Spinal Trauma

Last updated: September 5, 2017

Epidemiology

Etiopathophisiology, Pathology

Vertebro-Column

Regional aspects

Stability and spinal cord injury

Incidence of spinal cord injury

Predisposing Factors to spinal cord injury

SCI

Primary SCI

Pathology in Stages

Degree of SCI

Secondary SCI

Ischemia

Biochemical Cascade

Wave of apoptosis

Experimental Therapies

Neuroprotective Agents

Cell-based therapies

Clinical Features

History

Physical examination

Classification (VCT)

Scales (SCI-....)

ASIA

International Spinal Cord Injury Basic Pain Data Set

Prehospital Management

Airway

Hypotension correction

Spine Immobilization

Transportation

Diagnosis & Strategies for Immobilization

SCI without radiographic abnormalities (SCIWORA)

Cervical Spine

Awake asymptomatic ADULT patients

Awake asymptomatic PEDIATRIC patients

Awake patient + neck pain or tenderness

Obstund / untetable patient

Diagnostic Modalities

X-ray

Cervical Spine

Thoracic Spine

CT

CTA

MRI

CT myelography

Bone Scan

Somatosensory evoked potentials

Complications

Posttraumatic syrinx (s. traumatic syringomyelia)

Treatment

Neuroprotectants

Stereoids

GM-1 ganglioside (Sygen)

Naloxone, thyrotropin release hormone, tertilaazad

Minocycline

Riluzole

Cardiovascular care

Respiratory care

General Measures

Surgery

Spinal Canal Decompression

Timing of surgical intervention

Penetrating Wounds

Definitive Spine Reduction & Stabilization

Cervical VCT

tongs application

halo application

External cervical immobilization devices

Thoracolumbar VCT

Prognosis

Rehabilitation

Associated Injuries

Pediatric Aspects

Neonatal SCI

Specific Verterbral and Legamemous Injuries (incl. Pathologic Fractures) → see p. TiS9 >>

VCT – vertebral column trauma.

SCI – spinal cord injury.

SCI is devastating in both socioeonomic and psychological aspects (intensive initial hospital care, long-term rehabilitation, lifelong care).

Epidemiology

VCT causes = 6% traumatic hospitalizations

SCI causes = 1% traumatic hospitalizations.

Incidence

10.4/3 per million (15,000 new cases of SCI per year in the United States and Canada)

Mostly young and otherwise healthy (peak 20-24 yrs; 50% are < 23 yrs; 65% are < 35 yrs).

- male-to-female ratio – 3:20:1

- incidence highest during summer weekends.

- incidence is rising among blacks.

- quadriplegia occurs in 54.1% of cases, with complete SCI occurring in 55.6%.

Second wave – elderly (females = males, but males are less numerous in elderly populations)

Prevalence

721,906/1,000,000.

The most common TYPLOGY of SCI: motor vehicle collisions, followed by falls, gunshot wounds, and diving.
average age at time of injury has climbed substantially over the last five decades, from the age of 29 in the 1970s to the age of 42 currently (2017).

pediatric SCI for those 15-years-old or younger are rare (3.5%), while injuries in retirees are on the rise; particularly due to falls.

ETIOPATHOPhYSIOLOGY, PATHOLOGY

VERTEBRAL COLUMN

Causes

Causes (most commonly indirect force applied to vertebral column — sudden flexion, hyperextension, vertebral compression, or rotation):

1) motor vehicle accidents 50-90% (declining)
2) violence 5-29% (increasing)
3) falls 17-21% (esp. in elderly)
4) recreational activities 7-16% (esp. diving)
5) birth trauma (esp. cervical spine – breech delivery, “shaken baby”)

Evolutional aspects:

prehistoric humans suffered little spinal injury - semirecumbent posture, with shoulders hunched well forward, combined with well-developed posterior cervical muscles that extended head against pull of gravity, protected cervical spine against day-to-day trauma.

- in evolution, people assumed upright posture (so that hands could be used more effectively), shoulders dropped away from newly elevated head and previously hypertrophied psoas muscles atrophied - this provided head with tremendous range of motion but decidedly diminished protection of spine.

Spinal cord injury is characterized by BIPHASIC PATHOPHYSIOLOGY:

1. PRIMARY injury – mechanical
2. SECONDARY injury (can be more detrimental than primary injury) – ischemia, biochemical cascades, inflammation, apoptosis.

REGIONAL ASPECTS

1. Vertebral trauma

- 50% - cervical (30% occur at C4; 50% occur at C5;)
- 50% - thoracic + sacral (60-70% occur in T1-L1)

N.B. trauma most often affects junctions between fixed and mobile segments

2. Cervical column (very mobile segment that joins two large body masses – head with trunk) – CLOSED SPINAL INJURIES by hyperextension or flexion, etc.

- 42% cervical fractures are unstable
- 39% cervical fractures give neurologic deficit.
- 4-8% patients with head injury have concomitant cervical spine injury.

- cervical region is the most frequently injured spine segment in vehicular crashes, especially when shoulder and lap belt restraints are not worn.
- falls are the most common (70%) cause in elderly

3. Thoracic spine (longest segment) – high percentage of WRENTH INJURIES

- T3-T12 region has high stability (stabilizing effects of rib cage, almost-vertical orientation of articulating processes, slingo-like oblique arrangement of spinous processes) - significant force is required to cause fracture / dislocation.

- laminae are also protective – broad, sloping, overlapping.

- thoracic spinal canal is narrow, cord has poor vascular supply – vertebral injuries have high incidence of SCI (90% dislocations above T6 result in complete paraplegia!!)

4. Thoracolumbar union (rigid thoracic area transitions to mobile lumbar spine) – CLOSED SPINAL INJURIES by vertical compression with flexion (= compression fractures with anterior wedging), or rotation (= fracture dislocations), etc.

- lower mechanical stability - no stabilizing effect of rib cage (T1-T12 region has false ribs), spinous processes are more horizontal, disc height!

- injuries to T1-L1 can result in significant paralysis (conus medullaris).

5. Low lumbar spine - injuries are more forgiving - roots of cauda equina are smaller, more flexible, and more resistant to injury (as they are PNS) compared with conus medullaris (as it is CNS).

STABILITY and spinal cord injury

Holdsworth’s concept

ANTERIOR COLUMN - (90% of vertical strength), vertebral bodies and intervertebral disks - held in alignment by anterior & posterior longitudinal ligaments.

POSTERIOR COLUMN - articulating facets (provide remaining 20% of vertical strength), pedicles, transverse processes, laminae, spinous processes - held in alignment by mual ligament complex (supraspinous, interspinous, ligamentum flavum) capsular ligaments, ligamentum flavum.

If BOTH COLUMNS are dramatically disrupted at one level (spine moves as two separate pieces) - MECHANICALLY unstable injury - great risk of vertebral canal misalignment (= spinal cord injury) from even slight motion.

If only ONE COLUMN is disrupted (other column resists further movement) - likelihood of spinal cord injury resulting from neural motion depends on integrity of ligaments supporting involved column.

- failure of any of one columns may involve compression effect (failure of vertical strength) or failure of ligamentous strength (inability to oppose dislocation).

- a healthy canal compression is more frequent when anterior column is involved.

- any degree of subluxation must be treated as potentially unstable!!! (ligamentous disruption allows cord compression at moment of impact, but vertebral bodies return closer to their original stations afterward).

MECHANICALLY stable - fragments are not likely to move and cause neural damage when spine is physiologically loaded.

N.B. MECHANICALLY stable injuries may be NEUROLOGICALLY unstable - result in spinal cord damage from:

1) fracture fragments (bone splintering)
2) herniated intervertebral disks
3) epidural hematoma
4) spinal cord vascular compromise.
Denis (1983) divided ANTERIOR COLUMN of Holdsworth into two segments:

- ANTERIOR SEGMENT - anterior halves of vertebral bodies with intervening disks, anterior longitudinal ligament.
- MIDDLE SEGMENT - posterior halves of vertebral bodies and their intervertebral disks, posterior longitudinal ligament.

