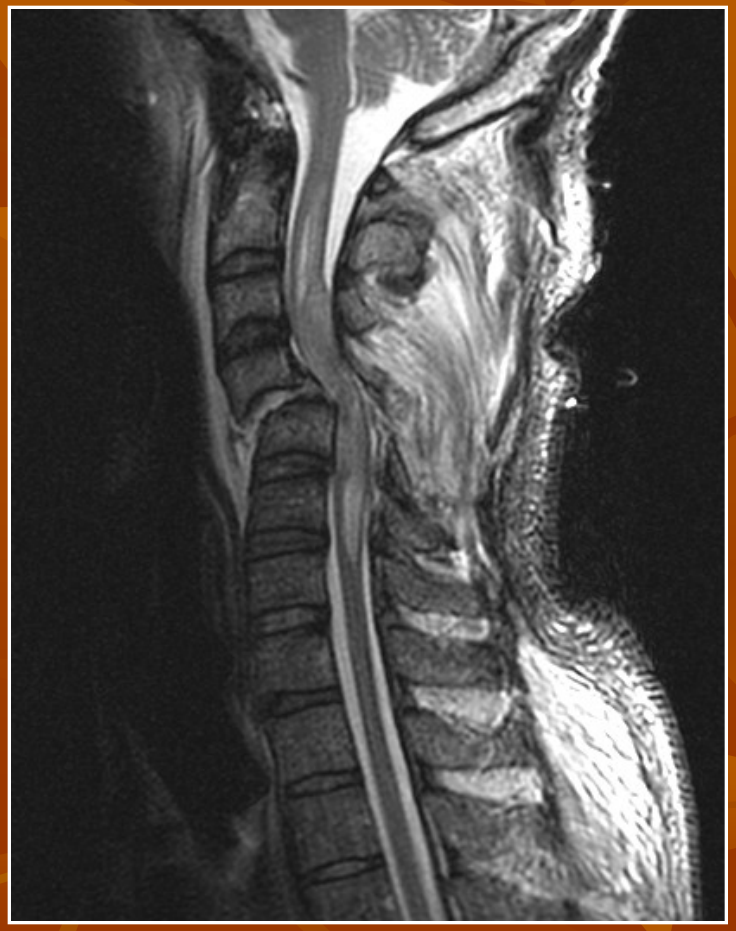
Arterial supply and venous drainage of the spinal cord



Spinal Cord Injury
pathophysiology

Primary injury

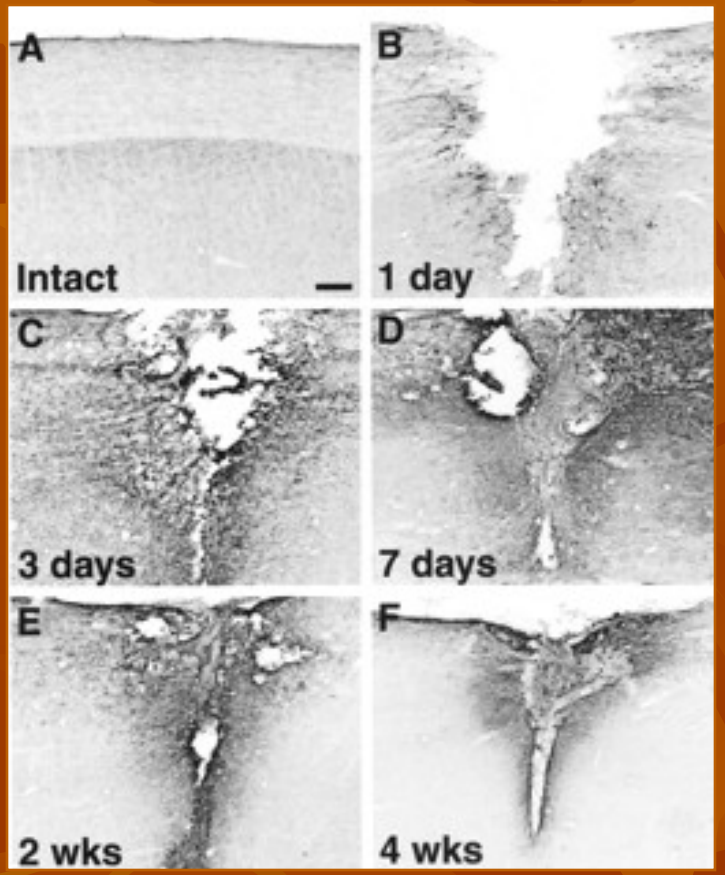
- Initial insult to cord
- Local deformation
- Energy transformation



