Head Injury/C-Spine/Spinal Cord Trauma

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HEAD INJURY/C-SPINE/SPINAL CORD TRAUMA

I. HEAD TRAUMA

A. Anatomy

1. Scalp (five layers) - pneumonic – S-C-A-L-P
   a. Skin, subCutaneous, galea Aponeurotica, Loose areolar and Periosteum
   b. Vascular with vessels between the dermis and galea
   c. Lacerations of scalp may cause significant or even fatal hemorrhages, especially in children
   d. Hematoma of the scalp occurs between galea and periosteum (subgaleal) or beneath the periosteum (subperiosteal).
   e. The sharp cutoff of a subperiosteal hemorrhage at a suture line may be mistaken for a fracture on palpation
2. Skull - Rigid brain case protecting neural structures
   a. Frontal, ethmoid, sphenoid, parietal (2), temporal (2), occipital bones
   b. Bones of skull have inner and outer tables with cancellous bone between
   c. Irregularity of floor or cranial vault increases problems with brain contusion
3. Meninges (Intracranial hemorrhages are named in relation to these layers)
   a. Dura Mater - tough outer covering, adheres to bone, folds compartmentalizes the brain
      i. Midline falx cerebri – separates brain into 2 cerebral hemispheres
      ii. Tentorium cerebelli – separates cerebellum & brainstem from cerebrum
      iii. Falx cerebelli – separates cerebellum hemispheres
      iv. Dural layers also separate to form dural sinuses
   b. Arachnoid - thin, filmy, veil-like membrane
   c. Pia Mater - thin membrane adherent to brain surface
4. CSF
   a. Production by choroid plexus in lateral, third and fourth ventricles
   b. Function
      i. Protection during trauma
      ii. Fluid pathway for delivery of substances to the brain & spinal column
      iii. pH influences respiratory drive & cerebral blood flow
   c. Pressure – 5 – 15 mmHg in ventricles
5. Blood
6. Brain
   a. Weighs 1400 g
b. Occupies 80% cranial cavity
c. Divisions – cerebrum, cerebellum, brainstem

B. Cerebral hemodynamics

1. Blood brain barrier (BBB) – maintains microenvironment
2. Cerebral Blood Flow (CBF)
   a. 15% of cardiac output
   b. Maintained by altering diameter of cerebral vessels
      i. HTN, alkalosis, hypocarbia – vasoconstriction
      ii. Hypotension, acidosis, hypercarbia, hypoxia – vasodilatation
      iii. Injured vessels lose responsiveness
   c. Constant at mean arterial pressure of 60 – 150 mmHg
   d. CBF increases linearly as MAP increases above 150 mmHg
3. Cerebral Perfusion Pressure (CPP) - Pressure gradient across the brain
   a. Estimated by MAP – ICP (intracranial pressure) = CPP
   b. CBF depends on CPP, CBF remains constant when CPP is 50-160mm Hg
   c. Autoregulation is lost when CPP < 40 mmHg

C. Pathophysiology

1. Primary brain damage
   a. Mechanical, irreversible damage occurs at time of trauma – lacerations, hemorrhages, etc
   b. Produces injury at the cellular level
2. Secondary brain damage – intracellular & extracellular pathological processes arise as a direct result of trauma. Damage to nerve axons is followed by chromatolysis. Followed by either dissolution of cell or subsequent recovery.
3. ICP closed box model - Intracranial pressure (ICP) is a function of the volume of these three components and any space occupying lesion. Increased ICP > 15 mmHg
   a. CSF
      i. 1st compensatory mechanism – displace CSF to spinal column
      ii. Can accommodate 50 to 100 ml lesion – after that small increases in volume produce large increases in ICP as can be seen by the diagram below
      iii. CSF production not increased in trauma unless a space occupying lesion obstructing CSF flow; usually not a major contributing factor
**Pressure-Volume Curve**

b. Blood - autoregulated  
   i. As ICP increases, CPP decreases, autoregulation is lost  

c. Brain parenchyma - Types of brain swelling  
   i. Congestive brain swelling – caused by increased blood volume  
   ii. Cerebral edema – increase in brain volume caused by fluid in cerebral cells  
      • Vasogenic Edema – altered permeability of blood-brain barrier  
      • Cytotoxic Edema – direct cellular injury (hypoxia) disables Na-ATP pump, allows sodium & water to accumulate intracellularly

D. Herniation Syndromes

1. **Uncal herniation (Transtentorial)**  
   a. Due to increasing volume in the temporal lobe or middle cranial fossa.  
   b. Uncus of temporal lobe herniates across incisura of the tentorium cerebelli and impacts against midbrain.  
   c. Ipsilateral CN III compressed by the shift resulting in **ipsilateral pupillary dilatation - aniscoria**, ptosis, impaired EOM, sluggish pupil, finally nonreactivity  
   d. Contralateral Babinski develops early  
   e. Weakness is usually contralateral  
   f. 25% have ipsilateral weakness due to compression of the contralateral cerebral peduncle against the opposite tentorium (**Kernohan's notch phenomena**) – gives false localizing signs  
   g. Impaired consciousness is NOT a reliable early sign but continued herniation results in agitation & restlessness → lethargy → coma  
   h. As herniation continues, contralateral pupil may dilate in the preterminal stage  
   i. Develop decerebrate posturing  
   j. Respiratory – normal → hyperventilation → atactic breathing → apnea  
   k. Cardiac – large fluctuations of blood pressure & cardiac
2. **Central Transtentorial herniation**
   a. Increased volume or pressure in the supratentorial compartment (usually frontal or occipital areas) causing bilateral or central pressure is exerted on brain above
   b. Usually more chronic than uncal herniation
   c. Results in brainstem ischemia or hemorrhage from compression and shear of perforating arteries from basilar artery (Duret hemorrhages)
   d. May be the etiology of the **Cushing response** (i.e., arterial hypertension, bradycardia & respiratory irregularities seen with increasing ICP)
   e. Decreased LOC
   f. Motor – bil. weakness, increased tone, bilateral Babinski’s
   g. Pupils – pinpoint, loss of light reflex
   h. Respiratory – yawns & signs → sustained hyperventilation → slow & irregular breaths → apnea
   i. Posturing – decorticate → decerebrate