Injuries involving 2 or 3 columns are unstable!

INCIDENCE of spinal cord injury:

Overall, only 10-15% VCTs result in neurologic deficits.

<table>
<thead>
<tr>
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<th>Percent with neurologic deficit</th>
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<tbody>
<tr>
<td>Fracture of vertebral body</td>
<td>3</td>
</tr>
<tr>
<td>Fracture of posterior element</td>
<td>19</td>
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<tr>
<td>Fracture of posterior elements and vertebral body</td>
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<td>56</td>
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<tr>
<td>Dislocation + fracture of posterior elements and vertebral body</td>
<td>61</td>
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</table>

PREDISPENSING FACTORS to spinal cord injury - preexisting vertebral column pathology (may allow SCI even without fracture or dislocation!!!):

1) instability of apophyseal joints (e.g. rheumatoid arthritis).
2) atlantoaxial instability (e.g. Down syndrome*, rheumatoid arthritis) → severe SCI after minor injury
   * laxity of transverse ligaments
3) decreased spinal canal - spondylosis (elderly predisposition to cervical SCI**), spinal stenosis, ligamentum flavum hypertrophy:
   ** forcible cervical extension → cord compression between arthritically enlarged anterior vertebral ridge and posteriorly located hypertrophic ligamentum flavum:

PRIMARY SCI - IMMEDIATE NEUROLOGIC DEFICIT due to STRUCTURAL DAMAGE (via traction / compression / laceration) of spinal cord.

VCT and SCI most often coexist, yet either can occur in isolation.

A. DIRECT injury to spinal cord (rare) - bypassed protection afforded by vertebrae: bullets / missiles, stabbing with sharp object.

B. INDIRECT injury to spinal cord - result of forces acting on vertebral column leading to:
a) VCT (fractures, dislocations).
   N.B. VCT need not be mechanically unstable to damage spinal cord (but not immobilized mechanically unstable VCT may lead to cord injury later!)
b) **hyperflexion / hyperextension** of vertebral column, esp. if spinal stenosis is present (e.g. 37% cervical SCI show no vertebral column injury);
   - violent neck hyperextension (e.g. pedestrian struck from rear by vehicle) may
     cause pons from medulla, or medulla from cervical cord → instantaneous death.
   - spinal cord is damaged by misalignment of vertebral canal, bone fragments (bone splitting), herniation of disc material, infolding of ligaments.

   it requires little direct force to cause significant injury to exposed spinal cord (spinal cord is much more vulnerable than cauda equina);

   2-3 cm weight dropped from height of only 10 cm onto monkey's surgically exposed spinal cord results in permanent neurologic dysfunction.

**PATHOLOGY IN STAGES**

- microhemorrhages*, exudation & massive swelling *in CENTRAL GRAY MATTER* occur within minutes** → progress over next few hours (extend from gray matter to white matter, progress longitudinally and taper several segments above & below level of injury; small hemorrhages may coalesce into hematomyelia) → cord fills whole spinal canal at injury level → intradural pressure† → secondary ischemia.

- *nurture of small venules

- **macroscopically cord looks swollen, reddish, and soft

- subependymal hemorrhage is rare, and any extracranial hemorrhages are small.

- hematomyelia is limited to central gray matter (LMN dysfunction); eventually absorbed, leaving centrally placed, smooth-walled cyst.

- within several weeks – edema subsides, hemorrhages are absorbed, acute exudate is replaced by macrophages (most prominent cell being *lipid phagocytes*), axons undergo wallerian degeneration.

- reparative stage may persist for up to 2 years → glial scar, fibrosis, syrinx-like caviation (in place of necrotic – hemoraghic areas).

- in ≤ 5 years after injury, cord becomes shrunken (replace by fibrous tissue); progressive proliferation of acellular connective tissue → chronic adhesive arachnoiditis.

**DEGREES OF SCI**

1) concussion / stretching (much less common than in brain)
   - **DIABETIC** neurologic dysfunction with recovery in minutes or hours (e.g. SYNDROME OF NEURALGIA after athletic injury - dramatic, although transient, neurological deficits including quadriplegia);

2) contusion (hemorrhagic softening) → **PERMANENT** neurologic dysfunction, surrounding compressed tissue suffers PROLONGED neurologic dysfunction.

3) laceration / crushing → **PERMANENT** neurologic dysfunction.

4) hemisection / transaction* → **PERMANENT** neurologic dysfunction.

*it is extremely rare for primary injury to transect spinal cord.

- ≥10% of remaining cross-sectional area of spinal cord is enough to support locomotion.

**SECONDARY SCI**

- **NEUROLOGIC DEFEIT PROGRESSES** over many hours due to ISCHEMIC / HYPOXIC / TOXIC DAMAGE to spinal cord – may eventually culminate in permanent neurological deficits – very important aspect in treatment† (even when complete transverse myelopathy is evident immediately after impact, it may be reversible)

- gray matter necrosis is evident within 4 h, at 8 hours necrosis becomes global (paralysis below level of lesion becomes irreversible).

**ISCHEMIA**

- very prominent feature of post-SCI events.

1. Blood vessel damage (leads to ischemia):
   1) loss of autoregulation (significant reduction in spinal cord blood flow within 2 hours of injury).
   2) cord compression by epidural hematoma.
   3) vascualar compression (e.g. by dissected vertebra) → thrombosis.

2. Spinal shock causes systemic hypotension → ischemia exacerbation.

**BIOCHEMICAL CASCADE**

Primary SCI initiates complex autodestructive progressive biochemical cascade, involving free radical-induced lipid peroxidation (destruction of membranes) → progressive edema & ischemia.

1. Massive release of GLUTAMATE (key element in excitotoxicity) leads to overstimulation of neighbor neurons → intracellular calcium & sodium+, extracellular potassium+ → production of free radicals → death of healthy neurons and oligodendrocytes (AMPA glutamate receptors play major role in oligodendrocyte damage).

2. Neuroinflammation

WAVE OF SYMPTOMS

- further affects oligodendrocytes up to 4 segments from trauma site days 0 weeks after initial trauma (syringomyelia may develop as outcome).

- even after severe conusive SCI, axons survive in the subpial rim of white matter, but show dysfunctional conduciton (because of changes in axonal K+ channel expression and distribution).
Key subacute event is **DEMYELINATION** (post-traumatic degeneration of white matter) – due to:
1) oligodendrocyte loss (death and limited renewal)
2) ↓ myelin gene expression.

**EXPERIMENTAL THERAPIES**

### NEUROPROTECTIVE AGENTS

1. **RILUPRIDE** – targets excitotoxicity. see p. Spin21 >>
2. **BA-210** (Cethrin®) – Rho antagonist; promotes neuroregeneration and neuroprotection.
4. **ANTIS-MONES** Nogo-A is myelin-associated neurite outgrowth inhibitory protein limiting recovery and plasticity after CNS injury.
5. **VEGF** – stimulates angiogenesis and limits apoptosis.

### CELL-BASED THERAPIES
- primarily target myelin repair by restoration of oligodendrocyte population. Spontaneous remyelination is limited!

#### A. Endogenous precursor cells
- within adult spinal cord - do not promote remyelination (even after infusion of exogenous growth factors)

#### B. Cell replacement therapy – potential cell types:
1) embryonic stem cells (ESCs) – ethical issues!!
2) adult brain-derived NEURAL PRECURSOR CELLS (NPCs) – extensive capacity for self-renewal and multipotency in vitro!
- NPCs reside in forebrain (subventricular zone) and spinal cord for life.
- NPCs can be isolated in vitro in the presence of growth factors → formation of clonally derived free-floating colonies (NEUROSPHERES).
3) activated macrophages
4) Schwann cells
5) olfactory ensheathing cells
6) mesenchymal stem cells
7) skin-derived precursor cells

### CLINICAL FEATURES

#### HISTORY

1. **Mechanism & forces** of injury.
2. **Site** and duration of any pain.
3. **Transient or persistent numbness, tingling, weakness** (or other neurologic problems).
   - Mortality of spinal cord injury is so significant that even transient minor symptoms should be investigated!
4. Any prior injuries / disorders involving spinal column or spinal cord.

