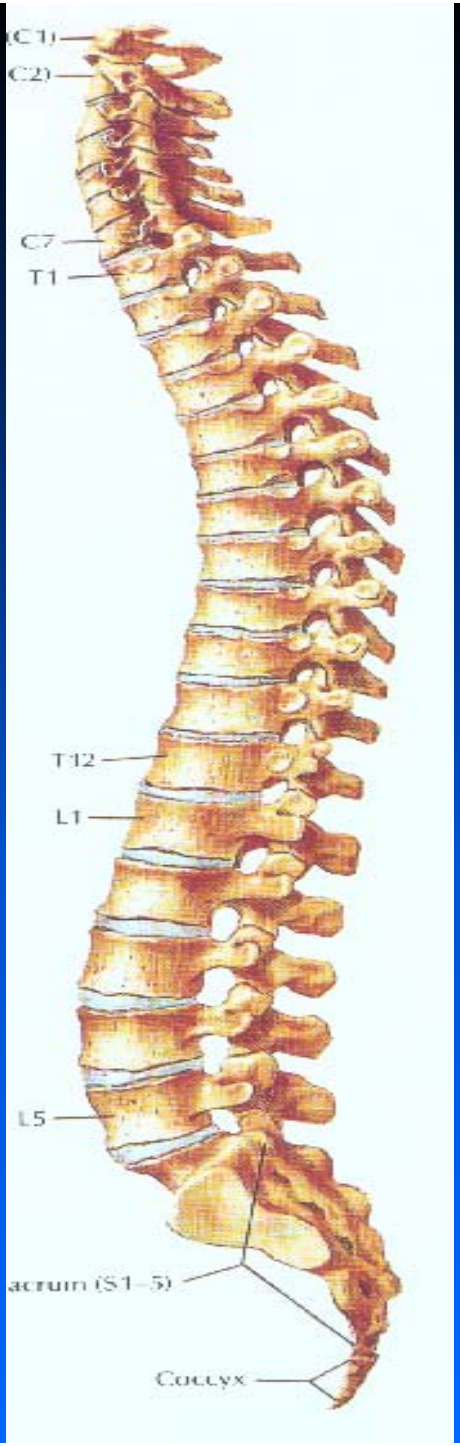
ACUTE SPINAL CORD INJURY



ACUTE SCI

PRIMARY INJURY

- High mortality and morbidity
- Sec changes:
  - Start soon after injury
  - Causes further damage to spinal cord
- Primary injury is associated with external compression
  - Longer the compression-poorer is prognosis

CAUSES OF ACUTE SCI

- Road traffic accidents
- Sports and recreational activities
- Work related accidents
- Fall from height
- Violence

ACUTE SPINAL CORD INJURY

- Very common in India
- More common in males
- Accidental fall is more common than any other cause

MECHANISM OF PRIMARY INJURY

- Rapid cord compression or transection by # dislocation or burst #.
- Acute distraction
- Shearing due to acceleration or deceleration
- Direct penetrating injuries

PRIMARY INJURY INITIATES Sec Changes (within few hours)

1. Vasospasm Ischaemia, haemorrhage, thrombosis, disrupted micro circulation, impaired autoregulation and neurogenic shock.
2. Ionic derangement
   - Increased intracellular calcium and sodium
   - Increased extracellular potassium

PRIMARY INJURY INITIATES Sec Changes (within few hours)

3. Accumulation of extracellular neuro transmiters
   - Serotonin, Catecholamines, Glutamate are toxic to cell
4. Endogenous opioids
5. Free radical accumulation
   - Arachidomic acid release
   - Lipid peroxidation
6. Oedema, inflammation
7. Loss of ATP dependant cellular process

ASSESSEMENT OF SPINAL CORD INJURY

1. Immediate assessment: Neurological function

2. During rehabilitation: Neurologic function + ADL (FIM= functional independence measure)

Comparison – Immediate and at one year.