3. **Cerebellotonsillar** or posterior fossa herniation
   a. When supratentorial compression continues unchecked, or when an expanding mass is situated in the posterior fossa or large central vertex mass
   b. Cerebellar tonsils prolapse through the foramen magnum
   c. Causes compression of the medulla and respiratory arrest. (Rapidly fatal)
   d. Pupils – pinpoint
   e. Motor – flaccid quadriplegia

4. **Upward Transtentorial**
   a. Caused by upward expanding posterior fossa lesion
   b. LOC declines rapidly
   c. Pupils – pinpoint
   d. Downward conjugate gaze

E. **Assessment**

1. **History**
   a. Mechanism of injury
   b. Loss of consciousness
   c. ETOH or drugs
   d. Complaints before the trauma
   e. Assessment & treatment on the scene
   f. Posttraumatic seizures
   g. Past medical history
      i. Comorbid conditions – Coagulopathy
      ii. Medications - Anticoagulants
   h. Return to normal mental status

2. **Vital signs**
   a. Cushing response - hypertension, bradycardia and respiratory
irregularities.

b. Hypotension from head injury occurs only as a preterminal event (brainstem regulatory collapse) or from intracranial bleeding into an expandable skull of a young child. Look for associated visceral injuries.

3. Neurologic exam
   a. Assess level of consciousness - single most important factor
   b. Examine pupils - Note size, shape, and reactivity
      i. **Unilateral fixed, dilated pupil**
         - With normal mental status, R/O drops, surgery or congenital
         - **Abnormal mental status, R/O increasing ICP and uncal herniation**
      ii. Pinpoint pupils
          - Pontine lesion
          - Narcotics
      iii. Pupils fixed and dilated bilaterally
         - Hypoxia/anoxia or hypotension
         - Atropine/cycloplegics
         - Brain death
         - Hypothermia
         - Herniation syndromes
   c. Check extra-ocular movements (EOM) for weakness
      i. Spontaneous eye opening suggests intact reticular activating system
      ii. Reflexes – test the brainstem
         - **Oculocephalic (Doll's eyes)** –(must r/o C-spine injury first)
           ** Turn patient’s head to stimulate vestibular apparatus - movement of eyes in opposite direction → intact brainstem**
           ** Eyes turn same direction as head → damaged**
         - Oculovestibular (caloric response) (Safe to test w/ C-spine injury)
           ** Need intact tympanic membrane**
           ** Cold water placed in ear canal results in movement of endolymph in semicircular canals.**
           ** Interpreted by pons as movement of head in opposite direction – eyes deviated toward the ear & nystagmus away → normal**
           ** Comatose – no nystagmus, just turn eyes towards cold water → intact brainstem**
         - These tests assess integrity of cranial nerves III, VI.
         - Plus nuclei in the midbrain and pons
   d. Assess cranial nerves II through XII
   e. Check movement and power of each extremity. Also examine sensation DTR’s and Babinski sign
f. Rectal sphincter tone, voluntary contraction and sensation

g. Carefully examine cervical, thoracic, and lumbar spine for tenderness

h. Glasgow Coma Scale
   i. Proposed by Teasdale and Jennett in 1974 as a means of providing uniformity for all examiners. 3 criteria

<table>
<thead>
<tr>
<th>Eye Opening</th>
<th>Verbal Stimuli</th>
<th>Motor Response</th>
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<tbody>
<tr>
<td>Spontaneously 4</td>
<td>Oriented, converses 5</td>
<td>Obeys verbal commands 6</td>
</tr>
<tr>
<td>To verbal 3</td>
<td>Disoriented, converse 4</td>
<td>Localizes pain 5</td>
</tr>
<tr>
<td>To pain 2</td>
<td>Inappropriate words 3</td>
<td>Flexion withdrawal 4</td>
</tr>
<tr>
<td>None 1</td>
<td>Incomprehensible 2</td>
<td>Abnormal flexion 3</td>
</tr>
<tr>
<td></td>
<td>No response 1</td>
<td>Extension 2</td>
</tr>
</tbody>
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   ii. Provides uniformity between examiners
   iii. Good for rapid serial exams - not a substitute for a careful neuro exam
   iv. Falsely lowered due to hypoxia, hypotension and intoxication, intubation, non-English speaking, etc.
   v. Risk severity
      • ≤ 8 for at least 6 hours → severe injury
      • 9 – 13 → moderate head injury
      • 14 – 15 → mild head injury
i. Posturing
   i. **Decorticate** – flexion of upper & extensions of lower ext, lesion of the midbrain
   ii. **Decerebrate** – extension of both upper & lower extremities - lesion more caudally, worse prognosis

F. Management of severe head trauma

1. Overview
   a. Normal ICP in adults is < 15-20 mm Hg.
   b. CT cannot estimate or predict ICP
   c. Fatal if uncontrolled > 25-30 mm HG.

2. Hyperventilation
   a. Fastest means of decreasing ICP. PCO2 should be reduced to 30-35 torr
   b. Onset of action < 30 sec, peak effect 8 min
   c. Should not be continued over 24 hours
   d. Hyperventilation causes cerebral vasoconstriction decreasing intracranial blood volume. May cause focal areas of ischemia.

