Spinal Trauma

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VCT – vertebral column trauma.
SCI – spinal cord injury.

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

EPIDEMIOLOGY

VCT causes ≈ 6% TRAUMATIC HOSPITALIZATIONS
SCI causes ≈ 1% TRAUMATIC HOSPITALIZATIONS.

INCIDENCE: 10.4-8.3 per million (15,000 new cases of SCI per year in the United States and Canada)
Majorly 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 (males = males, but males are less numerous in elderly populations).
The most common ETIOLOGY of SCI: motor vehicle collisions, followed by falls, gunshot wounds, and diving.

**MODERN TRENDS**
- 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 (most commonly indirect severe force) applied to vertebral column → sudden flexion, hyperextension, vertebral compression, or rotation:

1. motor vehicle accidents 30-50% (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 *paraspinal muscles atrophied* - this provided head with tremendous range of motion but considerably 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**

**Thoracic spine**
- 40% of all vertebral injuries (most commonly facet injury resulting from normal motion)
- 40% 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

**Thoracic spine (longest segment)** - high percentage of MOBILE INJURIES

- T1 region has high stability (stabilizing effects of rib cage, almost vertical-orientation of articulating processes, shingle-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 T2 result in complete paraplegia!)

**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).
- lower mechanical stability - no stabilizing effect of rib cage (T1-12 region has false ribs), spine is more horizontal, disc height.
- injuries to T2-12 can result in significant paralysis (conus medullaris).

Mid to 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**

![Hullsworth's concept: ANTERIOR COLUMN (80% of vertical strength) - vertebral bodies and interspinal disks - held in alignment by anterior & posterior longitudinal ligaments](image)

![Posterior columns - articulating facets (provide remaining 20% of vertical strength), pedicles, transverse processes, laminae, spinous processes - held in alignment by nuchal ligament complex](image)

If ROTTI COLUMNs are traumatically 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 normal motion depends on integrity of ligaments supporting involved column.
- Failure of any one of columns may involve compression effort failure (vertical compression) or failure of ligamentous strength (inability to oppose distraction).
- Spinal canal compromise 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).

N.B. MECHANICALLY stable injuries may be NEUROLOGICALLY unstable - result in spinal cord damage from:
- 1) fracture fragments (bone splintering)

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**PREVALENCE** 721-906/1,000,000.

**SPINAL TRAUMA**
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>
<th>Type of injury</th>
<th>Percent with neurologic deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fracture of vertebral body</td>
<td>3</td>
</tr>
<tr>
<td>Fracture of posterior element</td>
<td>19</td>
</tr>
<tr>
<td>Fracture of posterior elements and vertebral body</td>
<td>11</td>
</tr>
<tr>
<td>Dislocation only</td>
<td>17</td>
</tr>
<tr>
<td>Dislocation + fracture of posterior elements</td>
<td>27</td>
</tr>
<tr>
<td>Dislocation + fracture of vertebral body</td>
<td>56</td>
</tr>
<tr>
<td>Dislocation + fracture of posterior elements and vertebral body</td>
<td>61</td>
</tr>
</tbody>
</table>

**PREDISPOSING 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.
  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:

**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 SCIs show no vertebral column injury).
   - Violent neck hyperextension (e.g. pedestrian struck from rear by vehicle) may avulse tons from medulla, or medulla from cervical cord → instantaneous death.
   - spinal cord is damaged by misalignment of vertebral canal, bone fractures (bone splintering), 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);

   1. gm 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 is 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.

   *rupture of small venules
   **macroscopic cord looks swollen, reddish, and soft
   - subarachnoid hemorrhage is rare, and any extradural 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 phagocyte); axons undergo wallerian degeneration.

   • reparative stage may persist for up to 2 years – glial scar, fibrosis, spongiform (in place of necrotic – hemorrhagic areas) → healing and new myelination.

   • in 5-7 years after injury, cord becomes shrunken (replaced by fibrous tissue); progressive proliferation of acellular connective tissue → chronic adhesive arachnoiditis.

   **DEGREES OF SCI**

   1) concussion / stretching (much less common than in brain) → DEEP neurologic dysfunction with recovery in minutes or hours (e.g. SYNCOPE OF NEUROPRAXIA after athletic injury - ischaemic, though transient, neurologic 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 / transection* → 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 DEFICIT PROGRESSION 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) vasoconstriction (e.g. by dislocated vertebra) → thrombosis.