#### PHYSICAL EXAMINATION

1. **SIGNS OF TRAUMA** - provide clues to mechanism of injury:
   1) abrasions, contusions, swellings:
      - contusions about scapula suggest **rotation or flexion-rotation** injury of thoracic spine.
      - injury to gluteal region or feet and ankles suggests **compression** injury.
      - oblique anterior chest or transverse lower abdominal abrasions from automobile belts mandate search for spinal, intrathoracic, and intraabdominal injuries.
   2) visible / palpable:
      - spinal deformity, gap between spinous processes = unstable spinal injury.
      - kyphosis ≥ 30° indicates instability.
   3) local pain & tenderness (may be noted only in subacute phase, may be progressive).
      - examiner's hand is gently positioned under supine patient to palpate each spinal level:
        - level of spinal injury (e.g. burning hands syndrome = see below >)

2. **SENSORY SYMPTOMS** (lack of sensation below certain level, burning paresthesias, hemiseodynia loss sparing face):
   - conscious patient may experience severe pain in sensory dermatome corresponding to level of spinal injury (e.g. burning hands syndrome = see below >).
3) AUTONOMIC SYMPTOMS (urinary retention, constipation, ileus, gastroparesis, poliomyelitis, neurogenic shock*), Horner syndrome, sustained priapism**.
   *hypotension with bradycardia and flushed-dry-warm peripheral skin
   **loss of sympathetic tone (poor prognosis)

These combinations reflect ANATOMIC SPINAL SYNDROMES (according to transverse and longitudinal location of SCI):

A) SPINAL CORD TRANSSECTION - Myelitis traumatica transversa complete (COMPLETE SCI; others below are INCOMPLETE SCIs)

B) CENTRAL CORD SYNDROME** - most common INCOMPLETE SCI syndrome*; etiology:
   a) neck hyperextension (esp. in patients with cervical stenosis) - cord compression between bony bars anteriorly and thickened buckling ligamentum flavum posteriorly - cord hypoperfusion in watershed distribution (mostly central portion of cord - central gray and most central portions of pyramidal & spinothalamic tracts).
   b) hematoma (usually confined to central gray matter).

BURNING HANDS SYNDROME (variation of central cord syndrome first described in extension injuries at C6-C7 level in football players) - severe burning paresthesias in hands; no other neurological dysfunction; > 50% there is underlying spine fracture-dislocation.

C) ANTERIOR CORD SYNDROME** - (cervical flexion resulting in anterior cord congestion; large disc herniation or burst fracture compressing anterior cord; laceration or thrombosis of anterior spinal artery) - worst prognosis of incomplete SCI syndromes (only 10-20% recover motor function).

D) BROWNSQUEAR SYNDROME** (direct penetrating trauma, lateral mass fractures of cervical spine, locked facets) - best prognosis of incomplete SCI syndromes.

E) POSTERIOR CORD SYNDROME - (hyperextension injuries with fractures of vertebral arch) - rare.

F) CUNES MEDULLARIS SYNDROME (disc herniation or burst fracture of T12 body) - resultant in anterior cord contusion; no other neurological dysfunction.

G) CALAECUPE SYNDSMRE (N.B. abrupt SCI (complete or incomplete) may initially cause SPINAL SHOCK. see p. Spinal >>
   *three most common syndromes (together constitute 90% incomplete SCI cases)

Define NEUROLOGIC LEVEL - most caudal spinal segment with normal sensation and muscle strength of 3/5 or better.

Carefully document any motor / sensory FUNCTION BELOW LEVEL (incomplete SCI)

- sacral sparing may be only evidence that paralysis may not be complete - always test perineum sensation, voluntary and sphincter control / contraction / relaxation!!!

N.B. absent bulbocavernosus & anal wink reflexes = spinal shock is present (sacral sparing is not testable at this time – wait for return of above reflexes!!!)

- SCI completeness may be fully evaluated only after spinal shock (return of reflex activity below level, but no sensation or voluntary motor control below level = complete cord transsection).

- most cervical and thoracolumbar injuries are complete; lumbar injuries produce incomplete lesions.

- absent reflex above returned reflexes indicates L MNJ injury (e.g. spinal root or cauda equina lesion - often caused by surgically correctable lesion!)

CLASSIFICATION (VCT)

AO Mechanistic Classification - complex subdivisions to include most fractures:

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ASIA

AMERICAN SPINAL INJURY ASSOCIATION (ASIA) system for examination and classification of spinal cord injury:

- recommended by "Clinical Assessment Following Acute Cervical Spinal Cord Injury" guidelines (Level 2 evidence).

**ASIA Impairment Scale**

- **A** = Complete SCI - loss of motor and sensory function in S4-S5 segments.
- **B** = Incomplete SCI - sensory but not motor function is preserved.
- **C** = Incomplete SCI - motor function is preserved (> ½ of key muscles below neurological level have muscle grade < 3 [unable to resist gravity]).
- **D** = Incomplete SCI - motor function is preserved (> ½ of key muscles below neurological level have muscle grade ≥ 3).
- **E** = Normal.

Falsely localizing level of neurologic deficit:

- a) lower cervical dislocation may damage vertebral arteries → decreased flow through anterior spinal artery → neurologic level at C1-C2 level (or even brainstem).
- b) lumbar fracture or dislocation may compress great radicular artery of Adamkiewicz (sends branches as cephalad as T4 level) → neurologic level at T4.

In unconscious patient (TBI, drugs), only clues to significant SCI may be:

1) lack of facial grimacing to peripherally applied painful stimuli (sensory loss)
2) lack of arm/leg withdrawal to painful stimulation applied to head (motor loss).

Cephalad progression of deficit (esp. hypesthesia) is indicator of deterioration - neurologic examination is repeated at frequent intervals!

- 40% cervical SCI patients present in ASIA A.
use of separate upper- and lower extremity ASIA motor scores rather than a single total ASIA motor score will reduce measurement error when ASIA motor score is used as predictor of outcome.

flow effects among paraplegic patients (no measure of motor function between T1 and L1) and ceiling effect among quadriplegic patients (injury above measurable motor units).

ASIA is not applicable to pediatric patients.

**INTERNATIONAL SPINAL CORD INJURY BASIC PAIN DATA SET**

- recommended by “Clinical Assessment Following Acute Cervical Spinal Cord Injury” guidelines (Level 1 evidence) as additional scale to ASIA.
- pain can be horribly debilitating, hindering patient.
- PREVALENCE of chronic pain after SCI is 25-80%.
- chronic pain causes functional impairment beyond that expected from neurological injury, plus causes debilitating depression.

**PREHOSPITAL MANAGEMENT**

- nowhere in spectrum of emergency care can EMT have so great impact on patient’s destiny as in management of head and spine injures; proper management in field can make difference between normal existence or lifetime spent in total paralysis, and there are more than few people walking about today who owe their lives and their ability to move to treatment EMT gave them in field.

**QUADRIPLEGIA IS FOREVER**

All VCT patients should be treated as mechanically & neurologically unstable, until proven otherwise.

- formerly, up to 10% quadriplegia cases were iatrogenic (injudicious manipulation by paramedical personnel, examining physicians, or radiology technicians).

**AIRWAY**

- spontaneous respirations (suspicious C-spine injury)
- apneic
- child < 8 yrs OR bonygeal injury
- nasotracheal intubation
- failed
- positive
- lateral C-spine X-ray
- failed
- orotracheal intubation
- failed
- severe maxillofacial injury
- cricothyrotomy
- tracheostomy

N.B. in any way maintain in-line immobilization (not traction!- head and neck held in neutral position.