3. Osmotic agents
   a. Mannitol 20% solution
      i. 0.25 - 1 gm/kg IVPB
      ii. Results in decrease in brain volume – draws out tissue water
iii. Onset - minutes, peak 60 minutes, duration 6 – 8 hours
iv. Side effects – renal failure in large amt, paradoxical intracranial hemorrhage
v. Neuroprotective properties – blood volume expander reduces blood viscosity free radical scavenger
vi. No evidence that fluid restriction is beneficial. Patients should be kept normovolemic. Avoid over-hydration.
b. Hypertonic saline
   i. Some encouraging data, inconclusive
   ii. Side effects – renal failure, central pontine myelinoysis, rebound ICP elevation

4. Decompression
   a. Burr Holes – for herniation not responding to other methods
   b. Surgical decompression – no focal lesion, but massive edema
c. External ventricular drain EVD (ventriculostomy).
   i. In rare circumstances, a neurosurgeon may place a bedside or ED, E.V.D. for drainage of CSF
   ii. Controversial, but works immediately

5. Steroids of no benefit – high dose methylprednisolone detrimental

6. AVOID HYPOTENSION - Strive to maintain systolic BP of at least 90 mm Hg

7. Avoid other secondary insults such as hypoxia, hyperthermia, seizures or electrolytes imbalances

8. Seizure prophylaxis - indications
   a. Depressed skull fracture
   b. Paralyzed and intubated patient
   c. Seizure at time of injury or ED presentation
   d. GCS<8
   e. Acute subdural or epidural hematoma
   f. Acute ICH
   g. Prior history of seizures
   h. Penetrating brain injury

   a. Reduce cerebral metabolic demands
   b. Affects vascular tone
   c. Inhibits free radical-mediated cell membrane lipid peroxidation
   d. Used when other methods to reduce ICP unsuccessful

10. Antibiotics
   a. Indicated – open skull fx, penetrating head injury, complicated scalp lacerations
   b. Not indicated – presence of otorrhea or rhinorrhea

11. Factor VII – conflicting results, individualized

G. Lesions following head injury

1. Concussion
a. Transient loss of neurological function resulting in loss of consciousness, usually resolves < 6 hours
b. Amnesia for events before injury (retrograde) and after injury (anterograde) may be present
c. No significant gross anatomic, microscopic or radiographic damage demonstrable, PET scans show abnormalities
d. Adults - headache, confusion, amnesia
e. Peds – confusion, restlessness, lethargy, irritability
f. CT of brain if significant head injury or loss of consciousness, focal neurologic deficits, prolonged LOC, or persistent altered level of consciousness
g. Admit if unconscious for prolonged period or unstable home environment
h. Worrisome sports injury, development of several scoring systems, not any accepted as the system to use
i. Second impact syndrome- sustain second concussion before complete resolution of symptoms from first concussion, experiences rapid, usually fatal, neurologic decline
j. Recommendations for return to sports
   i. Must wait 1 week after becoming asymptomatic
   ii. Must wait 1 month if period of LOC or prolonged posttraumatic amnesia
k. Postconcussive syndrome – headache, sensory sensitivity, memory or concentration difficulties, irritability, sleep disturbances, or depression which persist for days to months after a concussion

2. Skull fractures
   a. Significance - Presence of skull fracture in a conscious patient increases risk of intracranial hemorrhage 400 times
   b. Clinically significant if:
      i. Result in intracranial air & pass through an air-filled space (sinus)
      ii. Associated with an overlying scalp laceration (open)
      iii. Depressed skull fx below the inner table
      iv. Overlie a major dural venous sinus or middle meningeal artery
   c. Types
      i. Open fractures violate skin, nasal sinuses, and/or middle ear
         • Requires hospital admission for observation, possible antibiotic coverage / may need debridement.
      ii. Closed fractures do not violate dura, skin, or sinus
      iii. Linear fractures do not have displacement of bone edges.
         • May have damaged meningeal vessels with resulting epidural hematoma.
         • If no intracranial hematoma is present, no specific E.D. treatment is required.
- Children with linear fractures should have repeat skull X-rays in 6 months to rule out a leptomeningeal cyst or growing fracture
  iv. **Sutural diastasis** – traumatic disruption of a cranial suture – coronal or lambdoid sutures
  v. **Commminuted skull fracture** – multiple linear fractures radiate from impact site
  vi. **Depressed skull fractures** - bone piece below the plane of the skull
    - Greater than the full thickness of the skull require surgical treatment whether open or closed
    - Greater incidence of underlying brain injury.
  vii. **Basilar skull fracture**
    - Fracture through one or more of the bones of the skull base - sphenoid, ethmoid, temporal or occipital bones.
    - Usually clinical diagnosis based on signs such as raccoon eyes (periorbital ecchymosis), Battle sign (mastoid ecchymosis), hemotympanum, otorrhea, or rhinorrhea.
    - Can injure CN III, IV, & V
    - Fx of sphenoid can cause carotid pseudoaneurysms or carotid venous fistulae
    - Rarely demonstrable on plain X-ray.
    - Routine CT with bone windows may or may not show fracture line – can increase yield with posterior fossa views and thin cuts < 5mm.
    - All require admission until cessation of CSF leak – usually 1 week
    - Increased risk of meningitis. Use of prophylactic antibiotics is controversial – usually not given in 1st wk