   2. Spinal shock causes systemic hypotension → ischemia exacerbation.

   **HYPERTHERMIA / HYPOXIA**

   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 oversaturation of neighbor neurons → intracellular calcium & sodium, extracellular potassium! → production of free radicals → death of healthy neurons and oligodendrocytes (AMP A receptor proteins play major role in oligodendrocyte damage).

   2. Neuroinflammation

   **WAVE OF APOTOPSIS** - further affects oligodendrocytes up to 4 segments from trauma site days → weeks after initial trauma (syngiomyelia may develop as outcome).

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

**EXPERIMENTAL THERAPIES**

**NEUROPROTECTIVE AGENTS**

1. **Riluzole** – targets excitotoxicity. see p. Spine21 >>
2. **BA-218** (Cethrin®) - Rho antagonist; promotes neuroregeneration and neuroprotection.

**CELL-BASED THERAPIES**

- primarily target **myelin repair** by restoration of oligodendrocyte population.

**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).

**PHYSICAL EXAMINATION**

I. **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 suggest compression injury.
      - oblique anterior chest or transverse lower abdominal abrasions from automobile belts mandate search for spinal, intrathoracic, and intraabdominal injuries.
  2. visible / palpable.
  3. local pain & tenderness (may be noted only in subacute phase, he be progressive).

II. **VARIATIONS OF SPINAL MANIFESTATIONS**

- examine patient's hand is gently positioned under supine patient to palpate...
- 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, poikilothermia, neurogenic shock*), Horner syndrome, sustained priapism**.

**Hyperreflexia 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):

**ASIA grade B recommendation demonstrated internal reliability and validity.

A) SPINAL CORD TRANSACTION - Myelita traumatica transversa completa (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).

N.B. central cord syndrome is ischemic lesion (frequently no radiologically identifiable fractures!!!) - neurologic changes tend to improve with time!

30% usually quadriparesis return level and bladder control, become continent, and regain some hand function

b) hematoma/hematoma (usually confined to central gray matter).

BURNING HANDS SYNDROME (variation of central cord syndrome first described in extension injuries at C5-C7 level in football players) - severe burning paresthesias in hands; no other neurologic dysfunction; > 50% initially quadriplegic patients return bowel and bladder control, become ambulatory, and regain some hand function

C) ANTERIOR CORD SYNDROME** (cervical flexion resulting in anterior cord compression; 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) BROWN-SQUIER 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) CONUS MEDULLARIS SYNDROME (disc herniation or burst fracture of T12 body).

G) CAUDA EQUINA SYNDROME

N.B. abrupt SCI (complete or incomplete) may initially cause SPINAL SHOCK, see p. TrS9 >>

*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+ 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 perineal sensation, voluntary anal sphincter contraction, and rectal tone;

N.B. absent bulbocavernous & 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 transaction).

– most cervical and thoracolumbar injuries are complete; lumbar injuries produce incomplete lesions.
– absent reflex above returned reflexes indicates LMIN injury (e.g. spinal root or cauda equina lesion - often caused by surgically correctable lesion!)

CNS Evidence-Based Guidelines for Thoracolumbar Spine Trauma (2019)

Grade C Recommendation (neurological assessment scales): numerous scales (Functional Independence Measures, Sunnybrook Cord Injury Scale, and Frankel Scale for Spinal Cord Injury) have demonstrated internal reliability and validity. ASIA impairment scale has not been specifically studied.

Grade B Recommendation - the following can be used to predict neurological function and outcome:

1) sensory (absence of pinprick sensation predicts poor bladder recovery

2) ankle spasticity - highly accurate in predicting neurogenic bladder dysfunction

3) urethral and rectal sphincter function – reappearance of sphincter function correlates with bladder recovery

4) abductor hallucis (AH) motor function (e.g. on EMG) – may be earliest and most accurate indicator of supraspinal influence and the recovery of neurologic function

CLASSIFICATION (VCT)

Cervical Spine – see p. TrS9 >>
Thoracolumbar Spine – see p. TrS9 >>

SCALES (SCI)

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!