- nasotracheal intubation gives least stress on cervical spine but often technically difficult to perform!
- careful orotracheal intubation (esp. with laryngoscope) with in-line spinal immobilization is preferred method of airway management even with evidence of spinal injury!
- cricothyroidotomy / tracheostomy may be preceded by temporizing needle cricothyroidotomy with jet insufflation.
- incising through cervical fascial planes could release previously contained hematoma – life-threatening hemorrhage.

All maneuvers (nasotracheal intubation, orotracheal intubation, cricothyroidotomy) appear to be safe - choice of technique should be determined by physician's experience and expertise with each.

**HYPOTENSION CORRECTION**

1. Leg elevation, Trendelenburg position
2. Fluids IV

N.B. severe hypotension itself is potential cause of spinal cord damage!

**SPINE IMMobilIZATION**

Collar + board + head packs + tape

Slowly and judiciously.

As soon as practical (often before extrication is complete; in sport injuries, immobilize athlete on field).

Exceptions – immobilization is not recommended:

a) awake, alert, not intoxicated patients without neck pain or tenderness, who do not have abnormal motor or sensory examination and who do not have any significant associated injury that might detract from their general evaluation.

b) patients with penetrating trauma (immobilization causes increased mortality from delayed resuscitation).

Maintain axial neck traction and do not flex; minimally disturb patient.

Patient is moved as single inflexible object - LOG ROLLING TECHNIQUE.

Semitropic cervical spine collar is applied; patient is placed on back board.

Protect it (spine) until you detect it (unstable spine injury)?

1. If victim is wearing helmet – helmet must be cautiously removed because it impedes immobilization (interferes with collar placement; helmet is glassy – if necessary, use adhesive tape), resuscitation, bleeding control, aspiration prevention.
   1) victim lies supine
   2) take off protective eyewear
   3) axial traction (palms on helmet rim, finger grasp under mandible)
   4) second rescuer unbucks helmet strap and takes over axial traction (on mandible and occiput)
   5) first rescuer then removes helmet: widening it at ears, lifting at nose.
   6) axial traction is taken over again by first rescuer (palms on ears).
Incorrect helmet removal can do harm!

2. One rescuer maintains axial traction (traction should not be applied, i.e. only minimal extension):

3. Other rescuer places semi-rigid cervical collar:
   - properly placed collar must admit (between it and neck) two fingers.
   - variety of effective spinal immobilization orthoses are commercially available (Hare extrication collar, Philadelphia collar, four-poster neck immobilizer, number of adjustable rigid plastic collars) - all appear to be effective, but none have been found to be more effective than traditional backboard with sandbags and tape.
   - soft cervical collars (normally prescribed for outpatient therapy of minor cervical disorders) are inadequate for this purpose!

Philadelphia collar – commercially available splint, comes in two halves that are secured by Velcro straps; posterior shell may be flattened and slid under patient’s neck (while assistant applies inline stabilization) → anterior shell is placed over front neck and chin → secure Velcro straps.
4. Victim (maintaining inline immobilization*) is transferred on backboard (rigid & flat) and immobilized. *very minimal traction

LOGROLLING is only acceptable way to turn patient suspected of spinal injury.

- body moves as whole - one segment does not precede or drag behind another.
- requires minimum of three people: one at head (to maintain longitudinal stabilization), one at side (to turn trunk), and one at feet (to turn legs).
- done in unison under control of one person at head, who directs operation.
- indications - placement on spinal board, placement of x-ray film, examination of patient's back.

- when on board, head must be extra immobilized* - rolled blankets or sandbags (alternative - head taping to rigid backboard).

Cervical collar prevents flexion/extension, side supports prevent rotation

*can be the only way to immobilize if collar is not available

Cervical spine can be immobilized by either "short" or "long" spine board ("short" board is dangerous - no guarantee that patient has no additional spinal injuries below).

Thoracic-lumbar spine is best immobilized on long board with taping patient to backboard above and below major joints.

- rolls are placed under flexures (neck, lumbar, popliteal areas).
- to prevent undue neck flexion, spine board for children ≤ 3 yr must have indentation for occiput.

Spinal immobilization using short board:

Spinal immobilization using long board:
When to suspect cervical spine injury

There is no immediate suspicion if becomes asymptomatic and

Treatment

Diagnostic work

Causes

Differential diagnosis of vertebral fractures:

Most of fractures missed on radiographs are:

- In young children, anterior corners of vertebral body may have small depression (represent epiphyseal margin).
- Horizontal residual epiphyseal margin - 10% chance of associated bony vertebral injury.
- if one fracture is found, other levels should be carefully checked for additional injury (10-15% incidence of multiple fractures).
- Fracture absence does not ensure spinal column stability! Small bone avulsion or slight malalignment of vertebrae may be only suggestion of gross ligamentous instability!
- differentiating new fractures from old may be difficult (H: CT, radionuclide bone scan, MRI).

IMMOBILIZATION

Most of fractures missed on radiographs are: spinous process fractures, transverse processes fractures, fractures in large patients.

Diagnostic differential of vertebral fractures:
1. Horizontal residual venous sinus grooves.
2. In young children, anterior corners of vertebral body may have small depression (represent epiphyseal margin).
3. Osteofracture centers at ends of transverse processes (may appear as fractures).
4. Spina bifida occulta.
5. T12 body slightly wedged anteriorly (physiologic wedging).
6. Asymmetry of pedicles of lower thoracic spine (7% persons).
7. Congenital butterfly vertebral body (appears as compression fracture in lateral projection).

SCI WITHOUT RADIOGRAPHIC ABNORMALITIES (SCIWORA)

Causes:
1. marked cervical spondylolysis and spinal stenosis.
2. spontaneously corrected dislocation.
3. children < 8 yrs - flexible spinal columns (greater ligamentous laxity & flexible spinal columns (greater ligamentous laxity - vertebral elements reduce spontaneously).

Diagnostic work-up: MRI if negative → flexion-extension XR, radiographic screening of entire spinal column.

TREATMENT: external immobilization of spinal segment of injury for up to 12 weeks (discontinue earlier if becomes asymptomatic and flexion-extension XR is negative).

avoid “high-risk” activities for up to 6 months following SCIWORA.

CERVICAL SPINE

Instability must be determined early!!

There is no imaging gold standard for cervical spinal instability, or for ligamentous injury!!!

When to suspect cervical spine injury

Spinal column and spinal cord injuries are more prevalent in blunt cervical trauma than in penetrating.

a) impaired consciousness (incl. even mild alcohol intoxication) and suspected craniofacial trauma
b) significant head or facial trauma
A. (cannot be assessed clinically) → cervical spine
B. noncontiguous spine injury
C. If C

Official guidelines

National Emergency X-Radiography Utilization Group (NEXUS): criteria:

1. no posterior midline cervical spine tenderness.
2. no intoxication.
3. no focal neurologic deficit.
4. no painful distracting injury.

CCR is more sensitive than NEXUS (100% vs. 90-99%).

Awake asymptomatic ADULT patients

"Clearing" = no imaging (i.e. low-risk patients - remove C-collar)

Official guidelines (Level 1) – all must be present:

1) awake
2) asymptomatic (no neck pain or tenderness, normal neurological examination)
3) no injury detracting from an accurate evaluation
4) able to complete a functional range of motion examination.

N.B. irrespective of the mechanism of potential injury!

Canadian C-Spine Rules (CCR):

1) high-risk clinical factor (age > 65 years, dangerous mechanism, paraplegia in the extremities) → imaging
2) any low-risk clinical factor (simple rear-end motor vehicle collision, sitting position in ED, amnibulatory at any time after accident, delayed onset of neck pain, absence of midline cervical spine tenderness) → assess for neck range of motion
   a) unable to axially rotate head > 45° in either direction → imaging.
   b) able to axially rotate head > 45° in both directions - do not require imaging

National Emergency X-Radiography Utilization Group (NEXUS) criteria:

1. no posterior midline cervical spine tenderness.
2. no intoxication.
3. no focal neurologic deficit.
4. no painful distracting injury.