Spinal Cord Injury
pathophysiology

Secondary injury

- **Biochemical cascade**
- **Cellular processes**



Theories for secondary injuries

- *Lipid Peroxidation and Free Radicals*
- *Abnormal Electrolyte Fluxes and Excitotoxicity*
- *Abnormal Vascular Perfusion*
- *Abnormal Intracellular Sodium Concentration*
- *Associated Inflammatory and Immune Response*

Level of Spinal injury

- Neurological level is at the most lowest segment with normal motor & sensory function

- Difficult to determine :

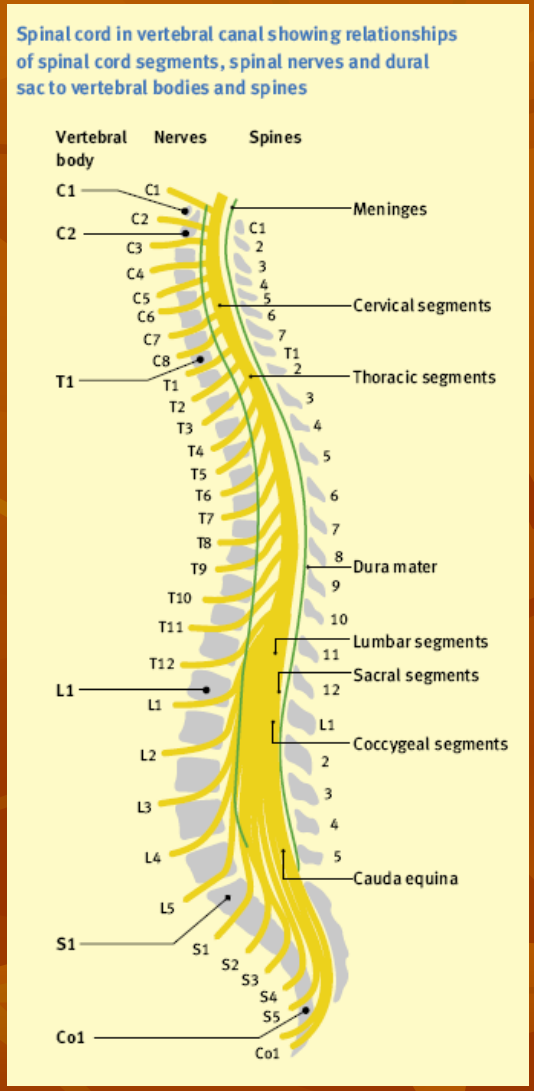
  - as most muscle efferents receive fibres from more than one level

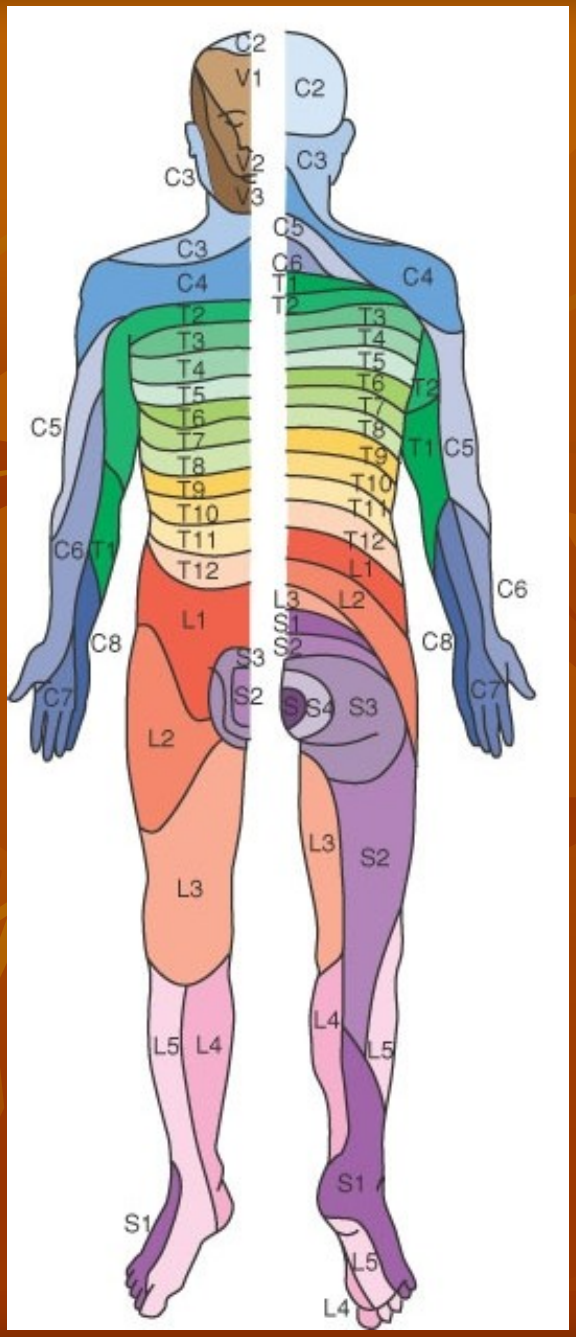
  - Closed cord lesions may extend over several cms.