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 injuries; 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% quadruplegia cases were iatrogenic (injudicious manipulation by paramedical personnel, examining physicians, or radiology technicians).
Airway

- spontaneous respirations
- apneic
- (suspected) C-spine injury
- lateral C-spine X-ray
- nasotracheal intubation
  - failed
  - positive
- orotracheal intubation
  - failed
- cricothyroidotomy

Spinal Trauma

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 fiberoptic camera) in in-line spinal immobilization is preferred method of airway management even with evidence of spinal injury!
- cricothyroidotomy / tracheostomy may be preceded by tempatizing needle cricothyroidotomy with jet insufflation.
- incising through cervical fascial planes could release previously contained hematoma → life-threatening hemorrhage.

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

Semirigid cervical spine collar is applied, and patient is placed on back board.

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).

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Hypotension:

- positive
- negative

C-spine injury

- child < 8 yrs OR
- laryngeal injury

Maxillofacial injury

- severe

1. Leg elevation, Trendelenburg position
2. Fluids IV

N.B. severe hypotension itself is potential cause of spinal cord damage!
Incorrect helmet removal can do harm!

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

- **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.

3. Other rescuer places *semirigid* cervical collar. *difference from rigid collar – patient can open mouth
- 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!

4. Victim (maintaining inline immobilization)* is transferred on backboard (rigid & flat) and immobilized. *very minimal traction

LOG ROLLING 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.
• 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:

• in general, neck should be placed in neutral position. However, if patient is awake and chooses to hold neck in unusual position, it should not be forced (some patients with cervical spine fractures have been made quadriplegic by ill-advised attempts to straighten neck) - immobilize patient in position in which he is found. Do not attempt to straighten out his back!

• in thoracolumbar dislocations with gibbus deformity and neurologic compromise, patient is placed in lateral decubitus position with knees flexed - this maximizes residual diameter of narrowed spinal canal (vs. supine position narrows spinal canal).

Do not use stable lateral NATO position:

• adequate suctioning device should be immediately available once patient has been immobilized to prevent possible aspiration of blood or vomitus.

• Foley catheter, nasogastric tube*, large-bore intravenous lines are always indicated in acute spine-injured patient *can rupture retropharyngeal hematoma (also gagging – stress on neck).

TRANSPORTATION

• it is desirable to tilt backboard onto left side, leaning slightly forward (to prevent aspiration):
• transportation must be rapid (10% patients suffer progressive cord l root damage between time of injury and beginning of appropriate treatment in hospital)
• insert patients left on hard backboards rapidly develop decubitis!!!
• Once in hospital, remove patient from board as soon as practical! (prolonged use is even counterproductive - uncomfortable patients may start moving on board)