Awake patient with neck pain or tenderness

⇒ CT (3-view XR only if CT unavailable):

Normal imaging:
1) Continue cervical immobilization until asymptomatic
2) Discontinue cervical immobilization following normal and adequate dynamic flexion/extension radiographs
3) Discontinue cervical immobilization following normal MRI obtained within 48 hours of injury
4) Discontinue cervical immobilization at discretion of treating physician

Several studies favor MRI (Level II) over dynamic radiographs (Level III), but may not be feasible or indicated in all situations.

Clavicle injury detected – continue immobilization and image whole spine (10% patients have noncontiguous spine injury)

• cooperative patient should be cautioned against attempted movement until radiographic studies have been performed.
• combative patient may require individual assigned to hold patient’s head in alignment with the longitudinal axis of body; sedation (± paralysis and intubation) may be required for patients who pose danger to themselves because of excessive movement.

Obtunded/unevaluable patient
(cannot be assessed clinically) → cervical spine CT – if reported as normal:

A. Continue cervical immobilization until asymptomatic
B. Discontinue cervical immobilization following normal MRI obtained within 48 hours of injury
C. Discontinue immobilization at discretion of treating physician.

David M. Panczykowski et al. “Comparative effectiveness of using computed tomography alone to exclude cervical spine injuries in obtunded or intubated patients: meta-analysis of 14,327 patients with blunt trauma”

Modern CT alone is sufficient to detect unstable cervical spine injuries in trauma patients.

Adherent imaging is unnecessary when CT was negative for acute injury. Cervical collar may feasi or indicated in all situations.
patients who have had serious SCI with permanent sequelae demonstrate abnormalities without moving patient.

**DIAGNOSTIC MODALITIES**

**X-RAY**

For X-ray evaluation principles → see p. D70 >>

- **Signs of instability** → also see p. D70 >>

- X-ray can miss some fractures (H. CT or MRI, dynamic radiographic views are also diagnostic but contraindicated in immediate setting).

**CERVICAL SERIES**

1) **fracture stable supine lateral view** (positive in 85-90% cervical spine injuries) – initial radiologic approach (also performed as part of head CT), may be inadequate as sole view!:
   - shoulders are depressed actively or passively by pulling patient’s hands toward feet using slow steady traction over 1-2 minute period (rather than sudden jerk).
   - head is stabilized but not actively distracted (can be disastrous in severe C1-C2 ligamentous injury).

2) **AP view**

3) **open-mouth (odontoid) view** → all three views (three-view trauma series) should be obtained before C-spine immobilization is discontinued

4) **swimmer’s (transaxial) view** → if above views do not visualize C7-T1 interspace:
   - a. large, muscular, obese patients.
   - b. paralasis of shoulder depressing muscles (caused by lesions that are, unfortunately, located in lower cervical region) - unopposed trapezius muscles elevate shoulders.
   - N.B. if shoulder girdle injury prohibits swimmer’s view → CT.

5) **supine oblique-neuronal animal view** → optimal view in confirming subluxations, posterior laminar fractures, unilateral facet dislocations!

6) **flexion & extension lateral films** - confirming stability vs. subluxations before clearing C-collar in alert patients with persistent neck pain and no signs of instability in other views

**Hazards**: - performs only in awake cooperative patient if above views are negative but ligamentous injury is possible (MRI is safer!!!)
   - *if subluxation happens, pain will stop patient from moving, therefore, study may be false negative because of severe muscle spasm prevents enough amplitude – wait 2 weeks and repeat study:
     - a) if spine is stable, neck pain subsided and patient will show full amplitude
     - b) if spine is unstable, pain is worse at 2 weeks
   - differentiate from pseudosubluxation → see p. D70 >>

- these views will identify > 99% unstable cervical injuries.

**TRUNK SERIES**

1) lateral view

2) AP view (ribs and transverse processes).

- **lateral flexion & extension studies** (standing if possible) - to look for gross instability.

- **fractures of upper thoracic vertebrae** → posterior mediastinal hemorrhage → ill-defined mediastinal widening (differentiate from traumatic aorta rupture - CTA or angiography).

**CT**

- extremely helpful for **bone definition** + also shows **soft tissue changes** + permits examination **without moving patient** from supine position.

Indications:
   - a) vertebral injuries (fractures, subluxations) on X-ray
   - b) normal vertebral X-ray, but clinical suspicion remains (e.g. persistent neck pain or neurologic deficit - but MRI is better)
   - c) **difficult to evaluate** vertebral X-ray

    N.B. unclear fractures / displacements on standard radiographs should be further evaluated by CT:
    - *thoracic fractures → CT with contrast (to exclude intrathoracic injuries – possible injury to aorta rupture - CTA or angiography).

- axial CT may fail to depict subtle **horizontally oriented injuries**, minimal **vertebral body compression fractures**; H: frontal and sagittal reformation, with very thin sections for primary images.

**CTA**

- **Indications - blunt cervical trauma + modified Denver Screening Criteria**

1. Fractures involving C1-6 transverse foramina

2. C1–3 subluxations

3. Facet dislocations

- MRI is recommended instead of CTA after blunt cervical trauma in patients with complete SCI or vertebral subluxations.

**MRI**

- **Direct visualization of cord injury** (MRI findings correlate with neurologic status; virtually all patients who have had serious SCI with permanent sequelae demonstrate abnormalities on MRI).

- **indicated when neurologic deficits are present** (esp. if level of deficits does not coincide with **bilateral Asymmetries**).

- **acute fractures - vertebral marrow edema** (not seen in chronic compression fractures!)

- **prognostic value**:
  - mild or transient loss of function usually is not accompanied by any signal changes.
  - cord hemorrhage (type 1 contusion) – poorer prognosis.
cord edema (type 2 contusion) – frequent significant neurologic improvement (but edema extending for > 1 spinal segment predicts poor prognosis).
N.B. the length of cord edema is the only independent predictor of recovery in SCI in multivariate analysis.
- MRI is incompatible with life-support, monitoring systems, and cervical traction devices (H: MRI-compatible support systems).
- when MRI is not available, CT myelography is best alternative.

**CT myelography**
- best performed by lateral C1-2 puncture (using fluoroscopy with patient supine).
- often all that is found is complete block near level of injury.
- cord compression may be shown (but is infrequent).
- delayed postmyelography CT should demonstrate most cavities in spinal cord.
N.B. abnormal contrast accumulation in cord may be due to myelomalacia (i.e., not cavity).

**Bone Scans**
- with Tc 99m hydroxydimethylpyrimidine.
- may detect occult/minor fractures (area of increased uptake) – scans become positive only > 12-24 hours after injury (or even as long as 72 hours after injury).
- In 111T-111-tagged WBCs are more specific in detection of abscess/osteomyelitis.

**Somatosensory evoked potentials**
- helpful in distinguishing complete or incomplete lesions (results are less sensitive or specific than MRI, but test can be repeated often).

**Importance of completely visualizing all seven cervical vertebrae:**
1. Inadequate cross-table lateral cervical spine radiograph (C7 not visualized).
2. Repeat lateral film demonstrates burst fracture of C7.

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COMPLICATIONS

GENERAL SPINAL COMPLICATIONS → see p. Spinal >>

- 30% SCI patients require hospital admission every year for complications.
- Depression (following initial period of denial) occurs in almost all patients and may be masked by jocularity.

PROGRESSIVE NEUROLOGIC DYSFUNCTION in previously neurologically stable patient:

1) undiagnosed disk or bone* impingement on spinal cord
2) myelomalacia
3) developing posttraumatic syrinx
4) subarachnoid cystic changes, post-traumatic pseudomeningoceles
   *e.g. post-traumatic deformity (such as angular kyphosis)
   - all are potentially operable conditions (with exception of myelomalacia).

POSTTRAUMATIC SYRINX (TRAUMATIC SYRINGOMYELIA) - residual intramedullary cysts (e.g. after hematomyelia resorption) become distended → progressive neurologic disorder (after several months - years of neurologic stability).
- neurologic progression is invariably rostral to original injury - POST-TRAUMATIC ASCENDING MYELOPATHY (occurs most frequently in cervical region).
- myelographic appearance similar to other forms of syringomyelia.