3. **Cerebral contusion**
  a. **Bruises** on surface of brain after direct local trauma or sudden acceleration / deceleration injuries
  b. Coup lesions occur at the site of impact. More common Contrecoup lesions occur opposite the site of impact.
  c. Contusions occur most frequently at the frontal and temporal polar regions, as well as over the irregular basal areas.
  d. Neurologic deficits reflect location of the contusion
  e. Can be ischemia of underlying brain tissue due to compression by blood
  f. Cerebral edema with increasing ICP likely reaching maximum at 48 to 72 hours
  g. Work-up includes CT, and admission for observation.
  h. Patient is at risk for seizures so anticonvulsant prophylaxis indicated.
4. **Diffuse axonal injury**
   a. Caused by mechanical shear force
   b. Damaged axons become edematous & separate from each other
   c. Effects range from mild confusion to death.
   d. **Macrosopic appearance may be normal** or show small hemorrhages usually located near third ventricle, within the white matter of the corpus callosum, internal capsule of the brainstem, better seen with MRI
   e. Management is directed at controlling ICP & supportive
   f. Clinically, all patients present in coma, and duration of coma indicates severity of injury.
      i. Mild – coma 6 – 24 hours, 15% mortality, recover have mild or no disabilities
      ii. Moderate – coma > 24 hours, 25% mortality, awake & have persistent moderate to severe cognitive deficits
      iii. Severe – coma > 24 hours, persistent brainstem & autonomic dysfunction, mortality high, few in persistent vegetative state

5. **Epidural hematoma**
   a. Bleeding between inner table of skull and dura.
   b. Rare in elderly & children under 2 yo
   c. Due to **laceration of meningeal vessel** (artery or vein) or dural sinus by a skull fracture.
   d. **Most common** site (70%) is the lateral aspect of the cerebral hemisphere by tears of the middle meningeal artery or vein due to fracture of **the temporal bone**
   e. Occurs in frontal pole, temporal fossa, and posterior fossa (10% each).
   f. Bleeding strips the tightly adherent dura from the bone, yielding a **bi-convex appearance** on CT.
   g. **Classical presentation** - patient is stunned or knocked unconscious by a blow to the head, recovers (lucid interval), then develops a progressive contralateral hemiparesis and lapses into unconsciousness as the clot expands
   h. Incidence - **1% - 2% of head injured pts** presenting to ED
   i. Mortality rate - 6% to 27%. Dependent on size of blood, amount of underlying brain injury, length of time to treatment
   j. Treatment - **surgical evacuation**
      i. > 30 cm in volume
      ii. Comatose w/ anisocoria – the sooner the better

6. **Subdural hematoma (SDH)**
   a. Bleeding in the potential space between the dura & brain
   b. Results from tearing of a cortical bridging vein to sagittal sinus with sudden acceleration/deceleration injuries
   c. Blood spreads paralleling both brain surface and inner table of skull characteristic crescent-shaped appearance on CT
   d. Often associated with significant underlying brain injury
e. Most common in alcoholics & elderly w/ atrophy
f. Classification
   i. **Acute subdural hematoma** - discovered within the first 24 hours of injury. Fresh clotted, high density blood on CT.
      - Have mental status changes and/or neurologic deficit.
      - Mortality - 25% to 60%.
      - Good functional recovery occurs 25% to 35% of cases
      - Surgical treatment: > 10 mm in size or > 5mm midline shift
   ii. **Subacute SDH** - symptomatic between 24 hours to 14 days post injury. These have improved prognosis.
      - Appears *isodense* on CT scan
      - Most require surgery
   iii. **Chronic SDH** - symptomatic greater than 2 weeks post injury
      - **Hypo-dense or isodense** on CT.
      - Current theory - continued small hemorrhages within the capsule lead to a gradual increase in size over time
      - If symptomatic – need surgery
      - Present with subtle symptoms H/A, dementia, lethargy, or stroke-like symptoms of focal weakness

7. **Intracerebral hemorrhage**
   a. Bleeding into the brain parenchyma from small blood vessel injury. May be a continuum from contusion to hemorrhage.
   b. Seen in patients on anticoagulant therapy.
   c. **Most common frontal and temporal lobe** (85%)
   d. Similar reactive brain swelling as with contusions at 48 to 72 hours
   e. May require surgical evacuation for control of ICP
   f. Mortality rate - 55%
   g. Presentation - *altered level of consciousness and focal deficits depending on size and location of hematoma*

8. **Traumatic subarachnoid hemorrhage**
   a. Trauma is the most common cause of blood in the subarachnoid space & CSF
   b. **Most common abnl seen on CT after head injury**
   c. Syx - headache, neck stiffness, photophobia
   d. Vasospasm occurs 48 hours to 2 weeks post injury
   e. **Treat calcium channel blockers (nimodipine & nicardipine)**
   f. No intervention needed. Admit for observation.

9. **Subdural hygroma**
   a. Collection of clear, xanthochromic blood-tinged fluid in the dural space
   b. Pathogenesis not clear
   c. Syx – *headache, nausea, vomiting, decreased LOC, focal*
motor deficits
d. Treatment – if asymptomatic → observation, symptomatic → surgery

H. Imaging and disposition

1. Classification of patients with **MINOR** head injury (GSC 14-15)
   a. Low risk
      i. Asymptomatic
      ii. No other injuries
      iii. No focality on exam
      iv. Normal pupils
      v. No change in consciousness
      vi. Intact orientation/memory
      vii. Initial Glasgow coma scale score of 15
      viii. Accurate history
      ix. Trivial mechanism
      x. Injury > 24 hours ago
      xi. No or mild headache
      xii. No vomiting
      xiii. No preexisting high-risk factors
   b. Medium risk
      i. Initial GCS score of 15
      ii. Brief loss of consciousness
      iii. Posttraumatic amnesia
      iv. Vomiting
      v. Headache
      vi. Intoxication
   c. High risk
      i. Focal neurologic findings
      ii. Asymmetric pupils
      iii. Skull fracture
      iv. Multiple trauma
      v. Serious, painful, distracting injuries
      vi. External signs of trauma above the clavicles
      vii. Initial GCS score of 14 or 15
      viii. Loss of consciousness
      ix. Posttraumatic confusion/anemia
      x. Progressively worsening headache
      xi. Vomiting
      xii. Posttraumatic seizure
      xiii. History of bleeding disorder/anticoagulation
      xiv. Recent ingestion of intoxicants
      xv. Unreliable/ unknown history of injury
      xvi. Previous neurologic diagnosis
      xvii. Previous epilepsy
      xviii. Suspected child abuse
      xix. Age >6 yr. & < 2 years
d. Guidelines for imaging  
   i. Low risk – neuroimaging not indicated, observe with competent person 12 – 24 hours  
   ii. Moderate risk – CT scan or prolonged observation  
   iii. High risk – CT scan  

e. Guidelines for disposition  
   i. Low risk – normal exam and obs for 4 – 6 hours  
   ii. Moderate risk – depends on CT results, exam, reliable observer, early follow-up  
   iii. High risk – if negative, clinical decision if discharge  