  - Dermatomes have imprecise boundaries.

Cord level

- C2 – C7 = add +1 for cord level
- T1 – T6 = add +2
- T7 – T9 = add +3
- T10 = L1, L2 level
- T11 = L3, L4 level
- L1 = sacro coccygeal segments




C2
V1
C2
C3
V2
C3
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C3
C6
C4
T1
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T11
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C8
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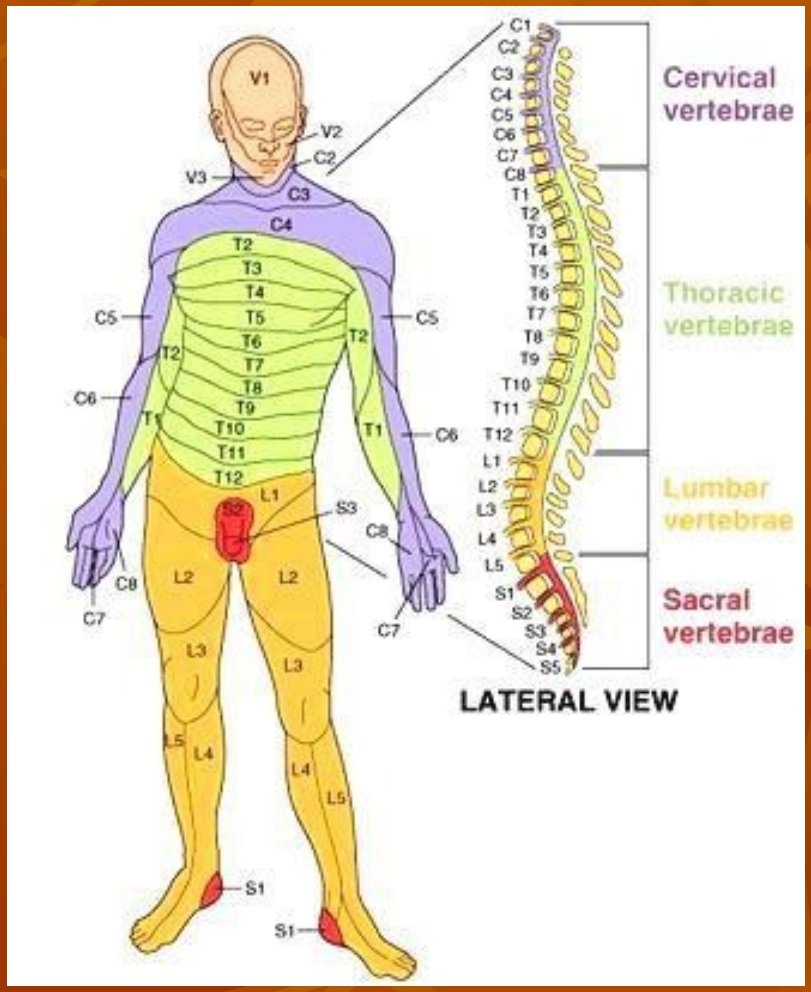
V1
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T12
L1
L2
L3
L4
L5
S1
S2
S3
S4
S5
C5
C6
C7
C8
T1
Cervical vertebrae
Thoracic vertebrae
Lumbar vertebrae
Sacral vertebrae
LATERAL VIEW