Posttraumatic myelomalacia:
A. Sagittal T1-MRI - mottled low signal at C4-C5 level.
B. Sagittal T2-MRI - high signal at same level.

TREATMENT

- primary goal is to limit secondary injury!
- critical factor is time from injury to therapy (complete axonal disruption from secondary phenomena precludes recovery).

Treatment encompasses 6 phases:
1. Emergency treatment with ABC, appropriate immobilization, transfer to specialized center.
2. Treatment of general medical problems (e.g. hypotension, poikilothermy, ileus, urinary retention).
3. Spinal alignment.
4. Surgical cord decompression (if indicated) – must be done ASAP if neurodeficits are progressing.
5. Spinal stabilization – more elective approach.
6. Rehabilitation.

NEUROPROTECTION

STEROIDS

2013 guidelines of Congress of Neurological Surgeons (CNS) and American Association of Neurological Surgeons (AANS) recommend against use of steroids early after acute spinal cord injury
- reduce lipid peroxidation & inflammatory response → improvement in function (but likelihood of clinically significant functional improvement is low).
- some studies show only improvement by one sensory level.
- steroids are not FDA approved for SCI.

There is no Class I or II evidence supporting clinical benefit of steroids in treatment of acute SCI

Class I, II, and III evidence exists that high-dose steroids are associated with harmful side effects** including death.

*scattered reports of Class III evidence claim inconsistent effects likely related to random chance or selection bias.
**1.5 times higher incidences of GI hemorrhage and pneumonia, 2 times higher incidence of wound infection, 3 times higher incidence of pulmonary embolism in MP-treated patients compared to controls; most compelling is Class I evidence from > 10 000 patients with TBI, indicating that high-dose MP leads to significantly higher mortality independent of injury severity.

**NASCIS (National Acute Spinal Cord Injury Study) I-III studies - the largest study investigating the effects of the steroid methylprednisolone in acute spinal cord injury. Outcome evaluated at six weeks, six months or one year.

**NASCIS I
- There was no difference between moderate-dose and low-dose MePred.
- There was a trend towards better outcome for moderate-dose MePred if given within 8 h.

**NASCIS II
- Patients who received MePred within 8 h of injury had a statistically significant improvement in motor and sensory function.
- There was no effect of naloxone.

**NASCIS III
- No statistically significant benefit was seen for continuing MePred treatment for 48 h (beyond 24 hrs).
- No statistically significant benefit was seen for ultra-early (< 3 hrs) administration of MePred.

**NASCIS Conclusions
MePred improves outcome of acute spinal cord injury (ASCIs) if given within 8 h of injury. NASCIS Critique
The positive results of the NASCIS trials pertain only to post-damage subgroup analyses. For example, NASCIS II only showed a benefit with MePred for the subgroup of patients who received it within 8 h of injury. It is imperative, therefore, to re-emphasize that the conclusions from this subgroup analysis cannot be extended to all patients within the trial.

The large range of the neurological scores used has meant that it is questionable whether small improvements are clinically relevant (Spencer and Bazarian, 2003 ).

**Steroids for acute spinal cord injury (COCHRANE Review)
Background
Animal experimentation with pharmacologic therapy for acute spinal cord injury started in the late 1960s (Ducker 1969), became more common in the 1970s and led, in the USA, to the first National Acute Spinal Cord Injury Study (NASCIS 1) started in 1979 and completed in 1984 (Bracken 1984/85).

As far as can be ascertained, this was the first randomized trial of any therapeutic modality for all aspects of spinal cord injury. The second National Acute Spinal Cord Injury Study followed (Bracken 1990/93). A multicenter trial from Japan (Otao 1994) and a single-center trial from France (Petitjean 1998) both evaluated one of the treatment arms of NASCIS 2 which represents the first replication of a trial in this area. The third NASCIS trial has been reported (Bracken 1997/98).

Main results
There are few trials in this area. One steroid has been extensively studied, methylprednisolone sodium succinate, which has been shown to improve neurologic outcome up to one year post-injury if administered within eight hours of injury and in a dose regimen of: bolus 30mg/kg over 15 minutes, with maintenance infusion of 5-4 mg/kg per hour infused for 23 hours. The initial North American trial results were replicated in a Japanese trial but not in the one from France. Data was obtained from the latter studies to permit appropriate meta-analysis of all three trials. This indicated significant recovery in motor function after methylprednisolone therapy, when administration commenced within eight hours of injury. A more recent trial indicates that, if methylprednisolone therapy is given for an additional 24 hours (a total of 48 hours), additional improvement in motor neurologic function and functional status are observed. This is particularly observed if treatment cannot be started until between three to eight hours after injury. The same methylprednisolone therapy has been found effective in whiplash injuries. A modified regimen was found to improve recovery after surgery for lumbar disc disease.

Authors' conclusions
High-dose methylprednisolone steroid therapy is the only pharmacologic therapy shown to have efficacy in a phase three randomized trial when administered within eight hours of injury. One trial indicates additional benefit by extending the maintenance dose from 24 to 48 hours, if start of treatment must be delayed to between three and eight hours after injury. There is an urgent need for more randomized trials of pharmacologic therapy for acute spinal cord injury (e.g. drug cocktails, sequential therapies).

**Historical standard of care for neurologic deficits (complete or incomplete SCI) after blunt trauma - BRACKEN protocol

<table>
<thead>
<tr>
<th>High doses of ACTIVITY PREDNISOLONE ( \times ) ASAP (no later than 8 hours; preferably start in field)</th>
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<tr>
<td>30 mg/kg IV bolus (over 15 min) → after 45 min, start IV 5.4 mg/kg/h over 23 h.</td>
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<tr>
<th>Alternative - Dexamethasone</th>
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<tr>
<td>10-100 mg IV ➔ 6-10 mg IV q6h</td>
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</table>

**Treatment duration: a) if steroid is started within 3 hours of injury, continue it for 24 h. b) if steroid is initiated 3-8 hours after injury, continue it for 48 hours. N.B. steroids beyond 24-48 hours are deleterious (late inflammation is necessary for healing processes)!

**Adverse effects:
1) gastric bleeding (GI prophylaxis is necessary!!)
2) wound infection.

**Contraindications:
1) > 8 hours after SCI (steroids worsen outcome)!
2) gunshot SCI
3) drug abusers

**IAT-i LuANGE UNBEiG SsYNDROME — not recommended!
- found in cell membranes of mammalian CNS tissue
- thought to have antieutoxotic activity, promote neurite sprouting, potentiate effects of nerve growth factor, and prevent apoptosis.
- although patients with ASIA grade C and D SCI treated with Sygen demonstrate statistically significant improvement in modified Ranaz grade compared to placebo-treated patients at 4 and 8 weeks after injury, advantage is lost at subsequent follow up visits - no difference between actively treated and placebo-treated patients was noted in any of outcome measures at 1 year.
Surgical therapy is limited to:

- attempts at local cooling in human SCI patients began in the 1970s. When using an epidural cooling system during the time of surgical decompression for cervical or thoracic ASIA A patients, 65 percent improved at least one ASIA grade. Of 14 patients in the cervical cohort, 5 patients converted to ASIA B, 3 to ASIA C, and 1 to ASIA D. Of 6 patients in the thoracic cohort, 1 converted to ASIA B, 2 to ASIA C, and 1 to ASIA D.
- systemic modest hypothermia, defined as cooling to 32–34°C via a central venous catheter, has recently been the focus of several clinical studies in SCI in 35 neurologically complete, cervical ASIA Impairment Scale (AIS) A, adult patients who received 48 hours of cooling starting at mean 5.8 hours after injury, 43 percent improved at least one AIS grade by last follow-up. 23 percent regained some motor function and 11 percent improved to AIS D or better.

CARDIOVASCULAR CARE

- Neurogenic shock = hypotension + bradycardia.
- Systemic hypothermia may exacerbate spinal cord injury (secondary insult).