2. Moderate head trauma  
   a. GCS of 9 – 14  
   b. All should receive CT scan  
   c. All should be admitted for observation regardless of CT scan  

3. Severe head trauma  
   a. GCS of < 8  
   b. All should receive CT scan  
   c. All should be admitted  

4. Transfer indications  
   a. Early transfer for patient with possible significant injury if without neurosurgical or diagnostic capabilities  
   b. Stabilize patient prior to transfer, if possible  
   c. R / O C-spine fracture or immobilize neck.  

I. Complications after head injury  

1. Seizures  
   a. Common in children  
   b. Acute – within 24 hours following injury, transient, not repeated  
   c. Subacute  
      i. 24 – 48 hours following injury  
      ii. Etiology: increasing cerebral edema, small hemorrhages, penetrating injuries  

2. Central nervous system infections  
   a. Meningitis after basilar skull fractures  
      i. Typical signs & syx of meningitis  
      ii. Organisms  
         • Pneumococci – within 3 days of injury, treat w/ Ceftriaxone or cefotaxime plus vancomycin  
         • Gram negatives - > 3 days after injury – Ceftriaxone or cefotaxime plus nafcillin and vancomycin  
   b. Brain abscess  
      i. Infrequent  
      ii. From penetrating injuries, depressed skull fx, fracture crosses paranasal sinus  
      iii. Syx – headache, nausea, vomiting, decrease LOC, focal neurologic deficit, nuchal rigidity, seizure
iv. Organisms – S. aureus & gram negatives  
v. Treatment – antibiotics and surgery  
c. **Cranial osteomyelitis**  
i. Due to penetrating injury  
ii. Syx – redness, swelling, warmth at site  
iii. Diagnosis – 50% seen on plain skull films, others bone scan and gallium scan  
iv. Treatment – surgical debridement & antibiotics  

3. **DIC**  
a. Develops within hours after injury  
b. Injured brain activates the extrinsic clotting system  
c. If stable, develops DIC, repeat the CT scan to find new hemorrhage  
d. Treatment – correct neurosurgical condition, treat with FFP and platelets as indicated  

4. **Neurogenic pulmonary edema**  
a. Develops minutes to days after injury  
b. Caused by altered hydrostatic forces & microvascular permeability  
c. Treatment – lower ICP  

5. **Cardiac Dysfunction**  
a. Variety of arrhythmias – most common is SVT  
b. Theory – cause is autonomic nervous system dysfunction and/or high level of catecholamines  
c. Treatment goals stop arrhythmia and support cardiac output  

II. **CERVICAL SPINE TRAUMA**  

A. **Immobilization**  

1. **Indications**  
a. Abnormal mental status of uncertain cause  
b. Any trauma victim, especially sports, MVA, falls  
c. Head or facial trauma  
d. Any neurologic deficit  
e. Neck or back pain  
f. Localized tenderness  
g. Unexplained hypotension  
h. Priapism or abdominal breathing  

2. **Techniques**  
a. Initially at site - spine board and extrication collar  
b. Immobilize head and body  
i. Anchor torso to spine board  
ii. Blocks along sides of head  
iii. Tape/strap across forehead and collar to board  
iv. Keep patient supine and immobile  
c. Have adequate suction available, and be able to tilt board, if needed.
B. Initial management & assessment

1. Primary survey
   a. Assure patient airway and ventilatory ability
      i. Lost either from phrenic nerve injury, (C3-C5) retro hematoma, or associated facial injuries
      ii. Recommended - In-line orotracheal intubation with RSI & in-line stabilization
      iii. Cricothyrotomy – failed orotracheal intubation, associated massive facial trauma, RSI contraindicated
   2. Circulation. Check adequacy
      a. **Spinal shock** – complete loss of motor, sensory, reflex, and sympathetic function below level of injury
         i. Occurs w/ complete cord transection or injury above sympathetic plexus. Rarely due to temporary spinal cord concussion.
         ii. Heart rate normal or bradycardic due to unopposed vagal tone. Bradycardia responds to atropine
         iii. No peripheral signs of vasoconstriction – patient usually warm and vasodilated
         iv. Diagnosis of exclusion – rule out blood loss
         v. Start treatment with crystalloids
         vi. High cervical lesions, severe hypotension (<70 mm Hg) – Trendelenburg position, vasopressors, cardiac pacing
         vii. Usually good urine output.
         viii. Cessation marked by return of reflexes or bulbocavernous reflex
   3. Secondary survey - assess completely motor & sensory functions
      a. Assess breathing pattern - abdominal breathing indicates lower cervical injury - low tidal volume breaths
      b. Look for muscle fasciculations
      c. Look for Horner's Syndrome – unilateral facial ptosis, miosis and anhydrosis - from C7-T2 level
      d. Look for painful dysesthesias
      e. Palpate entire spine and musculature
      f. Test DTRs – localizes and diagnostic
         i. Paralysis with intact DTRs – upper motor neuron (spinal cord) lesion
         ii. Paralysis with absent DTRs – lower motor neuron (nerve root) lesion or spinal shock
      g. Baseline assessment of sensation
         i. Light touch-posterior column
         ii. Pain (pin prick) – anterior spinothalamic tract
         iii. Proprioception – posterior column
         iv. Islands of sparing means patient has 50% chance of functional motor recovery. Must find early to facilitate
treatment.
v. Evidence of incomplete injury - sacral sparing, normal rectal tone, flexor toe movement