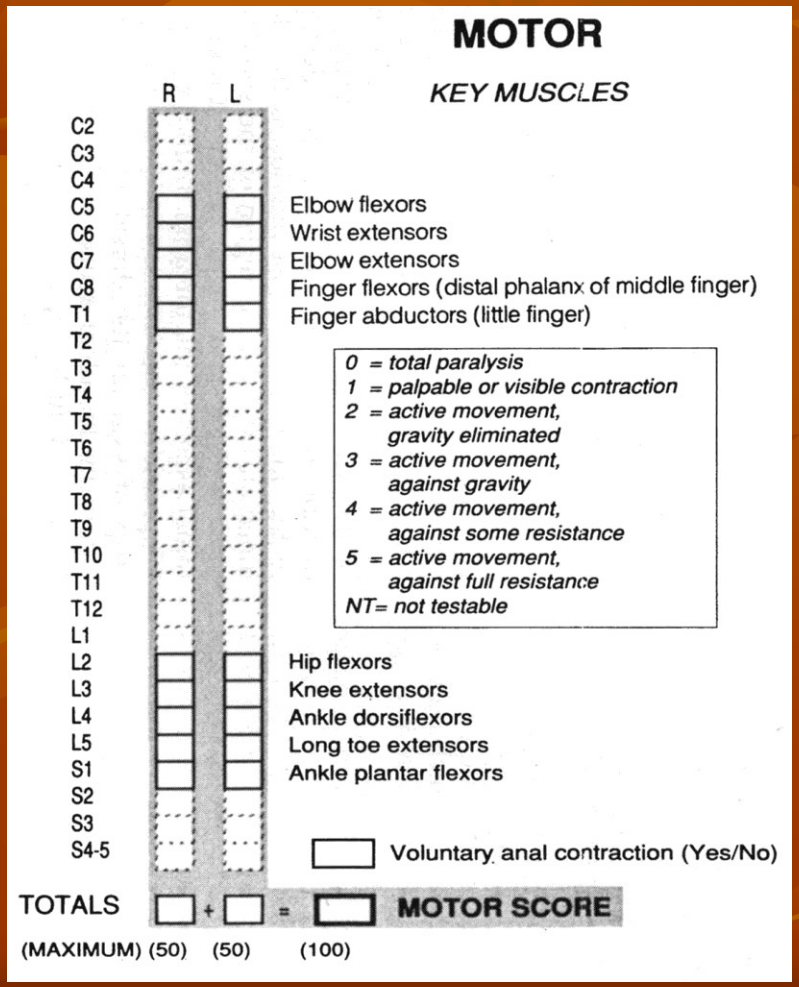
Table 3. Cervical And Lumbosacral Dermatomes And Myotomes.