DIAGNOSIS & STRATEGIES FOR IMMobilization
Specific categories of patients whose diagnoses are likely to be delayed:
1) patients with head injuries (level of consciousness)
2) patients with multiple injuries (competing pain distracts attention)
3) intoxicated patients (level of consciousness)
N.B. for these patients maintain spinal immobilization until unstable injury has been ruled out both by radiologic and clinical examination!
• other significant fractures (such as pelvic fractures, multiple rib fractures, scapular fractures) should heighten index of suspicion for vertebral fractures.
• 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 great ligamentous instability!!
• differentiating new fractures from old may be difficult (HE, radiouclide bone scan, STIR MRI).
Most of fractures missed on radiographs are: spinous process fractures, transverse processes fractures, fractures in large patients.
```text
Differential diagnosis 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. Osification centers at ends of transverse processes (may appear as fractures).
4. Spina bifida occulta.
5. Tz: 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)
Cases:
a) marked cervical spondylosis and spinal stenosis.
b) spontaneously corrected dislocation.
c) children < 8 yrs - 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
Scoliosis 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
c) suggestive mechanism of injury* associated with other painful injuries (difficult accurate clinical assessment)!
*diving trauma, fall from height, motor vehicle accident, lightning trauma
Patients with osteoporosis, arthritis, metastatic disease can develop spinal injuries as result of even very minor trauma (slips, falls, low-velocity motor vehicle crashes)
d) trauma signs above clavicles - NATO rule.
e) all trauma cases (and unable to exclude injury) – main rule!
f) suggestive clinical features:
  1) neck deformation / pain / tenderness / paravertebral muscle spasm
  2) unexplained hypotension (esp. with bradycardia, warm dry red skin)
  3) focal neurologic deficit and motor deficits in extremities
  4) diaphragmatic breathing
  5) ptiapism
  6) specific extremity position:

Numerous reports of occult cervical spine injuries + litigious environment in which medicine is practiced today → widespread practice of obtaining neck CT of all victims of MVC, falls, and sports-related injuries regardless of symptomatology!!

*questionable - numerous studies have failed to reveal spine injury, regardless of mechanism of injury, in any alert patient who did not complain of neck pain and who did not have another painful injury
Awake asymptomatic ADULT patients:

*Clearing* cervical without 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 distracting from an accurate assessment
- 4) able to complete a functional range of motion examination.

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

**Canadian C-Spine Rules (CCR):**
- any high-risk clinical factor (age >65 years, dangerous mechanism, paroxysms in the extremities) → imaging
- any low-risk clinical factor (simple rear-end motor vehicle collision, sitting position in ED, ambulatory 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-ray Utilization Group (NEXUS) criteria:**
- no posterior midline cervical spine tenderness.
- no intoxication.
- normal level of alertness.
- no focal neurologic deficit.
- no painful distracting injury.

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

Awake asymptomatic PEDIATRIC patients:

*Clearing* cervical without imaging (i.e. low-risk patients - remove C-collar):

**Official guidelines (Level 2)** - all must be present:
1) alert (GCS > 13)
2) asymptomatic (no neck pain or tenderness, normal neurological examination)
3) no painful distracting injury
4) no unplanned hypotension
5) not intoxicated.
For kids < 5 years old:
6) not motor vehicle collision (MVC)
7) not fall from > 10 feet
8) no non-accidental trauma

Awake patient + neck pain or tenderness

→ CT (3-view XR only if CT unavailable):
  * AP, lateral, and open-mouth odontoid (open-mouth is not recommended for kids < 9 yo)

**Normal imaging:**
1) Continue cervical immobilization until asymptomatic
2) Discontinue cervical immobilization following normal and adequate dynamic flexion/extension radiographs
3) Continue 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.

**C-spine injury detected** → continue immobilization and image whole spine (10% patients have incongruous-spine injury)
- cooperative patient should be cautioned against attempted movement until radiographic studies have been performed.
- obtunded or paralyzed patient may require individual assigned to hold patient’s head in alignment with longitudinal axis of body, sedation (± paralysis and intubation) may be required for patients who pose danger to themselves because of excessive movement.

**Obtunded / uncooperative 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.

**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 SPINE

1) cross-table spine lateral view* (positive in 85-90% cervical spine injuries) – initial radiologic approach (ratio 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*
Indications

- Somatosensory evoked potentials
  
  - Essential in awake cooperative patient*

  - If above views are negative but ligamentous injury is possibility (MRI is safer!!)

- 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 – pulmonary, vascular).