C. Spinal cord syndromes

1. Mechanisms
   a. Primary
      i. Transection of the neural elements
      ii. Compression between the vertebra
      iii. Primary vascular damage – Ischemia or compression
   b. Secondary – cascade of events caused by the primary event causing ischemia to the neurons

2. Complete spinal cord syndrome – nothing below injury
   a. If persists greater than 24 hours after injury, 99% will not have functional recovery.
   b. Check for persistent perianal sensation, rectal sphincter tone or slight flexor toe movement - indicates partial lesion with potential for functional recovery
   c. R/O spinal shock (see above) – absence of bulbocavernosus reflex indicates presence of spinal shock mimics complete spinal cord syndrome
   d. R/O intra-abdominal, etc. injuries as no sensation in abdomen

3. Incomplete spinal cord lesions
   a. Central Cord Syndrome (most common)
      i. Degenerative arthritis of neck w/ forceful hyperextension.
      ii. Hypertrophied ligamentum flavum buckles into cord contuses central portion (vascular injury).
      iii. Effects pyramidal and spinothalamic tracts, which are in center of cord, with variable sensory involvement.
      iv. Upper extremity deficits greater than lower extremity deficits
      v. Variable prognosis - 50% regain some function.
      vi. Urinary retention invariably found.
   b. Brown-Sequard syndrome
      i. Functional hemisection of cord.
      ii. Penetrating injury or lateral mass fractures of C-spine.
      iii. Ipsilateral paralysis, loss of proprioception, touch and vibration (posterior column) & contralateral loss of pain and temperature (spinothalamic tract)
      iv. Prognosis fair to good.
   c. Anterior cord syndrome
      i. Hyperflexion injuries, protrusion of bony fragments or herniated disc into anterior spinal canal, OR by laceration/thrombosis of anterior spinal artery.
      ii. Complete motor paralysis and hypalgesias below level injury with preservation of position, touch and vibration (posterior column).
d. **Horner's syndrome**
   i. Damage to cervical sympathetic chain
   ii. **Ipsilateral ptosis, miosis, anhydrosis**
   iii. Due to direct cervico-medullary trauma or vertebral artery occlusion

e. Posteroinferior cerebellar artery syndrome
   i. Dysphagia, dysphonia, hiccups, nausea, vomiting, dizziness or vertigo, cerebellar ataxia

f. Dejeune onion skin pattern
   i. Analgesia of the face
   ii. Damage to spinal trigeminal tract

g. Cauda equina syndrome
   i. Perineal or bilateral leg pain, bowel or bladder dysfx, perianal anesthesia, diminished rectal sphincter tone, lower extremity weakness

h. **SCIWORA**
   i. More common in peds
   ii. Mechanism is unclear
   iii. Brief episode of upper extremity weakness or paresthesias followed by neurologic deficits hours to days later
   iv. No radiographic evidence

D. **Cervical diagnostic evaluation**

1. **Indications**
   a. **Nexus criteria**
      i. No midline tenderness
      ii. No focal neurologic deficit
      iii. Normal alertness
      iv. No intoxication
      v. No painful, distracting injury
      vi. Sensivity 99% and specificity 12.9%
   b. **Canadian C-spine Rule**
      i. Any high risk factors?
         • > 65 years of age
         • Fall from height > 1 meter
         • Axial loading injury
         • High-speed motor vehicle crash > 100km/h
         • Rollover
         • Ejection
         • Motorized recreational vehicle
         • Bicycle collision
      ii. Any low-risk factors that allow safe assessment?
• Simple rear-end vehicle crashes
• Sitting position in the ED
• Ambulatory at any time
• Delayed onset of neck pain
• Absence of midline neck tenderness
  iii. Is the patient able to actively rotate his or her neck 45 degrees to the left and right?

2. Routine series (all can be done supine)
   a. Cross table lateral - all 7 cervical & the 1st thoracic vertebrae
      i. Methodically review – ABCs
      ii. Alignment – anterior & posterior contour lines and spinolaminar line for smooth lordotic curvature
      iii. Bony changes – look for fractures
      iv. Cartilage space assessment – anterior or posterior widening of the intervertebral space or interspinous space
      v. Soft tissue
         • Retropharyngeal – Ant border of C2 to posterior wall of of pharynx - <7mm in adults & children
         • Predental space should not exceed 3 mm in adults & 5 mm for children
         • At C3-C4 not greater than 5 mm, or ½ the width of the C2 body
         • Retrotracheal space – anterior body of C6 to posterior surface of the trachea - <22 mm in adults or <14 mm less than 15yo
   b. A-P film
      i. Views lower 5 cervical & upper thoracic vertebrae
      ii. Overlapping articular pillars create wavy margin
      iii. Spinous processes connected with straight line
      iv. Bodies intact and uniform
      v. Tracheal and laryngeal air shadows within midline
      vi. Intervertebral spaces roughly equal
      vii. Calcified thyroid cartilage mimics fractures
      viii. On A-P thoracic film - mediastinal stripe for bulging due to posterior mediastinal hemorrhage from vertebral fractures
   c. Odontoid view (usually open mouth)
      i. X-ray of Atlas and Axis (C1 and C2)
      ii. Check lateral masses
      iii. Check for alignment of lateral masses with odotoid
      iv. Patient cannot be rotated or tilted
      v. Spinous process midline
      vi. Inspect transverse processes for fracture
      vii. Odontoid fractures – three categories:
• Type I involves the distal portion of the dens (tip) – most stable of the three types