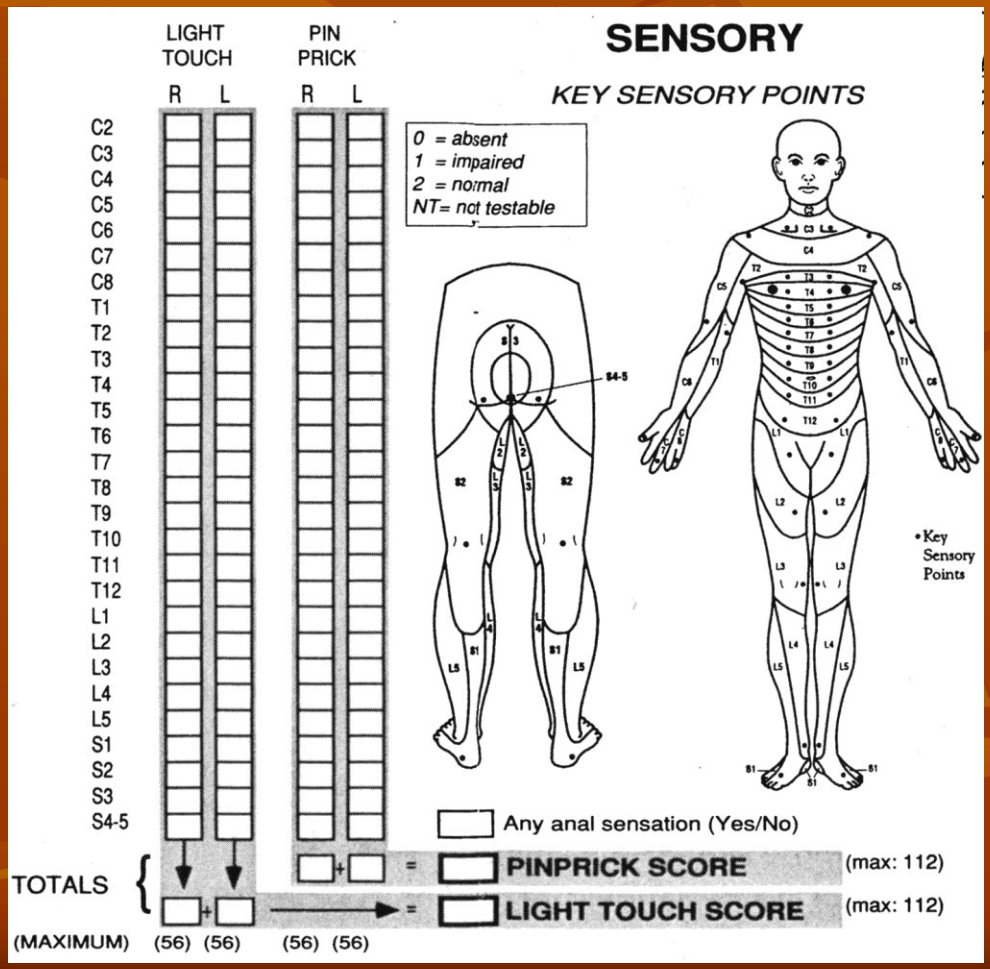
Spinal level	Key sensory area for dermatomal testing	Myotome
C5	Radial antecubital fossa	Elbow flexors (biceps*, brachialis, and brachioradialis*)
C6	Thumb	Wrist extensors (extensor carpi radialis longus and brevis)
C7	Middle finger	Elbow extensors (triceps*)
C8	Little finger	Finger flexors* (distal phalanx—flexor digitorum profundus)
T1	Ulnar antecubital fossa	Hand intrinsics (interossei)
L2	Mid-anterior thigh	Hip flexors (iliopsoas)
L3	Medial femoral condyle	Knee extensors* (quadriceps)
L4	Medial malleolus	Ankle dorsiflexors (tibialis anterior)
L5	Dorsal second/third toe web space	Long toe extensors (extensor hallucis longus)
S1	Lateral heel	Ankle plantar flexors* (gastrocnemius, soleus)

* Commonly tested reflexes

ASIA Score

- Based on key muscle strength & key sensory points
- Useful for following improvement or deterioration