Goal MAP 85-90 mmHg for 7 days

(transfer of class III evidence)

- Admission to ICU for 7 days is, thus, necessary for close hemodynamic monitoring (e.g. Swan-Ganz catheter) – patients frequently develop cardiovascular instability often despite initial stable cardiac function.
- Rule out hemorrhagic causes.
- For neurogenic shock: Fluid (IV) (avoid hypovolemia! If crystallines do not restore BP, administer colloids!)
  +pressor-inotrope (agent of choice = dobutamine) + noradrenaline + phenylephrine.

  Oral: DEXMEDETOMIDINE, MIDODRINE
  *too many c/v complications (thus, NORPHENYLEPHRINE is preferred)

  N.B. Problem is hypothermia + cardiac suppression - fluid resuscitation alone may result in pulmonary edema! HR cardiac support
  Avoid a-agonists (PHENYLEPHRINE) – exacerbates (reflexly) bradycardia!

  Spinal cardiac center is at T1-4, lesions
  a) below T6 – OK to use PHENYLEPHRINE
  b) above T6 –
  c) for severe bradycardia – titrate ATROPINE IV or temporary pacing
  + optimal blood viscosity (to increase cord perfusion) is achieved with hematocrit 33-37%.

RESPIRATORY CARE

- Direct relationship exists between level of cord injury and degree of respiratory dysfunction:
  a) High lesions (ie. C1 or C2), vital capacity is only 5-10% of normal, and cough is absent
  b) Lesions at C3-6, vital capacity is 20% of normal, and cough is weak and ineffective
  c) High thoracic cord injuries (T2-4), vital capacity is 30-50% of normal, and cough is weak
  d) Lower cord injuries, respiratory function improves
  e) Injuries at T1, respiratory dysfunction is minimal, vital capacity is essentially normal, and cough is strong

  N.B. respiratory failure may worsen due to ascending cord edema!

1/3 of patients with cervical spine injuries will require intubation (most in the first 24 h) - decreasing vital capacity and increasing respiratory rate or PCO2 are all indications for possible emergent or urgent intubation.

GENERAL MEASURES

- See p. S
- Warm to maintain core temperature at least 96°F
- Intermittent bladder catheterization (to prevent permanent bladder atony) – bladder retraining, anterol sacral stimulation, urine acidification, etc.
- Adequate early nutrition:
  - nasogastric tube for first 24-48 hours (longer if ileus persists), stress ucer prophylaxis → bowel retraining
  - Early enteral nutrition (initiated within 72 hours) is safe, but has not been shown to affect neurological outcome, length of stay or incidence of complications in acute SCI.
- Indirect calorimetry is used to determine caloric needs of SCI patients.
- Anticoagulant measures must be started within 72 hours (preferred – Enoxaparin 1SC)
- Skin care (pressure sores can develop in < 1 hour in SCI patients!) – use RotoRest bed
- Prapism is not usually treated.

Surgical therapy is limited to...
a) restoration of spinal canal anatomy
b) removal of foreign bodies
c) removal of any bone / disc / hematomata that may be compressing cord.

**SPINAL CANAL DECOMPRESSION**

**Complete SCI** - fixed and permanent (little hope for major recovery of distal function - **DECOMPRESSION** surgery is unlikely to be of benefit!)

**Incomplete SCI** - surgery is likely to be of benefit:

- The study was non-randomized and the patient selection decision in early versus late group was decided by the surgeon based on clinical factors.
- The mean time to surgery in the early and late groups was 14.2 and 48.3 hours, respectively.
- Patients demonstrated a 19.8% vs. 8.8% improvement of 2 AIS grades in the early and late groups, respectively.
- The study has several limitations that must be taken into consideration. First and foremost, were the two groups similar in terms of injury severity? In the early group there were 57.7% of patients with AIS A and B injuries versus 38.2% in the late group (p <0.01). This can produce a ceiling effect in the degree of improvement.
- N.B. converting preclinical evidence that early decompression in the setting of spinal cord injury (SCI) improves neurological outcomes; however, the effect of early surgical decompression remains uncertain.

- The biggest disadvantage of early surgery – hypotension during anesthesia induction (esp. elderly) and secondary cord insult – important to communicate with anesthesia team BP goals!
- In animal studies, early decompression makes difference in outcome only if done within 6 hours post-injury.
- The chances of adequate decompression in cervical injuries: ACD→F – only 6% correction – 75% 360 – 100%.

**PENETRATING WOUNDS**

- If missiles have passed through body cavities prior to penetration of spinal canal, body cavities are explored before considering spinal injury.
- If missiles have passed through large bowel → spinal injury is **debrided**.
- Surgery can be delayed / omitted if the missiles have traversed only soft tissues, stomach, or small bowel.
- Penetrating spine wounds are usually stable.
- Injury exploration is accomplished by laminectomy.
- If missile has destroyed **vertebral body** → debride body and implant graft or prosthesis:
  - in neck – through incision along medial border of sternocleidomastoid muscle; in chest – through thoracotomy; in lumbar area – through flank dissection.

**DEFINITIVE SPINE REDUCTION & STABILIZATION**

- Goals of stabilization surgery – to prevent:
  1) neurological loss
  2) deformations
  3) pain
- Stable injuries are treated with **rest, analgesics, and muscle-relaxing drugs**.
- For elderly patients, early mobilization is important.
- NSAIDS are discouraged (may reduce radiographic healing).
- Fractures heal by 2–3 months; full stabilization may take up to 2 years.
- After initial period of healing of 12-24 weeks, moderate flexion-extension movements are safe (instability and subluxation indicate primary failure of fusion surgery).
- Persistent neck pain indicates nonhealing with instability.

SPINAL CANAL DECOMPRESSION

**Incomplete SCI** (most improve with time) → sequential neurologic examinations:

- a) stable or improving → monitor further:
- b) deteriorating → emergency surgical intervention (spinal cord **DECOMPRESSION** → STABILIZATION).

N.B. laminectomy to explore sites of cord injury may exaggerate structural instability!

- There are no studies supporting the benefit of early surgery!
Malalignment is present (dislocated facets, listhesis) → closed reduction by SKELETAL TRACTION

If closed reduction fails → SURGICAL OPEN REDUCTION & STABILIZATION

• if choosing posterior approach, MRI is necessary to exclude herniated disk (that would require anterior approach).

TONGS application

Gardner-Wells tongs:

• shave (if time permits) and prep areas above both ears.
• local anesthesis, directly through skin (without skin incision); inject also under pericranium!
• patient's head is stabilized by assistant.
• Gardner-Wells tongs are applied symmetrically just above ears - in vertical line of tragus, 1 fingerbreadth (or 1 cm) above ear lobe, below temporal ridges (areas into which tongs are placed should be below maximal transparietal skull diameter);
  - pins that are placed too central will be in thinner bone and may cause a painful hematoma in temporalis muscle,
  - placement too cephalad → risk of tongs sliding off top of scalp,
  - placement anterior to external auditory meatus → traction causes spine extension (may be desirable when treating odontoid dislocations),
  - placement posterior to external auditory meatus → traction causes spine flexion (may be desirable when trying to "unlock" facets).
• position tongs with sterile points just touching skin.
• while assistant holds tongs in place, tighten both screws simultaneously until outer end of point just protrudes beyond flush end screw - indicates that sufficient tension has been applied.
• attach traction to loop at top of tongs.