• Type II involves the base of the dens – most common type, always unstable

• Type III involves the body of C2

  d. If technically adequate, fails to detect injury < 1% of the time

3. Modified views
   a. Swimmer's (transaxillary) helpful in trying to visualize lower C-spine
   b. Obliques
      i. Demonstrate pedicles and facets better – looking for unilateral facet dislocation or true subluxation
      ii. Laminae should look like overlapping roof shingles – disruption signals ligamentous injury
      iii. Laminar fractures seen well
      iv. Assess intervertebral foramina
   c. Flexion-extension views
      i. Indicated if concern for ligamentous injury
      ii. In awake, alert, cooperative patients without neurologic symptoms or deficits
      iii. Contraindicated if: non-dynamic view is suspicious for a fracture or subluxation, or if clinical signs of an acute spinal cord lesion are present
iv. Abnormal if: > 3.5 mm of horizontal displacement between the disks, displaced apophyseal joints, widened disk spaces, loss of greater than 30% of the disk height, presence of prevertebral hematoma
v. Probably more helpful if done 1–2 weeks following injury
vi. Replaced by CT & MRI

4. **Pediatric variations**
a. Pseudosubluxation of C2-C3
   i. 4% of kids less than 8 years
   ii. Immature muscles – hypermobile spine
   iii. If suspect injury, use posterior cervical line on lateral film line from anterior cortex of C1 spine to anterior cortex of C3 spine
   iv. If base of C2 spine is greater than 2 mm behind posterior cervical line, suspect Hangman's fracture
b. Retropharyngeal space
   i. Less than 2 year old may normally be widened on expiration, so use inspiratory films
c. Nonfusion of odontoid in kids mimics fracture

5. CT scan
   a. Indications
      i. High-energy injury mechanism
      ii. High-risk clinical parameter
      iii. Plain films reveal an injury, an area of suspicion, or are inadequate
      iv. High clinical suspicion of injury, despite normal plain films
   b. Advantages – patient lies supine, can evaluate injuries already diagnosed by x-ray, sensitivity is better than x-ray
   c. Disadvantages – cost & radiation

6. MRI C-spine
   a. Indications
      i. Complete or incomplete neurologic deficits with radiographic evidence of fracture or subluxation
      ii. Neurologic deficits not explained by plain films or CT – SCIWORA
      iii. Deterioration of neurologic function
      iv. Suspicion of ligamentous injury following inadequate or negative flex-ex films
   b. Contraindications – pacemaker, cerebral aneurysm clips, metallic foreign bodies
   c. CT & x-ray are still superior when evaluating bony structures

E. Anatomy of spinal column

1. Spine as two columns
   a. Anterior column
i. Vertebral bodies
ii. Intervertebral discs
iii. Anterior and posterior longitudinal ligaments.

b. Posterior column
i. Contains spinal canal
ii. Formed by: Pedicles, Transverse processes, Articulating facets, Laminae, & Spinous processes
iii. Held in alignment by: Nuchal ligament complex (Supraspinous ligaments, Interspinous ligaments, Infraspinous ligaments), Capsular ligaments & Ligamentum flava

F. Classification of spinal injuries (based on mechanism)

1. **Flexion** injuries
   a. Simple wedge fractures - of anterosuperior vertebral body
      i. Stable – rare nervous system damage
      ii. Impaction of vertebrae against subjacent vertebra.
      iii. Becomes unstable if multiple or involves > ½ of height

   b. Flexion teardrop fracture
      i. Wedge or chip-shaped fragment of antero-inferior vertebral body, displaced anteriorly
      ii. Common cause of anterior cord syndrome
      iii. Involves ligamentous injury and may have deficit
      iv. Unstable

   c. Clay-shoveler's fracture
      i. Now due to direct trauma to spinous processes or forced hyper-flexion
ii. Oblique fracture of spinous process base (C7, C6, T1)
iii. Stable no associated deficits.

d. Anterior subluxation
   i. Disruption of ligamentous complexes, starts posteriorly & proceeds anteriorly
   ii. Potentially unstable
   iii. Widening of interspinous or intervertebral spaces posteriorly
   iv. High incidence of delayed instability.

![Image of spine with subluxation]

e. Bilateral facet dislocation (locked facets)
   i. High incidence cord deficit
   ii. All anterior and posterior ligaments disrupted.
   iii. Extremely unstable
   iv. See anterior displacement of spine above level of injury (Greater than 2 A-P diameter of lower body).
   v. Requires traction or even surgery

![Image of spine with facet dislocation]

f. C1-C2 dislocations due to flexion injury
   i. Unstable due to location
   ii. Dislocation at atlantooccipital or atlantoaxial joints +/- odontoid fracture.
   iii. Greater than 4 mm between C1 and odontoid on lateral = atlantoaxial instability.

2. Flexion - rotation injuries
   a. Unilateral facet dislocation (locked facet)
i. Stable
ii. Symptom – torticollis (muscle spasm)  
iii. On lateral, see forward displacement of dislocated segment on vertebra below.  
iv. A-P and obliques helpful – lose "shingles on roof".  
v. If unilateral facet dislocation occurs at C1-C2, is unstable.  
   Seen as marked asymmetry between odontoid and lateral masses of C1 (rotary atlantoaxial dislocation)  
vi. Larger facets of lumbar region make this injury rare, but if one or both articular processes fracture, upper vertebra swings anterior.