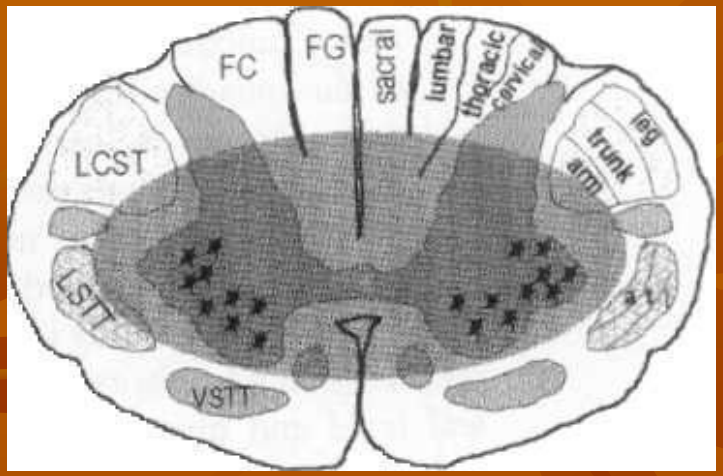




Degrees of injury

- Complete - flaccid paralysis + total loss of sensory & motor functions

- Incomplete - mixed loss
  - Anterior sc syndrome
  - Posterior sc syndrome
  - Central cord syndrome
  - Brown sequard's syndrome
  - Cauda equina syndrome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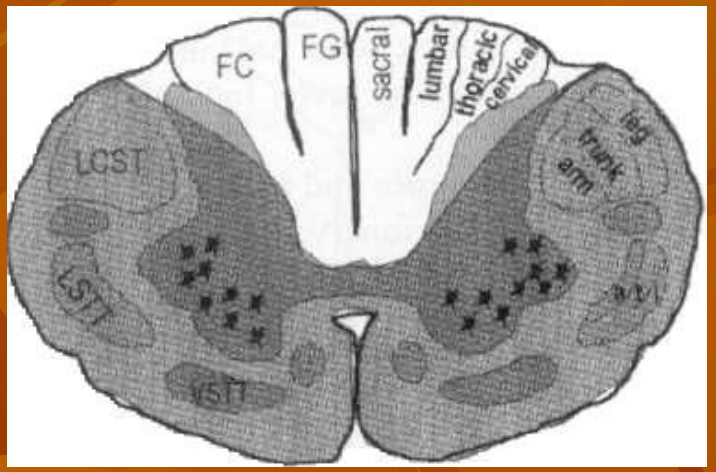
sacral
lumbar
thoracic
cervical
leg
trunk
arm
LCST
LSTT
VSTT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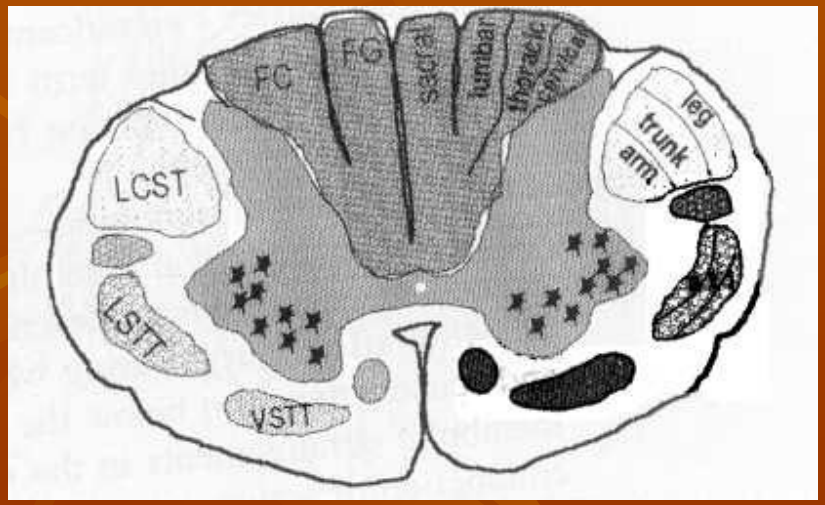
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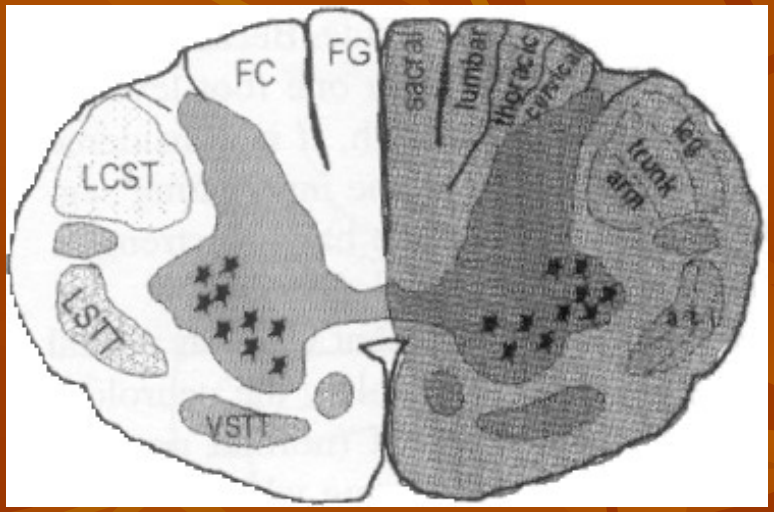
VSTT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lumbar
thoracic
cervical
leg
trunk
arm
LCST
LSTT
VSTT

Anterior spinal cord syndrome

- Flexion rotational force to spine
- Due to compression fracture of vertebral body or anterior dislocation
- Anterior spinal artery compression
- Loss of power, reduced pain and temperature below the lesion.

Posterior cord syndrome

- Hyperextension injuries
- Posterior vertebral body fracture
- Loss of proprioception and vibration sense
- Severe ataxia

Central cord syndrome

- Older age with cervical spondylosis
- Hyperextension with minor trauma

- Cord is compressed by osteophytes from vertebral body against thick ligamentum flavum.

- Damages the central cervical tract

Brown sequards syndrome

- Hemisection of the cord
- Stab injury and lateral mass fractures
- Uninjured side has good power but absent pinprick and temperature
- Ipsilateral weakness and position sense loss
- Contralateral pain and temperature loss

Neurogenic shock

- Lesions above Th6
- Minutes – hours (fall of catecholamines may take 24 hrs)
- Disruption of sympathetic outflow from Th1 - L2
- Unapposed vagal tone
- Peripheral vasodilatation
- Hypotension, Bradycardia & Hypothermia

- BUT consider haemmorhagic shock if – injury below Th6, other major injuries, hypotension with spinal fracture alone without neurological injury.