  - Axial CT may fail to depict subtle horizontally oriented injuries, minimal vertebral body compression fractures; H: frontal and sagittal reformations, 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 skeletal abnormalities). C0-C2 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. C0-C2 fractures (type 1 contusion) → poister prognosis. C0-C2 fractures (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 C0-C1 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 18F-fluoride or 99mTc-methylene diphosphonate. 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 99mTc-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:

**Lateral view (C7 not visualized)**  
**Tomography** - subluxation of C7 on T1 (arrow):  

A. Inadequate cross-table lateral cervical spine radiograph (C7 not visualized).  
B. Repeat lateral film demonstrates burst fracture of C7.  

Contusion of spinal cord due to hyperextension injury at C5-6 (sagittal T2-MRI):  
- swelling and mixed high and low signal in spinal cord from C3 to C7;  
- high signal in ruptured C5–6 intervertebral disc and prevertebral soft-tissue swelling.  

L1 burst fracture and conus contusion (T2-MRI):  
- wedging and retroinclination of L1 vertebral body fracture; conus is displaced and contains focus of uniformly increased signal that is several centimeters long (i.e., type 2 cord contusion).  

### COMPLICATIONS

**GENERAL SPINAL COMPLICATIONS**  
- 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 syringomyelia  
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 SYRINGOMYELIA (S. TRAUMATIC SYRINGOMYELIA):**  
- residual intramedullary cysts (e.g., after hematomyelia resorption) become distended → progressive neurologic disorder (after several months ± years of neurologic stability).
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, poliklothermy, 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

Studied drugs in SCI: GM-1 ganglioside (Sygen), rituximab, minocycline, methylprednisolone, tirilazad mesylate, naloxone, nimodipine, perfluorocarbons, ProCord (activated macrophage cell therapy), BA-210/VX-210/Cethrin (Rho inhibitor - delivered topically during decompression surgery), recombinant human erythropoietin, granulocyte colony-stimulating factor.

CNS Evidence-Based Guidelines for Thoracolumbar Spine Trauma (2019)

- N.B. task force selected 167 articles. Of these, all studies were rejected for not meeting inclusion criteria or for being off topic. No studies were selected for systematic review.

Ineffective evidence to make a recommendation for specific pharmacologic agent to improve clinical outcomes in TL fractures + SCI.
- the complication profile should be carefully considered when deciding on the administration of methylprednisolone.

STEROIDS

- reduce lipid peroxidation & inflammatory response → improvement in function (but likelihood of clinically significant functional improvement is low).
- some studies show only scattered reports of Class III evidence claim inconsistent effects likely related to random chance or selection bias.
- Class I, II, and III evidence exists that high-dose steroids are associated with harmful side effects* including death.
- there is no class I evidence supporting clinical benefit of steroids in treatment of acute SCI.*
- steroids are not FDA approved for SCI

2013 guidelines of Congress of Neurological Surgeons (CNS) and American Association of Neurological Surgeons (AANS) recommend against use of steroids early after acute cervical spinal cord injury

Steroids may be indicated when regaining 1-2 levels makes a big difference:
1) cervical SCI - improve mobility, avoid vent dependency.
2) autonomic dysreflexia - risk with SCI at Th6 and above.

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

<table>
<thead>
<tr>
<th>N ACS I</th>
<th>N ACS II</th>
<th>N ACS III</th>
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<tbody>
<tr>
<td>Class of evidence</td>
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<td>1</td>
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<tr>
<td>Randomization</td>
<td>Moderate-dose versus low-dose MePred</td>
<td>MePred versus naloxone versus placebo</td>
</tr>
<tr>
<td>Number of patients</td>
<td>330</td>
<td>487</td>
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</tbody>
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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.
Contraindications

Adverse effects

Treatment

Alternative

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

Steroids for acute spinal cord injury (COCHRANE Review)

Background

Animal experimentation with pharmacologic therapy for acute SCI 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 I) 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 causes of spinal cord injury. The second National Acute Spinal Cord Injury Study followed (Bracken 1990/93). A multicenter trial from Japan (Otani 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 whip lash 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 3 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).

NASCIS II

- Patients who received MePred within 8 h of injury had a statistically significant improvement in motor and sensory function at 6 months after injury, especially those with more severe injury, however, the sensory gains were lost at the 1-year endpoint.
- Again, there was also a trend toward higher rates of complications in the MePred group.