ACUTE SPINAL CORD INJURY

- Most important is clinical examination
- Imaging, electrophysiological studies come later
- Clinical examination should be accurate enough to be compared

Safe Assumptions

- Every patient with a head injury and every unconscious patient
- Every patient with multiple trauma
- Every motor-vehicle accident victim
- Every victim of a sports or recreational accident
- Every severly injured worker
- Every victim of a fall at home
- Every SCI has an unstable spinal column and any movement of the spinal column after trauma will cause further damage to the spinal cord

CO-EXISTENCE

- 15% of all head injuries have significant spinal injury
- Patient of SCI who is restless, hypoxic, uncooperate may have head injury

Assumptions in Impaired Consciousness

- Hypotension and bradycardia – spinal shock
- Paradoxical respiration
- Low body temperature and high skin temperature
- Priapism
- Bilateral paralysis of arms and legs, espically flaccid
- Bilateral paralysis of legs, especially flaccid
- Lack of response to painful stimuli
- Detection of an anatomical level in response to painful stimuli
- Painful stimulation produces only head movement or facial grimacing
- Sweating level
- Honer's syndrome
- Brown-Sequard syndrome

SPINAL SHOCK

- Spinal shock occurs in major SCI
- It is a source of confusion
- It is "Neurogenic shock"

INVOLVEMENT

Spinal shock involves

1. Loss of motor function
2. Loss of sensory function
3. Loss of sympathetic autonomic function

- Higher the lesion
- More severe the lesion

} greater is the severity & duration of spinal shock

FREQUENCY OF SPINAL SHOCK

- Most severe - complete cervical cord injuries
- Less severe - incomplete thoracic injuries
- Minimal - lumbar injuries

SOMATIC MOTOR COMPONENT

- Paralysis
- Flaccidity
- Areflexia
  - Deep tendon
  - Cutaneuos

SENSORY AND AUTONOMIC COMPONENT

- Sensory - Anaesthesia to all modalities
- Autonomic – Hypotension: skin hyperaemia and warmth (sympathetic) bradycardia (unopposed vagotonia)

MECHANISM OF SPINAL SHOCK

- Mechanism unknown
- May be temporary electrolyte or neurotransmitter effect on impulse conduction

DIFFICULTY

- Differentiate between
  - Physiological spinal shock
  - Pathological – SCI
- Variable duration of shock
  - Hours to weeks

CLINICAL GUIDELINE

- Motor and sensory components of spinal shock lasts only an hour or less
- By the time patient is examined these elements have terminated
- Most advanced countries: patient is examined between 1 & 4 hours
- Reflex and autonomic lasts days to months.
- Safe course: follow motor and sensory deficits
- The guideline does not undermine the value of PR examination periodically to know sphincter tone.

Difference between spinal and systemic shock

SPINAL SHOCK

- Hypotension
- Brady carde
- Warm ext

SYSTEMIC SHOCK (Hypovolaemic)

- Hypotension
- Tachicardia
- Cold extremities

WHIPLASH SYNDROME

- Cervical hyperextension injuries
- Described by A.G. Davis in 1945
- Cervical spine injuries in car accidents

WHIPLASH

- Trauma causing cervical muscle-ligamental strain
- Cause: acceleration/deceleration of head in relation to trunk in any plane
- Highest incidence: rear end accidents
- Seat belt: possibly increases the incidence

PATHOLOGY

- TYPICAL - Rear car accident - Hyperextension

  Tear of sternomartoid, ALL, ant. annulus, avulsion of disc, longer colli, oesophageal tear, laryngeal haematoma, brain stem contusion, sp.cord oedema

Avulsion of nerve roots

Damage to cervical sympathetic chain

MRI upto 4 months can detect injury.

ACCELERATION INJURY

SIGNS AND SYMPTOMS

- Pain in the neck:-muscle sprain and ligaments injury
- Headaches common: upto 6 months
- Thoraco lumbar backpain:-present in 40% of patients . Difficult to treat.
- Paraesthesiae in hands. Could be due to brachial plexus stretch.
- Dysphagia:- present in 15%. Takes long time to recover.
- Uncommon:- Dizzines, vertigo, visual and auditory disturbances.
- 20% show neurological signs.

RADIOLOGY

- Plain x-rays show muscle spasm
- Obliterated lordosis
- Spine is straight

X-RAY APPEARANCE IN WHIPLASH

MRI is useful to detect soft tissue injury

- PIVD in 20% age – 45-64 years
- PIVD in 57% age > 64

- Spinal cord impingement
  - ✓ 16% - 45-64
  - ✓ 26% - >64 years

PROGNOSIS

- Symptoms usually last 6 months

Treatment

- Rest 2 weeks
- Soft collar 2 weeks
- Analgesics when necessary

THANK YOU