Spinal shock

- Transient physiological reflex depression of cord function – **‘concussion of spinal cord’**
- Loss anal tone, reflexes, autonomic control within 24-72hr
- Flaccid paralysis bladder & bowel and sustained priapism
- Lasts even days till reflex neural arcs below the level recovers.

Assessment & Managemnt

- Failure to suspect leads to failure to detect injuries
- ABCDE – Logroll and remove the spinal board
- Look for markers of spinal injury
- Secondary survey
- Adequate Xray's
- Emergency treatment
- Surgery
- Definitive care & rehab.

Clinical features

- Pain in the neck or back radiating due to nerve root irritation
- Sensory disturbance distal to neurological level
- Weakness or flaccid paralysis below the level

Signs in an Unconcious patients

- Diaphragmatic breathing
- Neurological shock (Low BP & HR)
- Spinal shock - Flaccid areflexia
- Flexed upper limbs (loss of extensor innervation below C5)
- Responds to pain above the clavicle only
- Priapism

Signs of spinal injury

- Forehead wounds – think of hyperextension injury
- Localized bruise
- Deformities of spine - Gibbus, feel a step & Priapism

Emergeny Care

- Careful manual handling especially if unconcious
- Jaw thrust is safer
- Correct gross spinal deformities
- Call the anaesthetist if diaphragmatic paralysis or RR>35
- Use flexible fibreoptic scopes in unstable fractures
- Ryles tube if abdominal distension causes respiratory probl
- Cathetrize to avoid overstretching of detrusor
- IV fluids – paralytic ileus in first 48hrs.
- Passive movements to rule out fractures
- Small iv doses of opiates

Assessment

- Document the level of injury
- Rule out other injuries – CT/FAST in abdominal injuries as there is paralytic ileus and absent peritioneal irritation.
- Associated injuries in dorsal spine fracture are :
  - Renal injuries
  - Chest and Sternal injuries
  - Wide Mediatinum due to fracture haematoma.
  - Retroperitoneal injuries

Therapy

- *Pharmacologic Interventions*
- *Decompression*
- *Prospects of Spinal Cord Regeneration*

Emergency treatment

- ABCDE
- Keep warm
- Treat if BP<80mmHg & HR <50bpm
- H2 Antagonists & Heparin

The National Acute Spinal Cord Injury Study (NASCIS)

- Methylprednisolone 30mg/kg iv bolus over 15min immediately
- 45minutes after the bolus a 5.4mg/kg/h infusion over 23 hrs in first 3 hours after the injury.
- 5.4mg/kg/hr for 47hrs if 4 - 8hrs following the injury.

- *Steroids*
- Steroids mainly act by prevention of lipid peroxidation by free radicals and membrane stabilisation. They may help prevent apoptosis by checking calcium fluxes, improving vascular perfusion, and are thought to help reduce white matter oedema, and enhance Na/KATPase activity

- *Naloxone*
- Naloxone is an opiate receptor that was included in one treatment arm of the NASCIS studies
- Found to be effective in the subgroup of patients with incomplete spinal cord injuries

- *Gangliosides*
- These are glycosphingolipids at the outer cellular membranes of the central nervous system
- There is some evidence that gangliosides may have neuroprotective action, with more speedy recovery of motor and sensory function in partial cord injuries

- *Calcium Channel Blockers*
- Thought to work by improvement in blood flow via vessel dilatation

- *Antagonists of Glutamate Receptors*
- Works by prevention of excitotoxicity as a result of glutamate accumulation – helps to prevent abnormal sodium and calcium fluxes, which may prove lethal to cells

- *Others*
- Inhibition of cyclo-oxygenase
- Minocycline
- Sodium channel blockers
- Erythropoietin
- Cyclosporin

Acute Neuroprotective Agents

new areas of interest in household drugs

minocycline

DOI: 10.1093/brain/awg178 Advanced Access publication June 4, 2003 *Brain* (2003), **126,** 1628–1637

Neuroprotection by minocycline facilitates significant recovery from spinal cord injury in mice

Jennifer E. A. Wells,[1] R. John Hurlbert,[1] Michael G. Fehlings[2] and V. Wee Yong[1]