3. Extension injuries
      i. Occiput compresses onto spinous process of axis.  
      ii. Potentially unstable due to location.
   b. Hangman's fracture  
      i. Traumatic C2 spondylolysis  
      ii. Abrupt deceleration  
      iii. Unstable, yet cord damage usually minimal due to width of neural canal here.  
      v. Associated prevertebral swelling may cause airway obstruction.
   c. Extension teardrop fracture  
      i. Large teardrop-shaped fragment off anteroinferior corner of vertebral body (ligament pulls it off)  
      ii. Often occurs at C5-C7 in driving accidents  
      iii. Unstable  
      iv. High association with central cord syndrome
4. Vertical compression injuries
   a. Suspect with injury to gluteal region or feet
   b. Cervical and lumbar areas, capable of straightening at time of impact
   c. Burst fracture of vertebral body.
      i. Compressive forces from above or below force nuclear pulposus of disc into vertebral body
      ii. Causes burst fracture
      iii. Stable as all ligaments intact, although fracture segments can impinge into neural canal
      iv. Associated with anterior cord syndrome

d. Jefferson Fracture of Atlas (C1)
   i. Rare
   ii. Lateral masses driven laterally
   iii. See widening of predental space on lateral
   iv. Odontoid view differentiates this from fracture of posterior neural arch of C1
   v. Extremely unstable

5. Stability
   a. Stable
      i. Simple wedge fracture
      ii. Clay-shoveler’s fracture
      iii. Unilateral facet dislocation
      iv. Burst fracture
   b. Unstable
      i. Flexion teardrop
ii. Bilateral facet dislocation
iii. C1C2 dislocation
iv. Hangman’s fracture
v. Extension teardrop
vi. Jefferson fracture
c. Potentially unstable
i. Posterior neural arch fracture
ii. Anterior subluxation

G. Treatment (fracture, dislocation or deficit)

1. Strict immobilization
2. Rapid neurosurgical consultation
3. Surgery
   a. Emergency - Relieving impingement from herniated disks, foreign bodies, bony fragments, epidural hematoma
   b. Delayed – to stabilize fracture
4. Steroids
   c. High dose methylprednisolone
      i. Start within 3 hours of injury
      ii. Bolus dose over 15 minutes (30 mg/kg)
      iii. Maintenance dose over 48 hours (5.4 mg/kg/hr)
   b. Consortium of Spinal Cord Medicine – “no clinical evidence exists to definitively recommend the use of any neuroprotective pharmacologic agent including steroids”
   c. Cochrane Review – high dose methylprednisolone is the only pharmacologic agent that has been shown to have efficacy
   d. CONTROVERSIAL!!! CHECK WITH YOUR SPINAL CORD CENTER!
5. Transfer – regional spine center where all specialists are available
HEAD INJURY/C-SPINE/SPINAL CORD TRAUMA

PEARLS

1. Cerebral blood flow is maintained at a constant level by autoregulation.

2. Increased intracranial pressure occurs if there is an increase in brain parenchyma, intracranial blood or CSF.

3. Increased intracranial pressure may result in the Cushing response - hypertension and bradycardia.

4. Hyperventilation to a pCO2 of 30 - 35 torr is the fastest means of decreasing ICP but only lasts a short while.

5. There is no evidence that corticosteroids decrease ICP.

6. Uncal herniation is the most common, symptoms of ipsilateral papillary dilatation, contralateral Babinski, and contralateral muscle weakness.

7. Central Transtentorial herniation is the next most common, symptoms of decreased LOC, bilateral muscle weakness and Bibinski reflexes, sustained hyperventilation, possibly Cushing’s.

8. Pinpoint pupils and a brain injury is a pontine lesion.

9. A normal Doll’s eye is when the head is turned and the eyes turn in the opposite direction, that is an intact brainstem.

10. A Glasgow Coma Scale has 3 criteria: eye opening (4), Verbal Stimuli (5) and motor response (6).

11. Depressed skull fractures greater than the full thickness of the skull require surgical elevation.

12. Suspect a basilar skull fracture in a patient with Raccoon eyes (periorbital ecchymosis), Battle Sign (mastoid ecchymosis), hemotympanum or CSF rhinorrhea. The most common area is the temporal bone with hemotympanum.

13. Epidural hematomas appear at biconvex (lens)-shaped and subdural hematomas appear as crescent-shaped on CT scans.

14. Epidural hematomas are due to laceration of a meningeal artery (most commonly the middle meningeal) or a vein.
15. Epidurals are rare in children under 2 y.o.a. and in the elderly but subdurs occur in the very old, the very young and the very intoxicated.

16. Trauma is the most common cause of subarachnoid hemorrhage. Subarachnoid hemorrhage is the most common finding on a CT scan of a brain injured patient.

17. Intracerebral hemorrhage most commonly occurs in the frontal and temporal lobes.

18. Subacute subdural hematomas may not be seen well on a CT scan because they are isodense.

19. The treatment for most complications of increased ICP such as neurogenic pulmonary edema and DIC is to lower the ICP.

20. Spinal cord injury may result in spinal shock: hypotension and bradycardia but warm skin. It is a diagnosis of exclusion.

21. Central cord syndrome results in greater upper extremity weakness than lower extremity weakness.

22. Brown-Sequard Syndrome results in contralateral loss of pain and temperature and ipsilateral loss of proprioception, light touch and vibration.

23. Anterior cord syndrome results in motor paralysis and hypalgesia below the level of the injury with preservation of position, touch and vibration.

24. The biggest difference between NEXUS criteria and the Canadian C-spine rule is the addition of patients 65 years and older as high risk for injury and the ability to move the neck.

25. Adequately done C-spine x-rays miss significant injuries < 1 % of the time.

26. Bilateral C-spine facet dislocations (locked facets) are extremely unstable and require traction or surgery to reduce.

27. The Jefferson fracture of C1 is from a vertical compression resulting in an extremely unstable fracture.

28. Unilateral facet dislocations are stable. They are difficult to diagnose on x-ray. Be sure to look at the alignment of the transverse processes on the AP view.
29. High dose methylprednisolone is controversial.

30. Anterior subluxation is unstable due to the disruption of ligaments which begins posteriorly and moves anteriorly. High propensity of delayed stability.
REFERENCES


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