- 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.
- Post hoc review noted that patients receiving MePred bolus 3 to 8 h after injury had short-term neurologic improvement when given MePred for 48 h, but these gains were lost at 1 year.
- Patients undergoing 48-hour MePred infusion also had substantially higher infectious complications and a higher death rate than the 24-hour group.

NASCIS Conclusions

MePred improves outcome of acute SCI if given within 8 h of injury.

NASCIS Critique

The negative results of the NASCIS trials pertain only to post-hoc 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 neurologic scores used has meant that it is questionable whether small improvements are clinically relevant (Spencer and Bazarian, 2003).

GM-I (EUGENOS: SYGEN) - not recommended

- found in leptothoracic neurolymphatic tissue of mammalian CNS tissue
- thought to have antietoxic activity, promote neuritic 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 BENZEL 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.
Hypothermia
- 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 SCI, AYA 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.

- recommend cooling as an effective therapy for spinal cord injury due to its potential to increase neurologic recovery. The primary goal of cooling is to protect the spinal cord from further injury. Cooling has been shown to reduce ischemic injury, decrease cerebral metabolic rate, and improve oxygen delivery. Cooling also has been shown to reduce the incidence of deep vein thrombosis (DVT) and improve hemodynamic stability.

Cardiovascular Care
- Neurogenic shock = hypotension + bradycardia
  - systemic hypotension may exacerbate spinal cord injury (secondary insults).

- Goal MAP 85-90 mmHg for 7 days

- 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 minimal stable spinal function.

- rule out hemorrhagic causes.

- for neurogenic shock → fluids IV (avoid hypervolemia); if crystalloids do not restore BP, administer colloids + vasopressor (agent of choice – dopamine) + norepinephrine.

- Spinal cardiac center is at T1 above T6 – B=bradycardia; T=thoracic SCI; H=heart; C=cough.

- Spinal cardiac center is at T4 for severe bradycardia - titrate atropine IV or temporary pacing.

- optimal blood viscosity (no increase cord perfusion) is achieved with hematocrit 33-37%.

CNS Evidence-Based Guidelines for Thoracolumbar Spine Trauma (2019)
- insufficient evidence to recommend for or against the use of active maintenance of arterial blood pressure after thoracolumbar SCI.

- Consensus Statement by the Workgroup: in light of published data from pooled (cervical and thoracolumbar) SCI populations, clinicians may choose to maintain MAP > 85 mmHg in an attempt to improve neurological outcomes.

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 and weak;
  - b) lesions at C3,C4, vital capacity is 20% of normal, and cough is weak and ineffective;
  - c) high thoracic cord injuries (T2-L), 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 PaCO2 are all indications for possible emergent or urgent intubation.

Thromboembolism
- antiembolic measures must be started within 72 hours (preferred – ENOXAPARIN SC)

CNS Evidence-Based Guidelines for Thoracolumbar Spine Trauma (2019)
- Inefficient evidence to recommend for or against routine screening for deep venous thrombosis in setting PE (or venous thromboembolism-associated morbidity and mortality).

- Consensus Statement by the Workgroup: based on pooled data (cervical and thoracolumbar) SCI populations, the use of DVT prophylaxis is recommended.

- Several studies have shown the highest incidence of VTE events occurs among patients with thoracic segment SCI.

- studies suggest that early initiation of prophylaxis and continuation for a period of approximately 3-5 postinjury (or until transferred to wheelchair) are effective strategies for the prevention of VTE.

- several studies suggest the combination (chemical + mechanical prophylaxis) is better than either alone.

- Inefficient evidence to recommend a specific regime of DVT prophylaxis to prevent PE (or venous thromboembolism-associated morbidity and mortality).
Inefficient evidence to recommend a specific treatment regimen for documented venous thromboembolism that would provide fewer complications than other treatments.

**CERVICAL SCI**

**Guidelines for the Management of Cervical Spine and Spinal Cord Injuries offers more specific guidance:**

Level I Recommendation - use prophylaxis in patients with motor deficit caused by SCI.

Level I Recommendation - prophylaxis strategies:

1. low dose heparin— in combination with pneumatic compression stockings or electrical stimulation
2. LMWH, rotating beds, or a combination of modalities.

N.B. in N. low dose heparin alone or oral anticoagulation is not recommended (level II).

Early initiation (within 72 h) and a 3-month duration for prophylaxis are recommended (level III). The selective use of inferior vena cava filters is recommended for patients who either fail anticoagulation or are not candidates for pharmacologic or other mechanical modalities (level III).

**GENERAL MEASURES**

- warm to maintain core temperature of at least 96°F
- intermittent bladder catheterization (to prevent permanent bladder atony) → bladder re-training, anterior sacral stimulation, urine acidification, etc
- adequate early nutrition — nasogastric tube for first 24-48 hours (longer if ileus persists), stress ulcer prophylaxis — bowel re-training