HALO application

• system consist of:
  1) halo ring/crown
  2) pins
  3) plastic vest
  4) uprights
• uprights may be adjusted for proper alignment of halo.
• halo ring is applied:
  a) for traction
  b) for stabilization (after period of initial traction with tongs).
• four sites for pin placement are located:
  ≈ 1 cm above lateral* segments of eyebrows *to avoid supraorbital nerves posterior parietal skull (that ring will be 1 cm above pinnae of ears).
• patient is lying supine on thin narrow board that holds head.
• patient keeps eyes closed (if keeps open – may be unable to close due to eyebrow skin pinned to skull)
• sterilized pins are applied to skull through anesthetized scalp with torque screwdriver; tighten to = 8-10 lb and lock in place with hexagonal nuts.
• connecting bars are employed in such manner as to hold head in neutral position → control X-ray.
• pins should be tightened second time in 24 h.
  N.B. if pins become loose days* after application – likely due to infection – do not re-tighten as pins may go intracranially!
* vs. within 24 hours – safe to retighten!
• local pin care - HYDROGEN PEROXIDE 3 times daily.
EXTERNAL CERVICAL IMMobilIZATION DEVICES

a) **halo vest** – preferred in situations of considerable instability.

b) **four-poster (occipital-mandibular) brace**: uprights, chin piece, and occiput pad limit flexion and extension; addition of head band restricts rotation at C1–2; earlobe must be opposite shoulder tip.

c) **Lerman Minerva CTO brace**:

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**Thoracolumbar VCT**

- patient is kept supine or lateral decubitus on *rotating bed* until further decision is made.
- realignment (and reduction maintenance) *cannot be accomplished with external traction*!

A. **Stable injuries** (or instability in only one column) can be managed with immobilization only – thoracic-lumbar-sacral ORTHOSIS (TLSO):
  a) orthotic 3-point vest or brace - only partially effective (thoracolumbar junction is especially difficult to immobilize) - primarily serves as reminder to patient to minimize movement.
  b) body cast (modified polypropylene jacket) - more effective immobilization; very uncomfortable.

- observe for *development of deformity* (somewhat unstable fractures may develop progressive deformity despite use of orthotic brace) before allowing ambulation / PT with brace – upright X-rays with patient in brace – check for increasing vertebral body collapse / angulation.

B. **Unstable injuries** → **SURGICAL STABILIZATION**

Approach is guided by fracture anatomy and location of spinal canal encroachment.

**Posterior approach** - does not permit access to vertebral bodies (useful when decompression of spinal canal is not major consideration)
- fixation of posterior bony elements (with Harrington rods, Cotrel-Dubousset instrumentation or Weiss springs) *permits early stabilization* → early mobilization.
patient in prone position.

Postero lateral (transpedicular) approach - improved access to vertebral bodies (decompression of ventral impingement of canal is still very technically difficult - such reduction may be achieved by placing patient in extended position on Jackson table). 

may be combined with posterior stabilization procedure.

often used for high thoracic fractures (T1-4).

patient in prone or modified lateral decubitus position.

Anterior approach - allows access to vertebral bodies at multiple levels - useful for decompression!!!

transethoracic exposure is required to access vertebral bodies down to L3; lower fractures require transabdominal-retroperitoneal exposure.

vertebral bodies are often resected and replaced with bone graft (cortaneous or from bone bank) – N.B. this does not result in early stability (vs. posterior stabilization).

patient in supine position.

Decompression

historically, simplest approach for decompression has been to “unroot” spinal canal, i.e. laminectomy − relieves pressure on posterior elements, and allows spinal cord to move away from elements anterior to it.

laminectomy may not decompress spinal cord compromised by anterior mass (H: check with US, do ligamentotomy) and laminectomy may have devastating effects on structural integrity of spinal column when anterior and/or middle columns are compromised (H: fusion).

Types of stabilization procedures:

A) posterior intersegmental fusion (fixateur interne?)- least-invasive method - use of pedicle screws to obtain stability and promote fusion of 2 segments

B) posterior rods (Harrington, Cotrel-Dubousset rod system) - require extensive exposure; effective in stabilizing multiple or unstable fractures; rods are attached with pedicle screws, stainless steel wires, clips, clamps, hooks; rods prevent further deformity and deterioration of 5-7 segments!

C) cage

D) Z-plate anterior thoracodorsal plating (form of anterior arthrodesis) - used for burst fractures.

- three-column injuries generally require both anterior and posterior stabilization (360°)

- monitoring during course of spine instrumentation is appropriate:
  1) fluoroscopy - monitors anatomical changes.
  2) somatosensory evoked potentials or Stagnara “wake-up” test monitor neural function.

*awakens patient after each step in instrumentation and asking to move those parts that are potentially affected by instrumentation

- implanted instruments are left in place indefinitely – hony elements are often eroded.

N.B permanent fixation can be assured only if bony fusion is obtained (aligned laminae should routinely be decorticated, and cancellous bone, with or without finely divided cortical bone, is inserted over decorticated laminae and anterior mass).

- external orthosis* is applied at time of recovery from anesthesia → ambulation is started in immediate postoperative period (if neurological status permits; if severe paresis - physical therapy is begun immediately), orthosis is worn for 3 months (12 weeks).

*unecessary in cases of segmental fixation with Luque rods or pedicle screws and plates

PROGNOSIS

- see p. Spinal >>

- 40% patients with SCI die within 24 hours of accident.

- 94% patients with SCI survive initial hospitalization (vs. 80% mortality for World War I soldiers).

- yearly follow-up is necessary.

- life expectancy with SCI is shortened by 15-20 years.

REHABILITATION

- see p. Spinal >>

RESTORATIVE CELL TRANSPLANTATIONS

- see p. TRN 221

Candidate cells:

1. Swann cells

2. Stem cells - types used in at least phase I trials: human embryonic stem cells, neural stem cells, bone marrow mesenchymal stem cells.

ASSOCIATED INJURIES

N.B. 75% spinal patients have some other systemic injury. 10-15% have associated head injury.

All multi-traumatized patients or any patient with severe head injury have vertebral injury or SCI until proven otherwise!!

- A B cervical spine is much more severely associated with trauma injury than with facial injury!

- vertebral fractures are caused by significant forces - associated injuries of almost any must be suspected!

- during acute stage of SCI, both GI tract and bladder become atonic; abdominal examination is unreliable - be quick to obtain abdominal CT / ultrasound / peritoneal lavage.

- brain, thoracic, abdominal injuries take precedence over spinal injuries.

PEDIATRIC ASPECTS

Biomechanics of pediatric spine are fundamentally different from that of adult:

1) ligamentous laxity; if spine is fractured – it indicates significant force!

2) wedge-shaped vertebrae

3) horizontally-oriented facets

4) predental space up to 5 mm, wider prevertebral soft tissue space

5) pseudoluxation of C6 on C7 (as well as of C7 on C1)

6) immature neck muscles and proportionally large head - cervical spine acts like fulcnum (fulcrum starts in upper cervical levels and changes progressively to lower levels as pediatric
cervical spine matures, until it reaches adult level at C5–6; most injuries occur at C1–3 level in children (< 8 yrs).

Differences of pediatric injuries from adult injuries:

1. Disproportionate involvement of upper cervical spine:

<table>
<thead>
<tr>
<th></th>
<th>Adult</th>
<th>Pediatric</th>
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</thead>
<tbody>
<tr>
<td>C1-2</td>
<td>1–2%</td>
<td>80–87%</td>
</tr>
<tr>
<td>C4-7</td>
<td>85%</td>
<td>10–15%</td>
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2. High frequency of SCIWORA (spinal cord injury without radiographic abnormality) up to 50% pediatric SCI cases - related to direct spinal cord traction, spinal cord concussion, vascular injury, H: MRI.

3. High susceptibility to delayed onset of neurological deficits.
4. Higher proportion of complete SCI (potentially poor prognosis).
5. More frequent subluxations (vs. fracture-dislocations) due to horizontally-oriented cervical facets.

Neonatal SCI

Soft and lax neonatal spine is susceptible to:

A) hyperextension trauma - hyperextension of fetal neck in utero (“flying fetus”) 
B) traction injury during BREECH DELIVERY

- cord can be transected while soft and pliable spine remains intact.
- distraction injuries following breech deliveries are most common in lower cervical + upper thoracic regions.

N.B. more commonly brachial plexus is stretched during breech delivery.

- with appropriate care, most infants survive for many years; usual causes of death - recurring pneumonia and progressive loss of renal function.

BIBLIOGRAPHY for ch. “Spinal Trauma” — follow this link >>