Traumatic Spine Injury

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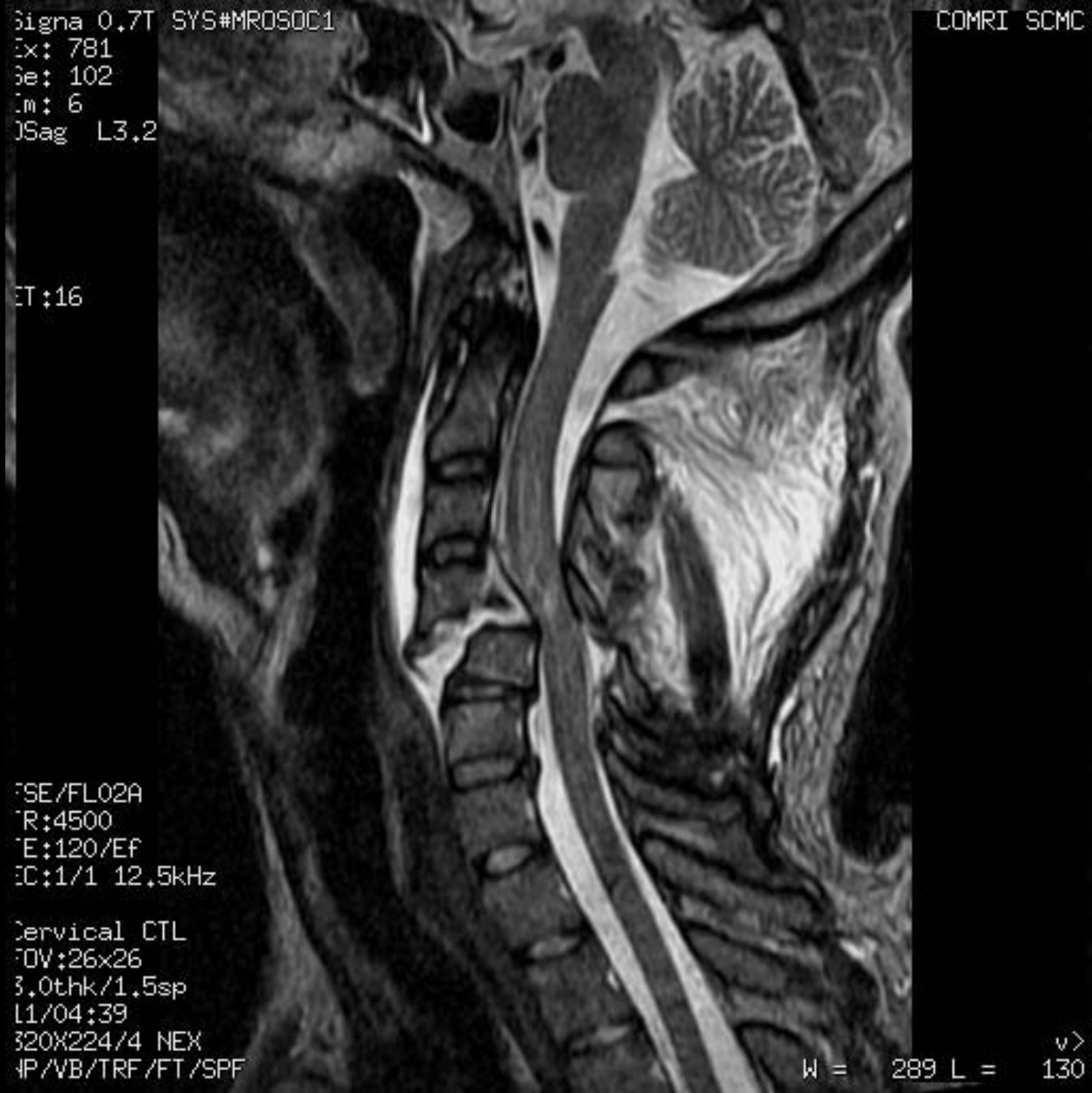
Overview

- Occur 1-3% of major trauma
- 16-30% die before hospital
- Areas of greatest mobility:
  - C_{5-7}
  - T_{12}-L_1

“Clearing” C-spine

Can rule out C-spine injury if all 5 absent:

1. neck pain / tenderness
2. neurologic deficits
3. impaired level of consciousness
4. intoxication
5. painful distracting injuries.