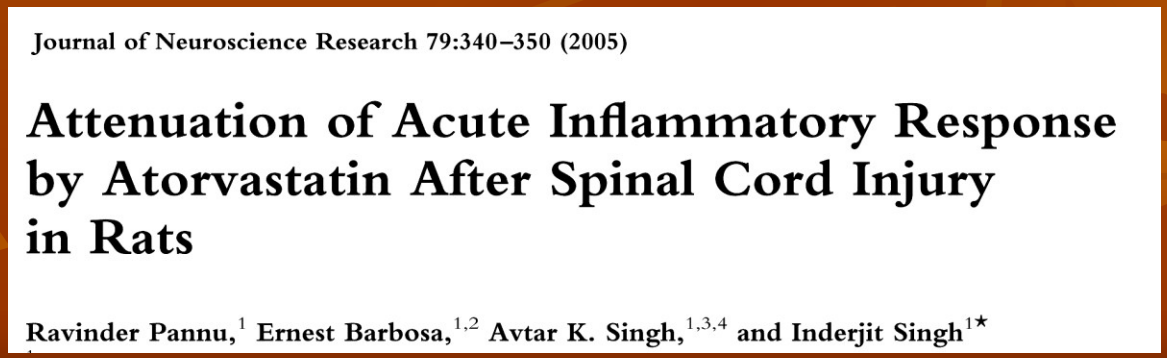
erythropoietin

PNAS | July 9, 2002 | vol. 99 | no. 14

Recombinant human erythropoietin counteracts secondary injury and markedly enhances neurological recovery from experimental spinal cord trauma

Alfredo Gorio*†, Necati Gokmen†‡, Serhat Erbayraktar§, Osman Yilmaz¶, Laura Madaschi*, Cinzia Cichetti*, Anna Maria Di Giulio*, Enver Vardar‖, Anthony Cerami**, and Michael Brines*†††

Lipitor



Journal of Neuroscience Research 79:340–350 (2005)

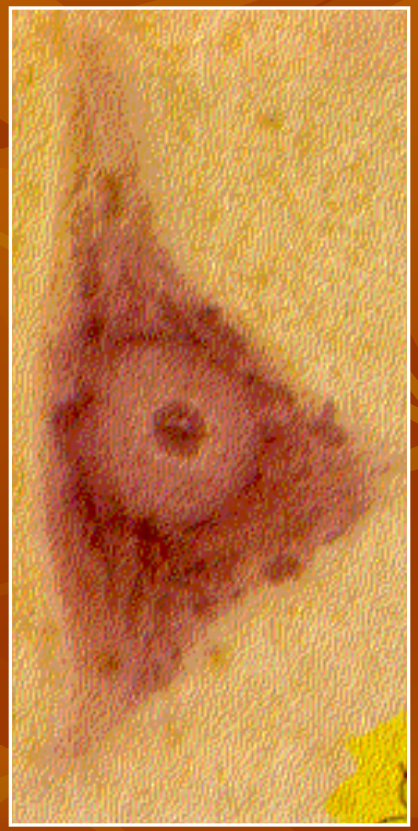
Attenuation of Acute Inflammatory Response by Atorvastatin After Spinal Cord Injury in Rats

Ravinder Pannu,[1] Ernest Barbosa,[1,2] Avtar K. Singh,[1,3,4] and Inderjit Singh[1]*

- *Prospects of Spinal Cord Regeneration*
- Regeneration is difficult and involves:
- Need to overcome the inhibitory environment inside the CNS
- Relative lack of regenerative capacity of CNS neurones
- Neurotropic factors to support axonal sprouts
- Bridging strategies across the zone of injury
- Presence of navigation molecules to let the axons grow into proper targets
- Finally, the re-grown axons must be functional and develop a synapse at the target tissue

Augmentation of Regenerative Ability of CNS Neurons
Neurotrophic Factors

- **Epidermal growth factor**
- **Fibroblast growth factor 2**
- **BDGF: brain derived growth factor**
- **Cyclic AMP**



Kojima, J Neurotrauma, 2002

Whiplash injury

- Sudden hyperextension and flexion
- Increasing neck pain for the first 24hours
- Associated headache, pain radiating to both shoulders and paraesthesia in hands
- Reduced lateral flexion
- Anterior longitudinal ligaments are torn causes dysphagia
- Forward flexion against resistance is painful
- 90% are asymptomatic after 2years
- 10% still have pain
- Some still claim money hence the need for proper documentations.