- 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 the best means to determine caloric needs of SCI patients
- skin care (pressure sores can develop in < 1 hour in SCI patients?) → use RotoRest bed
- priapism is not usually treated.

**SURGERY**

**DECOMPRESSION → REALIGNMENT → STABILIZATION**

Surgical therapy is limited to:

- restoration of spinal canal anatomy
- removal of foreign bodies
- removal of any bone / disc / hematoma that may be compressing cord.

- surgical management includes decompression of the injured spinal cord and fixation and fusion of the spine with prevention of secondary injury, but surgery does not directly address the initial insult.

**SPINAL CANAL DECOMPRESSIVE SURGERY**

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

CERVICAL-SPINE - change of single motor level has enormous impact on functional outcome (ventilatory function and upper extremity function) - ensure that level of injury does not ascend (if ascends from edema spread - anticipate impending respiratory failure).

THORACIC / LUMBAR - precise level is of less importance.

- *problem*, only rarely sure that SCI is complete in hyperacute stage — operate only if SCI is clearly even complete SCI, especially in ongoing cord compression?
- *Plus*, subacute STABILIZATION surgery may be performed to expedite rehabilitation

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

- a) stable or improving → monitor further.
- b) deteriorating → emergency? surgical intervention (spinal cord DECOMPRESSIVE → STABILIZATION).

- N.B. laminctomy to explore sites of cord injury may exaggerate structural instability!
- *there are no studies supporting the benefit of early surgery!*

**TIMING OF SURGICAL INTERVENTION**

A) incomplete SCI (esp. with neurological deterioration) in association with ongoing spinal cord compression (from disc fragments, hematoma, or unreduced subluxation) → most common emergency surgery indication!

In incomplete SCI, rapid intervention (DECOMPRESSIVE + STABILIZATION) is most appropriate (therefore, some advocate emergency surgery at midnight).

B) complete SCI - DECOMPRESSIVE surgery has little merit, but early spinal STABILIZATION prevents complications of long-term immobilization and allows for more effective rehabilitation; if complete SCI has perceived possibility for recovering some neurological function and there is ongoing cord compression → operate emergently.

- N.B. in cauda equina syndrome, surgical decompensation is recommended even with complete deficits - potential for recovery of peripheral nerves is great!

VCT should be fixed as early as practical, once patient is physiologically stable and no longer at risk to suffer neurologic deterioration (as result of cord manipulation).

**Aim for decompression within 24 hours (as soon as hemodynamically stable)**

**SURGICAL TIMING IN ACUTE SPINAL CORD INJURY STUDY (STASCIS) -** early versus delayed (cutoff 24 hours) decompression for traumatic cervical spinal cord injury


- prospective cohort study of 313 patients with cervical traumatic SCI comparing early and late decompressive surgery using a 24-hour cutoff.

- 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, corresponding to a 2.8 times higher odds in the early group.

- critique: early versus late surgery groups were not comparable - in the early surgery group there were 57.7% of patients with AIS A and B injury versus 38.2% in the late surgery group (p <0.01). This can produce a ceiling effect in the degree of improvement patients with AIS C and D injury can achieve.
Early (< 72 hr) vs. late (> 5 d) surgery for cervical SCI – randomized trial; no difference!

- the biggest disadvantage of early surgery – hypotension during anesthesia induction (esp. elderly) and secondary cord insult – important to communicate with anesthesia team BIP goals!
- in animal studies, early decompression makes difference in outcome only if done within 6 hours postinjury.
- chances of adequate decompression in cervical injuries:
  - ACDF – only 6%
  - corpectomy – 75%
  - 360 – 100%

CMS Evidence-Based Guidelines for Thoracolumbar Spine Trauma (2019)
Insufficient and conflicting evidence regarding the effect of timing of surgical intervention on neurological outcomes.

Grade B Recommendation - “early” surgery (< 72 h after injury) is an option to reduce length of stay and complications.
- some studies report higher mortality in early surgery group.

DEFINITIVE SPINE REDUCTION & STABILIZATION
- goals of stabilization surgery – to prevent:
  1) neurological injury
  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.

Cervical VCT
Malalignment is present (dislocated facets, listhesis) → closed reduction by SKELETAL TRACTION ASAP

If closed reduction fails → SURGICAL OPEN REDUCTION & STABILIZATION
- if choosing posterior approach, MRI is necessary to exclude hemiated disk (that would require anterior approach).

TONGS application
Gardner-Wells tongs:
- shave (if time permits) and prep areas above both ears.
- local anesthesia, 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 stapes, 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 ventral will be in thinner bone and may cause a painful hematoma in temporoparietal 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 flat end screw - indicates that sufficient tension has been applied.
- attach traction to loop at top of tongs.
HALO application