Signa 0.7T SYS#MROSOC1
COMRI SCMC
Ex: 781
Se: 102
Im: 6
OSag L3.2
ET:16
FSE/FL02A
TR:4500
TE:120/Ef
EC:1/1 12.5kHz
Cervical CTL
FOV:26x26
3.0thk/1.5sp
01/04:39
320X224/4 NEX
AP/VB/TRF/FT/SPF
W = 289 L = 130

Mechanism of injury ?

- Primary
  - Direct tissue damage (e.g. distraction, compression)
- Secondary
  - Vascular (e.g. vasospasm, edema)
  - Cellular (e.g. intracellular K, Ca)
  - Biochem (e.g. PGs, free radicals)

Secondary Injury

- Exacerbated by:
  - hypotension
  - hypoxemia
  - catecholamines
  - hypercoagulability
  - hypothermia
- Management of SCI is directed toward limiting secondary injury

Physiologic Sequelae - Pulmonary

- C_{5-7}
  - lose intercostal innervation (i.e. accessory m.s)
    $\Rightarrow$ volume reduction / atelectasis
- C_{3-5}
  - lose phrenic nerve
- above C_3:
  - lifelong ventilator

Physiologic Sequelae - Cardiovascular

- Complete Cx injury:
  - ventilatory dysfunction
  - hemodynamic instability
  - dysrhythmias (bradycardia, SVT, VT)
- Below T5:
  - functional sympathectomy ⇒ hypotension
- "Spinal shock"

Spinal Shock

- Hypotension & bradycardia
- Sympathetic : parasympathetic

  imbalance
  - Lose symp w/ high T or C disruption
  - Parasymp intact as exits from brainstem
- Duration
  - hours - days - weeks

Spinal Shock

- Hypotension ⇒ tachycardia
- Hypoxia ⇒ tachycardia
- Hypercarbia ⇒ tachycardia

Spinal Shock

- Treatment:
  - Fluids - *beware pulm edema*
  - Vasopressors w/ β-agonist properties :
    - *Dopamine / dobutamine*
  - *Beware potent α-agonists*
    (⇑ *sensitivity to exogenous catechol.s*)
- Maintain MAP > 70 mmHg
  - *theoretical* benefit to HTN - no evidence

Physiologic Sequelae - Cardiovascular

Remember other reasons for hypotension:

- bleeding
- tension pneumothorax
- myocardial injury
- tamponade
- sepsis.

Physiologic Sequelae

- Thermal control:
  - ∅ sweating / vasoconstriction
- GI – seen in 11% patients :
  - ileus
  - gastric atony / distension
  - PUD
  - pancreatitis
- GU – initial bladder flaccidity ⇒ spasticity
- Coagulation
  - DVT in 40% ⇒ PE in 10% (usually in 1st month)

Cord Protection

- High dose methylprednisone
  - in 1st 8h of injury shown to ⇓ 2° injury
- Hypothermia
  - Regional hypothermia protects CNS from ischemic insult - not systemic hypothermia
- Hypertension
  - theoretical benefit - little/no human data.

Indications for ETT

1. airway compromise
2. respiratory failure
3. GCS <8
4. intracranial hypertension
5. herniation

Intubation

1. Blind nasal intubation
2. Direct laryngoscopy w/ MILS
3. Awake FOI via nasal or oral route

Intubation

No evidence supports the superiority of any individual intubation technique.

Always consider operator experience & skill

Induction Drugs

- Succinylcholine
  - Safe for 1st 24h following injury
    - *no proliferation of end-plates yet*
  - Then ⇑ risk hyperkalemia
  - After 6-8 months safe again (*due to muscle atrophy*)
- Pentothal ?
  - Maintain MAP > 70 mmHg
- Inhaled agent ?
  - No evidence of any superiority
  - *Beware sensitivity to myocardial depressants*