- System consists 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
  = 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 retighten 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:

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)

- 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.
- 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.

**Posterolateral (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 (T1-3)
- patient in prone or modified lateral decubitus position.

**Anterior approach** - allows access to vertebral bodies at multiple levels - useful for **decompression**!
- transthoracic exposure is required to access vertebral bodies down to L3; lower fractures require transsubdiaphragmatic retroperitoneal exposure.
- vertebral bodies are often resected and replaced with bone graft (autologous 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 "unroof" 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 laminec 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 interspinous 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 rodding systems) - 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 thoracolumbar 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.
- *awaken 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 → bony elements are often eroded.
permanent fixation can be assured only if bony fusion is obtained (aligned laminae should routinely be decorticated, and can cellulose bone, with or without finely divided cortical bone, is implanted over decorticated laminae or vertebral bodies).

- external orthoses are 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); orthoses is worn for ~ 3 months (12 weeks).

*CNS Evidence-Based Guidelines for Thoracolumbar Spine Trauma (2019)

Choice of surgical approach (anterior, posterior, or combined anterior-posterior):

- Grade B Recommendation - selection of approach does not appear to impact clinical or neurological outcomes.

Conflicting evidence to recommend surgical approach for radiological outcomes or complications.

- Grade A Recommendation - both open and percutaneous pedicle screws give equivalent clinical outcomes.

- though regional motion was preserved in the nonfusion group, the nonfusion group also underwent additional surgery more often to remove the spinal implants due to screw loosening.

PROGNOSIS

- 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.
- improvements in the medical management of SCI now provide the opportunity for a near-normal life span (historically, life expectancy with SCI was shortened by 15-20 years).

Prediction of recovery according to AIS impairment scale:

<table>
<thead>
<tr>
<th>AIS grade at admission</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>First examination at 12 h</td>
<td>One year follow-up AIS grade</td>
<td>88%</td>
<td>5%</td>
<td>3%</td>
</tr>
<tr>
<td>First examination at 30 days</td>
<td>One year follow-up AIS grade</td>
<td>95%</td>
<td>3%</td>
<td>1%</td>
</tr>
</tbody>
</table>

Prediction of functional walking according to AIS impairment and other features:

<table>
<thead>
<tr>
<th>AIS level at admission</th>
<th>Functional walking (authors/references)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIS A/serious lesion</td>
<td>0% (Waters et al., 1993a,b)</td>
</tr>
<tr>
<td>AIS A/thoracic and lumbar lesions</td>
<td>5% (Waters et al., 1993a,b)</td>
</tr>
<tr>
<td>AIS at admission: recovery of lumbar ambulation at 1 year post-injury</td>
<td>1% (Cowan et al., 1991)</td>
</tr>
<tr>
<td>AIS B (only slight back preservation)</td>
<td>6% (Cowan et al., 1991)</td>
</tr>
<tr>
<td>AIS B (light touch + pin prick preservation)</td>
<td>0% (Cowan et al., 1991)</td>
</tr>
<tr>
<td>AIS C &lt; 30 years</td>
<td>0% (Ramos et al., 1997)</td>
</tr>
<tr>
<td>AIS C &lt; 50 years</td>
<td>5% (Ramos et al., 1997)</td>
</tr>
<tr>
<td>AIS D &lt; 50 years</td>
<td>1% (Ramos et al., 1997)</td>
</tr>
<tr>
<td>AIS D &gt; 50 years</td>
<td>1% (Ramos et al., 1997)</td>
</tr>
</tbody>
</table>

Prediction of recovery according to AIS impairment scale:

- see p. 515

- 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.
- improvements in the medical management of SCI now provide the opportunity for a near-normal life span (historically, life expectancy with SCI was shortened by 15-20 years).

REHABILITATION

- see p. 515

Candidate cells:

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

ASSOCIATED INJURIES

- see p. 505

RESTORATIVE CELL TRANSPLANTATIONS

- see p. 505
N.B. 75% spinal patients have some other systemic injury; 10-15% have associated head injury; 10-15% have associated head injury.

- vertebral fractures are caused by significant forces - associated injuries of almost any organ 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 vertebral
3) horizontally-oriented facets
4) predental space up to 5 mm, wider prevertebral soft tissue space
5) pseudoluxation of C2 on C3 (as well as of C3 on C4)
6) immature neck muscles and proportionally large head - cervical spine acts like fulcrum (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 C1-2 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>
</tr>
</thead>
<tbody>
<tr>
<td>C1-2</td>
<td>1-2%</td>
<td>60-87%</td>
</tr>
<tr>
<td>C2-3</td>
<td>85%</td>
<td>30%-40%</td>
</tr>
<tr>
<td>Thoracolumbar</td>
<td>10-15%</td>
<td>5%</td>
</tr>
</tbody>
</table>

2. High frequency of SCWORA - 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 >>