Emergence / Extubation

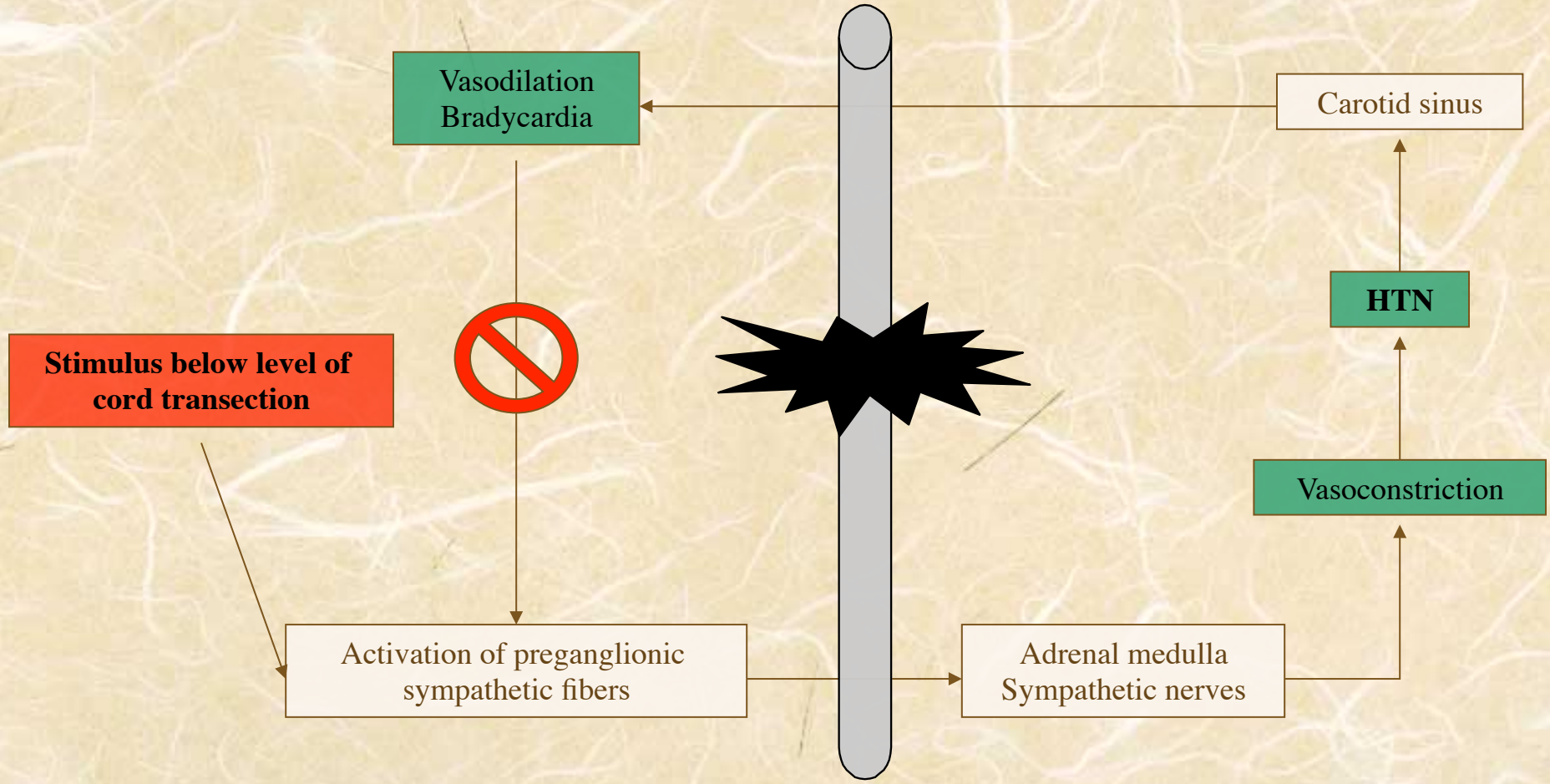
- Predisposition to hypoxemia:
  - ⇓ FRC

    (poor abdominal / intercostal m function)
- Aspiration risk
  - Gastric atony / ileus
  - Poor cough
  - Impaired airway reflexes

Leading causes of death

1. Septicemia
2. Pulmonary complications
   - atelectasis / aspiration pneumonitis / neurogenic pulmonary edema
3. Cardiac complications
4. PE
5. Suicide

Autonomic Hyperreflexia



Autonomic Hyperreflexia

- Tx / Prevention:
  - GA / regional both effective in preventing AH
  - Immediate withdrawal of trigger
  - Drugs :
    - *Vasodilators (e.g. SNP)*
    - *β-blockers (e.g. esmolol, labetolol)*
    - *Ca^{+}-channel blockers (e.g. nicardipine)*
    - *Symp blockers – e.g. trimethophan blocks nicotinic rec.s in autonomic ganglia ⇒ mixed auto blockade*
    - *Deep inh anes*

The End

Thank you